

MATHEMATICAL ANALYSIS OF PLANT DISEASE DISPERSION MODEL THAT INCORPORATES WIND STRENGTH AND INSECT VECTOR AT EQUILIBRIUM

Abstract

Numerous plant diseases caused by pathogens like bacteria, viruses, fungi protozoa and pathogenic nematodes are propagated through media such as water, wind and other intermediary carries called vectors, and are therefore referred to as vector borne plant diseases.

Insect vector borne plant diseases are currently a major concern due abundance of insects in the tropics which impacts negatively on food security, human health and world economies. Elimination or control of which can be achieved through understanding the process of propagation via Mathematical modeling. However existing models are linear and rarely incorporates climate change parameters to improve on their accuracy. Yields of plants can reduce significantly if they are infected by vectors borne diseases whose vectors have very short life span without necessarily inducing death to plants. Despite this, there is no reliable developed mathematical model to describe such dynamics.

This paper formulates and analyzes a dynamical nonlinear plant vector borne dispersion disease model that incorporates insect and plant population at equilibrium and wind as a parameter of climate change, to determine R_0 , local and global stability in addition to sensitivity analysis of the basic reproduction number R_0 .

Keywords: Basic reproduction number, sensitivity analysis, disease free equilibrium point (DFEP), endemic equilibrium point (EEP), local and global stability.

1. Introduction

Numerous plant diseases caused by pathogens namely: bacteria, viruses, fungi, protozoa and pathogenic nematodes are spread through media such as water, wind and other intermediary carriers called vectors, and are thus referred to as vector borne diseases (Agrios, 1997).

Diseases that spread from one host to another are called infectious diseases, most of which are very destructive to plants and animals. Vector borne plant diseases have important transmission properties which include: survival rate of the vector, reproduction rate of the vector, time of the year and vectors / insects activity level specifically biting rate, rate of development and reproduction of pathogen on the vector amongst others (Patz, 2008).

The most common vectors are insects such as aphids, whiteflies, plant hoppers and leafhoppers with aphids as the most important group. Aphids have been estimated to cause 70% of the vector borne plant diseases (Nault, 1997).

Vectors (insects) are susceptible to external environmental influences which include the climate change and affect the spread of plant diseases. The climate parameters that vary include temperature, precipitation (humidity) and wind.

Climate change could first affect disease directly by either decreasing or increasing the encounter rate between the vectors and hosts by changing the ranges of the two species.

38 Disease severity should be positively correlated with increase in virulence and aggressiveness
 39 of the vector. These two effects on disease will be mediated by host resistance and encounter
 40 rates which are potentially affected by climate change.

41 Mathematical models provide a powerful tool used to understand the dynamics of disease
 42 spread through a population and in decision making in regards to disease prediction and
 43 disease control (Zhou and Yao, 2014). Empirical models like regression models and
 44 deterministic models with climate variables as predictors and epidemic parameters as response
 45 variables are used to predict the success of the hosts and vectors across the range of conditions
 46 with a possibility of extrapolation when the mechanism of the relationship is clearly
 47 understood. Simulation models based on theoretical relationships are used to predict outcomes
 48 under a range of scenario.

49 Kermack and McKendrick (1927) developed a classical model for micro parasite host interactions in
 50 mammals which forms the basis of plant epidemiological models. The first models of temporal
 51 development of epidemic plant diseases were developed by Van der Plank (1960; 1963), and have
 52 formed the basis for plant disease modeling. Subsequently, various models have been developed.
 53 Earlier theory on vector- borne diseases by Edward and De Leo (2008) , Fitzibonn (1995) , Gourley
 54 (2007), Holt (1997) and Jeger (2004) highlight the importance of the direct interactions between the
 55 vector's dynamics and disease prevalence in the host. The focus in this work is on the dynamics of the
 56 vector host disease system without considering the dynamics of the enemies of the vector.

57 There have been many studies modeling specific crop diseases and biological controls. For example
 58 Tomato leave curl virus in india by Jeger (2004) and Cassava mosaic virus in sub – Saharan Africa by
 59 Otim (2006) and the climate change factor is not considered.

60 Other researchers studied the biological control of the vectors to reduce disease incidences using
 61 models. Gourley (2007)developed a model to investigate how pulsed application of biological
 62 larvicides or chemical insecticides on different life stages affects disease prevalence. Wei (2008)
 63 modeled vector borne diseases with horizontal transmission and time delay. Cui (2010) developed and
 64 analyzed a simple vector- host epidemic model with direct transmission.

65 The above two models were extended by Lashari and Zama (2011) in their study of the global and
 66 back bifurcation of a vector borne disease model with horizontal transmission in host population.
 67 Moore (2010) developed a model to determine how a predator of the vector affects the prevalence of
 68 a vector-borne disease in the absence of predators. Zhou (2014) improved on Moore (2010) model by

developing a model to study the disease control threshold and limit cycles with persistence of disease or without disease. However, both Moore and Zhou focus was on total host population dividing host and vector into susceptible and infectious. Lawrence and Wallace (2010) modeled the spatiotemporal dynamics of African Cassava Mosaic disease in which he incorporated wind but as an agent to the movement of the vector. Clearly the climate change variables have been modeled in isolation in any given model. We want to develop a general model to investigate the effect of a combination of the climate change factors on the spread of vector –borne plant diseases.

The research developed a Mathematical model incorporating the climatic change variables to be used to evaluate the dynamics of vector- borne diseases in plants. The climate change variables, temperature and precipitation influence the biting rate (a) are considered under the parameter for biting rate while wind will be factored as an agent to the movement of the vector, emigration or immigration hence incorporated in the model as $(\pm \theta)$. Climate induced death to insects was also examined. Since insects have very short lifespans, the study developed a deterministic general model for the perennial plant disease with assumption that vectors reproduce very fast and attain equilibrium. The plant population is constant. Section 2 describes model development. Section 3 describes model analysis where also basic reproduction will be determined using the next generation method and analytical global stability by the Lyapunov method. Finally Section 4 expounds results.

2. Model Development.

The model development begins with model description, model assumption, flow chart and listing of model equations.

2.1 Model description

Let $N_V(t)$ and $N_P(t)$ be the total population of the vectors and plants respectively. The plant population is subdivided into three compartment classes: susceptible plant class $S_P(t)$, exposed plant class $E_P(t)$ and infected plant class $I_P(t)$. The vector population is subdivided into two compartment classes: susceptible vector class $S_V(t)$ and infected plant class $I_V(t)$

The recruitment rate of plants and vectors are given by μ_p and π respectively. The rates at which $E_P(t)$ recover naturally to $S_P(t)$ is given by ω . Climate induced death occur at a rate δ in $S_V(t)$ and $I_V(t)$ respectively. The constant natural death rate in subclasses $S_P(t)$, $E_P(t)$ and $I_P(t)$ is given by μ_p . The rate at which $E_P(t)$ progresses to $I_P(t)$ is τ . The constant natural death rate in subclasses $S_V(t)$ and $I_V(t)$ is given by μ_v . The immigration and emigration rate of vector is given by θ , this implies that θ can be either be positive or negative. The infection rates for plants and vectors are β_1 and β_2 respectively while the vector biting rate is given by a .

