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Modeling Banana Xanthomonas Wilt with Protection

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Abstract. Banana Xanthomonas Wilt (BXW) is an infectious disease caused by Xanthomonas campestris pv. musacearum. The model incorporates a new class of protected banana plants into banana plant population. This new class are the susceptible banana plant that are treated with fertilizers. The basic reproduction number, R_o, is obtained using next generation matrix. The model analysis is done and equilibrium points are analysed to establish the local and global stability of disease-free and endemic equilibrium solution. It is shown that if the basic reproduction number, $R_o \leqslant 1$, then banana xanthomonas wilt is cleared from banana plantation and is globally asymptotically stable and if $R_o > 1$, the endemic equilibrium point is globally asymptotically stable and the disease persists in banana plant population. The impact of parameters in BXW model is investigated using sensitivity analysis. Numerical simulations are performed to justify the analytical findings.

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1. Introduction

Banana and plantain are perennial herbs, which belong to the Musa genius of the Musaceae family. They are cultivated in more than 120 countries throughout

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the tropics and subtropics [1].They are distributed mainly on margins of tropical rainforests [13]. Total annual world production of banana is estimated at 130 million tons and this makes it ranked the fourth most important food crop in the developing world after rice, wheat and maize [4].

Banana Xanthomonas Wilt is a vector-borne disease caused by Xanthomonas Campestris pv. Musacearum (xcm). The impact of BXW (Banana Xanthomonas Wilt) are both extreme and rapid, unlike those of other diseases that cause gradually increasing losses over years. The disease leads to absolute yield losses and death of the mother plant production cycles [12]. Following the first report of the disease in Uganda in 2001, a lot of research has been carried out and much data has been generated about the disease spread and control dynamics (See [6], [8], [10]) and references cited therein.

Mathematical models have been developed to model BXW dynamics in which one can predict through these models, the behaviour of the disease and control the particular epidemic. Nakakawa et al., [9] developed a deterministic model with optimal control in which the use of clean planting materials, debudding, disinfection of tools and roguing are taken into consideration in their model. They used Pontryagin's Maximum Principle to characterize and discuss possible control strategies that substantially reduce the infection levels of BXW within a plantation. In their paper, they did not consider a fraction of susceptible plant that are treated with fertilizers as this is an effective control strategy to reduce the BXW spread. Horub et al., [5] formulated a mathematical model for the vector transmission and control of Banana Xanthomonas Wilt by incorporating roguing of infected banana plants and replanting using healthy suckers without considering a new class of fraction of susceptible plants that are treated with fertilizers on the transmission dynamics.

This paper modifies and extends deterministic model developed by Horub et al., [5] by incorporating a new class of protected plants into the banana plant population. The new class of protected plants is the fraction of susceptible plants that are treated with fertilizers (Potassium, Calcium and Nitrogen). This is because exogenous application of potassium, calcium and nitrogen reduces susceptibility to xanthomonas wilt in banana plants [2]. The formulated model is then analysed theoretically using suitable Lyapunov function to establish both global stability of disease-free and endemic equilibrium points.

In addition to the introductory section, the paper has three more sections. Section 2 shows the mathematical formulation of the model. In Section 3, transformation of the model is presented. In Section 4, stability analysis of the model is carried out. Section 5 discusses the results and concludes the modeling work.

2. Materials and methods

In this section, a model for the spread of banana xanthomonas wilt in the banana plant population and vector population is formulated. The total plant population denoted by N_P is partitioned into three classes namely; the susceptible banana plants S_P, the infected banana plants I_P and the protected banana plants F_P and so that $N_P = S_P + I_P + F_P$. Also, the total vector population denoted by N_P, is sub-divided into two classes namely; the susceptible vector, S_V and the infected vector I_V. Thus the total population N_P and N_V for banana plants and vector population is given by $N_P = S_P + I_P + F_P$ and $N_V = S_V + I_V$. The parameters in Table 1 are chosen from the paper developed by Horub et al. [5].

Table 1. Summary of the parameters.

