Effect of Time Delay on Management and Control of Banana Xanthomonas Wilt on Banana Production and Yield

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Abstract: - Banana is a main crop in the livelihoods of numerous people in the Great Lakes region of East and Central Africa. In Uganda, banana plants have been threatened by Banana Xanthomonas Wilt (BXW), which has spread at varying rates over the past two decades. The disease attacks all banana cultivars and may result in 100 % yield loss at the farm level if existing control measures are not improved. In this paper, we propose a mathematical model to study the effect of time delay on the management and control of BXW on banana production and yield. Differential equations are developed and analyzed for disease-free and endemic equilibria. The formula of Cardino was used to find the roots for the third polynomial expression. The next-generation matrix method was used to compute the reproductive number for control. The results revealed that increased time delay in controlling the BXW leads to low banana production. Yet timely control of Banana Xanthomonas Wilt improves banana yield.

Key-Words: - Banana Xanthomonas Wilt, Production, Time delay, Stability analysis, Hopf bifurcation , formula

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1 Introduction

Banana is a food and cash crop that is vegetatively propagated. Banana (dessert bananas, cooked bananas and plantains) is the sixth on the list of staple crops in the world, with a global production of around 86 million tons per year [1]. Bananas' ability to produce fruits all year round makes it a vital food and cash crop in the tropics as it contributes to food safety and income generation to farmers [2], [3]. Banana Xanthomonas Wilt (BXW) was first reported in Uganda in September 2001 [4] and by August 2004, the disease had been confirmed in 21 districts in Northern, Eastern and Central Uganda. Recent reports indicate that BXW had destroyed 90% of the bananas in Uganda, in the past decade, with significant outbreaks reported in central and western regions which produce 70% of the bananas consumed in Uganda [5].

BXW is among banana bacterial diseases caused by Xanthomonas Campestris pv Musacearum (Xcm), the bacteria can survive in soil and plant debris [6], [7]. The BXW has been observed in nearly all cultivated banana cultivars. The bacterial disease is transmitted by birds like bats, insect vectors through the inflorescence or by soil-borne bacterial inoculum through the lower parts of the plant, unclean farming tools, and infected suckers used for setting up new plantings [8]. Disease symptoms include: yellowing and wilting of leaves, premature ripening with reddish brown discoloration in banana fingers, and rotting of fruit, male bud wilting, yellow ooze observed on the cross-section cut of the pseudostem, internal yellow discoloration of the vascular bundles, and finally complete death of the plant sucker [9], [10]. The fruits in affected plants cannot be eaten by either man or animals.

BXW is a big threat to both household and National food security in Uganda, where bananas are a major food crop and where 75% of the farmers are engaged in banana production [11]. BXW is an important emerging and non-curable infectious plant pathogen in Sub-Saharan Africa that can cause up to 100% yield loss at farm level if effective control measures are not put in place and Uganda loses up to $299.6m worth of banana output at farm get price annually due to BXW [12]

Despite the role played by bananas in providing food security and income to families that practice agriculture, production continues to be low due to some limitations, including diseases and pests. The BXW causes up to 100% yield loss, negatively impacting sustainable access to food and income to more than 100 million banana farmers globally. At this scale of loss, it implies that the disease has the potential to cause a total demise of the banana crop (an important feed for both man and animals) in Uganda.

Bacterial diseases like banana Xanthomonas wilt are difficult to control as no effective chemicals are available for their control and the use of antibiotics is not recommended [13]. Developing host-plant resistance has been the best and most cost-effective method of managing bacterial diseases since the use of transgenic technologies may provide a timely and cost-effective measure to address the dangers of the spread of BXW disease [13]. Management and control of the disease involve methods that reduce the inoculum's density and spread of the pathogen. Removal of the male bud (debudding) has proven to be very effective in preventing the disease incidence since the male bud appears to be the primary infection site [10].

According to Ochola et al.[14] the decline in banana productivity in western Kenya was exacerbated by Xanthomonas wilt caused by Xanthomonas campestris pv. musacearum. The study provided insight of household dynamics influencing efforts to eradicate the disease from Ugunja, Lunjre, Sidindi and Sigomere. Nkuba et al.[15] assessed the impact of BXW on farmers' livelihoods in the Kagera basin of Tanzania, Burundi and Rwanda. The banana production losses resulted in a significant reduction in household food security and incomes which forced most households to diversify into other food crops such as maize, cassava and sweet potatoes.

Mathematical models have proved to be powerful tools in examining infection propagation in plant populations. Of date research on plant diseases with time delay in the incubation and infectiousness has yielded good results in the control of pathogens for instance [16], [17]. To this end we propose a mathematical model of the form S-A-I (Susceptible, Asymptomatic and Symptomatic). A discrete delay is incorporated in the symptomatic class

2 Description of the Mathematical Model (S-A-I) with Time Delay in the Symptomatic Sub-population

We consider the Banana plant population that is divided into three classes namely: X(t), the number of healthy banana plant at time t, Y (t) the number of asymptomatic banana plant at time t and Z(t) the number of symptomatic banana plant at time t. We assume the continual harvesting rate h to apply to all banana plant subpopulations. The healthy plant sub-population is increased at a replenishment rate Λ of emerging suckers only. The healthy banana plants decrease through continuous harvesting at a constant harvesting rate h. Healthy banana plants are infected through contact with infected plants, insects and equipment, at saturation incidence rate of predator-prey Holling type II, since vectors that spread Xcm bacteria can only select/bite a limited number of plants [18]. We assume the first symptoms of Xcm bacteria take time, thus we incorporate the asymptomatic plant sub-population [19]. The study by Ocimati et al.[20] confirmed the systemicity of Xcm, with the pathogen able to live within the mat for long periods (5−16) months without causing disease. The asymptomatic sub-population is further reduced through a progression rate γ. The density of infected banana plants is assumed to reduce through disease-induced rate, δ and roguing rate, ν. Since it may take time for the bacteria to enter the plant cell and to spread in the plant and it takes time for farmers to remove the infected plants, a discrete time delay is considered; τ, the time it takes a farmer to rogue infected plants from the banana plantation. We assume an infected plant to be eradicated from the field by the farmer as soon as the disease symptoms manifest on the plant if the disease is to be eradicated.

