Journal of Advances in Mathematics and Computer Science

29(3): 1-17, 2018; Article no.JAMCS.44336

ISSN: 2456-9968

(Past name: British Journal of Mathematics & Computer Science, Past ISSN: 2231-0851)

Modeling the Dynamics of Banana *Xanthomonas* Wilt Transmission Incorporating Infectious Force in both Asymptomatic and Symptomatic Stages

Eliab Horub Kweyunga[1*], Julius Tumwiine[1] and Eldad Karamura[2]

[1] *Department of Mathematics, Mbarara University of Science and Technology, P.O.Box 1410, Mbarara, Uganda.*

[2] *Bioversity International, P.O.Box 24384, Plot 106, Katalima Road, Naguru, Kampala, Uganda.*

Authors' contributions

This work was carried out in collaboration between all authors. Author EHK designed the model, carried out the analysis and simulation,and wrote the first draft of the manuscript. Author JT carried out the literature review and checked the whole manuscript. Author EK provided the idea of the problem. All authors read and approved the final manuscript.

Article Information

DOI: 10.9734/JAMCS/2018/44336

Editor(s):

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Complete Peer review History: http://www.sciencedomain.org/review-history/26876

Original Research Article

Received: 15 July 2018

Accepted: 28 September 2018

Published: 27 October 2018

Abstract

Despite massive efforts by regional governments and partners in the East and Central African region towards fighting *Xanthomonas* Wilt of banana, the disease is reported to continue to spread to new areas and resurge in others it had been contained. The use of asymptomatic but infectious plants is hypothesized to play a leading role in the persistence of the disease and it's introduction to new areas. A model for the transmission of BXW by symptomless plants is proposed and

**Corresponding author: E-mail: hkweyunga@must.ac.ug*

analyzed. It incorporates both horizontal and vertical transmission modes and a dual source of inoculum in the force of infection. The basic reproduction number, $\mathcal{R}_0$, is obtained and it is found to completely determine the global dynamics of the model. By construction of a suitable Lyapunov function for the second additive compound system, the global stability of the endemic equilibrium is established.
Numerical simulation and sensitivity analysis of the basic reproduction number indicate that the disease is mainly driven by parameters involving asymptomatic plants rather than symptomatic ones.

Keywords: Asymptomatic plants; banana Xanthomonas wilt; basic reproduction number; Bendixson criterion; endemic equilibrium; global stability and vertical transmission.

2010 Mathematics Subject Classification: 92D30 (Primary), 34D20(Secondary)

1 Introduction

Banana is an important food and cash crop in the world and ranks the fourth most important after rice, wheat and maize. The East and Central African region is one of the most banana producing and consuming regions in Africa. With an estimated annual production of 10 million tonnes from 1.5 million hectares, Uganda is the second largest producer of bananas in the world after India [1]. However, the production and yield of banana in the region face a number of challenges mainly soil degradation, soil nutrient depletion, pests and diseases, availability of labor, population explosion vis-a vis reduced farms sizes, access to clean planting materials and market availability. Diseases of significance to banana production in the region include banana *Xanthomonas* wilt (BXW), black leaf streak, Fusarium wilt, Bugtok/Moko, blood disease, head rot and top bunch virus disease. Banana *Xanthomonas* wilt caused by the bacterium *Xanthomonas campestris pv. musacearum, (Xcm)*, is the most devastating of all the banana diseases in the East and Central African region and is non-discriminative of all *Musa* cultivars. The disease was first reported in Ethiopia in 1969 on enset, a close relative of banana that is native to the Ethiopian highlands [2]. Outside Ethiopia, BXW was first reported in Uganda and DR Congo in 2001 and later in the neighboring countries of Kenya, Tanzania, Rwanda and Burundi [3].

The disease is characterized by progressive yellowing and wilting of leaves, uneven and premature ripening of fruits, wilting of bracts, shriveling and rotting of male buds and a characteristic yellow ooze from the cut pseudo stem [4]. It attacks all cultivars grown in the region and no resistant varieties have so far been identified.

BXW is mainly spread by insect vectors through the male buds as they forage for nectar; contaminated farm tools used for crop husbandry practices; use of infected but symptomless suckers as planting material and via vertical transmission from mother banana plant to emerging suckers [5].

Based on experience with other banana bacterial wilt diseases such as Moko/Bugtok, the management strategies promoted in the region against BXW include the destruction and disposal (or roguing) of infected banana plants, disinfecting farm tools using chemicals or a flame of fire, using clean planting materials and prompt removal of male buds (debudding) using a forked stick , removal of infected banana mats, cutting of single diseased banana plants in mats and banana free fallows [6, 7, 8] are some of measures recommended. These measures, coupled with institutional actions such as quarantine, awareness creation and setting up of task forces at local, national and regional levels, where effectively implemented have registered tremendous success in curtailing the initial

outbreak.

Of late however, there have been extensive reports of resurgence and introduction of the disease in new areas possibly due to complacency by farmers and failure to fully apply the management options advocated for in the region [9].

Following the first report of the disease in Uganda in 2001, a lot of research has been carried out and much data generated about the disease spread and control dynamics (see [7],[10],[11],[12],[13] and references therein).

It was previously believed that once a banana plant in a banana mat becomes infected, all the attached banana plants and emerging suckers would also become infected. This was the basis for the recommendation to farmers to practice complete mat uprooting (CMU) upon identification of a single diseased pseudo-stem on a mat. However studies have since established that *Xcm* does not colonize all the lateral shoots and partial or incomplete systemicity results whereby some suckers from the same mat get infected while others escape infection [12].

Based on recent data generated by the numerous studies, new control options are being promoted such as single diseased stem removal (SDSR) as opposed to complete mat uprooting [11], suspension of crop husbandry practices such as de-bugging, de-suckering and de-leafing for a given period to address tool-based transmission, and cutting diseased pseudo-stems at ground level when symptoms are still limited to the male buds to prevent *Xcm* colonizing the banana corm tissues and attached suckers [12],[13].

Mathematical models for the dynamics of the transmission and control of BXW have been developed to gain insights on the disease dynamics. Kweyunga and Tumwiine [14] considered the dynamics of the vector transmission and control of BXW with roguing of infected plants and replanting with disease clean planting materials as control measures. It was established that at appropriate roguing and replanting rates, the disease can be contained. Nannyonga *et al* [15] used optimal control theory to study the dynamics of BXW within plantations with controls targeting transmission via vectors and contaminated tools. Their model incorporated vertical transmission of the disease from mother plant to emerging suckers. Vertical transmission of BXW and inflorescence infection were considered in [16] that incorporated incomplete systemicity. It was established that inflorescence infection and roguing rates had more impact on persistence threshold levels while vertical transmission parameters had an insignificant impact. Furthermore, an optimal control framework in which the use of clean planting materials, de-budding, disinfection of tools and roguing were considered as control measures of BXW within a plantation of multiple cultivars was designed in [17].

