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Modelling the transmission dynamics of banana xanthomonas wilt disease with control measures

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MODELLING THE TRANSMISSION DYNAMICS OF BANANA XANTHOMONAS WILT DISEASE WITH CONTROL MEASURES

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A Dissertation Submitted in Partial Fulfilment of the Requirements for the Degree of Master's in Mathematical and Computer Sciences and Engineering of the Nelson Mandela African Institution of Science and Technology

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ABSTRACT

Banana Xanthomonas Wilt disease (BXW) is a bacterial disease which highly threaten banana production in East and Central Africa. It is caused by a bacteria known as *Xanthomonas campestris pv. musacearum (Xcm)*. Mathematical modelling gives an insight on how to best understand the transmission dynamics and control of the disease. The existing mathematical models for the dynamics of BXW disease have not included contaminated soil, community farming education programmes and clearance of Xcm bacteria in the soil. This study formulated a model which includes contaminated soil. In analysis of the model, the existence and stability of the equilibrium points was checked, calculated the basic reproduction number and carried out sensitivity analysis of some model parameters. We further conducted numerical simulation to validate the results. The numerical simulations showed that the infection rate by contaminated farming tools (β_i and β_e), the infection rate by contaminated soil (ω_2), vertical disease transmission rate (θ), and the shedding rate of Xcm bacteria in the soil (ϕ) are positively sensitive to the basic reproduction number. While, the most negative sensitive parameters are the clearance rate of Xcm bacteria from the soil (μ_h), removal of infected plants from the farm (r), harvesting (α_p), and banana plants disease induced death rate (d). The result also showed that contaminated soil contributes to the transmission and persistence of BXW disease. Furthermore, the basic model was modified to include the control measures. Numerical simulations was conducted to examine the impact of the suggested control measures. It was observed that as Participatory community farming education programmes, timely removal of infected banana plants, clearance of Xcm bacteria in the soil and vertical transmission control measures increases it dramatically reduces the number of secondary infections hence greatly contribute to the control of the BXW disease. Therefore, It is recommend that, along with the existing control measures such as sterilization of farming tools, timely removal of the male bud using a forked stick and planting healthy suckers, scientist and technologist should carry out studies to find a way to reduce or avoid vertical disease transmission and increase the Xcm clearance rate in the soil. Furthermore, technology for early detection of infected plants should be brought down to the local farmers at affordable costs. This will help stakeholders to detect and remove the infected plants from the farm in time and hence reduce the number of secondary infections. Moreover, Participatory community farming education programmes such as Farmers field schools (FFS) should be emphasized and practised.

DECLARATION

I, John Joel Mapinda, do hereby declare to the Senate of Nelson Mandela African Institution of Science and Technology that this dissertation is my own original work and that it has neither been submitted nor presented for similar a award in any other institution.

John Joel Mapinda (Candidate)	Date

The above declaration is confirmed

Prof. Verdiana Grace Masanja (Supervisor 1)	Date
Dr. Gasper Godson Mwanga (Supervisor 2)	Date

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CERTIFICATION

The undersigned certify that they have read and hereby recommend for acceptance by the Nelson Mandela African Institution of Science and Technology the dissertation entitled: Modelling the Transmission dynamics of Banana Xanthomonas Wilt disease with Control measures, in fulfilment of the requirements for the degree of Master's in Mathematical and Computer Sciences and Engineering of the Nelson Mandela African Institution of Science and Technology.

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DEDICATION

I dedicate this work to my family (My wife and children).

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LIST OF ABBREVIATIONS AND SYMBOLS

ACRONYM	DEFINITION
ABCC	A-Avoid disease introduction, B-Break male buds, C-Cut down diseased plants and the last C- Clean tools
BXW	Banana Xanthomonas Wilt
COCSE	Communication and Computational Science and Engineering
DFE	Disease Free Equilibrium
EE	Endemic Equlibrium
E_p	Latently infected banana plant
FFS	Farmer Field School
I_p	Infected banana plant
NM-AIST	The Nelson Mandela Institution of Science and Technology
ODE	Ordinary Differential Equation
R_0	Basic Reproduction number
R_e	Effective Reproduction number
SDSR	Single Diseased Stem Removal
S_p	Susceptible banana plant
Xcm	Xanthomonas campestris pv.musacearum