Table 1. Description of state variables

Variable	Description
$X(t)$	Density of healthy banana plant at time, t
$Y(t)$	Density of asymptomatic banana plant at time, t
$Z(t)$	Density of symptomatic banana plant at time, t
$N(t)$	Density of banana population at time, t; $N(t) = X(t) + Y(t) + Z(t)$

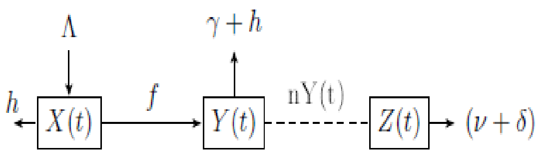


Fig. 1: A schematic diagram for Healthy-Asymptomatic-Symptomatic Model for Banana Plants

Table 2. Description of model parameters and their respective values

Parameter	Description	Value/unit	Ref
Λ	Replenishment of new banana suckers	2 day^{-1}	assumed
β	Contact rate of Xcm bacterium with healthy banana plant	2.032 day^{-1}	[21]
n	Proportion of Xcm bacteria released from asymptomatic banana	0.045 day^{-1}	assumed
γ	Progression rate to the symptomatic class	0.018 day^{-1}	[13]
a	Saturation constant of bacteria	8	assumed
h	Continual harvesting rate of banana plants	0.0714 day^{-1}	[22]
δ	Excess death due to Xcm bacteria	0.0167 day^{-1}	[23]
ν	Eradication rate of infected banana plants	1.0 day^{-1}	[24]
τ	Time delay in roguing infected banana plants	28 days	assumed

where $f = \frac{X(t)Z(t)}{a+X(t)}$ is a saturation functional incidence of Holling type II. In this mathematical model, the flows are governed by a system of delay differential equations that will numerically be integrated. This formulated model follows the mass-action law of kinetics normally applied in epidemiological models [25], thus the system of equations are given as:

$$
\begin{aligned}
\dot{X}(t) &= \Lambda - \frac{\beta \; X(t)Z(t)}{a+X(t)} - hX(t), \\
\dot{Y}(t) &= \frac{\beta \; X(t)Z(t)}{a+X(t)} - (\gamma+h)Y(t), \\
\dot{Z}(t) &= nY(t) - (\delta+\nu)Z(t-\tau), \\
\dot{N}(t) &= \Lambda - (hX(t) + AY(t) + BZ(t)) \qquad (1)
\end{aligned}
$$

.with A = γ + h − n, B = δ + ν and initial conditions

$X(\varphi) = g_1(\varphi) > 0,$ (2)

$Y(\varphi) = g_2(\varphi) > 0,$ (3)

$Z(\varphi) = g_3(\varphi) > 0.$ (4)

With $g_j(\varphi) \geq 0, \varphi \in [-\tau, 0]$, $g_j(0) > 0$, for j = 1,2,3.

Note: $(g_1(\phi), g_2(\phi), g_3(\phi) \in C([-\tau, 0], \mathbb{R}^3_+)$ where C is a Banach space of a continuous function mapping the interval [−τ, 0] into R^3. The feasible region of the model with the condition stated above is

$$\Phi = \{(X(t), Y(t), Z(t)) \in \mathbb{R}^3_+ | X(t) + Y(t) + Z(t) \leq \frac{\Lambda}{H}\}$$

,

we suppose H ≤ min{h, A, B}.

Lemma 2.1: The solutions to model system (1) with initial condition (4) remain positive and bounded for all

t ≥ 0.

Proof. Let (X (t), Y (t)), Z(t) be solutions to model system (1) with initial condition (4). Considering the first equation of system (1), we have

$$
\begin{aligned}
\dot{X}(t) &= \Lambda - \frac{\beta \; X(t)Z(t)}{a+X(t)} - hX(t) \\
\dot{X}(t) &> -(k+h)X(t). \qquad , \qquad (5)
\end{aligned}
$$

With $k = \frac{\beta \; Z(t)}{a+X(t)}$. From the second equation of system (3.12), the variables are separated and integrating both sides we obtain

$X(t) > X_0 e^{-(k+h)t} > 0.$ (6)

Therefore, from initial condition (4) X (t) > 0 for all t ≥ 0.

We also consider the second equation of the model system (1),

$$
\begin{aligned}
\dot{Y}(t) &= \frac{\beta \; X(t)Z(t)}{a+X(t)} - (\gamma+h)Y(t) \\
&> -(\gamma+h)Y(t).
\end{aligned}
$$

(7)

By separating the variables in equation (7) and using initial condition (4) , we have

$Y(t) > Y_0 e^{-(\gamma+h)t} > 0.$

Thus, from the initial condition (4), Y (t)>0 for all t≥0. To prove the solution of Z(t), let t_0 >0 be the first time such that Z(t0) ≡ 0. The first equation of the model system (1) shows that at t= t_0, we obtain

$\dot{Z}(t_0) = nY(t_0) - (\delta + \nu)Z(t_0 - \tau)$
$= nY(t_0) \geq 0$………………………… (8)

This implies that $Z(t) \geq 0$ for all $t \geq 0$ is always positive.