In this paper, we investigate the role of asymptomatic but infectious banana plants on the continued spread and possible resurgence of BXW in the East and Central African region. These banana plants, especially suckers, have been used as planting materials by farmers who want to start new farms and this could be responsible for the introduction of the disease to new areas. Moreover, in a situation where farmers are unable distinguish asymptomatic banana plants from symptomatic ones, the use of farms tools alternately between these banana plants results in disease transmission and may account for its persistence. Furthermore, vectors foraging for nectar and pollen may pick the pathogen from both asymptomatic and symptomatic plants and deposit it on healthy plants thereby aiding in disease transmission. Therefore, asymptomatic banana plants play a significant role in BXW transmission accounting for three major disease transmission modes namely via vectors, tools and infected planting materials. Our model incorporates both horizontal transmission (via tools, vectors and infected planting materials) and vertical transmission (from mother plant to emerging suckers) of the disease and introduces infectious force in both asymptomatic and symptomatic stages.

The paper is organized as follows: In Section 2, the model is formulated, the equilibrium points are obtained and their stability established. In Section 3, numerical simulation and sensitivity analyses are carried out while in Section 4, a brief discussion of the results is undertaken and conclusions given in Section 5.

2 Model Formulation and Analysis

BXW is a systemic disease in that it affects the entire plant which can then be used as a basic unit of modeling. The following assumptions are made:

- In absence of the pathogen, we consider monomolecular growth of the plant host population with carrying capacity K.
- The host plant size, $N(t)$, is divided into three categories namely; healthy plants, $H(t)$; asymptomatically infectious plants, $L(t)$ and symptomatically infectious banana plants, $I(t)$ with $N(t) = H(t) + L(t) + I(t)$.
- Healthy banana plants become infected when they get in contact either directly or indirectly (through tools, vectors or infected planting materials) with asymptomatically and symptomatically infected banana plants or via vertical transmission from the mother plant to its emerging suckers.
- We assume negligible latent period so that plants become infectious as soon as they are infected but symptom expression occurs later.
- Replenishment of host plants is via emergence of new suckers at a uniform rate λ in all plant categories.
- Of the suckers emerging from both asymptomatically and symptomatically infected plants, a fraction is diseased and joins the asymptomatically infected class while the remainder is healthy and joins the healthy plant category.
- Asymptomatically infected plants may be rogued otherwise they progress to the symptomatically infected plant category. Likewise, symptomatically infected banana plants may also be rogued otherwise they will die from the disease.
- Roguing rates are assumed to be different for both asymptomatically and symptomatically infected banana plants; given the farmers inability to distinguish between asymptomatically and symptomatically infected banana plants; their reluctance to rogue asymptomatically infected banana plants thinking they may bear fruits, which might be labor intensive.
- The incidence term is of the standard incidence form with a dual source of infection from both asymptomatically and symptomatically infected banana plants.

The model is specified by the following non-linear system of ordinary differential equations:

$$\begin{aligned} \frac{dH}{dt} &= \lambda(K-H) + (1-\delta)\lambda L + (1-\phi)\lambda I - \beta_1 H\frac{L}{K} - \beta_2 H\frac{I}{K} - \mu H, \\ \frac{dL}{dt} &= \beta_1 H\frac{L}{K} + \beta_2 H\frac{I}{K} + \delta\lambda I + \phi\lambda I - (\alpha + r_1)L, \\ \frac{dI}{dt} &= \alpha L - (r_2 + d)I, \end{aligned} \tag{2.1}$$

together with
$\frac{dN}{dt} = \lambda(K + L + I) - (\mu + \lambda)H - r_1 L - (r_2 + d)I$
which shows that the total host plant size in presence of the disease is variable while in absence of the disease, the plant population is given by $\frac{dN}{dt} = \lambda(K - H) - \mu H$.

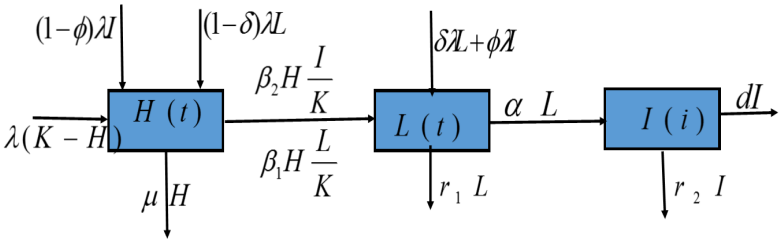

Fig. 1. Compartmental diagram representing the transfer of the disease among the different plant categories

Table 1. Parameter description and estimates for the model

Parameter	Description	Value	Reference
λ	sucker emergence rate	0.1667/day	[16]
ϕ	Suckers from asymptomatic plants	0.0143	estimated
δ	Suckers from symptomatic plants	0.0286	[12]
μ	Harvest rate of healthy plants	0.0022/day	estimated
β_1	Contact rate with asymptomatic plants	0.034/day	estimated
β_2	Contact rate with symptomatic plants	0.068/day	estimated
r_1	Roguing rate of asymptomatic plants	0.00525/day	estimated
r_2	Roguing rate of symptomatic plants	0.0105/day	[14]
α	Progression rate to symptomatic class	0.018/day	[18]
d	Death rate of infected plants	0.0167/day	[14]

2.1 Model Analysis

For simplicity, we let $h = \frac{H}{K}, l = \frac{L}{K}$ and $i = \frac{I}{K}$, then after differentiation and simplification, the following system of ordinary differential equations is obtained:

$$\begin{aligned} \frac{dh}{dt} &= \lambda(1-h) + (1-\delta)\lambda l + (1-\phi)\lambda i - \beta_1 hl - \beta_2 hi - uh, \\ \frac{dl}{dt} &= \beta_1 hl + \beta_2 hi + \delta\lambda l + \phi\lambda i - (\alpha + r_1)l, \\ \frac{di}{dt} &= \alpha l - (r_2 + d)i, \end{aligned} \tag{2.2}$$

together with $h(t) + l(t) + i(t) = 1$.

System (2.2) is equivalent to system (2.1), therefore we can study system (2.1) by studying system (2.2). For biological reasons, the model is analyzed in the feasible region

$\Gamma = \{(h, l, i) \in \Re^3_+ \mid h, l, i \geq 0, h + l + i \leq 1\}$,

where Γ is positively invariant with respect to system (2.2) and $\Re^3_+$ denotes a nonnegative cone of $\Re^3$ including its lower dimensional faces denoted by $\partial\Gamma$ and $\dot{\Gamma}$ the boundary and interior of Γ in $\Re^3$ respectively.

2.1.1 Equilibrium points

Equilibrium points are obtained by setting the derivatives of system (2.2) equal to zero.
Calculations show that system (2.2) admits two equilibria namely: the disease free equilibrium, $E_0(\frac{\lambda}{\lambda+\mu}, 0, 0)$, at which only the healthy plant population is present at its carrying capacity and the endemic or coexistence equilibrium point, $E_1(h^*, l^*, i^*)$ at which the proportion of healthy plants is depressed below the disease free value with

$$\begin{aligned} h^* &= \frac{(\alpha + r_1 - \delta\lambda)(r_2 + d) - \phi\lambda\alpha}{\beta_1(r_2 + d) + \beta_2\alpha} \\ l^* &= \frac{\lambda(r_2 + d)[\beta_1(r_2 + d) + \beta_2\alpha] - (\lambda + \mu)(r_2 + d)[(\alpha + r_1 - \delta\lambda)(r_2 + d) - \alpha\phi\lambda]}{[(\alpha + r_1)(r_2 + d) - \lambda(r_2 + d + \alpha)][\beta_1(r_2 + d) + \beta_2\alpha]} \\ i^* &= \frac{\lambda\alpha[\beta_1(r_2 + d) + \beta_2\alpha] - (\lambda + \mu)\alpha[(\alpha + r_1 - \delta\lambda)(r_2 + d) - \alpha\phi\lambda]}{[(\alpha + r_1)(r_2 + d) - \lambda(r_2 + d + \alpha)][\beta_1(r_2 + d) + \beta_2\alpha]} \end{aligned}$$

It is noted that the healthy plant proportion at E_0 depends only on the natural processes of sucker emergency at a rate λ and harvest at a rate μ whose reciprocal $\frac{1}{\mu}$ measures the length of the reproductive life time of the banana plant. Since all the parameters are non-negative constants, then $\frac{\lambda}{\lambda+\mu} < 1$, the carrying capacity.

