Journal of Mathematics Research; Vol. 9, No. 4; August 2017
ISSN 1916-9795 E-ISSN 1916-9809
Published by Canadian Center of Science and Education

A Mathematical Model for the Vector Transmission and Control of Banana Xanthomonas Wilt

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Received: April 24, 2017 Accepted: May 16, 2017 Online Published: July 19, 2017

doi:10.5539/jmr.v9n4p101 URL: https://doi.org/10.5539/jmr.v9n4p101

Abstract

Banana *Xanthomonas* wilt is currently wrecking havoc in East and Central Africa. In this paper, a novel theoretical model for the transmission of banana *Xanthomonas* wilt by insect vectors is formulated and analyzed. The model incorporates roguing of infected plants and replanting using healthy suckers. The model is analyzed for the existence and stability of the equilibrium points. The global stability of the disease-free equilibrium point was determined by using a Lyapunov function and LaSalle's invariance principle. For the global stability of the endemic equilibrium point, the theory of competitive systems, compound matrices and stability of periodic orbits were used. It was established that if the basic reproduction number satisfies $R_0 \leq 1$, the disease-free equilibrium point is globally stable and the disease will be wiped out and if $R_0 > 1$, the endemic equilibrium is stable and the disease persists. A numerical simulation of the model was also carried out. It was found out that at appropriate roguing and replanting, the disease can be contained.

Keywords: Banana *Xanthomonas wilt*, basic reproduction number, competitive systems, disease control, endemic equilibrium, roguing

1. Introduction

Banana is the world's fourth most important food crop after rice, wheat and maize. Approximately one-third of bananas produced globally are grown in sub-Saharan Africa. The great lakes region of Africa is the largest banana producing and consuming region in Africa with Uganda the second largest producer after India. Being a perennial crop with all year-round fruiting and harvesting abilities, banana plays a significant role as it represents a source of food and income for most Ugandans. It's estimated that banana production contributes up to 22% of national agricultural rural income in Uganda (Tripathi *et al,* 2008).

However the production of bananas in Uganda is threatened by the outbreak of Banana *Xanthomonas* wilt caused by *Xanthomonas campestris pv. musacearum*. The disease is mainly transmitted by insect vectors through the male buds (Tushemereirwe *et al,* 2004) and Rutikanga *et al.,* 2016). Contaminated farming tools, infected planting materials, nectar-sucking birds and bats are the other means of transmission of the bacterium (Tinzaara *et al.,* 2006; Buregyeya *et al.,* 2014). Insects pick up the bacterium externally on their body parts when they visit the banana inflorescence and transmit it to healthy plants while foraging for pollen and juice (Tinzaara *et al.,* 2006). It is characterized by progressive yellowing and wilting of leaves, uneven and premature ripening of fruit, wilting of bracts, shriveling and rotting of male buds and a characteristic yellow ooze from the cut pseudo stem (Eden-Green, 2004; Tushemereirwe *et al,* 2004).

Management measures include roguing of diseased banana plants, debudding or removal of the male buds using a forked stick, use of clean planting materials and sterilization of farm tools using chemicals or a flame of fire (Tinzaara *et al.,* 2016; Kubiriba *et al.,* 2014; Kubiriba & Tushemereirwe, 2014; Jogo *et al.,* 2013). In the absence of a cure (chemical or biological) and unavailability of resistant varieties, the disease continues to spread to new areas. It should however be noted that where these management options have been strictly applied, the disease has been contained .

Mathematical models for plant epidemics have been developed with the ultimate purposes of developing control strategies and assessing their effectiveness. Such models have been developed by Fishman *et al.* (1983), Fishman & Marcus (1984), Chan & Jeger (1994), Holt *et al.* (1997), Holt *et al.*(1999), Madden *et al.* (2000), Gibson *et al.* (2004), Jeger *et al.* (2004), Tang *et al.* (2010) , Zhonghua & Yaohong (2014), Nannyonga *et al.* (2015) and Nakakawa *et al.*(2016) among others. Holt *et al.* (1999) formulated an epidemiological model for identifying control strategies for tomato leaf curl virus disease in Southern India. Chan & Jeger (1994) developed an analytical model of plant virus disease dynamics with roguing and replanting of healthy plants and assessed the effectiveness of roguing. Fishman & Marcus (1984) formulated

and analyzed a model for the spread of a plant disease with periodic removal of infected plants while Gibson *et al.*(2004) developed a model to study the control strategies for sweet potato virus disease in Africa. Furthermore, epidemiological and economic models were formulated to study the spread and control of citrus tristeza virus disease (Fishman *et al.,* 1983); the models were used to assess the effectiveness of the eradication policy. An epidemiological model incorporating vector dynamics applied to African Cassava Mosaic virus disease was considered by Holt *et al.* (1997) who concluded that roguing of diseased plants had little effect on disease incidence. Jeger *et al.* (2004) modeled disease dynamics and control interventions for insect transmitted plant epidemics while Nannyonga *et al.*(2015) applied optimal control theory to study the vector and contaminated tool preventive methods to control the spread of banana *Xanthomonas wilt* within plantations. Clearly, literature is abundant on modeling the dynamics of many plant epidemics with the aim of gaining insights on epidemic development and control. Most of these models incorporate replanting and roguing of infected plants, see Tang *et al.* (2010) and Zhonghua & Yaohong (2014) and the references cited there in. However, models for the dynamics of banana *Xanthomonas* wilt transmitted by insect vectors are scarce in literature. The model proposed in this study also incorporates the roguing of infected banana plants and replanting using healthy suckers. The effectiveness and suitability of these measures is assessed.

This paper is organized as follows: In Section 2, we formulate the model, determine the equilibrium points and analyze their stability. In Section 3, a numerical simulation of the model is carried out while in Section 4, we discuss the results and make recommendations.