Parameters	Meaning	Value
μ_p	Death rate of infected banana plants	0.0167
ω	Emergence rate of new suckers	0.001
h	Harvesting rate of susceptible banana plants	0.0056
r	Roguing rate of infected banana plants which is also the replanting rate of healthy suckers	0.0105
ρ	Fraction of susceptible banana plants that are treated with fertilizer	0.5 (Assumed)
$(1-\rho)$	Remaining fraction of susceptible banana plants that are not treated with fertilizer	0.5 (Assumed)
α	Contact rate between susceptible banana plants and infected vector	0.021
θ	Contact rate between susceptible vector and infected banana plants	0.021
ω_v	Immigration or birth rate of vector	0.02
μ_v	Emigration or death rate of vector	0.02
m	The number of vector per banana plant	1

2.1 *Assumptions and decriptions of the model*

The following assumptions were made in order to formulate the equations of the model:

(a) The total banana plant population size is variable whereas the total vector population size is constant.
(b) There is no latency in both the host and vector populations and the transmission of the bacterium by the vectors is by non-circulative and non-persistent mode.
(c) The emigration and immigration rates of the vectors are equal so that the total vector population size is constant.
(d) The roguing rate of infected plant is balanced by the replanting rate of the healthy suckers.
(e) Fraction of susceptible banana plants that are treated with fertilizer move to the protected banana plant compartment.
(f) Remaining fraction of susceptible banana plants that are not treated with fertilizer move to the infected banana plant compartment.

Susceptible banana plants are recruited through two processes, namely, emergency of new suckers at a constant rate ω and replanting rate r using healthy suckers. Healthy plants are harvested at a rate h whose reciprocal is the life time of a healthy banana plant. Roguing of infected plant is done at a rate r which is also the replanting rate of healthy suckers. Infected banana plants die at a rate μ_p. Susceptible vector population is recruited through the immigration of vectors. The emigration rate of both classes of vectors is assumed to be equal to the immigration rate at a constant μ_v.

In the model, the term $\dfrac{\alpha S_P I_V}{N_P}$ denotes the rate at which the susceptible banana plants get infected by infected vector I_V and $\dfrac{\theta S_V I_P}{N_P}$ refers to the rate at which the susceptible vector are infected by the infected banana plant host.

Applying the assumptions, nomenclature of parameters and definitions of vari-

ables, the following system of ordinary differential equations is formulated:

$$\frac{dS_P}{dt} = \omega S_P - \frac{\alpha S_P I_V}{N_P} + rS_P - hS_P, \tag{1}$$

$$\frac{dI_P}{dt} = \frac{(1-\rho)\alpha S_P I_V}{N_P} - rI_P - \mu_P I_P, \tag{2}$$

$$\frac{dF_P}{dt} = \frac{\rho\alpha S_P I_V}{N_P}, \tag{3}$$

$$\frac{dS_V}{dt} = \omega_v N_v - \frac{\theta S_V I_P}{N_P} - \mu_v S_V, \tag{4}$$

$$\frac{dI_V}{dt} = \frac{\theta S_V I_P}{N_P} - \mu_v I_V. \tag{5}$$

3. Transformation of the model

It is convenient to use fraction of population instead of population number. This is done by dividing each population class by the total population and hence, we have

$$s_p = \frac{S_P}{N_P}, i_p = \frac{I_P}{N_P}, f_p = \frac{F_P}{N_P}, s_v = \frac{S_V}{N_V}, i_v = \frac{I_V}{N_V}, m = \frac{N_V}{N_P}.$$

Differentiating the fraction with respect to time t gives the following:

$$\frac{ds_p}{dt} = \phi s_p - \alpha m s_p i_v - \phi s_p^2 + \gamma s_p i_p, \tag{6}$$

$$\frac{di_p}{dt} = \alpha m s_p i_v - \rho\alpha m s_p i_v - \phi s_p i_p - \gamma i_p + \gamma i_p^2, \tag{7}$$

$$\frac{df_p}{dt} = \rho\alpha m s_p i_v - \phi s_p f_p + \gamma f_p i_p, \tag{8}$$

$$\frac{ds_v}{dt} = \omega_v(1 - s_v) - \theta i_p s_v, \tag{9}$$

$$\frac{di_v}{dt} = \theta i_p s_v - \omega_v i_v. \tag{10}$$

From the relation $s_p + i_p + f_p = 1$ and $s_v + i_v = 1$, it implies that $f_p = 1 - s_p - i_p$ and $s_v = 1 - i_v$ which reduces to the following system of differential equations:

$$\frac{ds_p}{dt} = \phi s_p - \alpha m s_p i_v - \phi s_p^2 + \gamma s_p i_p, \tag{11}$$

$$\frac{di_p}{dt} = \alpha m s_p i_v - \rho\alpha m s_p i_v - \phi s_p i_p - \gamma i_p + \gamma i_p^2, \tag{12}$$

$$\frac{di_v}{dt} = \theta i_p(1 - i_v) - \omega_v i_v. \tag{13}$$

where $\phi = \omega + r - h$ and $\gamma = r + \mu_p$.

For biological reasons, the model is analysed in the feasible region

$$T = \{(s_p, i_p, i_v) \in R_+^3 : s_p, i_p, i_v \leqslant 0, s_p + i_p = 1, 0 \leqslant i_v \leqslant 1\},$$

that can be shown to be positively invariant with respect to the system (11)-(13) where R^3_+ denotes the nonnegative cone of R^3 including its lower dimensional faces. We denote the boundary and the interior of T by ∂T and T respectively.