The impact of the disease is related to the proportion of healthy banana plants at the endemic equilibrium point. The parameters β_1 and β_2 appear only in the denominator while r_1 and the vertical transmission parameters δ and ϕ and the sucker emergence rate λ appear only in the numerator. The equilibrium healthy plant population therefore is proportional to the roguing rate of asymptomatic banana plants and inversely proportional to the contact rates β_1 and β_2 with asymptomatic and symptomatic banana plants respectively. The parameters α, r_2 and d appear in both the denominator and numerator and are therefore assumed to have limited influence on the equilibrium level of the healthy plants at the endemic equilibrium point.

2.1.2 Basic reproduction number

Since all the parameters have non-negative values, one condition holds for all the state variables (h^*, l^*, i^*) at the endemic equilibrium point to be biologically realistic namely:

$$\lambda[\beta_1(r_2 + d) + \beta_2\alpha] > (\lambda + \mu)[(\alpha + r_1 - \delta\lambda)(r_2 + d) - \alpha\phi\lambda]$$

or

$$\mathcal{R}_0 = \left(\frac{\lambda}{\lambda + \mu}\right) \frac{\beta_1(r_2 + d) + \beta_2\alpha}{[(\alpha + r_1 - \delta\lambda)(r_2 + d) - \alpha\phi\lambda]}. \tag{2.3}$$

Equation (2.3) is the expression for the basic reproduction number for *Xanthomonas campestris pv. musacearum*. According to Anderson and May [19], it is the number of secondary infectives generated by the introduction of a single infective in the population at the disease free equilibrium. When $\mathcal{R}_0 > 1$, the disease persists in the population when introduced otherwise it fails to invade. It can be noticed that when $\phi = \delta = 0$, vertical transmission is lost and the basic reproduction number becomes

$$\mathcal{R}_0^* = \left(\frac{\lambda}{\lambda + \mu}\right) \frac{[\beta_1(r_2 + d) + \beta_2\alpha]}{[(\alpha + r_1)(r_2 + d)]}. \tag{2.4}$$

Equation (2.4) can be re-arranged as

$$\mathcal{R}_0^* = h^* \left(\frac{\beta_1}{\alpha + r_1} + \frac{\beta_2}{r_2 + d} . \frac{\alpha}{\alpha + r_1}\right) \tag{2.5}$$

and can be partitioned into independent components representing contributions from the dual source of infection from both asymptomatic and symptomatic plants. Thus, $\mathcal{R}_0^* = \mathcal{R}_0^A + \mathcal{R}_0^S$ with

$\mathcal{R}_0^A = h^*(\frac{\beta_1}{\alpha+r_1})$ and $\mathcal{R}_0^S = h^*(\frac{\beta_2}{r_2+d}).\frac{\alpha}{\alpha+r_1}$. The expression $\mathcal{R}_0^A$ is the product of the proportion of healthy plants at the disease free equilibrium and the number of new infections per unit time arising out of contact with asymptomatic plants. Similarly, $\mathcal{R}_0^S$ is the product of the proportion of healthy plants at the disease free equilibrium, the number of new infections per unit time due to symptomatic plants and the probability that a plant reaches the symptomatic stage. The sum of these two terms represents the number of new infections arising out of one infectious plant introduced in the population at the disease free equilibrium. If this sum exceeds unity, the disease will invade the plantation otherwise it will die out.
The endemic equilibrium point can be expressed in terms of $\mathcal{R}_0$ as

$$\begin{aligned} h^* &= \left(\frac{\lambda}{\lambda+\mu}\right)\frac{1}{\mathcal{R}_0} \\ l^* &= \frac{\lambda(r_2+d)}{(\alpha+r_1)(r_2+d)-\lambda(r_2+d+\alpha)}\left(\frac{\mathcal{R}_0-1}{\mathcal{R}_0}\right) \\ i^* &= \frac{\alpha\lambda}{(\alpha+r_1)(r_2+d)-\lambda(r_2+d+\alpha)}\left(\frac{\mathcal{R}_0-1}{\mathcal{R}_0}\right) \end{aligned}$$

Clearly, the endemic equilibrium point, E_1, exists whenever $\mathcal{R}_0 > 1$, $(\alpha+r_1)(r_2+d) > \lambda(r_2+d+\alpha)$, $(\alpha+r_1) > \delta\lambda$ and $(r_2+d)(\alpha+r_1-\delta\lambda) > \alpha\phi\lambda$.

2.2 Local Stability of Equilibrium Points

In this section we establish the local stability of both the disease free and endemic equilibrium points.
The Jacobian matrix associated with the general solution of system (2.2) is

$$J = \begin{pmatrix} -(\lambda+\mu+\beta_1 l+\beta_2 i) & (1-\delta)\lambda-\beta_1 h & (1-\phi)\lambda-\beta_2 h \\ \beta_1 l+\beta_2 i & \beta_1 h+\delta\lambda-(\alpha+r_1) & \beta_2 h+\phi\lambda \\ 0 & \alpha & -(r_2+d) \end{pmatrix} \tag{2.6}$$

2.2.1 Local stability of the disease free equilibrium point

The local stability of the disease-free is evaluated At the disease free equilibrium point E_0, the deflated Jacobian matrix

$$J_{E_0} = \begin{pmatrix} -(\lambda+\mu) & (1-\delta)\lambda-\beta_1 h & (1-\phi)\lambda-\beta_2 h \\ 0 & \beta_1 h+\delta\lambda-(\alpha+r_1) & \beta_2 h+\phi\lambda \\ 0 & \alpha & -(r_2+d) \end{pmatrix}$$

which has three eigenvalues. Clearly one eigenvalue is $-(\lambda+\mu)$ and the rest are obtained from the submatrix

$$J'_{E_0} = \begin{pmatrix} \beta_1 h+\delta\lambda-(\alpha+r_1) & \beta_2 h+\phi\lambda \\ \alpha & -(r_2+d) \end{pmatrix}$$

whose $tr(J'_{E_0}) = \beta_1 h+\delta\lambda-(\alpha+r_1+r_2+d)$ and

$$det(J'_{E_0}) = [(\alpha+r_1-\delta\lambda)(r_2+d)-\alpha\phi\lambda] - \frac{\lambda}{\lambda+\mu}[\beta_1(r_2+d)+\beta_2\alpha]$$

By Routh-Hurwitz criteria [Britton [20] , E_0 is locally asymptotically stable if the $tr(J'_{E_0}) < 0$ and $det(J'_{E_0}) > 0$. Now $tr(J'_{E_0}) < 0$ if

$$\beta_1 h+\delta\lambda < (\alpha+r_1+r_2+d) \tag{2.7}$$

and $det(J'_{E_0}) > 0$ if

$$\mathcal{R}_0 = \left(\frac{\lambda}{\lambda+\mu}\right)\frac{[\beta_1(r_2+d)+\beta_2\alpha]}{(\alpha+r_1-\delta\lambda)(r_2+d)-\alpha\phi\lambda]} < 1$$

Thus, the disease free equilibrium point $E_0(\frac{\lambda}{\lambda+\mu}, 0, 0)$ is locally asymptotically stable if $\mathcal{R}_0 < 1$.