From the above expressions, we established that solutions of X(t),Y (t) and Z(t) are positive and biologically, there are chances of population survival at $t \geq 0$.

To prove for ultimate boundedness: If there is no disease in the population i.e. (Y (t) =Z (t) =0) the time derivative for the healthy banana plants is given as

$\dot{X}(t) = \Lambda - hX(t)$,…………………(9)

this implies that $\limsup_{t\to+\infty} X(t) \leq \frac{\Lambda}{h}$.

From equation four of model system (1), we have

$\dot{N}(t) = \Lambda - HN(t)$.

where $H = \min\{h, E, D\}$. Therefore $N(t) < \frac{\Lambda}{H} + \zeta$, for $\zeta > 0$ for all $t \geq 0$, implies that there exists a $q_0 > 0$ such that $Y(t) \leq q_0$. Then, the third equation of model system (1) is given as

$\dot{Z}(t) = nq_0 - (\delta + \nu)Z(t)$ ……… (10)

if $Y(t) \leq q_0$ and Z(t) is positive for $\tau = 0$, this implies that $\limsup_{t\to+\infty}(Z(t) \leq \frac{nq_0}{\delta+\nu})$.

Therefore, ∃ an $\omega > 0$ such that $X(t) < \omega$, $Y(t) < \omega$ and $Z(t) < t_0$.

This completes the proof.

3 Analysis of the Model

3.1 Analysis of the steady states

Proposition 3.1: The model system (1) has two non-negative steady states:

(i) The uninfected steady state

$E_0 = (X^* = \frac{\Lambda}{h}, Y^* = 0, Z^* = 0)$ exists biologically for $h > 0$

(ii) The infected steady state $E_1(X^*, Y^*, Z^*)$ where:

$$X = \frac{a}{\frac{n\beta}{(h+\lambda)(\nu+\delta)} - 1}$$

$$Y = \frac{n\beta\Lambda - (\delta+\nu)\big(\Lambda(h+\gamma) + \text{ha}(h+\nu)\big)}{(h+\gamma)\big(n\beta - (h+\gamma)(\nu+\delta)\big)}, \quad (11)$$

$$Z = \frac{n\left[\frac{n\beta\Lambda}{(\delta+\nu)(h+\gamma)} - (\Lambda + \text{ha})\right]}{n\beta - (h+\gamma)(\nu+\delta)}$$

The infected steady state biologically exists provided $(h + \gamma)(\nu + \delta) < n\beta$.

3.2 Uninfected steady state

In this Section, we give an overview of the local stability of the uninfected steady state E_0.

The banana bacterial free state occurs at a point where there is no infection within the plants, therefore, from model system (1) we obtain the uninfected state; $E_0 = (X^* = \frac{\Lambda}{h}, Y^* = 0, Z^* = 0)$. The biological interpretation of this steady state indicates that there is no disease propagation within the banana plantation. The linearized stability of the steady state of model system (1) is found by considering the Jacobian matrix $J|_{(E0)}$ which is an n × n matrix, and is obtained as

$$J|_{E_0} = \begin{pmatrix} -h & 0 & -\frac{\beta\ \Lambda}{\Lambda+ah} \\ 0 & -(\gamma+h) & \frac{\beta\ \Lambda}{\Lambda+ah} \\ 0 & n & -(\delta+\nu)e^{-\lambda\tau} \end{pmatrix} \quad (12)$$

From the Jacobian given in system (12), the third-degree transcendental polynomial is given as

$$L(\lambda,\tau) = \lambda^3 + a_2\lambda^2 - a_1\lambda + a_0 + (b_2\lambda^2 + b_1\lambda + b_0)e^{-\lambda\tau} = 0 \quad (13)$$

$$a_2 = (\lambda + 2h), a_1 = \frac{n\beta\Lambda}{\Lambda+\text{ah}},$$

$$a_0 = \frac{-\text{nh}\beta\Lambda}{\Lambda+\text{ah}}, b_2 = (\delta+\nu), b_1 = (\gamma+2h)(\delta+\nu),$$

$$b_0 = h(\delta+\gamma)(\gamma+h)$$

Clearly $a_j, b_j \in R$ (j = 0,1,2) and $\sum_{j=0}^{2} b_j^2 \neq 0$.

Theorem 3.1. The disease-free steady state E_0 is locally asymptotically stable if $R_0 < 1$ and unstable for $R_0 > 1$.

Proof. Suppose $\tau = 0$ then system (13) reduces to
$L(\lambda, \tau = 0) = \lambda^3 + m_2\lambda^2 - m_1\lambda + m_0 = 0$ (14 where:

$$m_0 = \frac{h}{\Lambda + \text{ah}}$$

Therefore system (14) has Eigenvalues given as;

$$\lambda_1 = -h$$

$$\lambda_2 = -\frac{1}{2}\big((\delta+\nu) + (\gamma+h)\big) - \frac{1}{2}\sqrt{\ }$$

(15)

The disease free steady state E_0 is locally stable since all eigenvalue solutions (14) have negative real roots and this will hold if $\lambda_3 < 0$ whenever $R_0 = \frac{n\beta\Lambda}{(\Lambda+\text{ah})(\delta+\nu)(\gamma+h)} < 1$. This implies that with $\lambda_{1,2,3} < 0$ then the disease-free steady state is locally stable. However, if atleast one of the eigenvalues $\lambda_3 > 0$ (positive) then $R_0 = \frac{n\beta\Lambda}{(\Lambda+\text{ah})(\delta+\nu)(\gamma+h)} > 1$ thus the disease-free steady state is unstable. This completes the proof.