2.2 Model assumptions

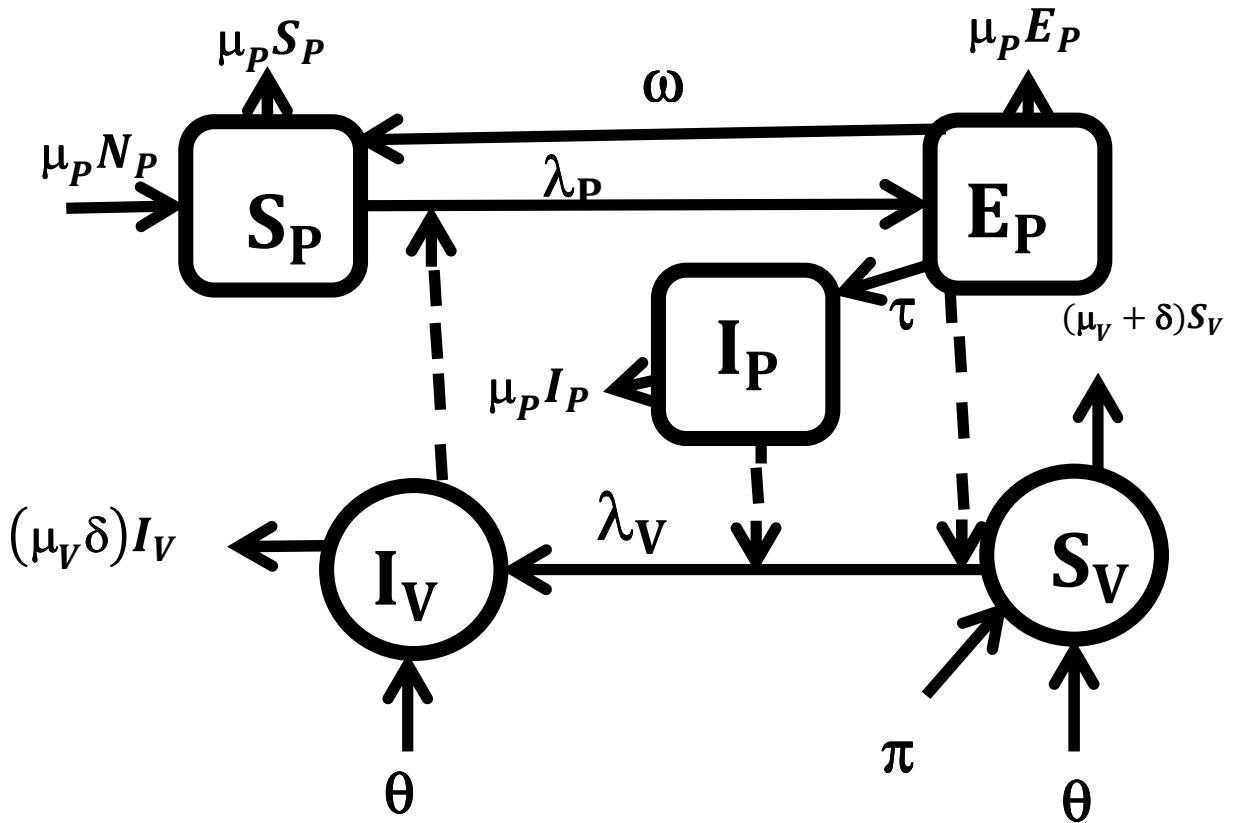
The model was formulated under the following assumptions;

- i. Homogeneous mixing of the plants and vectors.
 - ii. It is possible for exposed plants to recover from natural immunity.
 - iii. The plant population is constant while vector population is not constant.
 - iv. Rate of immigration and emigration is constant and can be estimated.
 - v. Vector population is assumed to increase very fast and then attain equilibrium.
 - vi. Effects of competitions from other vectors are assumed to be insignificant.
 - vii. The susceptible plants are infected when they come into contact with infected insect vectors.
 - viii. Susceptible insect vectors become infected only when they get in contact with an infected plant and exposed plants. There is no vertical infection through birth and from insect to insect directly.
- Modification parameter k is such that $k \geq 1$, implying that the climate factors increases force of infection

2.3 Flow chart diagram and the model equations

We considered the five state variables and parameters described in section 2.1 model assumptions listed in section 2.2 to represent the model flow chart diagram in the figure 1 in the next page;

Figure 1: Flow chart diagram of the $S E I S I$ model



125 The model was described by the following differential equations draw from the flow diagram

$$126 \quad \frac{dS_P}{dt} = \mu_P N_P + \omega E_P - (k\lambda_P + \mu_P) S_P \quad (1),$$

$$\frac{dE_P}{dt} = k\lambda_P S_P - (\omega + \tau + \mu_P) E_P \quad (2),$$

$$127 \quad \frac{dI_P}{dt} = \tau E_P - \mu_P I_P \quad (3),$$

$$\frac{dS_V}{dt} = \pi N_V \pm \theta S_V - (k\lambda_V + \delta + \mu_V) S_V \quad (4),$$

$$\frac{dI_V}{dt} = k\lambda_V S_V \pm \theta I_V - (\delta + \mu_V) I_V \quad (5),$$

128 where

$$N_V(t) = S_V(t) + I_V(t), N_P(t) = S_P(t) + E_P(t) + I_P(t), \lambda_P = a\beta_1 I_V \text{ and } \lambda_V = a\beta_2 (E_P + \eta I_P).$$

129 The insects have very short life span, they reproduce within a very short time and therefore it is
130 plausible to assume the dynamics of the insects analyzed at steady states. We equate equations

131 (4 – 5) to zero to obtain the expressions below at steady state,

$$N_V^* = S_V^* + I_V^*; S_V^* = \frac{\pi N_V^*}{k\lambda_V + \delta + \mu_V \pm \theta}; I_V^* = \frac{k\lambda_V \pi N_V^*}{(k\lambda_V + \delta + \mu_V \pm \theta)(\delta + \mu_V \pm \theta)};$$

$$\lambda_P^* = \frac{a\beta_1 k\lambda_V \pi N_V^*}{(k\lambda_V + \delta + \mu_V \pm \theta)(\delta + \mu_V \pm \theta)}$$

132 After substituting λ_P^* at steady state, the system of equations(1 – 5) reduces to;

$$133 \quad \frac{dS_P}{dt} = \mu_P N_P + \omega E_P - \left(\frac{a\beta_1 K^2 \pi N_V^* \lambda_V}{(k\lambda_V + \delta + \mu_V \pm \theta)(\delta + \mu_V \pm \theta)} + \mu_P \right) S_P \quad (6),$$

$$\frac{dE_P}{dt} = \frac{a\beta_1 K^2 \pi N_V^* \lambda_V S_P}{(k\lambda_V + \delta + \mu_V \pm \theta)(\delta + \mu_V \pm \theta)} - (\omega + \tau + \mu_P) E_P \quad (7),$$

$$134 \quad \frac{dI_P}{dt} = \tau E_P - \mu_P I_P \quad (8),$$

135 With initial conditions as, $S_P(0) = (S_P)_0$, $E_P(0) = (E_P)_0$ and $I_P(0) = (I_P)_0$. The sum of system
136 of equations[(6) – (8)] which is the rate of change of total population

137 is given by $\frac{dN_P}{dt} = 0$.

138 3. Model analysis

139 We analyzed the model by proving various theorems and carrying out algebraic computation
140 dealing with different attributes.

141

3.1. Positivity and boundedness of the solutions

We proved positivity and boundedness by stating and proving the theorem below.

Theorem1: The region Z given by $Z = \{S_P(t), E_P(t), I_P(t) \in \mathbb{R}_+^3 ; N_P = C\}$

is positively invariant and attracting with respect to model system[(6) – (8)],

Proof.

Let $\{S_P(t), E_P(t)$ and $I_P(t)\}$ be any solutions of the system with non-negative initial conditions

$\{S_P(0) \geq 0, E_P(0) \geq 0, I_P(0) \geq 0\}$.

Since, $\frac{dS_P}{dt} = \mu_P N_P + \omega E_P - \left(\frac{a\beta_1 K^2 \pi N_V^* \lambda_V}{(k\lambda_V + \delta + \mu_V \pm \theta)(\delta + \mu_V \pm \theta)} + \mu_P \right) S_P$, it follows that

$\frac{dS_P}{dt} = - \left(\frac{a\beta_1 K^2 \pi N_V^* \lambda_V}{(k\lambda_V + \delta + \mu_V \pm \theta)(\delta + \mu_V \pm \theta)} + \mu_P \right) S_P$. On integration, we obtained

$S_P(t) = e^{\int_0^t - \left(\frac{a\beta_1 K^2 \pi N_V^* \lambda_V(s)}{(k\lambda_V(s) + \delta + \mu_V \pm \theta)(\delta + \mu_V \pm \theta)} + \mu_P \right) dt} \geq 0$. Clearly, $S_P(t) = e^{\int_0^t - \left(\frac{a\beta_1 K^2 \pi N_V^* \lambda_V(s)}{(k\lambda_V(s) + \delta + \mu_V \pm \theta)(\delta + \mu_V \pm \theta)} + \mu_P \right) dt}$ is a non-negative function of t , thus $S_P(t)$ stays positive.