2.2.2 Local stability of the endemic equilibrium point

The local stability of endemic equilibrium points E_1 is investigated using the Routh-Hurwitz's criteria. That is, an equilibrium point is locally asymptotically stable if the characteristic equation of the Jacobian matrix evaluated at that point has all the coefficients being positive and that all of its roots have negative real parts. The deflated Jacobian matrix at the endemic equilibrium point, E_1, is given by

$$J_{(E_1)} = \begin{pmatrix} -(\lambda+\mu+\beta_1 l^*+\beta_2 i^*) & (1-\delta)\lambda-\beta_1 h^* & (1-\phi)\lambda-\beta_2 h^* \\ \beta_1 l^*+\beta_2 i^* & \beta_1 h^*+\delta\lambda-(\alpha+r_1) & \beta_2 h^*+\phi\lambda \\ 0 & \alpha & -(r_2+d) \end{pmatrix} \tag{2.8}$$

The eigenvalues are obtained from $det(J_{(E_1)} - mI) = 0$, where m represents the eigenvalues and I is a 3×3 unit matrix. The characteristic equation is of the form $m^3 + a_1m^2 + a_2m + a_3 = 0$, where

$$\begin{aligned} a_1 &= \lambda+\mu+\beta_1 l^*+\beta_2 i^*+\alpha+r_1+r_2+d-(\beta_1 h^*+\delta\lambda) \\ a_2 &= (\lambda+\mu+\beta_1 l^*+\beta_2 i^*)(\alpha+r_1+r_2+d-(\beta_1 h^*+\delta\lambda)) \\ &\quad +(\beta_1 l^*+\beta_2 i^*)(\beta_1 h^*+\delta\lambda-\lambda) \\ a_3 &= (\beta_1 l^*+\beta_2 i^*)[(\alpha+r_1)(r_2+d)-\lambda(\alpha+r_2+d)]. \end{aligned}$$

By Routh-Hurwitz criterion, E_1 is locally asymptotically stable when $a_1, a_2, a_3 > 0$ and $a_1a_2 > a_3$. By inequality (2.7), it is clear that $a_1 > 0$. In addition, $a_2 > 0$ provided $(\beta_1 h^* + \delta\lambda) > \lambda$ and $a_3 > 0$ provided $(\alpha + r_1)(r_2 + d) > \lambda(\alpha + r_2 + d)$. It is easy to show that $a_1a_2 > a_3$ if the conditions for the positivity of a_1, a_2 and a_3 are met.

Thus the endemic equilibrium point $E_1(h^*, l^*, i^*)$ is locally asymptotically stable whenever it exists. Since E_1 exists whenever $\mathcal{R}_0 > 1$, we conclude that the endemic equilibrium point is locally asymptotically stable whenever $\mathcal{R}_0 > 1$.

2.3 Global stability of the equilibrium points

We proceed to establish the global stability of the equilibrium points.

2.3.1 Global stability of the disease free equilibrium

Global stability of the system was analyzed by considering suitable Lyapunov function. Consider the following Lyapunov function candidate,

$$V = \alpha l + (\alpha + r_1 - \delta\lambda)i. \tag{2.9}$$

It is observed that the chosen Lyapunov function candidate $V(h; l; i)$ of (2.9) satisfy the conditions that $V(h^*; l^*; i^*) = 0$ and $V(h; l; i) > 0$ for all $(h; l; i) \neq (h^*; l^*; i^*)$. Moreover, $V(h; l; i)$ is radially unbounded.

We are now required to verify $V' \leq 0$.

$$\begin{aligned} V' &= \alpha[\beta_1 hl + \beta_2 hi + \delta\lambda l + \phi\lambda i - (\alpha + r_1)l] + (\alpha + r_1 - \delta\lambda)[\alpha l - (r_2 + d)i] \\ &= [(\beta_1(r_2 + d) + \beta_2)h + \alpha\phi\lambda - (r_2 + d)(\alpha + r_1) + \delta\lambda(r_2 + d)]i \\ &= [(\beta_1(r_2 + d) + \beta_2)h - [(\alpha + r_1 - \delta\lambda)(r_2 + d) - \alpha\phi\lambda]]i \\ &= [(\alpha + r_1 - \delta\lambda)(r_2 + d) - \alpha\phi\lambda]\left[\left(\frac{\lambda + \mu}{\mu}\right)h(\mathcal{R}_0 - 1\right)\right] i \\ &= [(\alpha + r_1 - \delta\lambda)(r_2 + d) - \alpha\phi\lambda](\mathcal{R}_0 - 1)i \\ &\leq 0 \end{aligned}$$

if $\mathcal{R}_0 < 1$.
In addition, $V' = 0$ if and only if $i = 0$ or $\mathcal{R}_0 = 1$ and $h = \frac{\lambda}{\lambda+\mu}$. Therefore, the largest compact invariant set in $\{(h, l, i) \in \Gamma : V' = 0\}$ is a singleton $\{E_0\}$. By LaSalle's invariance principle [21], we deduce then that E_0 is globally stable in Γ. Thus, the disease free equilibrium point, E_0 is globally asymptotically stable provided $\mathcal{R}_0 < 1$.

2.3.2 Global stability of the endemic equilibrium point

The global stability of the endemic equilibrium point is determined using the approach developed by Smith [22] and Li and Muldowney [23] for SEIR models. We first present a brief general mathematical framework for proving the global stability of the endemic equilibrium point:
Let $x \mapsto f(x)$ be a C^1 function for x in an open set $D \subset \Re^n$. Consider the differential equation:

$$x' = f(x) \tag{2.10}$$

Denote by $x(t, x_0)$ the solution to (2.10) such that $x(0, x_0) = x_0$.