2. Model Formulation and Analysis

We assume that there is no latency in both the host and vector populations and that the transmission of the bacterium by the vectors is by non-circulative non-persistent mode. We also assume that the emigration and immigration rates of the vectors are equal so that the total vector population size is constant. It is further assumed that the roguing rate of infected plants is balanced by the replanting rate by the healthy suckers. Banana *xanthomonas* wilt is a systematic disease in that the whole plant is infected and can therefore be used as a conceptual unit of modeling. The total banana plant population size at time t given by $N_H(t)$, is partitioned into subclasses of healthy and infected plants with sizes denoted by $S_H(t)$ and $I_H(t)$, respectively. Recruitment into the healthy plant population is via two processes namely, emergency of new suckers at a constant rate λ_1 and replanting using healthy suckers. Healthy plants are harvested at a rate μ whose reciprocal is the life time of a healthy banana plant. Roguing of infected plants is done at a rate r which is also the replanting rate of healthy suckers. The death rate of infected plants is assumed to be at a constant rate d. The vector population is divided into two classes; the susceptible and infective vectors whose sizes at time t are $S_V(t)$ and $I_V(t)$ respectively. Recruitment into the susceptible vector population is through the immigration of vectors which depends on the total vector population size $N_V(t)$. The emigration rate of both classes of vectors is assumed to be equal to the immigration rate at a constant g.

Healthy banana plants become infected through contact with infective vectors through a standard incidence term $\beta_1 S_H \frac{I_V}{N_H}$ where β_1 is the contact rate between infective vectors and healthy banana plants. Similarly, susceptible vectors acquire the bacterium through contact with infected banana plants through a standard incidence term $\beta_2 S_V \frac{I_H}{N_H}$ where β_2 is the contact rate between infected banana plants and non-infective vectors and $\beta_2 \frac{I_H}{N_H}$ is the force of infection.

The following system of ordinary differential equations based on the basic model assumptions, parameters and variables is derived to describe the dynamics of the vector transmission of banana *Xanthomonas* wilt and it's control:

$$\begin{aligned}
\frac{dS_H}{dt} &= (\lambda_1 + r - \mu)S_H - \beta_1 S_H \frac{I_V}{N_H}, \\
\frac{dI_H}{dt} &= \beta_1 S_H \frac{I_V}{N_H} - (r + d)I_H, \\
\frac{dS_V}{dt} &= gN_V - \beta_2 S_V \frac{I_H}{N_H} - gS_V, \\
\frac{dI_V}{dt} &= \beta_2 S_V \frac{I_H}{N_H} - gI_V,
\end{aligned} \tag{1}$$

where $N_H = S_H + I_H$ is the total host plant population and satisfies the equation $\frac{dN_H}{dt} = (\lambda_1 + r - \mu)S_H - (r + d)I_H$ and $N_V = S_V + I_V$ is the total vector population and satisfies the equation $\frac{dN_V}{dt} = 0$. It is noted that the total banana plant population size is variable whereas the total vector population size is constant. In the absence of the disease, the entire banana plantation consists of only the healthy plants and $r = 0$. The total population of the healthy plants grows exponentially when $\lambda_1 > \mu$, declines exponentially to extinction when $\lambda_1 < \mu$, and is constant for $\lambda_1 = \mu$. In this study we assume that $\lambda_1 > \mu$.

For convenience, we carry out a non–dimensionalization of the model as follows: Define $s_h = \frac{S_H}{N_H}$, $i_h = \frac{I_H}{N_H}$, $s_v = \frac{S_V}{N_V}$,

$i_v = \frac{I_V}{N_V}$ and $m = \frac{N_V}{N_H}$. By differentiating with respect to time and simplifying, we obtain the following system of equations:

$$\begin{aligned} \frac{ds_h}{dt} &= \alpha s_h - \beta_1 m s_h i_v - \alpha s_h^2 + \phi s_h i_h, \\ \frac{di_h}{dt} &= \beta_1 m s_h i_v - \alpha s_h i_h - \phi i_h + \phi i_h^2, \\ \frac{ds_v}{dt} &= g - \beta_2 s_v i_h - g s_v, \\ \frac{di_v}{dt} &= \beta_2 s_v i_h - g i_v, \end{aligned} \tag{2}$$

where $\alpha = \lambda_1 + r - \mu$ and $\phi = r + d$. Further, using the substitution $s_v = 1 - i_v$, system (2) can be reduced to the following system of three ordinary differential equations:

$$\begin{aligned} \frac{ds_h}{dt} &= \alpha s_h(1 - s_h) - \beta_1 m s_h i_v + \phi s_h i_h, \\ \frac{di_h}{dt} &= \beta_1 m s_h i_v - \alpha s_h i_h - \phi i_h(1 - i_h), \\ \frac{di_v}{dt} &= \beta_2 i_h(1 - i_v) - g i_v. \end{aligned} \tag{3}$$

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

$\Gamma = \{(s_h, i_h, i_v) \in \mathfrak{R}_+^3 \mid s_h, i_h, i_v \geq 0, s_h + i_h = 1, 0 \leq i_v \leq 1\}$,

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

2.1 Equilibrium Points and Basic Reproduction Number

To determine the equilibrium points, the right hand-side of system (3) is equated to zero. Calculations show that system (3) has four equilibrium points namely $E_0(0, 0, 0)$, $E_1(1, 0, 0)$, $E_2(0, 1, \frac{\beta_2}{\beta_2+g})$ and $E_3(s_h^*, i_v^*, i_h^*)$ where $s_h^* = \frac{(\alpha+\phi)(\beta_2+g)-\beta_1\beta_2 m}{\beta_2(\alpha+\phi)}$, $i_h^* = \frac{\beta_1\beta_2 m - g(\alpha+\phi)}{\beta_2(\alpha+\phi)}$ and $i_v^* = \frac{\beta_1\beta_2 m - g(\alpha+\phi)}{\beta_1\beta_2 m}$. In this study, the two equilibrium points of interest are E_1, the disease free equilibrium and E_3, the endemic equilibrium point.

The basic reproduction number, R_0, defined by Anderson & May (1991) as the number of secondary infectives arising out of one infective introduced in the disease free population, is determined using the approach by van den Driessche & Watmough (2002) and found to be given by the expression $R_0 = \sqrt{\frac{\beta_1\beta_2 m}{g(\lambda_1+2r+d-\mu)}}$.