The positivity of $E_P(t)$ and $I_P(t)$ was proved along the same lines hence considering equation(7) $\frac{dE_P}{dt} \geq -(\omega + \tau + \mu_P)E_P$ on integrating $E_P(t) = C_1 e^{-(\omega + \tau + \mu_P)t}$, where C_1 is a constant of integration. Applying initial condition we obtain $C_1 = E_P(0)$. Hence $E_P(t) = E_P(0)e^{-(\omega + \tau + \mu_P)t}$ and $E_P(t) = E_P(0)e^{-(\omega + \tau + \mu_P)t} \geq 0$.

Similarly, $I_P(t) = I_P(0)e^{-\mu_P t} \geq 0$.

Taking the time derivative of our total population along its solution path gives

$$\frac{dN_P}{dt} = \frac{dS_P}{dt} + \frac{dE_P}{dt} + \frac{dI_P}{dt} = 0,$$

This implies that, $N_P(t) = C$, where C is the constant of integration.

Hence, $\lim_{t \rightarrow \infty} N(t) = C$

This proves the boundedness of the solutions inside Z . This implies that all the solutions of our system[(6) – (8)], starting in Z and will remain in Z for all $t \geq 0$. Thus Z is positively invariant and attracting, and hence it is sufficient to consider the dynamics of our system in Z . This completes the proof.

3.2 Disease-free equilibrium point (DFEP)

The disease-free equilibrium point (DFEP) of the system[(6) – (8)], was obtained by setting all the E_P and I_P classes to zero to obtain $\mu N_P - \mu S_P^0 = 0$ which yields, $S_P^0 = N_P$

The DFEP for our system is given by $E^0 = (S_P^0, E_P^0, I_P^0) = (N_P, 0, 0)$.

The DFEP (E^0) is the infection free equilibrium point of the system[(6) – (8)], which indicates that in absence of insects, the system[(6) – (8)] will consist of one compartment class

3.3. The basic reproduction number (R_0)

We used the next-generation matrix method to determine the basic reproduction number (R_0) of the model (Carlos Castillo-Chavez, 2001). Using the notation f for a matrix of new infections terms and v for the matrix of the remaining transfer of infection terms in our system, we obtained

$$f = \begin{pmatrix} \frac{a\beta_1 K^2 \pi N_V^* \lambda_V S_P}{(k\lambda_V + \delta + \mu_V \pm \theta)(\delta + \mu_V \pm \theta)} \\ 0 \end{pmatrix} \text{ and } v = \begin{pmatrix} (\omega + \tau + \mu_P)E_P \\ -\tau E_P + \mu_P I_P \end{pmatrix}.$$

We then obtained the matrices F and V by finding the Jacobian matrices of f and v evaluated at DFEP respectively

$$F = \begin{pmatrix} \frac{a^2 \beta_1 \beta_2 K^2 \pi N_V^* N_P}{(\delta + \mu_V \pm \theta)^2} & \frac{a^2 \beta_1 \beta_2 K^2 \pi N_V^* \eta N_P}{(\delta + \mu_V \pm \theta)^2} \\ 0 & 0 \end{pmatrix} \text{ and } V = \begin{pmatrix} \omega + \tau + \mu_P & 0 \\ -\tau & \mu_P \end{pmatrix}.$$

We computed V^{-1} the inverse of V hence $V^{-1} = \begin{pmatrix} \frac{1}{\omega + \tau + \mu_P} & 0 \\ \frac{\tau}{\mu_P(\omega + \tau + \mu_P)} & \frac{1}{\mu_P} \end{pmatrix}.$

The eigenvalues of the matrix (FV^{-1}) are

$$q(1) = 0 \text{ and } q(2) = \frac{a^2 \beta_1 \beta_2 K^2 \pi N_V^* N_P}{(\delta + \mu_V \pm \theta)^2 (\omega + \tau + \mu_P)} + \frac{a^2 \beta_1 \beta_2 K^2 \pi N_V^* \eta \tau N_P}{\mu_P (\delta + \mu_V \pm \theta)^2 (\omega + \tau + \mu_P)}.$$

The basic reproduction number(R_0) is given by the spectral radius ζ (the dominant eigenvalue) of the matrix FV^{-1} denoted by $\zeta(FV^{-1})$ hence

$$R_0 = \zeta(FV^{-1}) = \frac{a^2 \beta_1 \beta_2 K^2 \pi N_V^* N_P}{(\delta + \mu_V \pm \theta)^2 (\omega + \tau + \mu_P)} \left\{ 1 + \frac{\eta \tau}{\mu_P} \right\}.$$

The basic reproduction number(R_0) is the average number of susceptible plants which can be infected by an infected insect in absence of interventions.

3.4. Existence of Endemic Equilibrium point for the model (EEP)

We state and prove the following theorem

Theorem 2: A positive endemic equilibrium exists whenever $R_0 > 1$.

Proof

Let the endemic equilibrium point be $E^* = (S_P^*, E_P^*, I_P^*)$ and the force of infection at equilibrium point be $\lambda_V^* = a\beta_2(E_P^* + \eta I_P^*)$. The system of equation (6)– (8) were equated to zero then solved in terms of λ^* to obtain;

$$\begin{aligned}
S_P^* &= \frac{\mu_P N_P [k\lambda_V^* (\delta + \mu_V \pm \theta) + (\delta + \mu_V \pm \theta)^2]}{a\beta_1 K^2 \pi N_V^* \lambda_V^* + \mu_P [k\lambda_V^* (\delta + \mu_V \pm \theta) + (\delta + \mu_V \pm \theta)^2]} \\
&\quad + \frac{[k\lambda_V^* (\delta + \mu_V \pm \theta) + (\delta + \mu_V \pm \theta)^2] \omega E_P^*}{a\beta_1 K^2 \pi N_V^* \lambda_V^* + \mu_P [k\lambda_V^* (\delta + \mu_V \pm \theta) + (\delta + \mu_V \pm \theta)^2]} \\
E_P^* &= \frac{a\beta_1 K^2 \pi N_V^* \lambda_V^* \mu_P N_P}{(\omega + \tau + \mu_P) \{a\beta_1 K^2 \pi N_V^* \lambda_V^* + \mu_P [k\lambda_V^* (\delta + \mu_V \pm \theta) + (\delta + \mu_V \pm \theta)^2]\} - a\beta_1 K^2 \pi N_V^* \lambda_V \omega}, \\
I_P^* &= \frac{\tau a\beta_1 K^2 \pi N_V^* \lambda_V^* \mu_P N_P}{\mu_P [(\omega + \tau + \mu_P) \{a\beta_1 K^2 \pi N_V^* \lambda_V^* + \mu_P [k\lambda_V^* (\delta + \mu_V \pm \theta) + (\delta + \mu_V \pm \theta)^2]\} - a\beta_1 K^2 \pi N_V^* \lambda_V \omega]}, \\
\lambda_V^* &= \frac{\lambda_V^* \mu_P R_0 (\delta + \mu_V \pm \theta)^2 (\omega + \tau + \mu_P)}{(\omega + \tau + \mu_P) \{a\beta_1 K^2 \pi N_V^* \lambda_V^* + \mu_P [k\lambda_V^* (\delta + \mu_V \pm \theta) + (\delta + \mu_V \pm \theta)^2]\} - a\beta_1 K^2 \pi N_V^* \lambda_V \omega}
\end{aligned}$$

192

193 $\lambda_V^* = 0$, Corresponds to DFE, while the expression for λ_V^* below correspond to endemic
194 equilibrium point

$$\lambda_V^* = \frac{\mu_P (\delta + \mu_V \pm \theta)^2 (\omega + \tau + \mu_P) (R_0 - 1)}{(\omega + \tau + \mu_P) \{a\beta_1 K^2 \pi N_V^* + \mu_P [k(\delta + \mu_V \pm \theta)]\} - a\beta_1 K^2 \pi N_V^* \omega}$$

195 Since $(\omega + \tau + \mu_P) \{a\beta_1 K^2 \pi N_V^* + \mu_P [k(\delta + \mu_V \pm \theta)]\} - a\beta_1 K^2 \pi N_V^* \omega > 0$, the condition necessary
196 and sufficient for $\lambda_V^* > 0$ is $\mu_P (\delta + \mu_V \pm \theta)^2 (\omega + \tau + \mu_P) (R_0 - 1) > 0$ that is $R_0 > 1$. That
197 completes the proof

198 The endemic equilibrium point (EEP) for our system $E^* = (S_P^*, E_P^*, I_P^*)$ is obtained by
199 substituting,

$$\lambda_V^* = \frac{\mu_P (\delta + \mu_V \pm \theta)^2 (\omega + \tau + \mu_P) (R_0 - 1)}{(\omega + \tau + \mu_P) \{a\beta_1 K^2 \pi N_V^* + \mu_P [k(\delta + \mu_V \pm \theta)]\} - a\beta_1 K^2 \pi N_V^* \omega} \text{ in } S_P^*, E_P^* \text{ and } I_P^*.$$

201

202 3.5. Local Stability of the disease free equilibrium point (DFE).