CHAPTER ONE

INTRODUCTION

1.1 Background of the problem

According to FAO statistics (2017), banana is one of the most important food crop in Tanzania after maize, cassava and sweet potatoes. Farmers use banana fruits as food and for commercial purposes to support their livelihoods. Banana to a lesser extent is used to make fiber, banana wine, banana beer and as ornamental plants. In 2017 the world produced 113 918 764 tonnes (113 918 kt), where 20 017 346 tonnes (20 017 kt) of bananas were produced in Africa (see Fig. 1) (FAO, 2018). In Africa bananas are largely grown in Tanzania, Kenya, Rwanda, Angola, Cameroon and Egypt. In spite of these successes, banana production in East and Central Africa is threatened by declining soil fertility, pests and diseases (Tripathi *et al.*, 2009). Dis-

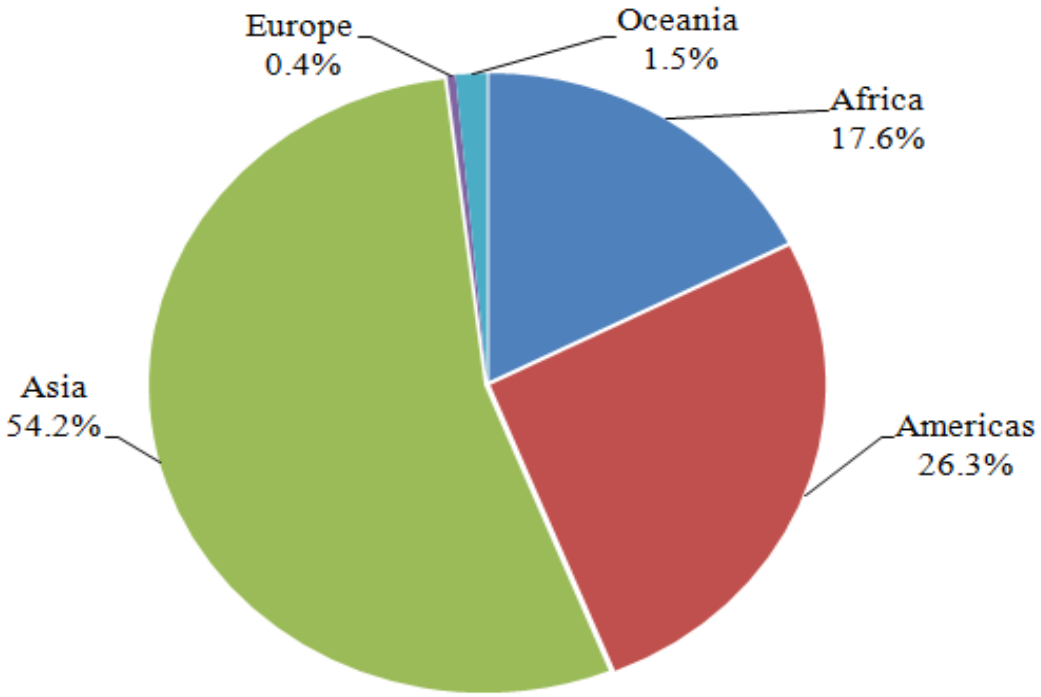


Figure 1: Banana production shares by region for the year 2017

eases affecting banana plants includes: Banana Xanthomonas Wilt (BXW); Banana weevils; Nematodes; and Sigatoka leaf spots. Banana Xanthomonas Wilt (BXW) has been reported to be the major disease which threatens banana farming in East Africa, which in turn affects the livelihood of the farmers and other people who depend on banana for their living Nakakawa *et al.* (2017).