2.2 Local Stability of Disease-free Equilibrium Point

The Jacobian matrix evaluated at the disease-free equilibrium point, $E_1 = (1, 0, 0)$, is

$$J_{E_1} = \begin{pmatrix} -\alpha & \phi & -\beta_1 m \\ 0 & -(\alpha+\phi) & \beta_1 m \\ 0 & \beta_2 & -g \end{pmatrix}, \tag{4}$$

which has three eigenvalues one being $-\alpha$ and the other two are obtained from the sub–matrix

$$J_{E_1'} = \begin{pmatrix} -(\alpha+\phi) & \beta_1 m \\ \beta_2 & -g \end{pmatrix},$$

whose trace $(J_{E_1'}) = -(\alpha + \phi + g)$ and determinant $(J_{(E_1')}) = (\alpha + \phi)g - \beta_1\beta_2 m$. For the disease free equilibrium point to be locally asymptotically stable, all the eigenvalues must have negative real parts. In this case, the trace $(J_{E_1'})$ is negative while the determinant should be positive. Clearly $trace < 0$ and $determinant > 0$ if $R_0 < 1$. Thus, the disease free equilibrium point is locally asymptotically stable if $R_0 < 1$.

2.3 Global Stability of Disease Free Equilibrium Point

The global stability of the disease–free equilibrium point is determined using Lyapunov's direct method (also called the second method of Lyapunov) as follows: Consider a dependent variable $L = gi_h + \beta_1 m i_v$ which is such that

1. $L \geq 0$ along the solutions of system (3)

2. $L = 0$ if and only if $i_h = 0$ and $i_v = 0$

The derivative of L along the solutions of system (3) is

$$\begin{aligned} L' &= g\beta_1 m s_h i_v - (\alpha+\phi)g s_h i_h + \beta_1\beta_2 m i_h(1-i_v) - g\beta_1 m i_v, \\ &\leq [-(\alpha+\phi)g s_h i_h + \beta_1\beta_2 m i_h(1-i_v)], \\ &= (\alpha+\phi)g i_h[\frac{\beta_1\beta_2 m(1-i_v)}{(\alpha+\phi)g} - s_h], \\ &= [R_0^2(1-i_v) - s_h](\alpha+\phi)g i_h, \end{aligned}$$

since $\beta_1\beta_2 m = (\alpha+\phi)gR_0^2$.

In the first inequality, the term $g\beta_1 m s_h i_v - \beta_1 m g i_v < 0$ in Γ and has been neglected.

In the case that $R_0 < 1$, then $L' = 0$ if and only if $i_h = 0$. For the case that $R_0 = 1$, then $L' = 0$ if and only if $i_h = 0$, $i_v = 0$ and $s_h = 1$. For the case when $R_0 > 1$, then $L' > 0$ for s_h sufficiently close to 1 except when $i_h = i_v = 0$. Solutions starting sufficiently close to E_1 leave a neighborhood E_1 except those on the invariant s_h- axis, on which system (3) reduces to $s_h' = \alpha s_h(1-s_h)$ and thus $s_h \longrightarrow 1$ as $t \longrightarrow \infty$. In all cases, the largest compact invariance set in $\{(s_h, i_h, i_v) \in \Gamma : L' = 0\}$ when $R_0 \leq 1$ is the singleton $\{E_1\}$. LaSalle's invariance principle (LaSalle, 1976) then implies that E_1 is globally asymptotically stable in Γ.

2.4 Local Stability of the Endemic Equilibrium Point

For simplification, the coordinates of the endemic equilibrium point can be expressed in terms of the basic reproduction number R_0 as $s_h^* = 1 - \frac{g}{\beta_2}\left(R_0^2 - 1\right)$, $i_h^* = \frac{g}{\beta_2}\left(R_0^2 - 1\right)$ and $i_v^* = \frac{R_0^2-1}{R_0^2}$. Clearly, the endemic equilibrium point E_3 exists only when $R_0 > 1$ otherwise it will not make biological sense.

The Jacobian matrix evaluated at the endemic equilibrium point is

$$J_{E_3} = \begin{pmatrix} -\alpha s_h^* & \phi s_h^* & -\beta_1 m s_h^* \\ \phi i_h^* & -\alpha s_h^* - \phi(1-2i_h^*) & \beta_1 m s_h^* \\ 0 & \frac{g i_v^*}{i_h^*} & -gR_0^2 \end{pmatrix}. \tag{5}$$

Let $p = \frac{g}{\beta_2}(R_0^2 - 1) = i_h^*$, then system (5) can be simplified to

$$J_{E_3} = \begin{pmatrix} -\alpha(1-p) & \phi(1-p) & -\beta_1 m(1-p) \\ \phi p & -\alpha(1-p) - \phi(1-2p) & \beta_1 m(1-p) \\ 0 & \frac{\beta_2}{R_0^2} & -gR_0^2 \end{pmatrix}.$$

The characteristic equation is obtained from $\det(J_{E_3} - \lambda I) = 0$, where λ represents the eigenvalues and I is the identity matrix and is given by

$$\lambda^3 + \lambda^2[gR_0^2 + 2\alpha(1-p) + \phi(1-2p)] + \lambda[gR_0^2\{\alpha(1-p) + \phi(1-2p)\} - g(\alpha+\phi)(1-p)$$

$$+\alpha(1-p)\{gR_0^2 + \alpha((1-p) + \phi(1-2p) - \phi^2 p(1-p)]$$

$$+gR_0^2\alpha(1-p)[\alpha(1-p) + \phi(1-2p)] - \alpha g(\alpha+\phi)(1-p)^2 + (\alpha+\phi)gp\phi(1-p) - gr_0^2 p\phi(1-p) = 0,$$

which is of the form

$$\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0,$$

where

$$\begin{aligned} a_1 &= gR_0^2 + \alpha(1-p) + \phi(1-2p) + \alpha(1-p), \\ &= gR_0^2 + (2\alpha+\phi)(1-p) - \phi p. \end{aligned}$$

$$a_2 = gR_0^2[\alpha(1-p) + \phi(1-2p)] - g(\alpha+\phi)(1-p) + \alpha(1-p)[gR_0^2 + \alpha(1-p) + \phi(1-2p)] - \phi^2 p(1-p),$$

$$a_2 = gR_0^2[(\alpha+\phi)(1-p) + \alpha(1-p) - p\phi] + (\alpha+\phi)(1-p)[\alpha(1-p) - g - p\phi].$$

and the constant term a_3 is given by

$$\begin{aligned} a_3 &= gR_0^2\alpha(1-p)[\alpha(1-p)+\phi(1-2p)] - \alpha g(\alpha+\phi)(1-p)^2 + (\alpha+\phi)gp\phi(1-p) - gR_0^2\phi^2 p(1-p), \\ &= g(1-p)(\alpha+\phi)(\alpha-p(\alpha+\phi))[R_0^2-1]. \end{aligned}$$

By the Routh-Hurwitz criteria, the endemic equilibrium point is locally asymptotically stable if $a_1, a_2, a_3 > 0$ and $a_1a_2 > a_3$. Now, $a_1 > 0$, $a_2 > 0$ and $a_3 > 0$ if $\alpha > p(\alpha+\phi)$ and $R_0 > 1$.