3.1 *Equilibrium point and basic reproduction number*

The equilibrium is gotten by setting the right hand side of (11)-(13) to zero and the system takes the form

$$\phi s_p - \alpha m s_p i_v - \phi s_p^2 + \gamma s_p i_p = 0 \tag{14}$$

$$\alpha m s_p i_v - \rho\alpha m s_p i_v - \phi s_p i_p - \gamma i_p + \gamma i_p^2 = 0 \tag{15}$$

$$\theta i_p(1 - i_v) - \omega_v i_v = 0 \tag{16}$$

where $E_o = (1, 0, 0)$.

The computation of the basic reproduction number R_o is needed in order to assess the local and global stability of disease-free equilibrium. This is obtained by expressing (6)-(10) as the difference between the rate of new infection in each infected compartment F and the rate of transfer between each infected compartment G. Hence, we have

$$\begin{bmatrix} \frac{di_p}{dt} \\ \frac{di_v}{dt} \end{bmatrix} = F - G = \begin{bmatrix} \alpha m s_p i_v - \rho\alpha m s_p i_v \\ \theta s_v i_p \end{bmatrix} - \begin{bmatrix} \gamma i_p + \phi s_p i_p + \gamma i_p^2 \\ \omega_v i_v \end{bmatrix}.$$

The Jacobian matrices J_F and J_G of F and G are found about E_0.

$$S = J_F J_G^{-1} = \begin{bmatrix} 0 & \frac{\theta}{\omega} \\ \frac{m\alpha - m\rho\alpha}{\phi + \gamma} & 0 \end{bmatrix}.$$

R_o is the maximum eigenvalue of S given as

$$R_o = \sqrt{\frac{\alpha m\theta - \rho\alpha m\theta}{K_T\omega_v}},$$

where $K_T = \gamma + \phi$, $\gamma = r + \mu_p$ and $\phi = \omega + r - h$.

3.2 *Local stability of disease-free equilibrium solution*

The Jacobian matrix of (14)-(16) is given as

$$J_E = \begin{bmatrix} -(\alpha m i_v + 2\phi s_p - \phi - \gamma i_p) & \gamma s_p & -\alpha m s_p \\ \alpha m i_v - \rho\alpha m i_v - \phi i_p & -A_T + 2\gamma i_p & \alpha m s_p - \rho\alpha m s_p \\ 0 & \theta(1 - i_v) & -(\theta i_p + \omega_v) \end{bmatrix} \tag{17}$$

where $A_T = \phi s_p + \gamma$
The Jacobian matrix evaluated at E_o is given by

$$J_{E_0} = \begin{bmatrix} -\phi & \gamma & -\alpha m \\ 0 & -(\phi+\gamma) & \alpha m - \rho\alpha m \\ 0 & \theta & -\omega_v \end{bmatrix} \tag{18}$$

One of the three eigenvalues are $-\phi$. The other two are obtained from the sub-matrix

$$J_{E_0} = \begin{bmatrix} -(\phi+\gamma) & \alpha m - \rho\alpha m \\ \theta & -\omega_v \end{bmatrix} \tag{19}$$

whose $trace(J_{E_0}) = -(K_T+\omega_v) < 0$ and $det(J_{E_0}) = 1 - \dfrac{\alpha m\theta - \rho\alpha m\theta}{K_T\omega_v} = 1 - R_o^2 > 0$ if $R_o < 1$.

Thus, E_o is locally asymptotically stable if and only if $R_o < 1$, and we have thus established the following Lemma.

Lemma 3.1 The disease-free equilibrium E_o is locally stable if $R_o < 1$ and unstable if $R_o > 1$.

3.3 *Global stability of disease-free equilibrium*

The following result investigates the global behaviour of the model as its solution trajectory approaches the equilibrium solution.

Theorem 3.1 The disease-free equilibrium E_o of (11)-(13) is globally asymptotically stable in T if $R_o \leqslant 1$ and unstable if $R_o > 1$.