Definition . A set K is said to be absorbing in D for the differential equation (2.10) if $x(t, k_1) \subset K$ for each compact $k_1 \subset D$ and t sufficiently large.
The following two basic assumptions are made;
(H_1): There exists a compact absorbing set $K \subset D$
(H_2): Equation (2.10) has a unique equilibrium point $\bar{\mathrm{x}}$ in D.
The equilibrium point $\bar{\mathrm{x}}$ is said to be globally stable in D if it is locally stable and all trajectories in D converge to $\bar{\mathrm{x}}$.
The assumptions (H_1) and (H_2) are satisfied if $\bar{\mathrm{x}}$ is globally stable in D.
For epidemic models and many other biological models where the feasible region is a bounded cone, (H_1) is equivalent to the uniform persistence of (2.10) Butler and Waltman [24].
The following global stability problem is formulated in Li and Muldowney [23].
Global stability problem: Under assumptions (H_1) and (H_2), find conditions on the vector field of equation (2.10) such that the local stability of $\bar{\mathrm{x}}$ implies its global stability in D.
For $n \geq 2$, by a *Bendixson* criterion, is meant a condition satisfied by f which precludes the existence of non-constant periodic solutions of equation (2.10).
A Bendixson criterion is said to be *robust under C' local perturbations of f at $x \in D$* if, for sufficiently small $\epsilon > 0$ and neighborhood U of x_1, it is also satisfied by $g \in C'(D \to \Re^n)$ such that the support$(f-g) \subset U$ and $|f-g| < \epsilon$, where $|f-g|_{C'} = sup\{|f(x)-g(x)|+|\frac{\partial f}{\partial x}(x)-\frac{\partial g}{\partial x}(x)| : x \in D\}$. Such g will be called the local $\epsilon - perturbations$ of f at x. The classical Bendixson's condition, $divf(x) < 0$ for $n = 2$ is robust under C' perturbations of f. For higher dimensional systems, the C' robust properties are discussed in Li and Muldowney [23].
Definition: A point x_0 is wandering for equation (2.10) if there exists a neighborhood U of x_0 and $T > 0$ such that $U \cap x(t, \cap)$ is empty for all $t > T$. As examples, all equilibria and limit points are non-wandering.

The following global stability result is proved in Li and Muldowney [23].
Theorem 1: Suppose

1. assumptions (H_1) and (H_2) hold;
2. System (2.10) satisfies the *Bendixson* criterion that is robust under C' local perturbations of f at all non-wandering points.

Then the unique equilibrium $\bar{x}$ is globally asymptotically stable in D.
A method for deriving the Bendixson criterion in $\Re^n$ is developed in Li and Muldowney [25] in which the main idea is to show that the second compound system equation

$$z'(t) = \frac{\partial f^{[2]}}{\partial x}(x(t, x_0))z(t), \tag{2.11}$$

with respect to a solution $x(t, x_0) \subset D$ to equation (2.10) is uniformly asymptotically stable. Here $\frac{\partial f}{\partial x}^{[2]}$ is the second additive compound matrix of the Jacobian matrix $\frac{\partial f}{\partial x}$. If D is simply connected, then the uniform asymptotic stability of (2.11) precludes the existence of any invariant simple closed rectifiable curve in D, including periodic orbits. The required uniform asymptotic stability of the linear system (2.11) can be proved by construction of a suitable Lyapunov function.
Let $x \mapsto P(x)(\ {}^{n}_{2}\ ) \times (\ {}^{n}_{2}\ )$ be a matrix- valued function that is C' for $x \in D$. Assume that P^{-1} exists and is continuous for $x \in K$, the compact absorbing set. A quantity $\bar{q}_2$ defined as $\bar{q}_2$

$$= lim_{t\to\infty} \sup \sup_{x_0 \in K} \frac{1}{t} \int_0^t \rho(B(x(s, x_0)))ds \tag{2.12}$$

where $B = P_f P^{-1} + PJ^{[2]}P^{-1}$. The matrix P_f is obtained by replacing each entry P_{ij} of P by it's derivative in the direction of f, p_{ij} and the quantity $\rho(B)$ is the Lozinskiĭ measure of B with respect to a vector norm $|.|$ in $\Re^N$, $N = (\ {}^{n}_{2}\ )$, and is defined by

$$\rho(B) = \inf_{h\to 0^+} \frac{|I + hB| - 1}{h}$$

For a simply connected region D, the condition $\bar{q}_2 < 0$ rules out the presence of any orbit that may give rise to a simple closed rectifiable curve that is invariant for equation (2.10) such as closed orbits, homoclinic orbits and heteroclinic cycles. Moreover, it is robust under C' local perturbations of f near any non-equilibrium point that is non-wandering.
The following global stability result is established in Theorem (3.5) in Li and Muldowney [23],

Theorem 2: Assume D is simply connected and that assumptions (H_1) and H_2 hold. Then the unique equilibrium $\bar{x}$ is globally stable in D if $\bar{q}_2 < 0$.
This is the approach we adopt to establish the global stability of the endemic equilibrium point, E_1.
It has already been established in Section 2 that system (2.2) has a unique equilibrium E_1 which is locally stable whenever $\mathcal{R}_0 > 1$.
Let $x = (h, l, i)$ and let $f(x)$ denote the vector field of system (2.2). The second additive compound matrix associated with the Jacobian matrix (2.6) of the general solution $x(t)$ of system (2.2) is given by

$$J^{[2]} = \begin{pmatrix} J_{11} & \beta_2 h + \phi\lambda & \beta_2 h + \phi\lambda - \lambda \\ \alpha & J_{22} & (1-\delta)\lambda - \beta_1 h \\ 0 & \beta_1 l + \beta_2 i & J_{33} \end{pmatrix}, \tag{2.13}$$

where $J_{11} = -(\lambda + \mu + \beta_1 l + \beta_2 i + \alpha + r_1) + \beta_1 h + \delta\lambda$
$J_{22} = -(\lambda + \mu + \beta_1 l + \beta_2 i + r_2 + d)$ and
$J_{33} = \beta_1 h + \delta\lambda - (\alpha + r_1 + r_2 + d)$.

We choose a vector $|.|$ in $\Re^3$ and a 3×3 matrix-valued function $P(x)$ such that the quantity $\bar{q}_2 < 0$. We set P as $P(h,l,i) = diag(1, \frac{l}{i}, \frac{l}{i})$, then $P^{-1} = diag(1, \frac{i}{l}, \frac{i}{l})$. Now, $P_f P^{-1} = diag(0, \frac{l'}{l} - \frac{i'}{i}, \frac{l'}{l} - \frac{i'}{i})$ and $PJ^{[2]}P^{-1}$ is given by

$$PJ^{[2]}P^{-1} = \begin{pmatrix} J_{11} & \frac{i}{l}[\beta_2 h + \phi\lambda] & \frac{i}{l}[\beta_2 h + \phi\lambda - \lambda] \\ \alpha\frac{l}{i} & J_{22} & \lambda - (\delta\lambda + \beta_1 h) \\ 0 & \beta_1 l + \beta_2 i & J_{33} \end{pmatrix}$$

such that $B = P_f P^{-1} + PJ^{[2]}P^{-1}$ is given by

$$B = \begin{pmatrix} J_{11} & \frac{i}{l}[\beta_2 h + \phi\lambda] & \frac{i}{l}[\beta_2 h + \phi\lambda - \lambda] \\ \alpha\frac{l}{i} & \frac{l'}{l} - \frac{i'}{i} + J_{22} & \lambda - (\delta\lambda + \beta_1 h) \\ 0 & \beta_1 l + \beta_2 i & \frac{l'}{l} - \frac{i'}{i} + J_{33} \end{pmatrix}$$

which can be written in block form as

$$B = \begin{pmatrix} B_{11} & B_{12} \\ B_{21} & B_{22} \end{pmatrix}$$

with
$B_{11} = J_{11} = -(\lambda + \mu + \beta_1 + \beta_2 + \alpha + r_1) + \beta_1 h + \delta\lambda$
$B_{12} = [\frac{i}{l}(\beta_2 h + \phi\lambda), \frac{i}{l}(\beta_2 h + \phi\lambda - \lambda)]$

$$B_{21} = \begin{pmatrix} \alpha\frac{l}{i} \\ 0 \end{pmatrix}$$

and

$$B_{22} = \begin{pmatrix} \frac{l'}{l} - \frac{i'}{i} + J_{22} & \lambda - (\delta\lambda + \beta_1 h) \\ \beta_1 l + \beta_2 i & \frac{l'}{l} - \frac{i'}{i} + J_{33} \end{pmatrix}$$