1.1.1 Banana xanthomonas wilt disease causes and transmission

BXW is a devastating disease caused by a bacterium called *Xanthomonas campestris pv.musacearum (Xcm)* Ssekiwoko (2007). As shown in Fig. 2 Xcm bacterium is transmitted by insect vectors, contaminated farming tools, contaminated soil, infected planting suckers and by the transportation of latently infected plants. The vectors such as birds, bats and other flying insects such as bees are the carrying agent of the Xcm bacteria from an infected banana plant to a susceptible plant. Birds transmit the Xcm bacteria after feeding on ripe banana fruits of an infected banana plant to the male buds of a susceptible plant Buregyeya *et al.* (2014). Bats can transmit the disease through feeding on nectar or ripe banana fruits of an infected banana plant to the healthy banana plant Buregyeya *et al.* (2014). Also, other vectors such as bees transmit the disease to a susceptible banana plant when contaminated with Xcm bacteria from a male bud of an infected plant Rutikanga *et al.* (2016), feeding on unsafe disposed remove banana plants and rarely from an inoculated farming tool. A susceptible plant can be infected through contaminated farming tool through farming activities such as weeding, pruning, removing access suckers, harvesting and male bud removal Blomme *et al.* (2014). A susceptible banana plant can acquire Xcm bacteria from the contaminated soil through mechanical injuries caused by farming activities such as weeding and organisms found in the soil such as nematodes and insects found in the lower parts of the plant such as roots (Mwebaze *et al.*, 2006; Hashim, 2013; Shehabu *et al.*, 2010; Sivirihauma *et al.*, 2017). Also, banana plant can be vertically infected from a diseased mother plant to the lateral shoots (Ocimati *et al.*, 2013*a*).

1.1.2 Banana xanthomonas wilt disease symptoms and control

The common symptoms of BXW include: yellowing and wilting of leaves; premature ripening and rotting of fruits; blackening and shrivelling of male bud flower; and yellow ooze observed on the cross section cut of the pseudo stem and eventually death of the entire plant (Kubiriba and Tushemereirwe, 2014; Nakato *et al.*, 2018). Figure 3 subplot a,b and c show the symptoms of leaves, stem and fruits of the banana plant affected by the BXW disease. Debudding, sterilization of the farming tools, roguing, timely removal of the male bud and burning of infected banana plants are some of the common measures used to control the BXW disease. Mathematical models have played and continue to play a crucial role in the understanding of the transmission dynamics of BXW disease leading to the best combination of the control measures to contain its devastating effects.

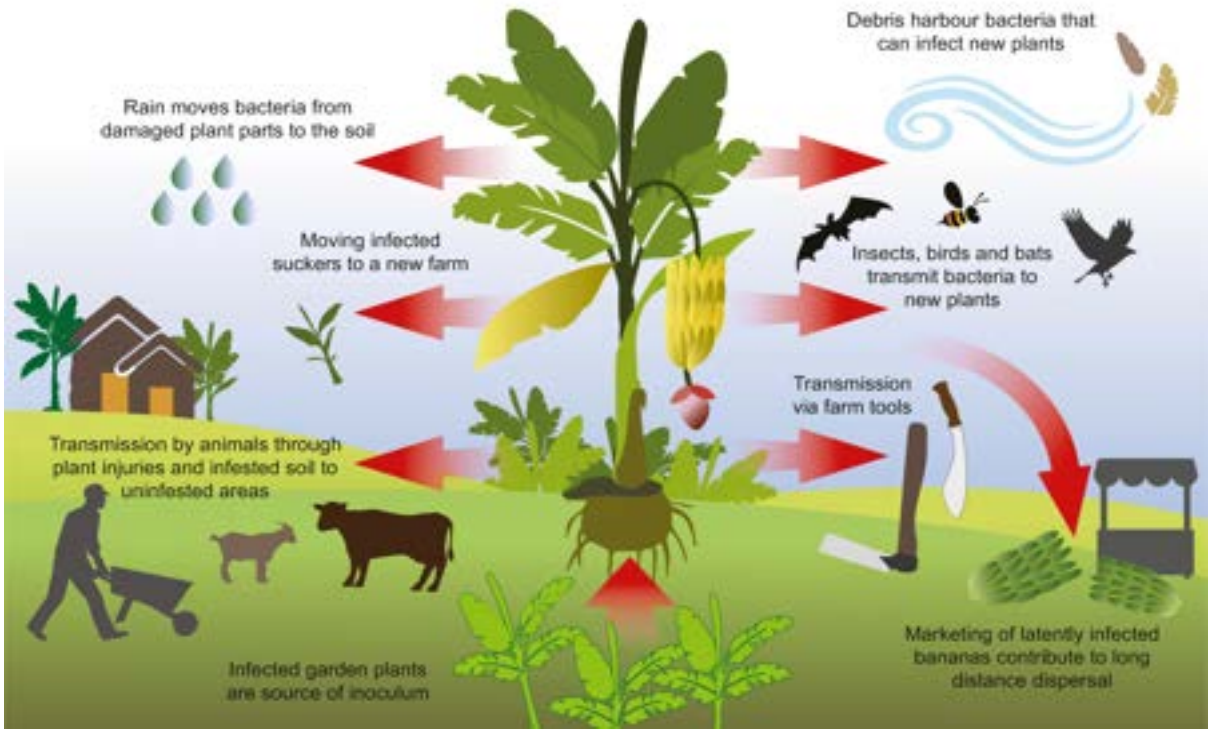


Figure 2: Infection pathways for BXW disease (Uwamahoro *et al.*, 2019)