3.3 Third-degree transcendental polynomial, τ >0

From the third-degree transcendental polynomial (13), we define
Q(λ,τ) = L(λ,τ) and we state the lemma.

Lemma 3.1:

For any time delay, τ > 0 we let J(τ) = #{λ : R(λ) ≥ 0,L(λ,τ) = 0} to denote the number of roots with positive real part. Let $0 \leq \tau_1 < \tau_2$ with $\tau \in [\tau_1,\tau_2]$ then there would be no roots to the system (13) on the imaginary axis. Therefore $J(\tau_1) = J(\tau_2)$.

We want to find out if the real part of a specific root $\lambda = \pm i\beta_0$, with $\beta_0 > 0$, may increase and ultimately become positive as time delay τ is varied. Taking $\lambda = i\beta_0$ as a root to system (13) then with the use of Euler formula we get

$$(a_2\beta_0^2 + a_0 + A\cos\beta_0\tau + B\sin\beta_0\tau) + i(B\cos\beta_0\tau - A\sin\beta_0\tau - a_1\beta_0 + \beta_0^3) = 0 \quad (16)$$

Where: $A = b_0 - b_2\beta_0^2$ and $B = b_1\beta_0$. By separating the imaginary and real parts of system (16) gives

$$\begin{aligned} A\cos\beta_0\tau + B\sin\beta_0\tau &= -(a_0 + a_2\beta_0) \\ B\cos\beta_0\tau - A\sin\beta_0\tau &= a_1\beta_0 + \beta_0^3 \end{aligned} \quad (17)$$

A Hopf frequency β_0 is got by squaring and summing up equations in system (17) thus we obtain

(18)

Where
$c_2 = a_2^2 - b_2^2, c_1 = 2(a_0a_1 + b_0b_1 + a_1) + (a_1^2 + b_1^2), c_0 = a_0^2 - b_0^2$
.Introducing an auxiliary value $u = \beta_0$ to system (18) we obtain

$$f(u) = u^3 + c_2u^2 + c_1u + c_0 = 0 \quad (19)$$

3.4 Finding roots to Eq.19 using Cardano's formula [26]

We reduce Eq.19 by removing the quadratic term by letting $u = \Phi - \frac{c_2}{3}$ and substituting u we get
$(\Phi - \frac{c_2}{3})^3 + c_2(\Phi - \frac{c_2}{3})^2 + c_1(\Phi - \frac{c_2}{3}) + c_0 = 0$
upon expansion we have

$$\Phi^3 + 3s\Phi + r = 0, \quad (20)$$

Where $s = \frac{3c_1 - c_2^2}{9}$ and $r = \frac{2c_2^3}{27} + c_0 - \frac{c_1c_2}{3}$.
Solving Eq.20, let Φ = x + y, x, y ∈ C and making a substitution we get

$$(x + y)^3 + p(x + y) + r = 0,$$

where p = 3s. Upon expansion, we get

$$g(x,y)=(x^3 + y^3 + r)(x + y)^3 + (3xy + p)(x + y) = 0 \quad (21)$$

The function $g(x,y) = 0$ if

$$x^3 + y^3 + r = 0, \quad (22)$$

$$3xy + p = 0. \quad (23)$$

Solving system (22) & (23) for x, by substitution method Eq.22 reduces to

$$27(y^3)^2 + 27ry^3 - p^3 = 0 \quad (24)$$

hence,

$$x^3 = -\frac{r}{2} \mp \sqrt{\frac{r^2}{4} + \frac{p^3}{27}},$$
$$y^3 = -\frac{r}{2} \pm \sqrt{\frac{r^2}{4} + \frac{p^3}{27}} \quad (25)$$

(26)

and

$$x = \sqrt[3]{-\frac{r}{2} \mp \sqrt{\frac{r^2}{4} + \frac{p^3}{27}}}, \ and \ y = \sqrt[3]{-\frac{r}{2} \pm \sqrt{\frac{r^2}{4} + \frac{p^3}{27}}}$$

It should be noted that x and y are principal roots of Eq.22, we claim a pair of primitive roots from Table 3. as solutions thus
$\{\Phi_1 = x_0+y_0, \Phi_2 = x_0\theta+y_0\theta^2, \Phi_3 = x_0\theta^2 + y_0\theta\}$, with $\theta = e^{\frac{2\pi i}{3}}$ and.$\theta^2 = e^{\frac{4\pi}{3}i}$

Table 3. Description of all possible solutions to Eq.21

x_0	$x_0\theta$	$x_0\theta^2$
(y_0,x_0)	$(y_0,x_0\theta)$	$(y_0,x_0\theta^2)$
$(y_0\theta,x_0)$	$(y_0\theta,x_0\theta)$	$(y_0\theta,x_0\theta^2)$
$(y_0\theta^2,x_0)$	$(y_0\theta^2,x_0\theta)$	$(y_0\theta^2,x_0\theta^2)$

Lemma 2. Let condition $\frac{\beta_0(b_1+b_2)}{b_0} < 1$ hold, the transcendental Eq.13 associated with a duo of purely imaginary roots $\lambda = \pm\beta_0 i$ at a series of critical values is given by
$\tau_c^k = \frac{1}{\beta_0}\left[\arctan(\frac{D_0}{D}) + (k-1)\pi\right] \ c = (1,2,3); k \in \mathbb{N}$.
where$D_0 = \frac{B(a_0+a_2\beta_0)+A\beta_0(a_1+\beta_0^2)}{A^2-B^2}$
and$D = \frac{B(a_0+a_2\beta_0)+A\beta_0(a_1+\beta_0^2)}{A^2-B^2}$.