Following Li and Muldowney [23], we let (u,v,w) denote the vectors in $\Re^3 \cong \Re^{\begin{pmatrix} 3 \\ 2 \end{pmatrix}}$ for the norm $|.|$ in $\Re^3$ chosen as $|(u,v,w)| = max\{|u|, |v| + |w|\}$ and let ρ denote the Lozinskiĭ measure with respect to this norm. The estimate for the Lozinskii measure $\rho(B)$ with respect to this norm is given by $\rho(B) \leq sup\{g_1, g_2\}$ where $g_1 = \rho_1(B_{11}) + |B_{12}|$ and $g_2 = |B_{21}| + \rho_1(B_{22})$. It should be noted that $|B_{12}|$ and $|B_{21}|$ are operator norms of B_{12} and B_{21} with respect to the l_1 vector norm when they are regarded as mappings from $\Re^2$ to $\Re$ and $\Re^2$ to $\Re$ respectively. $\rho_1(B_{22})$ denotes the Lozinskiĭ measure of the 2×2 matrix B_{22} with respect to the l_1 norm in $\Re_2$. To compute $\rho_1(B_{22})$, we add the absolute value of the off-diagonal elements to the diagonal one in each column of B_{22} and take the maximum of the two sums.
Now, $\rho_1(B_{11}) = -(\lambda + \mu + \beta_1 l + \beta_2 i + \alpha + r_1) + \beta_1 h + \delta\lambda$,

$$\begin{aligned} \rho_1(B_{22}) &= max\{\frac{l'}{l} - \frac{i'}{i} + J_{22} + \beta_1 l + \beta_2 i; \frac{l'}{l} - \frac{i'}{i} + J_{33} + \lambda - \delta\lambda - \beta_2 h\} \\ &= \max\{\frac{l'}{l} - \frac{i'}{i} - (\lambda + \mu + r_2 + d); \frac{l'}{l} - \frac{i'}{i} + \lambda - (\alpha + r_1 + r_2 + d)\} \\ &= \frac{l'}{l} - \frac{i'}{i} - (\lambda + \mu + r_2 + d), \end{aligned}$$

$|B_{21}| = max\{\frac{i}{l}[\beta_2 h + \phi\lambda]; \frac{i}{l}[\beta_2 h + \phi\lambda - \lambda] = \frac{i}{l}[\beta_2 h + \phi\lambda]$ and $|B_{21}| = \alpha\frac{l}{i}$.
Thus for $t > \bar{t}$,

$$\begin{aligned} g_1 &= \rho(B_{11}) + |B_{22}| \\ &= -(\lambda + \mu + \beta_1 l + \beta_2 i + \alpha + r_1) + \beta_1 h + \delta\lambda + \frac{i}{l}(\beta_2 h + \phi\lambda) \end{aligned} \tag{2.14}$$

and

$$\begin{aligned} g_2 &= |B_{21}| + \rho_1(B_{22}) \\ &= \frac{l'}{l} - \frac{i'}{i} + \alpha\frac{l}{i} - (\lambda + \mu + r_2 + d) \end{aligned} \tag{2.15}$$

From the second and third equations of system (2.2), we have

$$\frac{l'}{l} + (\alpha + r_1) - (\beta_1 h + \delta\lambda) = (\beta_2 h + \phi\lambda)\frac{i}{l} \tag{2.16}$$

$$\frac{i'}{i} = \alpha\frac{l}{i} - (r_2 + d) \tag{2.17}$$

Substituting equation (2.16) into equation (2.14) and equation (2.17) into equation (2.15) gives:

$$g_1 = \frac{l'}{l} - (\lambda + \mu + \beta_1 l + \beta_2 i), \tag{2.18}$$

$$g_2 = \frac{l'}{l} - (\lambda + \mu). \tag{2.19}$$

Now,

$$\begin{aligned} \rho(B) &\leq sup\{g_1, g_2\} \\ &\leq \frac{l'}{l} - (\lambda + \mu) \\ &= \frac{l'}{l} - M, \end{aligned}$$

where $M = (\lambda + \mu)$. We then have

$$\frac{1}{t}\int_0^t \rho(B)ds \leq \frac{1}{t}\log\frac{l'}{l} - M$$

which implies $\bar{\mathrm{q}}_2 \leq -\frac{M}{2} < 0$.

This completes the proof. Since the endemic equilibrium E_1 exists whenever $\mathcal{R}_0 > 1$, we therefore conclude that the endemic equilibrium point, E_1, is globally stable whenever $\mathcal{R}_0 > 1$.

The stability results in this section can be summarized in the following theorem:

Theorem 3: System (2.2) admits two equilibrium points; the disease free equilibrium point, E_0, and the endemic equilibrium point, E_1, which are such that:

(a) When $\mathcal{R}_0 < 1$, E_0 is the only equilibrium in the feasible region Γ and is both locally and globally stable.

(b) When $\mathcal{R}_0 > 1$, then E_0 is unstable and there exists a unique endemic equilibrium point E_1, that is both locally and globally stable. Furthermore, all solutions starting in Γ and sufficiently close to E_0 move away from E_0 if $\mathcal{R}_0 > 1$.

3 Numerical Simulation

The system was simulated using ODE solvers coded in Matlab programming language. The estimated parameter values given in Table 2 were used. The numerical results compare with the analytical results obtained in Section 2. The variations of plant proportions with time when $\mathcal{R}_0 < 1$

are shown in Fig. 2 while the situation where $\mathcal{R}_0 > 1$ at which the disease persists in the population when introduced is depicted in Fig. 3. The relationship between the basic reproduction number, $\mathcal{R}_0$ and the contact (or transmission) rates β_1 and β_2 is captured in Fig. 3 where it is shown that $\mathcal{R}_0$ increases with the contact rates.