203 To determine the local stability of the disease free equilibrium point we stated and proved the
204 following theorem.

205 **Theorem 3:** The DFEP of the system [(6) – (8)] is locally asymptotically stable $R_0 < 1$ and
206 unstable otherwise.

207 Proof

208 To establish the local stability of the system [(6) – (8)], we used the Jacobian of the model
209 evaluated at E^0 . Stability of this steady state was then determined based on the signs
210 eigenvalues of the corresponding Jacobian which were functions of the model parameters. The
211 Jacobian matrix evaluated at disease free equilibrium point E^0 is obtained as

$$J(E^0) = \begin{pmatrix} -\frac{a\beta_1 K^2 \pi N_V^* N_P}{(\delta + \mu_V \pm \theta)^2} + \mu_P & -\frac{a^2 \beta_1 \beta_2 K^2 \pi N_V^* N_P}{(\delta + \mu_V \pm \theta)^2} + \omega & -\frac{a^2 \beta_1 \beta_2 \eta K^2 \pi N_V^* N_P}{(\delta + \mu_V \pm \theta)^2} \\ 0 & \frac{a^2 \beta_1 \beta_2 K^2 \pi N_V^* N_P}{(\delta + \mu_V \pm \theta)^2} - (\omega + \tau + \mu_P) & \frac{a^2 \beta_1 \beta_2 \eta K^2 \pi N_V^* N_P}{(\delta + \mu_V \pm \theta)^2} \\ 0 & \tau & -\mu_P \end{pmatrix}$$

212 Using the Mathematica software the eigenvalues of the matrix $J(E^0)$ are;

$$q(1) = -\frac{a\beta_1 K^2 \pi N_V^* N_P}{(\delta + \mu_V \pm \theta)^2} + \mu_P,$$

$$q(2) = \frac{1}{2}(-\tau - \omega - 2\mu_P + \frac{R_0 \mu_P (\tau + \omega + \mu_P)}{\eta \tau + \mu_P} - \sqrt{(\tau + \omega)^2 + \frac{2((-1 + 2\eta)\tau - \omega)R_0 \mu_P (\tau + \omega + \mu_P)}{\eta \tau + \mu_P} + \frac{R_0^2 \mu_P^2 (\tau + \omega + \mu_P)^2}{(\eta \tau + \mu_P)^2}}),$$

$$q(3) = \frac{1}{2}(-\tau - \omega - 2\mu_P + \frac{R_0 \mu_P (\tau + \omega + \mu_P)}{\eta \tau + \mu_P} + \sqrt{(\tau + \omega)^2 + \frac{2((-1 + 2\eta)\tau - \omega)R_0 \mu_P (\tau + \omega + \mu_P)}{\eta \tau + \mu_P} + \frac{R_0^2 \mu_P^2 (\tau + \omega + \mu_P)^2}{(\eta \tau + \mu_P)^2}})$$

213

214 Clearly, $q(1) < 0$. We used Mathematica software to make R_0 the subject of the expressions for
215 $q(2)$ and $q(3)$ as below,

$$\frac{1}{2}(-\tau - \omega - 2\mu_P + \frac{R_0 \mu_P (\tau + \omega + \mu_P)}{\eta \tau + \mu_P}) < \pm \sqrt{(\tau + \omega)^2 + \frac{2((-1 + 2\eta)\tau - \omega)R_0 \mu_P (\tau + \omega + \mu_P)}{\eta \tau + \mu_P} + \frac{R_0^2 \mu_P^2 (\tau + \omega + \mu_P)^2}{(\eta \tau + \mu_P)^2}}$$

216 We found that conditions necessary and sufficient for $q(2)$ and $q(3)$ to be less than zero is

217 $R_0 < 1$. This completed the proof.

218 3.6. Global stability of the disease free point.

219 To prove the global stability, we stated and proved the following theorem.

220 **Theorem 4:** The conditions necessary and sufficient for the DFE to be globally asymptotically

221 stable in Lyapunov sense are $\left\{\frac{\mu_P}{\tau} + \eta\right\} \leq 0$ or $\frac{a\beta_1 K^2 \pi N_V^* S_P^0 I_P}{(k(E_P + \eta I_P) + \delta + \mu_V \pm \theta)(\delta + \mu_V \pm \theta)} \left\{\frac{\mu_P}{\tau} + \eta\right\} < \mu_P \frac{(S_P - S_P^0)^2}{S_P} +$

222 $\mu_P I_P + I_P \frac{\mu_P^2}{\tau} + \mu_P N_P \frac{S_P^0}{S_P} + \omega E_P \frac{S_P^0}{S_P}$ and unstable otherwise

223 **Proof**

224 We proposed the following Lyapunov function for the system [(6) – (8)]

$$225 \quad L(S_P, E_P, I_P) = S_P - S_P^0 - S_P^0 \ln \frac{S_P}{S_P^0} + E_P + X_1 I_P \quad (9)$$

226 $L(S_P, E_P, I_P)$ is positive definite and satisfies the conditions. For $\frac{dL(S_P, E_P, I_P)}{dt}$ to be negative
 227 definite, it must satisfy

$$\frac{dL(S_P^0, E_P^0, I_P^0)}{dt} = 0 \text{ and } \frac{dL(S_P, E_P, I_P)}{dt} < 0$$

228 Where, X_1 and X_2 were positive constants to be determined. The time derivative of the Lyapunov
 229 function is obtained as,

$$230 \quad \frac{dL(S_P, E_P, I_P)}{dt} = \left(1 - \frac{S_P^0}{S_P}\right) \frac{dS_P}{dt} + X_1 \left(1 - \frac{E_P^0}{E_P}\right) \frac{dE_P}{dt} + X_2 \frac{dI_P}{dt}, \quad (10)$$

231 On substituting for $\frac{dS_P}{dt}$, $\frac{dE_P}{dt}$ and $\frac{dI_P}{dt}$ in equations (6) – (8) and considering $\mu_P N_P = \mu_P S_P^0$ we
 232 obtained;

$$\begin{aligned} \frac{dL(S_P, E_P, I_P)}{dt} = & -\mu_P \frac{(S_P - S_P^0)^2}{S_P} - (\tau + \mu_P) E_P + X_1 \{\tau E_P - \mu_P I_P\} - \mu_P N_P \frac{S_P^0}{S_P} - \omega E_P \frac{S_P^0}{S_P} \\ & + \left(\frac{a\beta_1 K^2 \pi N_V^* (E_P + \eta I_P)}{(k(E_P + \eta I_P) + \delta + \mu_V \pm \theta)(\delta + \mu_V \pm \theta)} \right) S_P^0 \end{aligned}$$

233 Setting E_P to zero we obtain

$$-(\tau + \mu_P) + \frac{a\beta_1 K^2 \pi N_V^* S_P^0}{(k(E_P + \eta I_P) + \delta + \mu_V \pm \theta)(\delta + \mu_V \pm \theta)} + X_1 \tau = 0.$$

$$X_1 = 1 + \frac{\mu_P}{\tau} - \frac{a\beta_1 K^2 \pi N_V^* S_P^0}{\tau(k(E_P + \eta I_P) + \delta + \mu_V \pm \theta)(\delta + \mu_V \pm \theta)}$$

234 Then

$$\begin{aligned} \frac{dL(S_P, E_P, I_P)}{dt} = & -\mu_P \frac{(S_P - S_P^0)^2}{S_P} - \mu_P I_P - I_P \frac{\mu_P^2}{\tau} - \mu_P N_P \frac{S_P^0}{S_P} - \omega E_P \frac{S_P^0}{S_P} \\ & + \frac{a\beta_1 K^2 \pi N_V^* S_P^0 I_P}{(k(E_P + \eta I_P) + \delta + \mu_V \pm \theta)(\delta + \mu_V \pm \theta)} \left\{ \frac{\mu_P}{\tau} + \eta \right\}, \end{aligned}$$

235 The conditions necessary and sufficient for $\frac{dL(S_P, E_P, I_P)}{dt} < 0$ are

$$\begin{aligned} 236 \quad \left\{ \frac{\mu_P}{\tau} + \eta \right\} \leq 0 \text{ or } & + \frac{a\beta_1 K^2 \pi N_V^* S_P^0 I_P}{(k(E_P + \eta I_P) + \delta + \mu_V \pm \theta)(\delta + \mu_V \pm \theta)} \left\{ \frac{\mu_P}{\tau} + \eta \right\} < \mu_P \frac{(S_P - S_P^0)^2}{S_P} + \mu_P I_P + I_P \frac{\mu_P^2}{\tau} + \mu_P N_P \frac{S_P^0}{S_P} + \\ 237 \quad & \omega E_P \frac{S_P^0}{S_P}. \text{ This completed the proof.} \end{aligned}$$

238 3.7 Bifurcation Analysis.