Proof.

We solve Eq.17 for $\sin\beta_0\tau$ and $\cos\beta_0\tau$ by elimination method to obtain

$$\begin{aligned} \sin\beta_0\tau &= \frac{B(a_0+a_2\beta_0)+A\beta_0(a_1+\beta_0^2)}{A^2-B^2} \\ \cos\beta_0\tau &= -\frac{B(a_0+a_2\beta_0+\mathrm{BD})}{A} \end{aligned} \quad (27)$$

From system 27 we have (3.12)
$\tau_c^k = \frac{1}{\beta_0}\left[\arctan(\frac{D_0}{D}) + (k-1)\pi\right], c = (1,2,3); k \in \mathbb{N}.$

We note that for τ_c^k to be defined, then $A^2 > B^2$ substituting for A (3.13) and B we get $b_0 - b_2\beta_0 > b_1\beta_0$ that reduces to $\frac{\beta_0(b_1+b_2)}{b_0} < 1$.

3.4 Reproduction Number, R_0 for BXW

The basic reproduction number, R_0 is the average number of secondary infection cases produced by an infectious banana plant throughout its entire infectious period. The next generation matrix method is used to compute the well-known threshold R_o. We have only two infected sub-populations ie the asymptomatic sub-population and the symptomatic population.

$$F = \begin{pmatrix} 0 & a_1 \\ 0 & 0 \end{pmatrix}, V = \begin{pmatrix} b & 0 \\ c & d \end{pmatrix}, V^{-1} = \begin{pmatrix} \frac{1}{b} & 0 \\ -\frac{c}{bd} & \frac{1}{d} \end{pmatrix}$$

with

$a_1 = \frac{\beta\ x^*}{a+x^*}, b = \gamma + h, c = -n, d = (\delta + \nu)$.

The reproduction number

$$R_0 = \rho(\mathrm{FV}^{-1}) = \frac{n\beta\Lambda}{(\mathrm{ah}+\Lambda)(\gamma+h)(\delta+\nu)}. \quad (28)$$

When $R_0 < 1$, the disease-free state is stable, and BXW will die out. However, if $R_0 > 1$, an endemic equilibrium is established, and BXW will persist. To mitigate this, farmers should prioritize continuous removal of infected plants to reduce R_0 and prevent disease spread on the farm.

4 Sensitivity analysis of R_0

In this section, a sensitivity analysis of the basic reproduction number with respect to the parameter values is computed in Appendix I. We observe that sensitive numbers are; Δ_Λ,Δ_β,Δ_n are positive, whereas Δ_ν,Δ_δ,Δ_γ,Δ_h,Δ_a are negative.

Table 4. Sensitivity indices of R0 in Eq.28 to parameters for the H-A-S model

Parameter	Sensitivity index
h	-1.0208
β	1.0000
n	1.0000
ν	-0.9836
a	-0.2222
Λ	0.2221
γ	-0.2013
δ	-0.0164

The parameters are well-ordered from utmost sensitive to least. Overall, the utmost sensitive parameter is the continual harvesting rate of banana plants, h and the least sensitive parameter is excess death due to Xcm bacteria δ.

Given $\Delta\beta$, $\Delta_n = 1.0000$, R_0 is equally sensitive to transmission rate and Xcm bacteria release, increasing by 100% with a 1% rise in both parameters. To mitigate disease spread, farmers should reduce transmission via the removal of infected plants, vector control (e.g., weevils), and use of resistant banana varieties. We observe that sensitive numbers are; Δ_Λ and, Δ_δ,

Considering $\Delta_\nu = -0.9836$ implies 1% increase in the eradication rate of infected banana plants leads to 98.36% decrease in R_0. This is a key BXW control that is highly influential in producing high quality yields in banana production. A 1% increase in γ (roguing rate) decreases R_0 by 20.13% ($\Delta_\gamma = -0.2013$), highlighting the importance of promptly removing symptomatic plants to curb disease spread. Swift roguing is key to reducing transmission

Δ_h, a sensitivity index of -1.0208 indicates that a 1% increase in harvesting rate reduces R_0 by 102.08%, suggesting that removing infected banana plants significantly curbs disease spread. Early symptom detection, safe disposal of infected plants, and regular field monitoring are crucial. The negative Δ_h values support the impact of harvesting on reducing disease transmission.

A sensitivity index of Δ_a is negative, meaning an increase of 1% indicates R_0 decreases by 22% which means less bacterial growth and implication of less disease spread.

R_0 is very sensitive to the release of bacteria which means if proportion of infectious asymptomatic bananas increase at 1%, then R_0 increases at 100%. This is a big impact that requires intervention, such a situation is very tricky because no symptoms are seen. There is need for regular surveillance of the farm so that there is early detection of the affected plants. Additional consistently removing infected plants as soon as possible and checking farms on a weekly basis could reduce on disease spread.

5 Numerical Simulation

With parameter values shown in Table 1, the endemic equilibrium is shown. Figure 2 below shows the banana trajectory with initial values of the plantation ((X(t), Y(t), Z(t)) at (100, 20, 10)).