Proof Consider the Lyapunov function $L = \theta i_p + K_T i_v$. Its time derivative is

$$\begin{aligned}
L' &= \theta\frac{di_p}{dt} + K_T\frac{di_v}{dt} \\
L' &= \theta(\alpha m s_p i_v - \rho\alpha m s_p i_v - \phi s_p i_p - \gamma i_p + \gamma i_p^2) + K_T(\theta i_p(1-i_v) - \omega_v i_v) \\
L' &= \alpha m\theta s_p i_v - \rho\alpha m\theta s_p i_v - K_T\omega_v i_v + \gamma\theta i_p^2 - \gamma\theta i_p + K_T\theta i_p - K_T\theta i_p i_v - \theta\phi s_p i_p \\
L' &= \alpha m\theta s_p i_v - \rho\alpha m\theta s_p i_v - K_T\omega_v i_v + \theta i_p(\gamma i_p - \gamma) + \theta i_p(K_T - K_T i_v) - \theta\phi s_p i_p \\
L' &= K_T\omega_v i_v\left(\frac{\alpha m\theta s_p - \rho\alpha m\theta s_p}{K_T\omega_v} - 1\right) + \theta i_p(\gamma i_p - \gamma) + \theta i_p(K_T - K_T i_v) - \theta\phi s_p i_p \\
L' &= K_T\omega_v i_v(R_o^2 s_p - 1) - \theta i_p(\gamma - \gamma i_p) - \theta i_p(K_T i_v - K_T) - \theta\phi s_p i_p \\
&\leqslant K_T\omega_v i_v(R_o^2 s_p - 1) \leqslant 0 \quad \text{if} \quad R_0 \leqslant 1
\end{aligned}$$

Therefore, $L' \leqslant 0$ for $R_o \leqslant 1$. One sees further that $(s_p, i_p, i_v) \to (1, 0, 0)$ as $t \to \infty$. Consequently, the largest compact invariant set in $\{(s_p, i_p, i_v) \in \Gamma : L' = 0\}$ is the singleton E_0 and by Lyapunov-Lasalle's Theorem [7], the disease-free equilibrium point is globally asymptotically stable in Γ if $R_o \leqslant 1$ and this completes the proof of the Theorem. ■

3.4 *Local stability of endemic equilibrium*

The dynamics of the model on a small scale when the pathogen is sustained in the population is examined by deriving the Jacobian matrix for the system (14)-(16) at the endemic equilibrium E^*. The matrix is derived as

$$J_{E^*} = \begin{bmatrix} \phi - \alpha m i_v^* - 2\phi s_p^* + \gamma i_p^* & \gamma s_p^* & -\alpha m s_p^* \\ \alpha m i_v^* - \rho\alpha m i_v^* - \phi i_p^* & -\phi s_p^* - \gamma + 2\gamma i_p^* & \alpha m s_p^* - \rho\alpha m s_p^* \\ 0 & \theta - \theta i_v^* & -\theta i_p^* - \omega_v \end{bmatrix}, \tag{20}$$

where s_p^*, i_p^* and $^*_v$ denote the quantities of susceptible bananas, the infected bananas and the amount of the infected vectors when the ecosystem is invaded by the pathogen.

Theorem 3.2 The endemic equilibrium of the model (14)-(16) is locally asymptotically stable in T if $R_o > 1$ and is unstable if otherwise.

Proof The endemic equilibrium of the system is locally asymptotically stable if all the eigenvalues of (19) have negative real parts. The characteristic polynomial in λ of (19) is given as

$$|J_{E^*} I - \lambda| = \begin{vmatrix} m_1 - \lambda & m_2 & -m_3 \\ m_4 & m_5 - \lambda & m_6 \\ 0 & m_7 & m_8 - \lambda \end{vmatrix}, \tag{21}$$

where $m_1 = \phi - \alpha m i_v^* - 2\phi s_p^* + \gamma i_p^*$, $m_2 = \gamma s_p^*$, $m_3 = \alpha m s_p^*$, $m_4 = \alpha m i_v^* - \rho\alpha m i_v^* - \phi i_p^*$, $m_5 = -\phi s_p^* - \gamma + 2\gamma i_p^*$, $m_6 = \alpha m s_p^* - \rho\alpha m s_p^*$, $m_7 = \theta - \theta i_v^*$, $m_8 = -\theta i_p^* - \omega_v$, and is evaluated as

$$a_0\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0, \tag{22}$$

where $a_0 = 1$, $a_1 = -m_1 - m_5 - m_8$, $a_2 = m_2(m_5 - m_8) + m_5 m_8 - m_2 m_4 - m_6 m_7$, $a_3 = m_2 m_4 m_8 + m_1 m_6 m_7 + m_4 m_7 m_8 - m_1 m_5 m_8$.

Following Routh-Hurwitz stability criteria outlined in [11], the endemic equilibrium of the system (3.9)-(3.11) is locally asymptotically stable if $a_1 > 0$, $a_2 > 0$, $a_3 > 0$, and $a_1 a_2 - a_0 a_3 > 0$. ■

3.5 *Global stability of endemic equilibrium*

To investigate the dynamics of banana xanthomonas wilt on a large scale when all the variables of the system coexist for the solution of the model, the analysis will be extended beyond only a small region around the equilibrium by examining the global stability of the non-zero equilibrium.