It remains to show whether $a_1a_2 > a_3$. Now,

$$\begin{aligned} a_1a_2 - a_3 = [gR_0^2 + (\alpha+\phi)(1-p) + \alpha - p(\alpha+\phi)][gr_0^2\{(\alpha+\phi)(1-p) + \alpha - p(\alpha+\phi)\} + (\alpha+\phi)(1-p)\{\alpha - p(\alpha+\phi)\}] \\ -gR_0^2(1-p)(\alpha+\phi)\{\alpha - p(\alpha+\phi)\} + g(\alpha+\phi)(1-p)\{\alpha - p(\alpha+\phi)\} \\ = (gR_0^2)^2[(\alpha+\phi)(1-p) + \alpha - p(\alpha+\phi)] + (\alpha+\phi)(1-p)gR_0^2[\alpha - p(\alpha+\phi)] \\ +gR_0^2(\alpha+\phi)(1-p)[(\alpha+\phi)(1-p) + \alpha - p(\alpha+\phi)] \\ +(\alpha+\phi)^2(1-p)^2[\alpha - p(\alpha+\phi)] + gR_0^2(\alpha - p(\alpha+\phi))[(\alpha+\phi)(1-p) + \alpha - p(\alpha+\phi)] \\ +(\alpha - p(\alpha+\phi))(\alpha+\phi)(1-p)[\alpha - p(\alpha+\phi)] - gR_0^2(1-p)(\alpha+\phi)[\alpha - p(\alpha+\phi)] \\ +g(\alpha+\phi)(1-p)[\alpha - p(\alpha+\phi)]. \end{aligned}$$

Thus,

$$\begin{aligned} a_1a_2 - a_3 = (gR_0^2)^2[(\alpha+\phi)(1-p) + \alpha - p(\alpha+\phi)] + gR_0^2(\alpha+\phi)(1-p)[(1-p)(\alpha+\phi) + \alpha - p(\alpha+\phi)] \\ +(\alpha+\phi)^2(1-p)^2[\alpha - p(\alpha+)] + gR_0^2(\alpha - P(\alpha+\phi))[(\alpha+\phi)(1-p) + \alpha - p(\alpha+\phi)] \\ (\alpha+\phi)(1-p)(\alpha - p(\alpha+\phi))[\alpha - p(\alpha+\phi) - g] + \\ g(\alpha+\phi)(1-p)[\alpha - p(\alpha+\phi)] - g^2R_0^2(\alpha+\phi)(1-p). \end{aligned}$$

It is clear that $a_1a_2 > a_3$ when $\alpha > p(\alpha+\phi)$.

Since $a_1, a_2, a_3 > 0$ and $a_1a_2 > a_3$, then by the Routh-Hurwitz criteria for the characteristic polynomial of degree 3, the endemic equilibrium point is locally asymptotically stable when $R_0 > 1$.

2.5 Global Stability of the Endemic Equilibrium Point

The global stability of the endemic equilibrium point is determined using the approach developed by Li & Muldowney (1995) for SEIR models. We start with the preliminaries.

Let $x \mapsto f(x)$ be a C' function for x an open set $D \subset \mathfrak{R}^n$.

Consider the differential equation

$$x' = f(x). \tag{6}$$

Denote by $x(t, x_0)$ the solution to equation (6) such that $x(0, x_0) = x_0$.

Definition 1 *A set K is said to be absorbing in D for equation (6) 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 (6) has a unique equilibrium point $\bar{x}$ in D.

The equilibrium point $\bar{x}$ is said to be globally stable if it is locally stable and all trajectories in D converge to $\bar{x}$. The assumptions H_1 and H_2 are satisfied if $\bar{x}$ 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 (6) (Butler & Waltman, 1986).

The following global stability problem is formulated in Li & Muldowney (1996).

Global stability problem: Under assumptions H_1 and H_2, find conditions on the vector field of equation (6) such that the local stability of $\bar{x}$ implies its global stability in D.

System (6) is said to satisfy the Poincaré-Bendixson property if any non-empty compact omega set of equation (6) that contains no equilibria is a closed orbit.

Any autonomous system (6) in the plane satisfies the Poincaré-Bendixson property (Hale,1969). In addition, a three-dimensional competitive system satisfies the Poincaré-Bendixson property in a convex region (Hirsch,1990).

The following global stability result is proved in Li & Muldowney (1995).

Theorem 1 *Assume that*

1. *assumptions* (H_1) *and* (H_2) *hold;*
2. $\bar{x}$ *is locally asymptotically stable*
3. *System (6) satisfies the Poincaré- Bendixson property;*
4. *each periodic orbit of (6) in D is orbitally asymptotically stable.*

Then the unique equilibrium $\bar{x}$ is globally asymptotically stable in D. Assumption (3) is satisfied if D is a convex region in $\mathfrak{R}^3$ and (6) is a competitive system in D.

The orbital stability of periodic solutions in $\mathfrak{R}^n$ $(n \geq 2)$ can be verified using the following result of Muldowney (1990).
Theorem 2 *A periodic orbit* $\Omega = \{p(t) : 0 \leq t < \omega\}$ *is orbitally asymptotically stable with asymptotic phase if the linear system*

$$z'(t) = \frac{\partial f^{[2]}}{\partial x}(p(t))z(t) \tag{7}$$

is asymptotically stable where $\frac{\partial f^{[2]}}{\partial x}$ is the second additive compound matrix $\frac{\partial f}{\partial x}$ of f.

Definition 2 *A matrix is stable if all it's eigenvalues have negative real parts.*

Theorem 3 *An* $n \times n$ *real matrix A is stable if and only if* $A^{[2]}$ *is stable and* $(-1)^n det(A) > 0$. (Li & Wang,1998)

Using the theorems (1), (2), (3) above, the following result is used to establish the global stability of the endemic equilibrium point.