239 This bifurcation was explored using the Centre Manifold theory (Junli et al. 2011). The change of
 240 variables was made first for simplicity. Let $S_P = y_1$, $E_P = y_2$ and $I_P = y_4$, so that,

$$N_P = y_1 + y_2 + y_3.$$

Further, by using vector notation, $y = (y_1, y_2, y_3)^T$, the insect vector disease model[(6) – (8)] was written in the form $\frac{dy}{dt} = F(y)$, with $F = (p_1, p_2, p_3)^T$, as follows:

$$\dot{y}_1 = p_1 = \mu_P N_P + \omega y_2 - \left(\frac{a\beta_1 K^2 \pi N_V^* \lambda_V^{**}}{(k\lambda_V^{**} + \delta + \mu_V \pm \theta)(\delta + \mu_V \pm \theta)} + \mu_P \right) y_1, \quad (11)$$

$$\dot{y}_2 = p_2 = \frac{a\beta_1 K^2 \pi N_V^* \lambda_V^{**} y_1}{(k\lambda_V^{**} + \delta + \mu_V \pm \theta)(\delta + \mu_V \pm \theta)} - (\omega + \tau + \mu_P) y_2 \quad (12)$$

$$\dot{y}_3 = p_3 = \tau y_2 - \mu_P y_3 \quad (13)$$

With $\lambda_V^{**} = a\beta_2(y_2 + \eta y_3)$

The method entailed evaluating the Jacobian of the system [(9) – (11)] at the disease free equilibrium point, by $J(E_*)$, has eigenvalues $\left(-\frac{(\omega + \tau + \mu_P)\mu_P}{a^2\beta_2(\mu_P + \eta\tau)} + \mu_P, -\frac{(\omega + \tau + \mu_P)\eta\tau + \eta\tau\mu_P + \mu_P^2}{\mu_P + \eta\tau}, 0 \right)$, denoted by $J(E_*)$. This gave:

$$J(E_*) = \begin{pmatrix} -\frac{a\beta_1^* K^2 \pi N_V^* N_P}{(\delta + \mu_V \pm \theta)^2} + \mu_P & -\frac{a^2\beta_1^* \beta_2 K^2 \pi N_V^* N_P}{(\delta + \mu_V \pm \theta)^2} + \omega & -\frac{a^2\beta_1^* \beta_2 \eta K^2 \pi N_V^* N_P}{(\delta + \mu_V \pm \theta)^2} \\ 0 & \frac{a^2\beta_1^* \beta_2 K^2 \pi N_V^* N_P}{(\delta + \mu_V \pm \theta)^2} - (\omega + \tau + \mu_P) & \frac{a^2\beta_1^* \beta_2 \eta K^2 \pi N_V^* N_P}{(\delta + \mu_V \pm \theta)^2} \\ 0 & \tau & -\mu_P \end{pmatrix}$$

We considered the case where $R_0 = 1$. Suppose, further, that $\beta_1 = \beta_1^*$ is chosen as a bifurcation parameter. Solving for β^* from $R_C^* = 1$ gives

$$\beta_1^* = \frac{(\delta + \mu_V \pm \theta)^2 (\omega + \tau + \mu_P) \mu_P}{a^2 \beta_2 K^2 \pi N_V^* N_P (\mu_P + \eta\tau)}$$

Using Mathematica software the Jacobian of $\frac{dy}{dt} = F(y)$ at the disease free equilibrium point,

with $\beta = \beta^*$, denoted by $J(E_*)$, has eigenvalues $\left(-\frac{(\omega + \tau + \mu_P)\mu_P}{a^2\beta_2(\mu_P + \eta\tau)} + \mu_P, -\frac{(\omega + \tau + \mu_P)\eta\tau + \eta\tau\mu_P + \mu_P^2}{\mu_P + \eta\tau}, 0 \right)$.

We obtained one zero eigenvalue and two negative eigenvalues hence, the Centre Manifold theory was used to analyze the dynamics of the model (Junli Liu, et.al 2011).

Eigenvectors of J_{β^*} : For the case, when $R_0 = 1$ it can be shown that the Jacobian $[J(E_*)]$ at $\beta_1 = \beta_1^*$ (denoted by J_{β^*}) has a right eigenvector given by $u = [u_1, u_2, u_3]^T$, where,

260

$$u_1 = \frac{-\frac{a^2\beta_1^* \beta_2 K^2 \pi N_V^* N_P u_2}{(\delta + \mu_V \pm \theta)^2} - \frac{a^2\beta_1^* \beta_2 \eta K^2 \pi N_V^* N_P u_3}{(\delta + \mu_V \pm \theta)^2} + \omega u_2}{\frac{a\beta_1^* K^2 \pi N_V^* N_P}{(\delta + \mu_V \pm \theta)^2} - \mu_P} < 0, u_2 = u_2 > 0, u_3 = \frac{\tau u_2}{\mu_P} > 0,$$

Further, J_{β^*} has a left eigenvector ($v = [v_1, v_2, v_3]$), where,

$$v_1 = 0, \quad v_2 = v_2 > 0, \quad v_3 = \frac{\frac{a^2 \beta_1^* \beta_2 \eta K^2 \pi N_V^* N_P v_2}{(\delta + \mu_V \pm \theta)^2}}{\mu_P} > 0.$$

Since ($v_1 = 0$), we only need to compute the partial derivatives of p_2 and p_3 (at the disease free equilibrium point). For the system [(9) – (11)] the associated non-zero partial derivative of f_3 (at the disease free equilibrium) is given by

$$\frac{\partial^2 p_2}{\partial y_1 \partial y_2} = \frac{\partial^2 p_2}{\partial p_2 \partial y_1} = \frac{a^2 \beta_1 \beta_2 K^2 \pi N_V^*}{(\delta + \mu_V \pm \theta)^2} \text{ and } \frac{\partial^2 p_3}{\partial y_1 \partial y_3} = \frac{\partial^2 p_3}{\partial y_3 \partial y_1} = \frac{a^2 \beta_1 \beta_2 \eta K^2 \pi N_V^*}{(\delta + \mu_V \pm \theta)^2}.$$

This implies that,

$$r = \sum_{i,j=1}^3 v_2 u_i u_j \frac{\partial^2 p_2}{\partial x_i \partial x_j},$$

$$r = \left\{ v_2 u_1 u_2 \frac{a^2 \beta_1 \beta_2 K^2 \pi N_V^*}{(\delta + \mu_V \pm \theta)^2} + v_2 u_2 u_1 \frac{a^2 \beta_1 \beta_2 K^2 \pi N_V^*}{(\delta + \mu_V \pm \theta)^2} + v_2 u_1 u_3 \frac{a^2 \beta_1 \beta_2 \eta K^2 \pi N_V^*}{(\delta + \mu_V \pm \theta)^2} + v_2 u_3 u_1 \frac{a^2 \beta_1 \beta_2 \eta K^2 \pi N_V^*}{(\delta + \mu_V \pm \theta)^2} \right\}.$$

Since u_1 are less than zero, it follows that,

$$r = 2v_2 \left\{ u_1 u_2 \frac{\partial^2 p_2}{\partial y_1 \partial y_2} + u_1 u_3 \frac{\partial^2 p_2}{\partial y_1 \partial y_3} \right\} < 0.$$