1.1.3 Mathematical models

Mathematical models have played a great role in understanding the transmission dynamics of the infectious disease and making the right decision on disease control options. Mathematical models also helps in planning, implementing and evaluating the disease detection, control and prevention measures (Ma and Xia, 2009; Siettos and Russo, 2013). Researchers (Nakakawa *et al.*, 2017, 2016; Horub and Julius, 2017; Kweyunga *et al.*, 2018; Nannyonga *et al.*, 2015) among others, have developed mathematical models to study the transmission dynamics of BXW. Most of these models have not included infections resulting from soil inoculum. It is well documented that Xcm soil inoculum has a role to play in the persistence of BXW disease in the field (Sivirihauma *et al.*, 2017; Shimwela *et al.*, 2016; Nakato *et al.*, 2018). Thus inclusion of soil inoculum in the mathematical model will improve our understanding of BXW disease transmission dynamics. This dissertation aimed at formulating a mathematical model for the transmission dynamics of BXW that takes into consideration the soil inoculum as well as the control measures applied to curb the BXW disease.

(a) Yellowing of banana leaves

(b) Yellow ooze

(c) Yellow ooze

Figure 3: Symptoms of the BXW Disease. http://www.promusa.org/

1.2 Statement of the problem

Despite the great contribution of bananas in food security, its production in East and Central Africa is being threatened by various challenges. The major challenge being the BXW disease. Scholars (Nakakawa *et al.*, 2017, 2016; Horub and Julius, 2017; Kweyunga *et al.*, 2018; Nannyonga *et al.*, 2015) have developed mathematical models to study the transmission dynamics of BXW and its controls, but there is little information on the disease transmission dynamics especially when soil inoculum is included in the model. Therefore, this study focuses on developing and analyzing a mathematical model to study the transmission dynamics of BXW which among its key features are the inclusion of the soil inoculum and participatory community education programmes, clearance of Xcm bacteria in the soil, single diseased stem removal and control of vertical transmission as the control measures.

1.3 Rationale of the study

Diseases are the major threats that hinder the production of banana in Africa. Banana xanthomonas wilt disease is one of the diseases that cause huge loss to farmers hence affects their livelihood. There is the need to contain the disease so as to increase banana production in Africa. In order to best control the disease, there is a need to understand the transmission dynamics of banana xanthomonas wilt disease.

Mathematical models have played a great role in understanding the transmission dynamics of infectious diseases and making the right decision on disease control options.Mathematical

models also helps in planning, implementing and evaluating the disease detection, control and prevention measures. Soil inoculum is one of the means by which the disease may spread from one point to another. The models available in the literature does not include contaminated soil. This study aimed to develop a mathematical model which takes into consideration contaminated soil, this will increase the awareness to stakeholders on the transmission dynamics and control of Banana Xanthomonas wilt disease.

1.4 Objectives

1.4.1 General objective

The main objective of this study is to develop and analyze a mathematical model for the BXW disease which among its novelties are inclusion of soil inoculum of the Xcm bacteria causing BXW disease and participatory community education programmes, clearance of Xcm bacteria in the soil, single diseased stem removal and control of vertical transmission as the control measures.

1.4.2 Specific objectives

This study will be guided by the following specific objectives:

(i) To formulate a deterministic mathematical model for analyzing the transmission dynamics of the BXW disease, which include the soil inoculum, vertical disease transmission component and control measures.

(ii) To carry out the sensitivity analysis of the model parameters by using forward sensitivity index.

(iii) To determine the conditions for existence and stability of the equilibrium points.

(iv) To assess the impact of control measures on the transmission dynamics of BXW disease.

1.5 Research questions

This research intended to answer the following questions:

(i) How can a mathematical model for the BXW disease dynamics be formulated?

(ii) What are the most sensitive parameters of the model?

(iii) How can equilibrium points be derived?

(iv) What is the impacts of control measures on the dynamics of BXW disease?