Theorem 4 *Assume that*

1. *assumptions* (H_1) *and* (H_2) *hold;*
2. *system (6) satisfies a Poincaré-Bendixson property;*
3. *For each periodic solution* $x = p(t)$ *to (6) with* $p(0) \in D$*, system (6) is asymptotically stable;*
4. $(-1)^n det(\frac{\partial f}{\partial x}(\bar{x}))> 0$

Then the unique equilibrium $\bar{x}$ *is globally asymptotically stable in D.*

This approach is used in order to establish the global stability of the endemic equilibrium point. System (3) is uniformly persistent, (see Proposition (3.3) in Li *et al.* (1999)), if there exists a constant $0 < c < 1$ such that any solutions $(s_h(t), i_h(t), i_v(t))$ with $(s_h(0), i_h(0), i_v(0)) \in \dot{\Gamma}$ satisfies

$$min\{\lim_{t\longrightarrow\infty} inf s_h(t), \lim_{t\longrightarrow\infty} inf i_h(t), \lim_{t\longrightarrow\infty} inf i_v(t)\} > c. \tag{8}$$

The boundedness of Γ and condition (8) imply that (3) has a compact absorbing set $K \subset \dot{\Gamma}$ [Butler & Waltman,1986].

Definition 3 *A differential equation (6) is said to be competitive in D if for some diagonal matrix* $H = diag(e_1, e_2, e_3.....e_n)$*, where each* e_i *is either* -1 *or* $+1$*, then* $H(\frac{\partial f}{\partial x})H$ *has non-positive off diagonal elements for all* $x \in D$*. If D is convex, the flow of a competitive system preserves for* $t < 0$*, the partial ordering in* $\mathfrak{R}^n$ *defined by the orthant* $K = \{(x_1, x_2, ...x_n) \in \mathfrak{R}^n : e_i x_i \geq 0\}$.

To show that system (3) is a competitive system in the convex region $\dot{\Gamma}$, consider a partial ordering defined by the orthant $\{(s_h, i_h, i_v) \in \mathfrak{R}^n : s_h \leq 0, i_h \geq 0, i_v \leq 0\}$ and choose

$$H = \begin{pmatrix} -1 & 0 & 0 \\ 0 & 1 & 0 \\ 0 & 0 & -1 \end{pmatrix} \tag{9}$$

and using (5), then it can easily be verified that $HJ_{E_3}H$ has no non-positive diagonal elements for all $x \in D$. Thus, system (3) is competitive in ω, with respect to the partial ordering defined by the orthant $\{(s_h, i_h, i_v) \in \Re^n : s_h \leq 0, i_h \geq 0, i_v \leq 0\}$. The competitiveness of system (3) in a convex region $\dot{\Gamma}$ implies that system (3) satisfies a Poincaré-Bendixson property and thus conditions (1) and (2) hold. Using the substitution $s_h = 1 - i_h$, system (3) can be re-written as

$$\begin{aligned} s_h' &= (\alpha+\phi)s_h i_h - \beta_1 m s_h i_v, \\ i_h' &= \beta_1 m s_h i_v - (\alpha+\phi)s_h i_h, \\ i_v' &= \beta_2 i_h(1-i_v) - g i_v. \end{aligned} \tag{10}$$

whose Jacobian matrix is

$$J_{E_3} = \begin{pmatrix} (\alpha+\phi)i_h - \beta_1 m i_v & (\alpha+\phi)s_h & -\beta_1 m s_h \\ \beta_1 m i_v - (\alpha+\phi)i_h & -(\alpha+\phi)s_h & \beta_1 m s_h \\ 0 & \beta_2(1-i_v) & -\beta_2 i_h - g \end{pmatrix}. \tag{11}$$

The second compound matrix of the Jacobian matrix (11)

$$J_{E_3}^{[2]} = \begin{pmatrix} (\alpha+\phi)(i_h - s_h) - \beta_1 m i_v & \beta_1 m s_h & \beta_1 m s_h \\ \beta_2(1-i_v) & (\alpha+\phi)i_h - \beta_1 m i_v - \beta_2 i_h - g & (\alpha+\phi)s_h \\ 0 & \beta_1 m i_v - (\alpha+\phi)i_h & -(\alpha+\phi)s_h - \beta_2 i_h - g \end{pmatrix} \tag{12}$$

And the second compound matrix of system (11) along a periodic solution $(s_h(t), i_h(t), i_v(t))$ is

$$\begin{aligned} X' &= [(\alpha+\phi)(i_h - s_h) - \beta_1 m i_v]X + \beta_1 m s_h Y + \beta_1 m s_h Z, \\ Y' &= \beta_2(1-i_v)X + [(\alpha+\phi)i_h - \beta_1 m i_v - \beta_2 i_h - g]Y + (\alpha+\phi)s_h Z, \\ Z' &= [\beta_1 m i_v - (\alpha+\phi)i_h]Y + [-(\alpha+\phi)s_h - \beta_2 i_h - g]Z. \end{aligned} \tag{13}$$

To show that system (13) is asymptotically stable, consider the following Lyapunov function

$$V(X, Y, Z; s_h, i_h, i_v) = \sup\left[\mid X \mid, \frac{i_h}{i_v}(\mid Y \mid + \mid Z \mid) \right]. \tag{14}$$

Then, the orbit ψ of the periodic solution $(s_h(t), i_h(t), i_v(t))$ is at a positive distance from the boundary $\partial\Gamma$ by uniform persistence. Thus, there exists a constant $c_1 > 0$ such that

$$V(X, Y, Z; s_h, i_h, i_v) \geq c_1 \sup\{\mid X \mid, \frac{i_h}{i_v}(\mid Y \mid + \mid Z \mid)\} \tag{15}$$

for all $(X, Y, Z) \in \Re^3$ and $(s_h, i_h, i_v) \in \psi$. The left hand derivative of V along a solution $(X(t), Y(t), Z(t))$ to system (13) and (s_h, i_h, i_v) can be estimated as follows:

$$\begin{aligned} D_+ \mid X(t) \mid &\leq [(\alpha+\phi)(i_h - s_h) - \beta_1 m i_v] \mid X(t) \mid + \beta_1 m s_h \mid Y(t) \mid + \beta_1 m s_h \mid Z(t) \mid, \\ &= [(\alpha+\phi)(i_h - s_h) - \beta_1 m i_v] \mid X(t) \mid + \beta_1 m s_h(\mid X(t) \mid + \mid Z(t) \mid), \\ D_+ \mid X(t) \mid &= [(\alpha+\phi)(i_h - s_h) - \beta_1 m i_v] \mid X(t) \mid + \frac{\beta_1 m s_h i_v}{i_h}\frac{i_h}{i_v}[\mid Y(t) \mid + \mid Z(t) \mid], \end{aligned} \tag{16}$$