Theorem 3.3 The non-zero equilibrium of the system is globally asymptotically stable in T if $R_o > 1$.

Proof Suppose $s_p = s_p - s_p^*$, $i_p = i_p - i_p^*$ and $i_v = i_v - i_v^*$. Define a quadratic Lyapunov function k as

$$k(s_p, i_p, i_v) = b_1(s_p - s_p^*)^2 + b_2(i_p - i_p^*)^2 + b_3(i_v - i_v^*)^2, \tag{23}$$

where $b_1 > 0$, $b_2 > 0$, and $b_3 > 0$.

$$\frac{dk}{dt} = 2b_1(s_p - s_p^*)\frac{ds_p}{dt} + 2b_2(i_p - i_p^*)\frac{di_p}{dt} + 2b_3(i_v - i_v^*)\frac{di_v}{dt}. \tag{24}$$

We aim to show that $\frac{dk}{dt} < 0 \in T$ to establish that $R_0 > 1$.

$$\begin{aligned}\frac{dk}{dt} &= 2b_1(s_p - s_p^*)[\phi s_p - \alpha m s_p i_v - \phi s_p^2 + \gamma s_p i_p] \\ &+ 2b_2(i_p - i_p^*)[\alpha m s_p i_v - \rho\alpha m s_p i_v - \phi s_p i_p - \gamma i_p + \gamma i_p^2] \\ &+ 2b_3(i_v - i_v^*)[\theta i_p(1 - i_v) - \omega_v i_v]\end{aligned} \tag{25}$$

$$\begin{aligned}\frac{dk}{dt} &= [2b_1\beta(s_p - s_p^*)s_p + 2b_1\gamma(s_p - s_p^*)s_p i_p \\ &+ 2b_2(i_p - i_p^*)\alpha m s_p i_v + 2b_2\gamma(i_p - i_p^*)i_p^2 + 2b_3(i_v - i_v^*)\theta i_p] \\ &- [2b_1(s_p - s_p^*)\alpha m s_p i_v + 2b_1(s_p - s_p^*)\phi s_p^2 \\ &+ 2b_2(i_p - i_p^*)\rho\alpha m s_p i_v + 2b_2(i_p - i_p^*)\phi s_p i_p + 2b_2(i_p - i_p^*)\gamma i_p \\ &+ 2b_3(i_v - i_v^*)\theta i_p i_v + 2b_3(i_v - i_v^*)\omega_v i_v]\end{aligned} \tag{26}$$

$$\begin{aligned}\frac{dk}{dt} &= [2b_1\beta(s_p - s_p^*)^2 + 2b_1\gamma(s_p - s_p^*)^2(i_p - i_p^*) \\ &+ 2b_2\alpha m(i_p - i_p^*)(s_p - s_p^*)(i_v - i_v^*) + 2b_2\gamma(i_p - i_p^*)^3 + 2b_3\theta(i_v - i_v^*)(i_p - i_p^*)] \\ &- [2b_1\alpha m(s_p - s_p^*)^2(i_v - i_v^*) + 2b_1\phi(s_p - s_p^*)^3 \\ &+ 2b_2\rho\alpha m(i_p - i_p^*)(s_p - s_p^*)(i_v - i_v^*) + 2b_2\phi(s_p - s_p^*)(i_p - i_p^*)^2 + 2b_2\gamma(i_p - i_p^*)^2 \\ &+ 2b_3\theta(i_p - i_p^*)(i_v - i_v^*)^2 + 2b_3\omega_v(i_v - i_v^*)^2]\end{aligned} \tag{27}$$

If $\frac{dk}{dt} = X - Y$ then,

$$\begin{aligned}X =& 2b_1\beta(s_p - s_p^*)^2 + 2b_1\gamma(s_p - s_p^*)^2(i_p - i_p^*) + 2b_2\alpha m(i_p - i_p^*)(s_p - s_p^*)(i_v - i_v^*) \\ &+ 2b_2\gamma(i_p - i_p^*)^3 + 2b_3\theta(i_v - i_v^*)(i_p - i_p^*)\end{aligned}$$

and

$$\begin{aligned}Y =& 2b_1\alpha m(s_p - s_p^*)^2(i_v - i_v^*) + 2b_1\phi(s_p - s_p^*)^3 + 2b_2\rho\alpha m(i_p - i_p^*)(s_p - s_p^*)(i_v - i_v^*) \\ &+ 2b_2\phi(s_p - s_p^*)(i_p - i_p^*)^2 + 2b_2\gamma(i_p - i_p^*)^2 + 2b_3\theta(i_p - i_p^*)(i_v - i_v^*)^2 + 2b_3\omega_v(i_v - i_v^*)^2\end{aligned}$$