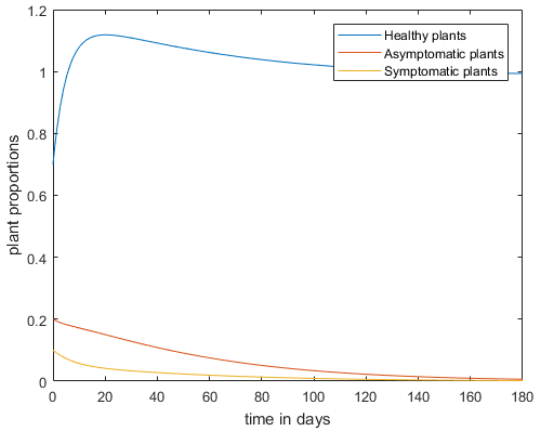

Fig. 2. Proportions of plant sizes at the disease free equilibrium point for $\mathcal{R}_0 = 0.67 < 1$ with $\lambda = 0.1667, \delta = 0.0286, \phi = 0.0143, \beta_1 = 0.034, \beta_2 = 0.068, \mu = 0.0022, \alpha = 0.0265, r_1 = 0.0525, r_2 = 0.105, d = 0.0167$ and $h = 0.7, l = 0.2, i = 0.1$

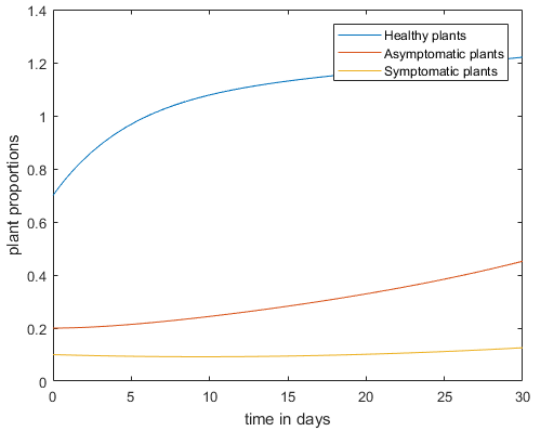

Fig. 3. Proportions of plant sizes at the endemic equilibrium point equilibrium point for $\mathcal{R}_0 = 1.25$ with $\lambda = 0.1667, \delta = 0.0286, \phi = 0.0286, \beta_1 = 0.068, \beta_2 = 0.068, \mu = 0.0022, \alpha = 0.0265, r_1 = 0.0525, r_2 = 0.0525, d = 0.0167$ and $h = 0.7, l = 0.2, i = 0.1$

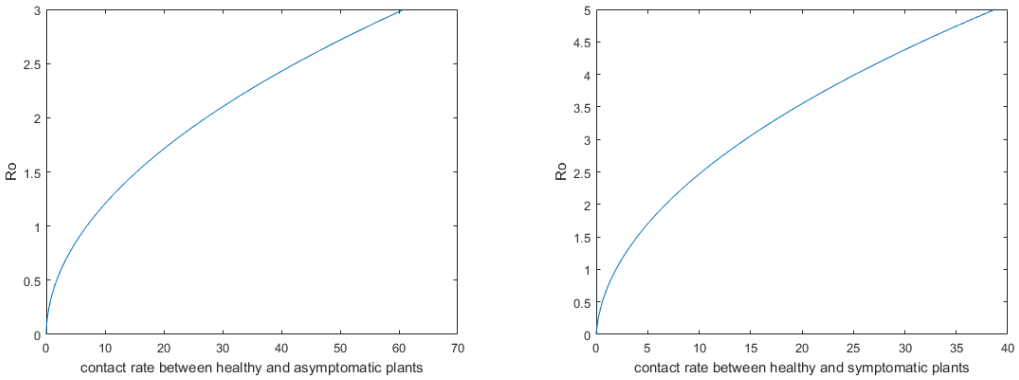

Fig. 4. Variation of $\mathcal{R}_0$ with β_1 and β_2 for parameter values in Table 1

3.1 Sensitivity Analysis of $\mathcal{R}_0$

The sensitivity analysis of the basic reproduction number gives a measure of how different parameters influence it. The higher the magnitude of the sensitivity index, the greater is the parameter's influence on $\mathcal{R}_0$. Following Arriola and Hyman [26], we define sensitivity analysis, χ_p, by the expression $\chi_p = \frac{\partial \mathcal{R}_0}{\partial P} \times \frac{P}{\mathcal{R}_0}$, where P is the parameter under consideration. When the sensitivity index of a particular parameter is positive, it implies that an increase in the parameter results in a corresponding increase in the value of $\mathcal{R}_0$ and vice versa. We performed a sensitivity analysis of the basic reproduction number for parameter values given in Table 2 and obtained the results shown in Table 3.1. It can be deduced from the results for sensitivity analysis that $\mathcal{R}_0$ is highly sensitive

Table 2. Expression and values for sensitivity analysis of $\mathcal{R}_0$

Parameter, P	Expression for sensitivity Analysis, χ_P	Value of χ_P
β_1	$\frac{\beta_1(r_2+d)}{\beta_1(r_2+d)+\beta_2\alpha}$	0.723
β_2	$\frac{\beta_2\alpha}{\beta_1(r_2+d)+\beta_2\alpha}$	0.277
r_1	$\frac{-r_1(r_2+d)}{(\alpha+r_1-\delta\lambda)(r_2+d)-\alpha\phi\lambda}$	-0.725
r_2	$\frac{-\alpha r_2[\phi\lambda\beta_1+\beta_2(\alpha+r_1-\delta\lambda)]}{[(\alpha+r_1-\delta\lambda)(r_2+d)-\alpha\phi\lambda][\beta_1(r_2+d)+\beta_2\alpha]}$	-0.217
λ	$1-(\frac{\lambda}{\lambda+\mu})[\frac{(\alpha+r_1-2\delta\lambda-\mu\delta)(r_2+d)-\alpha\phi(2\lambda+\mu)}{(\alpha+r_1-\delta\lambda)(r_2+d)-\alpha\phi\lambda}]$	0.1041
μ	$-\frac{\mu}{(\lambda+\mu)}$	-0.013
α	$\frac{\alpha(r_2+d)[\beta_2(r_1-\delta\lambda)+\beta_1(\phi\lambda-(r_2+d))]}{[(\alpha+r_1-\delta\lambda)(r_2+d)-\alpha\phi\lambda][\beta_1(r_2+d)(\beta_2\alpha)]}$	-0.064
d	$\frac{d\alpha[\alpha\phi\beta_1+\beta_2(\alpha+r_1-\delta\lambda)]}{[(\alpha+r_1-\delta\lambda)(r_2+d)-\phi\alpha\lambda][\beta_1(r_2+d)+\beta_2\alpha]}$	0.069
ϕ	$\frac{\alpha\phi\lambda}{(\alpha+r_1-\delta\lambda)(r_2+d)-\alpha\phi\lambda}$	0.025
δ	$\frac{\lambda\delta(r_2+d)}{(\alpha+r_1-\delta\lambda)(r_2+d)-\alpha\phi\lambda}$	0.0658

to the parameters β_1 and r_1 representing the contact with asymptomatically infected plants and their roguing rate; is moderately sensitive to the parameters β_2 and r_2 representing contact with symptomatically infected plants and their roguing respectively. For example, a 10% increment in the transmission rate due to contact with asymptomatically infected plants would translate in a 7.2% increment in the basic reproduction number. Likewise, a 10% increase in the rouging rate of asymptomatically infected banana plants would result in 7.3% decrease in the value of $\mathcal{R}_0$. It is also clear that $\mathcal{R}_0$ is least sensitive to the vertical transmission parameters δ and ϕ in addition to

parameters representing natural processes μ, λ and d as well as the parameter α, whose reciprocal is the average length of the incubation period. Management measures therefore should target the parameters with a high sensitivity index in particular reducing the contact rate with asymptomatic plants and intensifying their roguing.