(v) What is the role of soil inoculum on the persistence and spread of BXW disease?

1.6 Significance of the study

The findings of this study will:

(i) Add new knowledge pertaining to the contribution of soil inoculum on the transmission dynamics of the BXW to the existing knowledge and form a base for other researchers working on modelling the infectious disease in plants.

(ii) Provide evidence based information on how to control the BXW disease when the soil inoculum is included in the transmission dynamics and control measures. This information will aid policy and decision making as well as practices at farm level on the best control measures to contain and possibly eliminate the BXW disease.

1.7 Delineation of the study

Modelling the transmission dynamics of banana xanthomonas wilt disease is a broad field. This research is not intended to cover the entire domain of the transmission dynamics of banana xanthomonas wilt disease. Rather, it focuses on modelling the transmission dynamics of banana xanthomonas wilt disease taking into consideration the soil contaminated with Xcm bacteria. Furthermore, the study does not include all the control measures used to contain the BXW disease. Rather, it includes only Participatory community education programmes, Clearance of Xcm bacteria in the soil, Vertical transmission control, and Single diseased stem removal.

CHAPTER TWO

LITERATURE REVIEW

2.1 Introduction

The Banana Xanthomonas Wilt (BXW) is an infectious disease that affects all types of banana plants cultivar in the field Kubiriba and Tushemereirwe (2014). The BXW disease causes a huge loss to farmers due to its high transmission rate which may be through contaminated insect vectors, contaminated farming tools, contaminated soil, infected planting materials and transportation of latently infected banana plants. There are literature on the mathematical model dedicated to study the transmission dynamics of the disease but most of them did not include soil inoculum in the dynamics of the disease. This section briefly reviews the existing literature on mathematical modelling of BXW disease by emphasizing on their relevance, contributions, and gaps intended to be filled by this study.

2.2 Mathematical models for BXW disease

Horub and Julius (2017) developed a Susceptible Infectious (SI) model of BXW disease to study the vector transmission dynamics and control. The study showed that reducing the contact between vector and banana plant by timely removing the male bud using a forked stick have an impact on the BXW transmission dynamics. Furthermore, the study showed that reducing the transmission dynamics of BXW can be done by increase in rogueing and planting rate of healthy suckers. This study adopted the parameter values for harvesting, removal of infected banana plants and death of banana plants due to BXW disease from Horub and Julius (2017). However, Horub and Julius (2017) did not take into consideration the role of asymptomatic infected banana plants, soil inoculum, the disease transmission through infected farming tools and vertical transmission in the dynamics of BXW disease.

Nannyonga *et al.* (2015) assessed the impact of using contaminated tools in the occurrence of BXW. The model considered vectors and the use of contaminated tools as the mode of BXW transmission and their control measures. The study indicated that without controls all susceptible banana plants become infected within 100 days from the onset of the disease, unlike when control measures are applied it takes almost a year for healthy plants to be infected. Furthermore, it was observed that when the plant host inoculation rate per tool exceeds 37.2% more

efforts and resources are needed for a long time to eradicate the disease. This study adopted the parameter values for infection rate through farming tool contaminated by symptomatic banana plants, contact rates between banana plants and vectors, infection rates from infected banana plant to susceptible vectors and from contaminated vectors to susceptible banana plants from Nannyonga *et al.* (2015). Nevertheless, Nannyonga *et al.* (2015) did not consider the vertical transmission, soil inoculum and asymptomatic infected plants which are important components of the dynamics of BXW disease.

Nakakawa *et al.* (2017) formulated an SI mathematical model to study the role of debudding and roguing as BXW infection control within a mixed cultivar plantation. Findings from this study pointed out that the number of infected banana plants declines rapidly when debudding, disinfection of farming tools and Roguing as control measures are regularly applied. Moreover, it was revealed that withdrawal of control measures when the infection is less than 1% quickly increases the rate of infection toward the endemic equilibrium. Therefore, the study recommended that unceasing monitoring should be emphasized even when infection levels are unnoticeable. However, the study did not take into account the role of soil inoculum and asymptomatic infected plants in the transmission dynamics of BXW disease. Therefore, in order to understand better the transmission dynamics of BXW, there is a need to develop a mathematical model which take into consideration the soil inoculum and incorporating the asymptomatic infected plants.