$$D_+ \mid Y(t) \mid \leq \beta_2(1-i_v) \mid X(t) \mid + [(\alpha+\phi)i_h - \beta_1 m i_v - \beta_2 i_h - g] \mid Y(t) \mid + [(\alpha+\phi)s_h] \mid Z(t) \mid, \tag{17}$$

$$D_+ \mid Z(t) \mid \leq [\beta_1 m i_v - (\alpha+\phi)i_h] \mid Y(t) \mid - [(\alpha+\phi)s_h + \beta_2 i_h + g] \mid Z(t) \mid. \tag{18}$$

Now, from inequalities (17) and (18), the following is obtained

$$D_+(\mid Y(t) \mid + D_+ \mid Z(t) \mid) \leq \beta_2(1-i_v) \mid X(t) \mid - (\beta_2 i_h + g)(\mid Y(t) \mid + \mid Z(t) \mid), \tag{19}$$

such that

$$D_+ \frac{i_h}{i_v}(\mid Y(t) \mid + \mid Z(t) \mid) \leq \beta_2 \frac{i_h}{i_v}(1-i_v) \mid X(t) \mid + (\frac{i_h'}{i_h} - \frac{i_v'}{i_v} - \beta_2 i_h - g)\frac{i_h}{i_v}(\mid Y(t) \mid + \mid Z(t) \mid) \tag{20}$$

Equations (16) and (20) lead to

$$D_+ V(t) \leq max\{g_1(t), g_2(t)\}V(t), \tag{21}$$

where

$$g_1(t) = (\alpha + \phi)(i_h - s_h) - \beta_1 m i_v + \frac{\beta_1 m i_v s_h}{i_h}, \tag{22}$$

$$g_2(t) = \frac{\beta_2 i_h(1 - i_v)}{i_v} + (\frac{i_h'}{i_h} - \frac{i_v'}{i_v} - \beta_2 i_h - g). \tag{23}$$

From system (3),

$$\begin{aligned} i_h' &= \beta_1 m s_h i_v - (\alpha + \phi) s_h i_h, \\ \frac{\beta_1 m s_h i_v}{i_h} &= \frac{i_h'}{i_h} + (\alpha + \phi) s_h. \end{aligned} \tag{24}$$

Substituting equation (24) into equation (22) and using the expressions for i_v and i_h at the endemic equilibrium point gives

$$\begin{aligned} g_1(t) &= \frac{i_h'}{i_h} - \beta_1 m i_v + (\alpha + \phi) i_h, \\ &= \frac{i_h'}{i_h} - \beta_1 m \frac{(R_0^2 - 1)}{R_0^2} + (\alpha + \phi)(R_0^2 - 1)\frac{g}{\beta_2}, \\ &= \frac{i_h'}{i_h} - \beta_1 m \frac{(R_0^2 - 1)}{R_0^2} + \frac{\beta_1 \beta_2 m}{R_0^2}\frac{(R_0^2 - 1)}{\beta_2}, \\ &= \frac{i_h'}{i_h}. \end{aligned}$$

Similarly, from system (3),

$$\begin{aligned} i_v' &= \beta_2 i_h(1 - i_v) - g i_v, \\ \frac{\beta_2 i_h(1 - i_v)}{i_v} &= \frac{i_v'}{i_v} + g. \end{aligned} \tag{25}$$

Substituting equation (25) into equation (23) gives

$$\begin{aligned} g_2(t) &= \frac{i_h'}{i_h} - \beta_2 i_h, \\ &= \frac{i_h'}{i_h} - g(R_0^2 - 1). \end{aligned}$$

Thus,

$$\begin{aligned} D_+ V(t) &\leq max\{g_1(t), g_2(t)\} V(t) \\ &\leq \frac{i_h'}{i_h} \end{aligned}$$

Therefore,

$$\begin{aligned} \int_0^\omega D_+ V(t) &\leq \log i_h(t) \,|_0^\omega, \\ &= 0, \end{aligned}$$

since $i_h(t)$ is periodic of minimal period ω. This relation together with (21) above imply that, $V(t) \longrightarrow 0$ as $t \longrightarrow \infty$ and in turn that $(X(t), Y(t), Z(t)) \longrightarrow 0$ as $t \longrightarrow \infty$ by (15). As a result, the second compound system (13) is asymptotically stable if the minimal period $\omega > 0$. The same estimate also holds when $\omega \longrightarrow 0$. This verifies condition (3).

It now remains to show whether (5) is stable. Recall $p = \frac{g}{\beta_2}(R_0^2 - 1) = i_h$ such that $s_h = 1 - p$. Then, J_{E_3} can be re-written as

$$J_{E_3} = \begin{pmatrix} -\alpha(1-p) & \phi(1-p) & -\beta_1 m(1-p) \\ \phi p & -\alpha(1-p) - \phi(1-2p) & \beta_1 m(1-p) \\ 0 & \frac{\beta_2}{R_0^2} & -gR_0^2 \end{pmatrix}.$$

Now,

$$det(J_{(E_3)}) \quad =$$

$$\begin{aligned}
&= -\alpha(1-p)\{[\alpha(1-p)+\phi(1-2p)][gR_0^2] - \beta_1 m(1-p)\frac{\beta_2}{R_0^2}\} + \phi(1-p)(gR_0^2 p\phi) - \beta_1 m(1-p)\phi p(\frac{\beta_2}{R_0^2}),\\
&= -\alpha(1-p)\{gR_0^2[\alpha(1-p)+\phi(1-p)-\phi p] - g(\alpha+\phi)(1-p)\} + \phi^2 p(1-p)gR_0^2 - gp\phi(\alpha+\phi)(1-p),\\
&= -\alpha(1-p)gR_0^2[(\alpha+\phi)(1-p)-p\phi] + g\alpha(\alpha+\phi)(1-p)^2 + \phi^2 pg(1-p)R_0^2 - gp\phi(\alpha+\phi)(1-p),\\
&= g(1-p)R_0^2[-\alpha(\alpha+\phi)(1-p)+\alpha p\phi + p\phi^2] + g(\alpha+\phi)(1-p)[\alpha(1-p)-p\phi],\\
&= g(1-p)(\alpha+\phi)R_0^2[-\alpha(1-p)+p\phi] + g(\alpha+\phi)(1-p)[\alpha - p(\alpha+\phi)],\\
&= g(1-p)(\alpha+\phi)R_0^2[p(\alpha+\phi)] - g(\alpha+\phi)(1-p)[p(\alpha+\phi)-\alpha],\\
&= g(1-p)(\alpha+\phi)[p(\alpha+\phi)-\alpha]\{R_0^2-1\}.
\end{aligned}$$