Also,

$$q = \sum_{k,i,j=1}^n v_k u_i \frac{\partial^2 p_k}{\partial y_i \partial \beta^*} (0,0)$$

$$\frac{\partial^2 p_2}{\partial y_2 \partial \beta_1^*} = \frac{a^2 \beta_2 K^2 \pi N_V^* N_P}{(\delta + \mu_V \pm \theta)^2}; \quad \frac{\partial^2 p_2}{\partial y_3 \partial \beta_1^*} = \frac{a^2 \beta_2 K^2 \pi \eta N_V^* N_P}{(\delta + \mu_V \pm \theta)^2}$$

$$q = \sum_{i=1}^3 v_2 u_i \frac{\partial^2 p_2}{\partial y_i \partial \beta_1^*} = v_2 u_2 \frac{\partial^2 p_2}{\partial y_2 \partial \beta_1^*} + v_2 u_3 \frac{\partial^2 p_2}{\partial y_3 \partial \beta_1^*},$$

$$q = 2v_2 \left\{ u_2 \frac{a^2 \beta_2 K^2 \pi N_V^* N_P}{(\delta + \mu_V \pm \theta)^2} + u_3 \frac{a^2 \beta_2 K^2 \pi \eta N_V^* N_P}{(\delta + \mu_V \pm \theta)^2} \right\}.$$

Since v_2, u_2 and u_3 were greater than zero it followed that, $q > 0$.

Hence, it followed (from theorem of Castillo-Chavez and Song, considered the following general system of ordinary differential equations with a parameter, β_1^*) that when $\beta_1^* < 0$ with $|\beta_1^*| \ll 1$, (0,0) is unstable, and there exists a negative and locally asymptotically stable equilibrium; when $0 < \beta_1^* \ll 1$, (0,0) is stable and there exists a positive unstable equilibrium.

3.8. Global stability of the Endemic Equilibrium point (EEP)

276 To determine global stability we stated and proved the following theorem.

277 **Theorem 6:** The conditions necessary and sufficient for the EEP to be globally asymptotically
 278 stable in Lyapunov sense is $\frac{dK(S_P, E_P, I_P)}{dt} \leq 0$ is $P > Q$ and unstable otherwise, where,

$$P = \mu_P \frac{(S_P - S_P^*)^2}{S_P} + \left\{1 + \frac{\mu_P}{\tau}\right\} \mu_P I_P + \frac{a\beta_1 K^2 \pi N_V^* (E_P + \eta I_P) S_P}{(k\lambda_V + \delta + \mu_V \pm \theta)(\delta + \mu_V \pm \theta)} \\ + \left(\frac{a^2 \beta_1 \beta_2 K^2 \pi N_V^* (E_P^* + \eta I_P^*)}{(k\alpha\beta_2 (E_P^* + \eta I_P^*) + \delta + \mu_V \pm \theta)(\delta + \mu_V \pm \theta)} \right) S_P^* \frac{S_P}{S_P} + \omega \frac{S_P^*}{S_P} E_P + \left\{1 + \frac{\mu_P}{\tau}\right\} \tau \frac{I_P^* E_P}{I_P},$$

279

$$Q = \frac{a^2 \beta_1 \beta_2 K^2 \pi N_V^* S_P^*}{(k\alpha\beta_2 (E_P + \eta I_P) + \delta + \mu_V \pm \theta)(\delta + \mu_V \pm \theta)} \left\{ E_P + \eta I_P \frac{S_P E_P^*}{E_P} + \omega \frac{S_P^* E_P^*}{S_P} + \left\{1 + \frac{\mu_P}{\tau}\right\} \mu_P I_P^* \right. \\ \left. + (\tau + \mu_P) E_P^* + \frac{a^2 \beta_1 \beta_2 K^2 \pi N_V^* (E_P^* + \eta I_P^*) S_P^*}{(k\alpha\beta_2 (E_P^* + \eta I_P^*) + \delta + \mu_V \pm \theta)(\delta + \mu_V \pm \theta)} \right\}$$

280 **Proof**

281 We proposed the following Lyapunov function,

$$K(S_P, E_P, I_P) = S_P - S_P^* - S_P^* \ln \frac{S_P}{S_P^*} + \left(E_P - E_P^* - E_P^* \ln \frac{E_P}{E_P^*} \right) + X_1 \left(I_P - I_P^* - I_P^* \ln \frac{I_P}{I_P^*} \right), \\ E^* = (S_P^*, E_P^*, I_P^*)$$

282 where, X_1 is a positive constant to be determined. The Lyapunov function $K(S_P, E_P, I_P)$ satisfies
 283 the conditions, $K(S_P^*, E_P^*, I_P^*) = 0$ and $K(S_P, E_P, I_P) > 0$, hence it is positive definite. For
 284 $\frac{dK(S_P, E_P, I_P)}{dt}$ to be negative definite, it must satisfy,

$$\frac{dK(S_P^*, E_P^*, I_P^*)}{dt} = 0 \quad \text{and} \quad \frac{dK(S_P, E_P, I_P)}{dt} < 0.$$

285 Determining the time derivative of the lyapunov equation we obtain,

$$\frac{dK(S_P, E_P, I_P)}{dt} = \left(1 - \frac{S_P^*}{S_P} \right) \frac{dS_P}{dt} + \left(1 - \frac{E_P^*}{E_P} \right) \frac{dE_P}{dt} + X_1 \left(1 - \frac{I_P^*}{I_P} \right) \frac{dI_P}{dt},$$

286 Substituting for $\frac{dS_P}{dt}$, $\frac{dE_P}{dt}$ and $\frac{dI_P}{dt}$ and considering that at endemic equilibrium point $E^* =$
 287 (S_P^*, E_P^*, I_P^*) for the system satisfies equations (6) - (8)

$$288 \mu_P N_P = -\omega E_P^* + \left(\frac{a\beta_1 K^2 \pi N_V^* \lambda_V^*}{(k\lambda_V^* + \delta + \mu_V \pm \theta)(\delta + \mu_V \pm \theta)} + \mu_P \right) S_P^*,$$

$$\frac{a\beta_1 K^2 \pi N_V^* \lambda_V^* S_P^*}{(k\lambda_V^* + \delta + \mu_V \pm \theta)(\delta + \mu_V \pm \theta)} = (\omega + \tau + \mu_P) E_P^*,$$

289 $\tau E_P^* = \mu_P I_P^*$ and $\lambda_V^* = a\beta_2(E_P^* + \eta I_P^*)$, we obtained,

$$\begin{aligned} \frac{dK(S_P, E_P, I_P)}{dt} = & -\mu_P \frac{(S_P - S_P^*)^2}{S_P} + \{-(\tau + \mu_P) + X_1 \tau\} E_P \\ & + \frac{a^2 \beta_1 \beta_2 K^2 \pi N_V^* (E_P^* + \eta I_P^*) S_P^*}{(ka\beta_2(E_P^* + \eta I_P^*) + \delta + \mu_V \pm \theta)(\delta + \mu_V \pm \theta)} - X_1 \mu_P I_P \\ & + \frac{a^2 \beta_1 \beta_2 K^2 \pi N_V^* S_P^*}{(ka\beta_2(E_P + \eta I_P) + \delta + \mu_V \pm \theta)(\delta + \mu_V \pm \theta)} \{E_P + \eta I_P\} \\ & - \frac{a\beta_1 K^2 \pi N_V^* (E_P + \eta I_P) S_P}{(k\lambda_V + \delta + \mu_V \pm \theta)(\delta + \mu_V \pm \theta)} \frac{S_P E_P^*}{E_P} \\ & - \left(\frac{a^2 \beta_1 \beta_2 K^2 \pi N_V^* (E_P^* + \eta I_P^*)}{(ka\beta_2(E_P^* + \eta I_P^*) + \delta + \mu_V \pm \theta)(\delta + \mu_V \pm \theta)} \right) S_P^* \frac{S_P^*}{S_P} - \omega \frac{S_P^*}{S_P} E_P - X_1 \tau \frac{I_P^* E_P}{I_P} \\ & + \omega \frac{S_P^* E_P^*}{S_P} + X_1 \mu_P I_P^* + (\tau + \mu_P) E_P^*, \end{aligned}$$