Nakakawa *et al.* (2016) studied the transmission dynamics of BXW by taking into consideration the vertical and insect vectors mode of transmission. In their study, the Susceptible plants, latently infected plants and Infected plants (SEI) model was developed. It was observed that if the disease spread is mainly due to inflorescence infection then single stem removal is a reasonable approach for eradication of the disease. The study further indicated that controlling inflorescence infection and roguing regularly lead to the elimination of BXW in the farmstead setting; with the assumption that there is no tool-based transmission and no soil inoculum transmission. It was suggested that debudding and regular monitoring with roguing through single stem removal are active control measures for BXW eradication. However, neglecting the BXW transmission through infected farming tools is not realistic since debudding, roguing and other farming practices involve the use of farming tools. Therefore, this study has adopted the recruitment rate of susceptible banana plants from Nakakawa *et al.* (2016) and included both infection by contaminated farming tools and through contaminated soil in studying the dynamics of BXW disease.

Kweyunga *et al.* (2018) developed a mathematical model which includes infection forces from both asymptomatic infected and symptomatic infected banana plants. The study involved both horizontal and vertical modes of transmission. It was observed that the parameters which involves an asymptomatic infected plants were the most sensitive to the basic reproduction number. It was recommended that attention should be paid to the asymptomatically infected banana plants for effective control of the disease. However, the study did not consider contaminated soil in the dynamics of BXW disease. This study adopted the model formulated by Kweyunga *et al.* (2018) and modified it to include soil inoculum in the dynamics of BXW disease.

Basing on the reviewed literature about Mathematical Models for BXW disease, there is no study that has included soil inoculum in studying the dynamics of the BXW disease. But, Sivirihauma *et al.* (2017), Shimwela *et al.* (2016) and Nakato *et al.* (2018) argued that soil inoculum play an important role in the dynamics of BXW disease. Therefore, this study aimed to develop a deterministic mathematical model which includes asymptomatic infected banana plants, soil inoculum, contaminated farming tools infections and vertical transmission. Soil inoculum was the main new idea included in the study to improve the understanding of the BXW disease dynamics.

2.3 Control measures for BXW disease

According to Maina *et al.* (2006) removal of the male bud is an effective control measure to the spread of the disease by vector insect, especially when conducted using a forked stick to avoid disease transmission caused by infected farming tools. Maina *et al.* (2006) recommended that in order to avoid the spread of BXW disease during the incubation period, farmers should suspend the use of cutting tools in infected farms. The challenge here is how to determine that the banana plant has been infected when it is in an incubation period. Also, suspending farming activities is a difficult choice to some of the families who depend much on banana production for their living.

Buregyeya (2010) in his study on long distance spread of BXW, observed that the bacterial (Xcm) causing BXW in a room temperature can stay on cutting tools for up to 22 days. The study revealed that disinfection of tools or stopping to use cutting tools for pruning the plant in an infected field are effective ways to eliminate the mechanical spread of BXW by cutting tools. On the other hand, it is very difficult to avoid using the farming tools, as most of the farming activities including pruning and harvesting require the use of cutting tools. This study

aimed to investigate the impact of timely identifying and removing the infected banana plant.

The use of education campaigns to raise the farmers' awareness of different BXW control measures is essential for BXW disease management. Community awareness programs such as Farmer Field Schools (FFS) in Uganda played a big role in controlling banana diseases by practically teaching farmers about farm management skills which includes: understanding the modes of transmission; disease symptoms identification; disease control measures and the best ways to apply the suggested control strategies (Kubiriba *et al.*, 2012). In these schools, Farmers regularly meet with facilitators for different training and practical sessions. However, most of the existing models have not included community education programs as one of the control measures. Therefore, this study has included the participatory community education parameter to investigate its impact on the BXW transmission dynamics.

Kikulwe *et al.* (2019) conducted a study on BXW disease management with the aim of assessing the impactof adopting cultural practices to control the BXW disease. The results shows that training women farmers and having right information about BXW disease control measures increased the adoption of the control measures and reduced disease incidences. Furthermore, it was observed that farmers who fully adopt the control measures had reduced disease incidences and increased production compared to farmers who partially adopt the suggested control measures. Therefore, the study recommended that women should be involved in the training and information sharing among farmers and should be encouraged through participatory approaches. This study has included community education programmes in the model as one of the control strategies, though gender was not considered.