Since $\alpha > p(\alpha+\phi)$, then it is clear that determinant $J_{E_3} < 0$ if $R_0 > 1$ and therefore J_{E_3} is stable. This satisfies condition (4) above. Since all conditions of **Theorem 4** have been verified, the endemic equilibrium point E_3 is globally asymptotically stable if $R_0 > 1$.

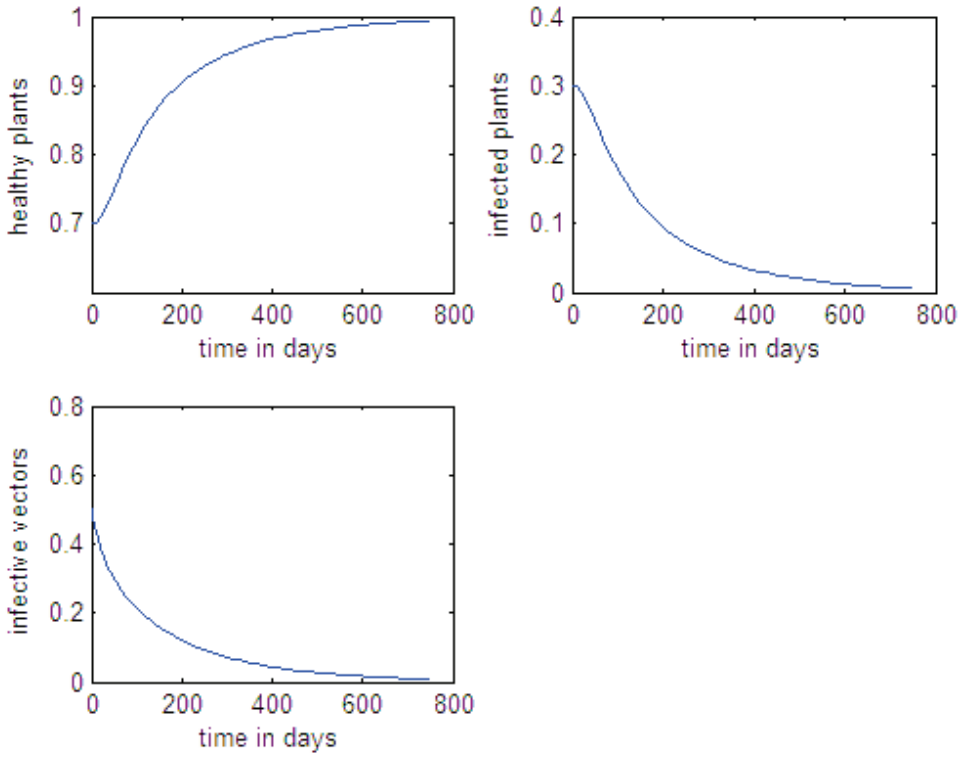

Figure 1. Variation of plant and vector proportions at the disease free equilibrium with initial values $s_h = 0.7, i_h = 0.3, i_v = 0.5$ and parameters $\lambda_1 = 0.001, \mu = 0.0056, r = 0.0105, d = 0.0167, g = 0.02, \beta_1 = \beta_2 = 0.021, m = 1$

3. Numerical Simulation

In this section, we provide a numerical analysis of the model to support the theoretical analysis carried out in Section 2. A set of estimated parameter values are obtained from literature. The model is simulated using Matlab ode45 solver. We simulate the model both in the absence and presence of the disease, and without control methods in place.

4. Discussion

In the paper, a mathematical model for the vector transmission and control of banana *Xanthomonas* wilt incorporating roguing of infected plants and replanting using healthy or disease free suckers was formulated. The steady states were obtained and their stability established. The basic reproduction number was determined using the next generation matrix

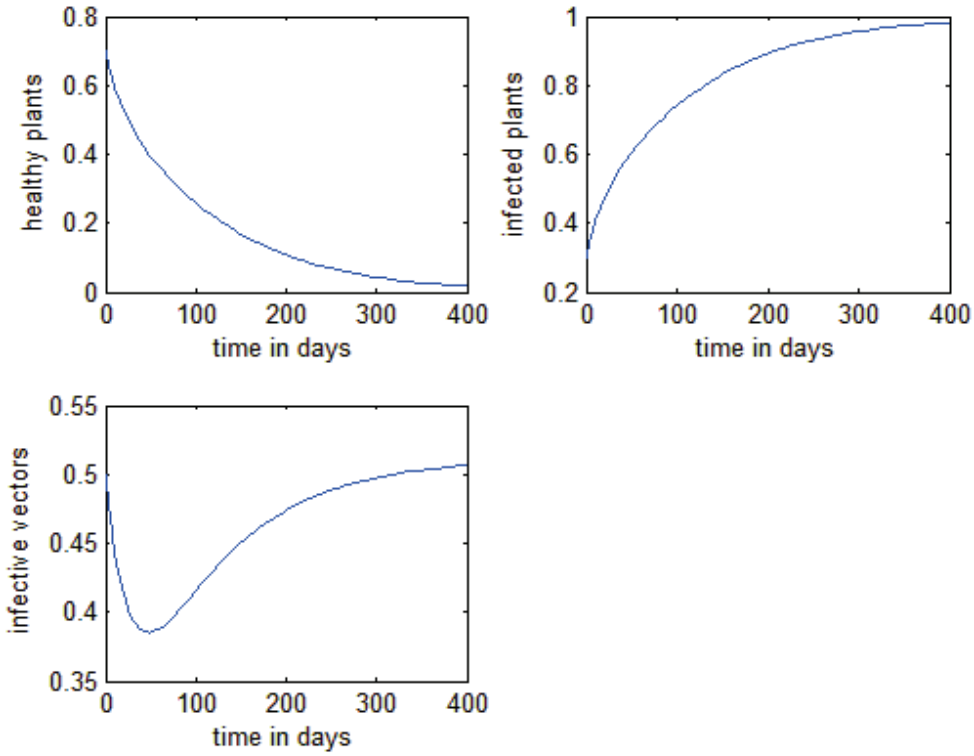

Figure 2. Variation of plant and vector proportions with neither roguing of infected plants nor replanting using healthy suckers