290 Setting E_P to zero we obtained the following equation,

$$-(\tau + \mu_P) + X_1 \tau = 0; \quad X_1 = \{1 + \frac{\mu_P}{\tau}\}.$$

291 Hence,

$$\begin{aligned} \frac{dK(S_P, E_P, I_P)}{dt} = & -\mu_P \frac{(S_P - S_P^*)^2}{S_P} - \{1 + \frac{\mu_P}{\tau}\} \mu_P I_P - \frac{a\beta_1 K^2 \pi N_V^* (E_P + \eta I_P) S_P}{(k\lambda_V + \delta + \mu_V \pm \theta)(\delta + \mu_V \pm \theta)} \\ & - \left(\frac{a^2 \beta_1 \beta_2 K^2 \pi N_V^* (E_P^* + \eta I_P^*)}{(ka\beta_2(E_P^* + \eta I_P^*) + \delta + \mu_V \pm \theta)(\delta + \mu_V \pm \theta)} \right) S_P^* \frac{S_P^*}{S_P} - \omega \frac{S_P^*}{S_P} E_P - \{1 \\ & + \frac{\mu_P}{\tau}\} \tau \frac{I_P^* E_P}{I_P} + \frac{a^2 \beta_1 \beta_2 K^2 \pi N_V^* S_P^*}{(ka\beta_2(E_P + \eta I_P) + \delta + \mu_V \pm \theta)(\delta + \mu_V \pm \theta)} \{E_P + \eta I_P\} \frac{S_P E_P^*}{E_P} \\ & + \omega \frac{S_P^* E_P^*}{S_P} + \{1 + \frac{\mu_P}{\tau}\} \mu_P I_P^* + (\tau + \mu_P) E_P^* \\ & + \frac{a^2 \beta_1 \beta_2 K^2 \pi N_V^* (E_P^* + \eta I_P^*) S_P^*}{(ka\beta_2(E_P^* + \eta I_P^*) + \delta + \mu_V \pm \theta)(\delta + \mu_V \pm \theta)}, \end{aligned}$$

292

293 Let,

$$\begin{aligned} P = & \mu_P \frac{(S_P - S_P^*)^2}{S_P} + \{1 + \frac{\mu_P}{\tau}\} \mu_P I_P + \frac{a\beta_1 K^2 \pi N_V^* (E_P + \eta I_P) S_P}{(k\lambda_V + \delta + \mu_V \pm \theta)(\delta + \mu_V \pm \theta)} \\ & + \left(\frac{a^2 \beta_1 \beta_2 K^2 \pi N_V^* (E_P^* + \eta I_P^*)}{(ka\beta_2(E_P^* + \eta I_P^*) + \delta + \mu_V \pm \theta)(\delta + \mu_V \pm \theta)} \right) S_P^* \frac{S_P^*}{S_P} + \omega \frac{S_P^*}{S_P} E_P + \{1 \\ & + \frac{\mu_P}{\tau}\} \tau \frac{I_P^* E_P}{I_P}, \end{aligned}$$

$$Q = \frac{a^2\beta_1\beta_2K^2\pi N_V^*S_P^*}{(ka\beta_2(E_P + \eta I_P) + \delta + \mu_V \pm \theta)(\delta + \mu_V \pm \theta)} \left\{ E_P + \eta I_P \frac{S_P E_P^*}{E_P} + \omega \frac{S_P^* E_P^*}{S_P} + \left\{ 1 + \frac{\mu_P}{\tau} \right\} \mu_P I_P^* \right. \\ \left. + (\tau + \mu_P) E_P^* + \frac{a^2\beta_1\beta_2K^2\pi N_V^*(E_P^* + \eta I_P^*)S_P^*}{(ka\beta_2(E_P^* + \eta I_P^*) + \delta + \mu_V \pm \theta)(\delta + \mu_V \pm \theta)} \right\},$$

$$\frac{dK(S_P, E_P, I_P)}{dt} = -P + Q,$$

294 The conditions necessary and sufficient for the EEP to be globally asymptotically stable in
 295 Lyapunov sense that is $\frac{dK(S_P, E_P, I_P)}{dt} \leq 0$ is $P > Q$. This completes the proof.

296 3.9 : SENSITIVITY ANALYSIS

297 The analytical sensitivity analysis of R_0 with various parameters was obtained by partial
 298 differentiation as follows,

$$\frac{dR_0}{d\omega} = -\frac{a^2\beta_1\beta_2K^2\pi N_V^*N_P}{(\delta + \mu_V \pm \theta)^2(\omega + \tau + \mu_P)^2} \left\{ 1 + \frac{\eta\tau}{\mu_P} \right\} < 0; \frac{dR_0}{d\delta} \\ = -\frac{2a^2\beta_1\beta_2K^2\pi N_V^*N_P}{(\delta + \mu_V \pm \theta)^3(\omega + \tau + \mu_P)^2} \left\{ 1 + \frac{\eta\tau}{\mu_P} \right\} < 0$$

299 When $\theta > 0$, $\frac{dR_0}{d\theta} = -\frac{a^2\beta_1\beta_2K^2\pi N_V^*N_P}{(\delta + \mu_V + \theta)^2(\omega + \tau + \mu_P)^2} \left\{ 1 + \frac{\eta\tau}{\mu_P} \right\} < 0$ and when $\theta < 0$ then

$$\frac{dR_0}{d\theta} = \frac{a^2\beta_1\beta_2K^2\pi N_V^*N_P}{(\delta + \mu_V + \theta)^2(\omega + \tau + \mu_P)^2} \left\{ 1 + \frac{\eta\tau}{\mu_P} \right\} > 0; \frac{dR_0}{d\tau} = \frac{a^2\beta_1\beta_2K^2\pi N_V^*N_P(\eta\omega + \mu_P[\eta - 1])}{\mu_P(\delta + \mu_V \pm \theta)^2(\omega + \tau + \mu_P)^2} > 0$$

$$\frac{dR_0}{d\beta_1} = \frac{a^2\beta_2K^2\pi N_V^*N_P}{(\delta + \mu_V \pm \theta)^2(\omega + \tau + \mu_P)} \left\{ 1 + \frac{\eta\tau}{\mu_P} \right\} > 0; \frac{dR_0}{d\beta_2} = \frac{a^2\beta_1K^2\pi N_V^*N_P}{(\delta + \mu_V \pm \theta)^2(\omega + \tau + \mu_P)} \left\{ 1 + \frac{\eta\tau}{\mu_P} \right\} \\ > 0$$

$$\frac{dR_0}{dK} = 2 \frac{a^2\beta_1\beta_2K\pi N_V^*N_P}{(\delta + \mu_V \pm \theta)^2(\omega + \tau + \mu_P)} \left\{ 1 + \frac{\eta\tau}{\mu_P} \right\} > 0; \frac{dR_0}{da} = 2 \frac{a\beta_1\beta_2K^2\pi N_V^*N_P}{(\delta + \mu_V \pm \theta)^2(\omega + \tau + \mu_P)} \left\{ 1 + \frac{\eta\tau}{\mu_P} \right\} \\ > 0$$

300 The parameters which are greater than one are directly proportional to R_0 while those that are
 301 less than zero are inversely proportional to R_0 . Climatic factors affect immigration or
 302 emigration of insects vectors, their biting rate, infection rates and death induced rates.

303 4. Results and discussion

304 The expression for basic reproduction number(R_0) obtained in model analysis estimates the
 305 number of secondary exposed plants and/or infectious plant that would be obtained if one
 306 infectious plant or exposed plant is introduced into the completely susceptible plant population.
 307 If $R_0 > 1$, the disease would invade the plant population and reach endemic equilibrium
 308 point(EEP)and should worry stakeholders. If $R_0 < 1$,the disease would die out.If $R_0 = 1$, the
 309 number of infected plants would remain constant.

The DFEP was found to be locally stable whenever $R_0 < 1$, which means that any starting values are near DFEP, they would move toward DFE asymptotically over time. The conditions necessary for global stability of DFEP and EEP were obtained, inferring that if those conditions are satisfied, any values they take would move toward equilibrium points asymptotically over time.