Hence, $\frac{dk}{dt} < 0$ and $R_0 > 1$ if $X < Y$. Also, $\frac{dk}{dt} < 0$ if $s_p = s_p^*$, $i_p - i_p^*$, and $i_v - i_v^*$. Therefore, the maximum invariant set in $[(s_p, i_p, i_v) : \frac{dk}{dt} = 0]$ is the singleton E^* and by LaSalle's invariant principle as in [3], E^* is globally asymptotically stable in T where E^* is the endemic equilibrium of the model. ■

4. Sensitivity analysis

In this section, we carried out sensitivity analysis of parameters of the model system (11)-(13) in order to determine the relative importance of the model parameters on the disease infection. To determine how best to reduce the infection, it is necessary to know the relative importance of the different factors responsible for the infections.

Sensitivity indices could be computed numerically so as to figure out parameters that have high impact on basic reproduction number R_0 and which of the parameters should be given preferential treatment by intervention strategies.

Analytically, sensitivity analysis on all parameters which account for disease dynamics is done using Chitnis et al (2008) approach, we compute sensitivity indices of the R_0 which measures initial disease infection and allow us to measure relative change in a state variable when a variable changes.

The normalized forward sensitivity index of a variable to a parameter is the ratio of the relative change in the variable to the relative change in the parameter. When the variable is a differentiable function of the parameter, the sensitivity index may be alternatively defined using partial derivatives.

Definition 4.1 The normalized forward sensitivity index of a variable, u, that depends differentiably on a parameter, p, is defined as:

$$N_p^u = \frac{\partial u}{\partial p} \times \frac{p}{u}$$

for $u \neq 0$.

Consequently, we derive analytical expression for the sensitivity index of R_0 as

$$N_{p_i}^{R_0} = \frac{\partial R_0}{\partial p_i} \times \frac{p_i}{R_0}$$

where $p_i, i \in \mathbb{N}$ denotes each parameter involved in R_0.
Using $R_o = \sqrt{\dfrac{\alpha m \theta - \rho \alpha m \theta}{K_T \omega_v}}$ where $K_T = \gamma + \phi$, $\gamma = r + \mu_p$ and $\phi = \omega + r - h$, we compute sensitivity index of each parameter with respect to the R_0, for instance:

$$N_\alpha^{R_0} = \frac{\partial R_0}{\partial \alpha} \times \frac{\alpha}{R_0} = 0.125546425 \times 0.036386811 = 0.004568234$$

We have Table 2 which summarizes the sensitivity indices of R_0 with respect to parameters $N_\theta^{R_0}, N_m^{R_0}, N_\rho^{R_0}, N_r^{R_0}, N_{\mu_p}^{R_0}, N_w^{R_0}, N_h^{R_0}$, and $N_{\omega_v}^{R_0}$.

Interpretation of sensitivity indices obtained in Table 2

The computed sensitivity indices on R_0 with respect to the involved parameters give insights to the model system proposed. Provided all parameters remain constant, most sensitive parameter is m (number of vector per banana plant) being the highest positive index. The indication is that if m increases by 100%, then R_0 increases by 150%. Thus, as R_0 continues to be higher, epidemic of the disease infection tends to occur. Similarly, sensitivity indices of α, θ show direct variation with respect to R_0. Precisely, increase in α, θ i.e (contact rate between susceptible banana plants and infected vector,contact rate between susceptible vector and

Table 2. Numerical values of sensitivity indices of R_0 with respect to parameter involved.

Parameter symbol	Sensitivity Index
α	+ 0.004568
m	+1.501134
θ	+0.004568
ρ	-0.00162
r	Complex number
μ_p	Complex number
w	Complex number
h	0.001529
ω_v	Complex number

infected banana plants respectively) increases R_0 more than h. There is decrease in R_0 when the ρ(fraction of susceptible banana plant that are treated with fertilizer) increases. On the other angle, r, μ_p, w, ω_v give complex number which is an indication that their sensitivity index is complicated due to some biological or environmental factors.

5. Numerical simulations of the model

In order to understand the overall picture of the disease behaviour, this section provides numerical simulations of each of the population classes using a Maple software package for plant and vector population. In addition, with the aid of figures, the results of the simulations are discussed. The parameter values used in the simulations are found in Table 1.