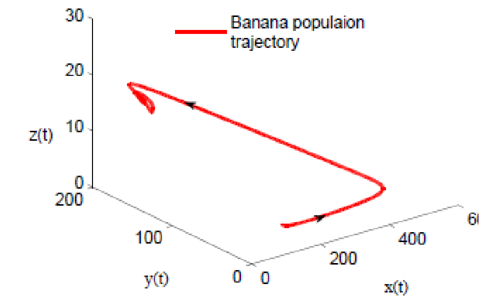
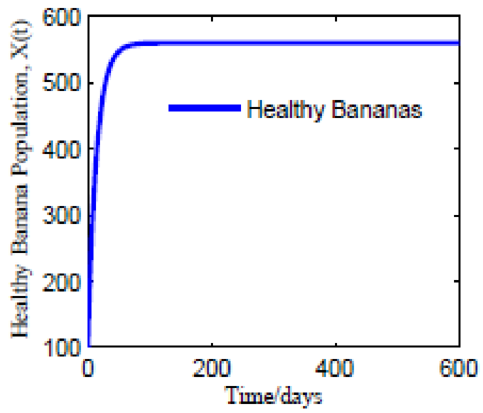
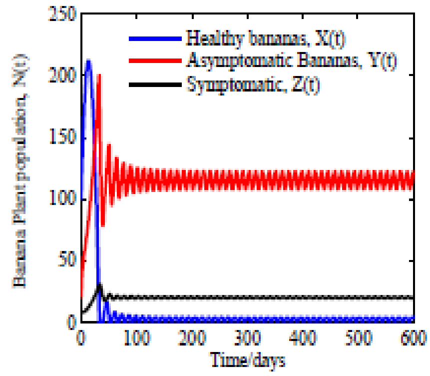


Fig. 2: A spiral stability in a 3D phase portrait for the subpopulations in the modelFig. 2: A spiral stability in a 3D phase portrait for the subpopulations in the modelA spiral pattern indicates stable dynamics with damped oscillations converging to equilibrium. The system's resilience causes perturbations to trigger oscillations that spiral back to a stable state, reflecting disease dynamics with time delays oscillations, and control measures or natural processes damping them over time(τ)causing oscillations, and control measures or natural processes damping them over time



(a)



(b)

Fig. 3: Simulation of model (1), (a) Infection free steady state and (b) endemic steady state, with variables $I_v = 10$,$I_b = I_{vb} = 10^3$,$B = 10^4$,$V = 10^3$. The rest of the parameters are as in Table 2.

Figure 3 shows that without BXW infection, healthy plants increase steadily before 100 days, then stabilize in the longrun. This reflects initial rapid replenishment (Λ) outpacing infection, followed by equilibrium as growth balances removal (harvesting) in the absence of disease pressure. Figure 3 shows the impact of BXW disease: healthy plants decline as infection rises, with asymptomatic plants reaching high numbers. The system's stability changes (Hopf bifurcations) likely stem from time delays in disease dynamics. Given these complex interactions, farmers should prioritize control measures like roguing and vector management to protect yields.

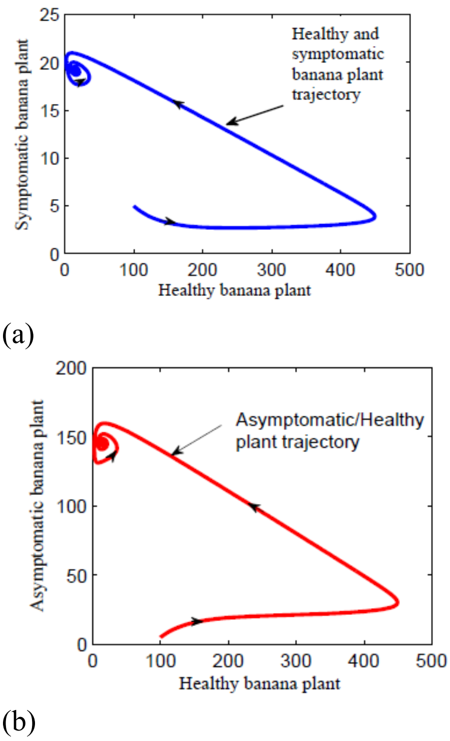


Fig. 4: (a) Simulation of model (1), Trajectories for the Healthy, asymptomatic and symptomatic banana plant with parameter ν =0.324 and (b) Endemic steady state, with variables I_v =10,I_b = I_{vb} =10^3,B =10^4,V =10^3. The rest of the parameter in Table 2

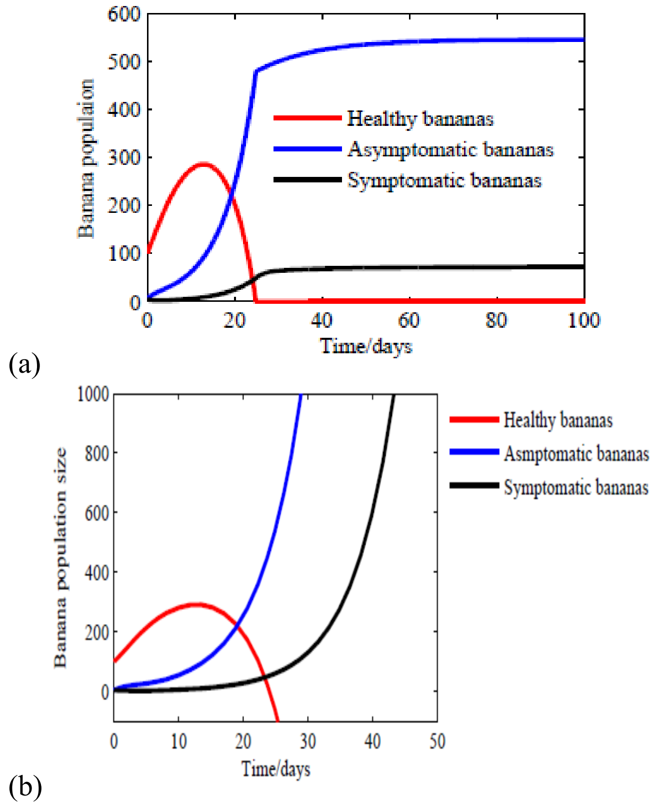


Fig. 5: Simulation of model (1) with time delay=1, 2 and γ = 0.0018. the rest of the parameters remain as indicated in Table 2.