5. Conclusion

In this paper, we formulated a non-linear deterministic model for plant vector borne diseases by incorporating temperature and precipitation under the biting rate and wind as an agent of immigration and emigration, obtained the conditions for positivity and boundedness, local and global stability of the solution. The expression of vector population at equilibrium point was obtained and incorporated in model to consider shorter life span of vector. The expression of basic reproduction number (R_0) was obtained. Basic reproduction number (R_0) estimates the number of secondary infection when one infected individual is introduced in completely susceptible population, this study carried sensitivity analysis to determine how various parameters in the model affects R_0 . Estimated numerical results, normalized sensitivity analysis and simulations to validate the model will be in our next paper. This study developed a deterministic model, future studies can consider stochastic model.

REFERENCES

- S.Z.Rida,M.Khalil,H.A.Hosham and S.Gadallah,Mathematical model of vector-Borne Plant Disease with Memory on the Host and the Vector.J.Progr.Fract.Differ.Appl.2, No.4,277-285(2016)
- F.Zhoi, and H.Yao, Global Dynamics of a Host-Vector-Predator Mathematical Model.J. of App: Mathematics vol 2014;59
- E.Miller and A.Huppert; The effects of Host Diversity on Vector-Borne Diseases: The condition under which diversity will amplify or dilute the disecive risk PLOS ONE 8 (11): e80279,doi:10.1371/journal.pone.0080279
- C.Petzoldt and Abby Seaman, climate change effects on insects Pathogens
- K.W.Okamoto and P.Amarasekare, The biological control of disease vectors.J. of Theoretical Biology 309(2012)47-57
- D.Fargette and K.Vie',Modeling of Temporal Primary Spread of African Cassava Mosaic Virus into Plantings,
- R.P.P Almeida, Ecology of Emerging Vector-Borne plant diseases
- Z.Lawrence and D.I.Wallace,The Spatio temporal dynamics of African Cassava Mosaic Disease
- A. A. Lashari and G.Zaman, "Global dynamics of vector-borne diseases with horizontal transmission in host population," computers Mathematics with Applications, Vol. 61, no> 4, pp.745-754, 2011.
- C.Petzoldt and Abby Seaman, climate change effects on insects Pathogens

347 D.Fargette and K.Vie', Modeling of Temporal Primary Spread of African Cassava Mosaic Virus
348 into Plantings,

349 E.Miller and A.Huppert; The effects of Host Diversity on Vector-Borne Diseases: The condition
350 under which diversity will amplify or dilute the disease risk PLOS ONE 8 (11): e80279,doi:10.
351 1371/journal.pone.0080279

352 F. Y. Zhou and H. X. Yao, "Dynamics and biocontrol: the indirect effects of a predator population
353 on a host-vector disease model," Abstract and Applied Analysis, Vol. 2014, Article ID 252718,14
354 pages, 2014

355 F.Zhoi, and H.Yao, Global Dynamics of a Host-Vector-Predator Mathematical Model.J. of App:
356 Mathematics vol 2014;59

357 H. Wei, X. Li, and M. Martchev, "An epidemic model of a vector borne disease with direct
358 transmission and time delay," Journal of Mathematical Analysis and Applications, Vol 342, no.2,
359 pp. 895-908, 2008.

360 Holt, J., et al., 1997. An epidemiological model incorporating vector population dynamics applied
361 to African cassava mosaic virus disease. J . Appl.Ecol.34, 793-806.

362 Jeger MJ,1983b. Analysing epidemics in time and space. Plant pathology32, 5-11.

363 Jeger MJ,1987a. Modelling the dynamic of pathogen populations. In: Wolfe MS, Caten CE, eds.
364 Population of plant pathogens: Their Dynamic and Genetics. Oxford, UK: Blackwell Scientific
365 Publications, 91-107.

366 Jeger MJ,1990. Mathematical analysis and modelling of spatial aspects of plants disease
367 epidemics. In: Kranz J, ed. Epidemics of plants Diseases. Berlin, Germany: Springer-Verlag
368 (Ecological studies, vol. 13, 2nd edn), 53-95

369 Jones DG,ed. , 1998. The epidemiology of plant diseases. Dordrecht. The Netherlands. Kluwer
370 Academic Publishers

371 K. W. Okamoto and P.Amarasekare, "The biological control of disease vectors," Journal of
372 Theoretical Biology, Vol. 309, pp. 47-57, 2012.

373 K.W.Okamoto and P.Amarasekare, The biological control of disease vectors.J. of Theoretical
374 Biology 309(2012)47-57

375 Kermack, W.O. and McKendrick, A.G 1927. A contribution to the mathematical theory of
376 epidemics. Proc. R.Soc.,Ser. A, 115:700-721.

377 Kranz J, ed. 1974, Epidemics of Plant Diseases: Mathematical Analysis and modelling. Berlin,
378 Germany:Spring-verlag.

379 L.Cai and X. Li, "Analysis of a simple vector-host epidemic model with direct transmission,"
380 Discrete Dynamics in Nature and society, vol. 2010, Article ID 679613, 12 pages, 2010.

381 Michael E, 1993. Mathematical Modelling of disease epidemiology. Parasitology Today 9, 397-9

382 Minogue KP, Fry WE, 1983a. Models for the spread of disease: model of description.
 383 Phytopathology 73, 1168-73

384 R.P.P Almeida, Ecology of Emerging Vector-Borne plant diseases

385 S. Moore, E. T. Borer and P. R. Hosseini, "Predators indirectly control vector-borne disease:
 386 Linking predator-prey and host pathogen models," Journal of the Royal Society Interface, vol.7,
 387 no. 42, pp. 161-176, 2009.

388 S.Z.Rida, M.Khalil, H.A.Hosham and S.Gadallah, Mathematical model of vector-Borne Plant Disease
 389 with Memory on the Host and the Vector. J.Progr.Fract.Differ.Appl.2, No.4, 277-285 (2016)

390 Otim-Nape GW/ Bua A, Thresh JM, Baguma Y, Ogwals,

391 Van der Plank J E, 1965, Dynamics of epidemics of plant disease Science 147, 120-4

392 Van der Plank J E, 1963, Plant disease: Epidemics and control, New York, USA: Academics press

393 Fitzgibbon, W.E., et al, 1995: Diffusion epidemic models with incubation and crisscross
 394 dynamics. Math.Biosci, 128, 131-155

395 Gourley, S.A. et al, 2007. Eradicating Vector-borne disease via age-structured culling. J.Math.Book
 396 54, 309-335

397 Junli Lui, Tailen Zhang, 2011. Global stability for a tuberculosis model computer modelling, 54
 398 (2011) 836-845.

399 Appendix 1: List of Variables and Parameters

Variables	Descriptions
$N_V(t)$	The total population of the vectors
$N_P(t)$	The total population of the plants
$S_P(t)$,	The population of susceptible plant
$E_P(t)$	The population of exposed plant
$I_P(t)$.	The population of infected plant
$S_V(t)$	The population of susceptible vectors
$I_V(t)$	The population of infected vectors
Parameters	Descriptions
π	The recruitment rate of vectors
μ_P	The recruitment rate of plants
ω	The rates at which $E_P(t)$ recover naturally to $S_P(t)$.
δ	The rate at which climate induced death occur in both $S_V(t)$ and $I_V(t)$
μ_P	The constant natural death rate in subclasses $S_P(t)$, $E_P(t)$ and $I_P(t)$
τ	The rate at which $E_P(t)$ progresses to $I_P(t)$.
μ_V	The constant natural death rate in subclasses $S_V(t)$ and $I_V(t)$
θ	The immigration and emigration rate of vector (this implies that the rate can be either be positive or negative).
β_1	The infection rates for plants
β_2	The infection rates for vectors
a	The vector biting rate

	λ_P	The number of individual plants which become infected at given time(force of infection of plants)
	λ_V	The number of individual plants which become infected at given time(force of infection of plants)
400	k	The rate of change of force of infection due to climatic factors. Assumption is $K \geq 1$

401 **Appendix 2: Definition of terms**

Term	Definition
Susceptible population	The populations who are free of infection but are at a risk of contracting the infection.
Exposed population	The populations that has contracted the infection but are at a lower risk of transmitting it and/or with no signs of infection
Infected population	The population with the disease causing pathogen and capable of transmitting the infection to other plants or vectors on contact
Basic reproduction number/ratio	The number of cases one generates on average over the course of infectious period in a completely susceptible population
Bifurcation	It occurs when a smooth change is made to parameter values causes the system to change its behavior
Disease free equilibrium point	It is infection free point
Endemic equilibrium point	The stationary point at which the disease has completely invaded the population

402