We perform the numerical simulations of the system of differential equations of the susceptible plants, infected plants and protected plants to determine the changes in the various populations of these compartments with time. There is a sharp decrease in the population of susceptible banana plant as the population of protected banana plant (fraction of susceptible plants that are treated with fertilizers) increases with time. Our findings show an inverse relationship between the susceptible banana plant population and protected banana plant population as shown in the diagram. This means that the susceptible banana plant population decreases as a result of increase in the population of protected banana plant. Protecting the susceptible banana plant population through treatment with fertilizers reduces their susceptibility to BXW. Also, there is a decrease in the magnitude of the infected banana plant population as the population of protected banana plant increases. This could be attributed to the population of susceptible banana plant and population of protected banana plant having an inverse relationship. Moreover, the susceptible vector population increases as a result of decrease in the magnitude of the infected vector population as indicated in the diagram.

6. Conclusion

We present a compartmental mathematical model describing the transmission of BXW between the interacting banana plant and vector populations. The model incorporates a new class of protected banana plant(fraction of susceptible banana plant that are treated with fertilizer), denoted by F_p, into the banana plant compartment. The disease-free and endemic equilibria are determined and their

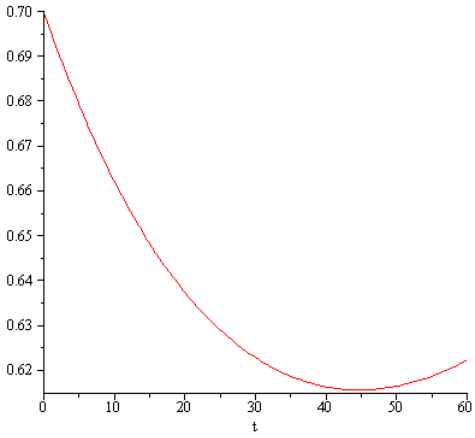

Figure 1. Graph of susceptible banana plant.

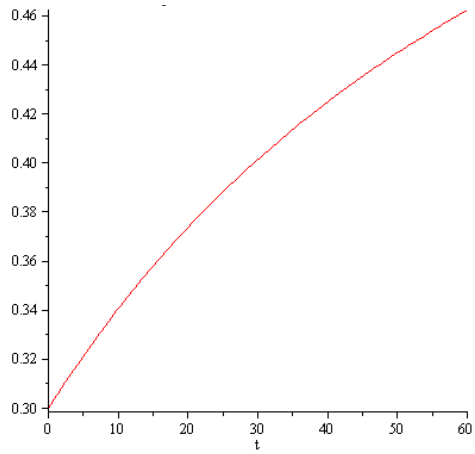

Figure 2. Graph of protected banana plant.

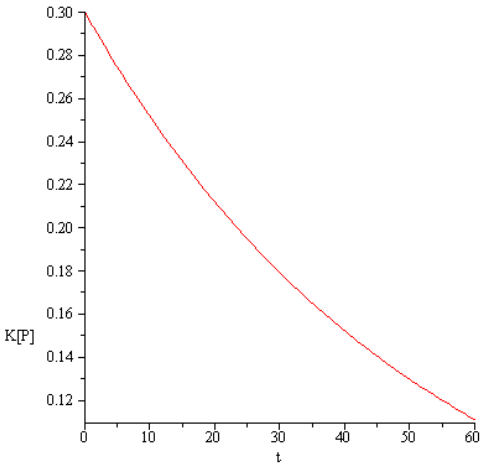

Figure 3. Graph of infected banana plant.

stability properties are investigated through an explicit formula for a threshold parameter, known as the basic reproduction number. In addition, sensitivity analysis of the model is carried out with a view to examining the factors most

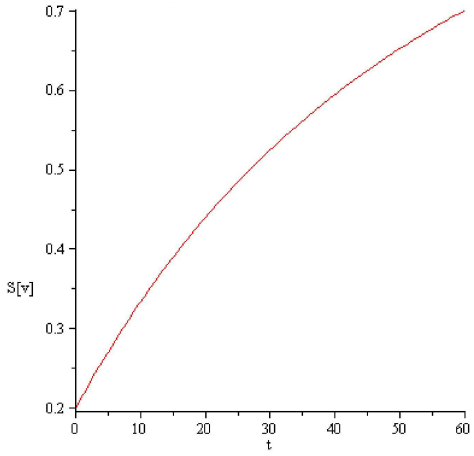

Figure 4. Graph of susceptible vector.