4 Discussion

In this paper, a model for the dynamics of banana *Xanthomonas* wilt incorporating infectious force in both the asymptomatic and symptomatic stages has been formulated and analyzed. The model also incorporates both horizontal and vertical transmission. The motivation was the need to investigate the contribution of asymptomatically infected banana plants on the reported resurgence and persistence of the disease in the East and Central African region. The equilibrium points of the model were obtained and their stability established. Numerical simulation as well as sensitivity analysis of the basic reproduction number were carried out. It was revealed that the dynamics of the models are completely determined by the basic reproduction number,$\mathcal{R}_0$; that both the disease-free and the endemic equilibrium points are asymptotically stable whenever $\mathcal{R}_0 > 1$. It was also found out that $\mathcal{R}_0$ is more sensitivity to the parameters involving asymptomatically infected plants than those concerning symptomatically infected plants specifically β_1 and r_1 vis-a-vis β_2 and r_2. It was further revealed that vertical transmission parameters ϕ and δ had a nominal effect which is not surprising considering that BXW is a fast killing disease.

5 Conclusion

Previous efforts have emphasized the roguing of symptomatically infected plants but this study recommends that similar attention should equally be placed on the asymptomatically infected plants as well in order to address the reported re-occurrence and persistence of the disease. Attempts should be made by regional governments to provide clean planting to farmers for re-establishment of destroyed fields and those who want start new plantations. Farmers should be discouraged to use apparently healthy looking suckers which may unfortunately be infectious as planting materials. In addition, cheaper and accessible technologies need to be developed to enable farmers positively identify infectious but asymptomatic plants rather than relying on visible symptoms to identify diseased plants. Roguing of both asymptomatic and symptomatic plants is crucial in managing BXW as it reduces the inoculum load thereby reducing opportunities for the further spread of the pathogen.

Acknowledgements

The authors are grateful to Bioversity International for the financial support and the reviewers for their valuable comments that improved the quality of the manuscript.

Competing Interests

The authors declare that no conflict of interests took place during the preparation of the manuscript.

References

[1] Nowakunda K, Tushemereirwe W. Farmer acceptance of introduced banana genotypes in Uganda. African Crop Science Journal. 2004;12:1-6.

[2] Yirgou D, Bradbury JF. A note on wilt of banana caused by the enset wilt organism *Xanthomonas musacearum*. East African Agricultural and Forestry Journal. 1974;40:111-114.

[3] Tushemereirwe WK, Kangire A, Smith JJ, Ssekiwoko F, Nakyanzi M, Kataama D, Musiitwa C, Karyeija R. An outbreak of bacterial wilt on banana in Uganda. Info Musa. 2003;12:6-8.

[4] Eden-Green SJ. How can the advance of banana Xanthomonas campestris pv.musacearum wilt be halted? Infomusa. 2004;13(2):38-41.

[5] Tinzaara W, Gold CS, Ssekiwoko F, Bandyopadhyay R, Abera A, Eden-Green, SJ. Role of insects in the transmission of banana bacterial wilt. African Crop Science Journal. 2006;14:105-110.

[6] Blomme G, Ocimati W, Sivirihauma C, Vutseme L, Mariamu B, Kamira M, van Schagen B, Ekboir J, Ntamwira J. A control package revolving around the removal of single diseased banana stems is effective for the restoration of *Xanthomonas* wilt infected fields. European Journal of Plant Pathology. 2017;149(2):385-400.

[7] Karamura E, Turyagyenda, FL, Tinzaara W, Blomme G, Molina A, Markham R. *Xanthomonas* wilt (*Xanthomonas campestrispv. musacearum*) of Bananas in East and Central Africa. Diagnostic and Management Guide. Bioversity International, Kampala, Uganda; 2008.

[8] Turyagyenda LF, Blomme G, Ssekiwoko F, Karamura E, Mpiira S, Eden-Green S. Rehabilitation of banana farms destroyed by *Xanthomonas* wilt in Uganda. Journal of Applied Biosciences. 2008;8(1):230-235.

[9] Tinzaara W, Karamura EB, Blomme G, Jogo W, Ocimati W, Rietveld A, Kubiriba J, Opio F. Why sustainable management of *Xanthomonas* wilt of banana in east and central Africa has been elusive. Acta Horticulture. 2013;986:157-164.

[10] Mwebaze JM, Tusiime G, Tushemerweire WK, Maina M. Development of a semi-selective medium for *Xanthomonas campestris pv.musacearum*. African Crop Science Journal. 2006;14:129-133.

[11] Nakato VG, Ocimati W, Blomme G, Fiaboe KKM, Beed F. Comparative importance of infection routes for Banana *Xanthomonas* wilt and implications on disease epidemiology and management. Canadian Journal of Plant Pathology; 2014. DOI: 10.1080/07060661.2014.959059.

[12] Ocimati W, Sekiwoko F, Karamura E, Tinzaara W, Eden-Green S, Blomme G. Systemicity of *Xanthomonas campestris pv. musacearum* and time to disease expression after inflorescence infection in East African highland and Pisang Awak bananas in Uganda. Plant Pathology. 2013;62:777-785.

[13] Ssekiwoko F, Turyagyenda LF, Mukasa H, Eden-Green SJ, Blomme G. Spread of *Xanthomonas campestris pv.musacearum* in banana (Musaspp.) plants following infection of the male inflorescence. Acta Horticulture. 2010;879: 349-358.

[14] Kweyunga EH, Tumwiine J. A mathematical model for the vector transmission and control of banana *Xanthomonas* wilt. Journal of Mathematics Research. 2017;9(4):101-113.

[15] Nannyonga B, Luboobi LS, Tushemereirwe P, Jablonska-Sabuka M. International Journal of Biomathematics. 2015;8(5):1550065. DOI:10.1142/S1793524515500655

[16] Nakakawa J, Mugisha JYT, Shaw MW, Karamura E. A mathematical model for the dynamics of banana *Xanthomonas* wilt with vertical transmission and inflorescence infection. Journal of Biological Systems. 2016;24(1):147-165.

[17] Nakakawa J, Mugisha JYT, Shaw MW, Tinzaara W, Karamura E. 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. DOI:10.1155/2017/4865015

[18] Tripathi L, Tripathi JN, Tushemereirwe WK. Rapid and efficient production of transgenic East African highland banana using intercalary meristematic tissues. African Journal of Biotechnology. 2008;7:1438-1445.

[19] Anderson RM, May RM. Infectious diseases of humans. Oxford University Press, Oxford; 1991.

[20] Britton NF. Mathematical biology. London, UK: Springer; 2003.

[21] LaSalle JP. The stability of dynamical systems. CBMS-NSF Regional Conference Series in Applied Mathematics, SIAM, Philadelphia. 1976;25.

[22] Smith RA. Some applications of Hausdorff dimension inequalities for ordinary differential equations. Proceedings of Royal Society. 1986;104:235-259.

[23] Li MY, Muldowney JS. A geometric approach to global- stability problems, SIAM. Journal of Mathematical Analysis. 1996;27:1070-1083.

[24] Butler GJ, Waltman P. Persistence of in dynamical systems. Proceedings of American Mathematical Society. 1986;96:425-430.

[25] Li MY, Muldowney JS. On Bendixson's criterion. Journal of Differential Equations. 1994;106:27-39.

[26] Arriola LM, Hyman JM. Being sensitive to uncertainity. Journal of Computing and Engineering; 2007. DOI:10.1109/MCSE.2007.27

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