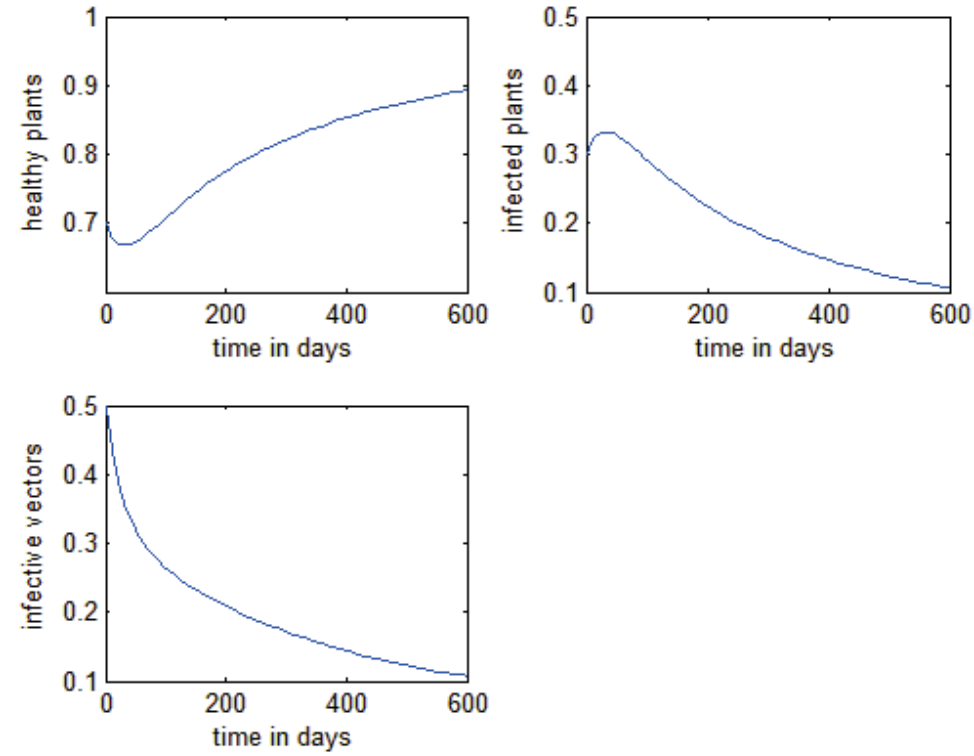

Figure 3. Variation of plant and vector proportions at the endemic equilibrium.

and its implications for disease management analyzed. It was revealed that R_0 is a combination of two basic reproduction numbers namely $R_0' = \frac{\beta_1 m}{\lambda_1+2r+d-\mu}$ for banana plants, that is, the number of secondary infected banana plants arising from one infected banana plant introduced in a plantation of completely healthy plants and $R_0'' = \frac{\beta_2}{g}$ the basic reproduction number for the vectors, that is, the average number of secondary infective vectors that arise out of one infective vector introduced into a population of completely susceptible or pathogen-free vectors. When $R_0' > 1$ the disease grows in the plant hosts and when $R_0'' > 1$ then pathogen spreads in the vector population. The two conditions enable the net reproduction number R_0 to be greater than unity. It was also observed that R_0 is directly proportional to the transmission or contact rates β_1, β_2 and the vector-plant ratio, m. R_0 is inversely proportional to vector immigration/emigration rate, g, the sucker emergence rate, λ_1 , the death rate d and roguing rate r of the infected plants.

Disease management is concerned with lowering the basic reproduction number to a value less than unity. In this case, reduction of the basic reproduction number, R_0 to a value less than unity involves reducing the contact rates β_1, β_2 between the banana plants and the vectors and reducing the vector-plant ratio,m. Contact between the vector and the banana plant is made at the male buds and therefore removal of the male buds is key. Removal of male buds also ensures that the number of insect vectors visiting the plant is also reduced thereby reducing the vector-plant ratio, m. Reducing R_0 to a value less than unity can also be achieved by increasing the roguing rate, r, of infected plants, increasing the emigration rate g of vectors, increasing the replanting rate of healthy suckers λ_1 and increasing the disease-induced death rate, d, of infected plants.

Increasing the roguing rate, r, of infected plants and the replanting rate using healthy suckers are crucial in managing the outbreak of banana *Xanthomonas* wilt. At low contact rates and high roguing rate, the disease can be averted as shown in Figure 1 while in absence of roguing and replanting, the entire healthy plant population is wiped out in finite time as shown in Figure 2. This calls for regular inspection of the banana plantation and prompt roguing of identified diseased plants. It should be noted that where this practice has been strictly applied in Uganda, the disease has been contained. The challenge with replanting using healthy or disease free suckers is absence of certified healthy suckers for replanting. No formal supply of disease free planting materials is available in Uganda within the areas currently affected by banana *Xanthomonas* wilt and consequently, most planting materials are sourced locally by farmers from their own or neighbors fields. Planting materials sourced in this way may appear to be healthy when in fact they are already diseased. This is suspected to be the major method of disease introduction to new areas.

Lastly, increasing the death rate of diseased plants ensures reduction in the inoculum load present. There is need to destroy the infected plants as soon as they are identified thereby killing the bacterium in the process. The greatest challenge is what happens to the infected plants removed by roguing? Unless they are completely destroyed especially by burning them, they can act as a source of the bacterium themselves. In Uganda, farmers have been encouraged to bury the plants removed by roguing but it is feared this may introduce the bacterium in the soil. In the end they are just left lying in plantation which poses a great danger. This calls for more research.

Acknowledgements

Eliab Horub Kweyunga is grateful for the financial support from the Belgium Technical Cooperation (BTC) Ref No: L09UGA/108 in the production of this manuscript. The authors thank the reviewers for their helpful comments.

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