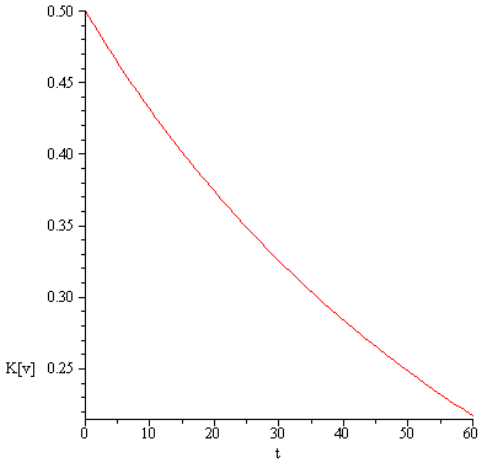

Figure 5. Graph of infected vector.

responsible for the transmission and spread of BXW. It is found that R_o is most sensitive to ρ (fraction of banana plants that are treated with fertilizers, i.e protected banana plants) in a negative sense. This means that increase in the fraction of susceptible banana plants that are treated with fertilizer, will bring R_o below unity, thereby curtailing the spread of BXW.

Previous efforts have focused attention on the roguing of symptomatically and asymptomatically infected plants but this study recommends that protecting the susceptible banana plants from being infected by applying fertilizers, is more effective in order to stop the spread of BXW. Efforts should be made by regional governments to produce fertilizers which can be sold to farmers at a cheaper price so that they can apply to susceptible banana plants which will protect them from being infected. Protection of susceptible banana plants, through application of fertilizers, is crucial in managing BXW as it reduces the spread of the pathogen.

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Appendix

The algorithms of the method used in carrying out the numerical simulations of the formulated model, in this manuscript, are displayed below:

```
> restart :
>
> a := 0.0167 : b := 0.5 : c := 0.02 : d := 0.021 : theta := 0.021 : f
      := 0.02 : g := 0.001 : h := 0.0056 : r := 0.0105 :
>
```

$$
\begin{aligned}
ODE1 := \Bigg(\Bigg[& \frac{\mathrm{d}}{\mathrm{d}t} S[P](t) = g - \frac{d \cdot S[P](t) \cdot K[v](t)}{S[P](t) + F[P](t) + K[P](t)} + r \\
& \cdot S[P](t) - h \cdot S[P](t), \frac{\mathrm{d}}{\mathrm{d}t} F[P](t) \\
& = \frac{b \cdot d \cdot S[P](t) \cdot K[v](t)}{S[P](t) + F[P](t) + K[P](t)}, \frac{\mathrm{d}}{\mathrm{d}t} K[P](t) \\
& = \frac{(1-b) \cdot d \cdot S[P](t) \cdot K[v](t)}{S[P](t) + F[P](t) + K[P](t)} - r \cdot K[P](t) - a \cdot K[P](t), \\
& \frac{\mathrm{d}}{\mathrm{d}t} S[v](t) = c - \frac{\mathrm{theta} \cdot S[v](t) \cdot K[P](t)}{S[P](t) + F[P](t) + K[P](t)} - f \cdot S[v](t) \\
& \frac{\mathrm{d}}{\mathrm{d}t} K[v](t) = \frac{\mathrm{theta} \cdot S[v](t) \cdot K[P](t)}{S[P](t) + F[P](t) + K[P](t)} - f \cdot K[v](t), \\
& S[P](0) = 0.7, F[P](0) = 0.3, K[P](0) = 0.3, S[v](0) = 0.2, \\
& K[v](0) = 0.8 \Bigg], numeric \Bigg) :
\end{aligned}
$$

```
> SOL := dsolve(ODE1, numeric, output = array([0, 0.2, 0.4, 0.6, 0.8,
      1, 400])) ;
```

$$SOL := \left[\left[\left[\begin{array}{cccccc} t & F_P(t) & K_P(t) & K_v(t) & S_P(t) & S_v(t) \end{array}\right]\right]\right],$$

```
[[[0., 0.29999999999999998, 0.29999999999999998,
0.80000000000000042, 0.69999999999999954,
0.20000000000000017],
[0.20000000000000001, 0.30090258341546688,
0.29927256200059670, 0.79700112189801948,
0.69908038196681554, 0.20299887810198022],
[0.40000000000000002, 0.30180112143933257,
0.29854503612409261, 0.79401673940305184,
0.69816795701972600, 0.20598326059694821],
[0.59999999999999997, 0.30269564207027971,
0.29781745106171603, 0.79104676874721668,
0.69726267623008997, 0.20895323125278339],
[0.80000000000000042, 0.30358617329464837,
0.29708983553308293, 0.78809112642833945,
0.69636449067237715, 0.21190887357166082],
[1., 0.30447274196496965, 0.29636221627978215,
0.78514973012230926, 0.69547335353723993,
0.21485026987769126],
[400., 0.58714278471261938, 0.00134954324679231372,
0.00194902796026884674, 0.20495878100991227,
0.99805097203973136]]]
```

```
> with(plots):
> SOL1 := dsolve(ODE1, numeric, range = 0..60):
> odeplot(SOL1, [[t, S[P](t)], [t, F[P](t)], [t, K[P](t)]]);
```