Figure 5 shows that minimal time delays in control measures can effectively mitigate BXW spread, boosting yields. In contrast, Figure 5 indicates rapid progression to symptomatic stages drives exponential growth of infected plants, risking crop failure

6 Conclusion

Field trials in Rwanda showed removing single diseased stems effectively controls BXW, outperforming whole-mat removal [28]. We recommend this targeted approach to farmers globally to boost yields and revenue. Early detection and removal of infected plants are critical. Therefore, farmers should not wait; they should uproot symptomatic plants as soon as possible! Our model supports this strategy, emphasizing timely intervention to prevent disease spread and protect banana crops. The basic reproduction number threshold clearly showed that control measures can be achieved whenever R_0 is below unity. Prioritize rapid response and targeted removal to keep production high.

Data availability

Data supporting this time delay model are from previously published research articles, and parameter values are cited in Table 2.

Future Study

To enrich knowledge in this study, we recommend future research in the following key areas:

(i) Incorporate distributed delays, stochasticity, or spatial heterogeneity into the model,

(ii) Investigate BXW's economic impact on banana production and evaluate cost-effective controls and

(iii) Integrate climate change and farmer behavior factors into BXW dynamics models.

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Conflict of interests

The authors declare that there is no.

References:

[1] FAO, Plant health and food security, FAO, Rome. Available at: http://www.fao.org/3/a-i7829e.pdf, (2017). Accessed on: December 5th, 2019.

[2] F. Uwamahoro, Banana xanthomonas wilt and potato bacterial wilt in Rwanda (Doctoral dissertation), (2019).

[3] P.J. Van Asten and C. Staver, The importance of banana (Musa sp.) for food and nutrition security, Impacts of climate change on the agricultural and aquatic systems and natural resources within the CGIAR's mandate, Working Paper, 23(2012), pp15-22.

[4] G. Blomme, W. Ocimati, C. Sivirihauma, L. Vutseme, B. Mariamu, M. Kamira, B. van Schagen, J. Ekboir, J. Ntamwira, A control package revolving around the removal of single diseased banana stems is eective for the restoration of Xanthomonas wilt infected elds, European Journal of Plant Pathology, 149(2), 2017, pp385-400 .

[5] New Vision, 2019. How to control the banana bacterial wilt. Available at: https://www.newvision.co.ug/news/1505431/control-bananabacterial-wilt. Accessed on December 20th, 2019.

[6] A. Z. Geberewold, Review on impact of banana bacterial wilt (Xhantomonas campestris pv. Musacerum) in East and Central Africa, Cogent Food & Agriculture, 5(1586075), 2019, pp1-15.

[7] J.M. Mwebaze, G. Tusiime, W.K. Teshemereirwe, and J. Kubiriba, The survival of Xanthomonas campestris pv. musacearum in soil and plant debris, Journal, 14(2), 2006, pp121-127.

[8] J. Nakakawa, J.Y.T. Mugisha, M.W. Shaw, W. Tinzaara and E. Karamura, Banana Xanthomonas wilt infection: The role of debudding and roguing as control options within a mixed cultivar plantation, International Journal of Mathematics and Mathematical Sciences, 2017 (2017), pp1-13.

[9] G. Blomme, M. Dita, K.S. Jacobsen, L. PØrez Vicente, A. Molina, W. Ocimati, S. Poussier and P. Prior, Diseases of bananas and enset: current state of knowledge and integrated approaches toward sustainable management, Frontiers in plant science, 8 (2017), pp1-25.

[10] M. Biruma, M. Pillay, L. Tripathi, G. Blomme, S. Abele, M. Mwangi, R. Bandyopadhyay, P. Muchunguzi, S. Kassim, M. Nyine, L. Turyagyenda, and S. Eden-Green Banana Xanthomonas wilt: a review of the disease, management strategies and future research directions, African Journal of Biotechnology, 6(8), 2007, pp954-962.

[11] Crop Area and Production Report, December 2010. Available at: https:// www.google.com/search?client = refox -b-d & q=Crop+Area+and+Production+Report %2C+December+2010, Accessed on: November 18th, 2019.

[12] E.M. Kikulwe, J.L. Kyanjo, E. Kato, R.T. Ssali, R. Erima, S. Mpiira, W. Ocimati, W. Tinzaara, J. Kubiriba, E. Gotor, S. Dietmar and E. Karamura, Management of Banana Xanthomonas Wilt: Evidence from Impact of Adoption of Cultural Control Practices in Uganda, Sustainability, 11(9) 2019, pp1-18.

[13] L. Tripathi, J.N. Tripathi, W.K. and Tushemereirwe, Control of banana xanthomonas wilt disease using genetic engineering, IV International Symposium on Banana: International Conference on Banana and Plantain in Africa: Harnessing International 879, 2008, pp649-657.

[14] D. Ochola, W. Jogo, M. Odongo, W. Tinzaara, M. Onyango and E. Karamura,

Household dynamics inuencing eective eradication of Xanthomonas wilt in small holder banana systems in Ugunja divisionKenya, African Journal of Agricultural Research, 9(26),2014, pp 2031-2040.

[15] J. Nkuba, W. Tinzaara, G. Night, N. Niko, W. Jogo, I. Ndyetabula, L. Mukandala, P. Ndayihazamaso, C. Niyongere, S. Gaidashova, I. Rwomushana, F. Opio, and E. Karamura, Adverse impact of Banana Xanthomonas Wilt on farmers' livelihoods in Eastern and Central Africa, African Journal of Plant Science, 9(7), 2015, pp279-286.

[16] L. Junli and Z. Tailei, Stability and Hopf Bifurcation Analysis of a Plant Virus Propagation Model with Two Delays, Discrete Dynamics in Nature and Society, 2018 (2018), pp1-12.

[17] B. Buonomo. The eect of time delay in plant-pathogen interactions with host demography, Mathematical Biosciences and Engineering, 12(3), (2015), pp473-490.

[18] L. J. S. Allen, An Introduction to Mathematical Biology, Pearson-Prentice Hall, Upper Saddle River, New Jersey, USA, 2007.

[19] M. Jackson and B. M. Chen-Charpentier,"Modeling plant virus propagation with delays," Journal of Computational and Applied Mathematics, 309 (2017), pp. 611-621.

[20] W. Ocimati, F. Ssekiwoko, E. Karamura, W. Tinzaara, S. Eden-Green, S and G. Blomme, Systemicity of Xanthomonas campestris pv. musacearum and time to disease expression after inorescence infection in East African highland and Pisang Awak bananas in Uganda, Plant Pathology, 62(4), 2013, pp777-785.

[21] J. Nakakawa, J.Y.T. Mugisha, M.W. Shaw and E. Karamura, A mathematical model for the dynamics of Banana xanthomonas wilt with vertical transmission and inorescence infection, Journal of Biological Systems, 24(1), 2016, pp147-165.

[22] Banana Plant Info, Available at http://www.bananaplants.net/bananainfo.html. Accessed on February 20th, 2020.

[23] E.H. Kweyunga, J. Tumwiine. A mathematical model for the vector transmission and control of banana Xanthomonas wilt. Journal of Mathematics Research. 9(4), 2017, pp101-113.

[24] Oluwafemi AB, Inuence of number of sucker per plant on the growth, yield and yield components of plantain (Musa sp) in Ado-ekiti, Nigeria, Agricultural Science Research Journal 3, 2013, 45-49, .

[25] F. Brauer, C. Castillo-Chavez, Mathematical Models in Population Biology and Epidemiology; Springer: Berlin, Germamy, 2001.

[26] K.E. Lestari, U.S. Pasaribu, S.W. Indratno, & H. Garminia. Generating roots of cubic polynomials by Cardano's approach on correspondence analysis. Heliyon, 6(6), 2020, pp1-8.

[27] O. Diekmann, J.A.P. Heesterbeek and M.G. Roberts, The construction of next-generation matrices for compartmental epidemic models, Journal of the Royal Society Interface, 7(47), 2009, pp873-885.

[28] A. Rietveld, B. Guy & SV. Gaidashova. A superior technology to control Banana Xanthomonas Wilt (BXW) in Rwanda.

APPENDIX I

Sensitivity Analysis of R_0 for BXW Disease: Method of Perturbation of Fixed Point Estimation.
In Eq.28, the basic reproduction number is a function with eight (8) parameters: n,β,Λ, a, h, γ, δ and ν.
Choosing a small perturbation ζ, $0 < \zeta << 1$ to a parameter Φ the resulting change in R_0 as ∂R is expressed as:

$$\partial R_0 = R_0(\Phi+\zeta) - R_0(\Phi) = \frac{R_0(\Phi+\zeta) - R_0(\Phi)}{\zeta} \times \zeta \approx \zeta \times \frac{\partial R_0}{\partial \Phi}$$

The normalized sensitivity index

$$\Delta_\Phi = \frac{\partial R_0}{R_0} \div \frac{\partial \Phi}{\Phi} = \frac{\Phi}{R_0} \times \frac{\partial R_0}{\partial \Phi} \quad (29)$$

Using the essential tool for mathematics and modeling software (Maple 13), the normalized sensitivity indices for the eight (8) parameter in Table 4 are given as follows:

$$\begin{aligned}
\Delta_\nu &= \frac{\nu}{R_0} \times \frac{\partial R_0}{\partial \nu} = -\frac{\nu}{\nu+\delta} = -0.9836,\\
\Delta_\Lambda &= \frac{\Lambda}{R_0} \times \frac{\partial R_0}{\partial \Lambda} = \frac{ah}{ah+\Lambda} = 0.2221,\\
\Delta_\delta &= \frac{\delta}{R_0} \times \frac{\partial R_0}{\partial \delta} = -\frac{\delta}{\delta+\nu} = -0.0164,\\
\Delta_\gamma &= \frac{\gamma}{R_0} \times \frac{\partial R_0}{\partial \gamma} = -\frac{\gamma}{\gamma+h} = -0.2013,\\
\Delta_h &= \frac{h}{R_0} \times \frac{\partial R_0}{\partial h} = -\frac{-h[(a(\gamma+h)+(ah+\Lambda))]}{(ah+\Lambda)(\gamma+h)} = -1.0208\\
\Delta_a &= \frac{a}{R_0} \times \frac{\partial R_0}{\partial a} = -\frac{ah}{ah+\Lambda} = -0.2222,\\
\Delta_\beta &= \frac{\beta}{R_0} \times \frac{\partial R_0}{\partial \beta} = 1.0000,\\
\Delta_n &= \frac{n}{R_0} \times \frac{\partial R_0}{\partial n} = 1.0000.
\end{aligned}$$