Previously removal of the whole mat where an infected plant arose was highly recommended to avoid further transmission of the disease. It was later observed that it is possible to remove the infected plant and let the healthy plants to grow (Ocimati *et al.*, 2015; Blomme *et al.*, 2017*a*). Ntamwira *et al.* (2019*a*) observed that timely removal of a diseased plant from the mat reduces BXW disease incidences and yield losses compared to removing the whole mat. Blomme *et al.* (2017*a*) revealed that delaying in cutting the disease plant results to the increase in Xcm bacterium inoculum and may results into the transmission of the disease to the attached shoots. Also Ntamwira *et al.* (2019*a*) added that large suckers where the diseased plant have being observed are more susceptible to the disease that the small shoots. This study has included a Single Diseased Stem Removal (SDSR) to contain and possibly eliminate the BXW disease.

Uwamahoro *et al.* (2019) studied the BXW disease control measures in Rwanda. It was pointed out that control measures should address BXW disease factors such as farming practices, spac-

ing between banana plants in the field, choice of banana types and farmers knowledge on BXW disease dynamics. It was recommended that there is a need to make sure that the right BXW disease information reaches the farmers. In this study all the mentioned factors are included in the Community education programs that involves extension officers and researchers.

According to Ochola *et al.* (2015), the factors that hinder the adoption of the ABCC (A-avoid disease introduction, B-Break male buds, C-Cut down diseased plants and the last C- clean tools) BXW disease management package as suggested by Karamura *et al.* (2006) and Tinzaara *et al.* (2009) includes the family size, farming experience, availability of disease information and participation in FFS. Jogo *et al.* (2011) observed that most of farmers are aware of the BXW disease, its symptoms and control measures but very few know how to correctly apply the suggested control measures. Farmers receiving different information from different sources affects the rate of adopting the control measures. Therefore for effective learning and adoption of BXW disease control measures participatory approaches such as farmer groups, FFS and community extension groups should be encouraged.

Basing on the reviewed literature, there is no study that has included the community education programmes, control of vertical BXW disease transmission or clearance of Xcm bacteria in the soil as control strategies to contain the BXW disease. This study has included these parameters to assess their impact to the control of the BXW disease.

CHAPTER THREE

MATERIALS AND METHODS

3.1 Formulation of the basic model

In formulating the model, the model by Kweyunga *et al.* (2018) is modified to include the contaminated soil and single diseased stem removal approach. The model involves two populations, banana plants population and vector population. Each population is then divided into sub populations depending on their infection status. From the defined sub populations, their interactions and assumptions made, a system of differential equations is formulated.

3.2 Theories

In this study, Theorem 2 of Van den Driessche and Watmough (2002) is used to prove the local stability of the disease free equilibrium point. Theorem 2.1 of Shuai and van den Driessche (2013) is used to identify the lyapunov function.

3.3 Next generation method

Basic reproduction number is the average number of new infections generated by the introduction of one infected individual in the population of completely susceptible individuals. Next generation method by Van den Driessche and Watmough (2002) is used to calculate the basic reproduction number. In this method, the model system $\dot{x_i} = f_i(x)$ is divided in two categories thus,

$$f_i(x) = \mathscr{F}_i(x) - \mathscr{V}_i(x), i = 1, ..., n. \tag{3.1}$$

where, $\mathscr{F}_i(x)$ is the transmission part and $\mathscr{V}_i(x)$ is a transition part. The transition part can be expressed as $\mathscr{V}_i(x) = \mathscr{V}_i^-(x) - \mathscr{V}_i^+(x)$, where $\mathscr{V}_i^+(x)$ is the rate of transfer of individuals into a compartment i and $\mathscr{V}_i^-(x)$ is the rate of transfer of individuals out of compartment i. If x_0 is the disease free equilibrium point of the model system, then

$$\frac{dx_i}{dt} = F - V, \tag{3.2}$$

where $F = \frac{\partial \mathscr{F}_i(x)}{\partial x_i}$ and $V = \frac{\partial \mathscr{V}_i(x)}{\partial x_i}$. Therefore the basic reproduction number is obtained by calculating the dominant eigenvalue of the matrix FV^{-1}, thus

$$R_0 = \rho(FV^{-1}), \tag{3.3}$$

where V^{-1} is the average time an individual spends in compartment j during its lifetime and F is the rate at which infected individuals in compartment j produce new infections in compartment i.

3.4 Normalized forward sensitivity index method

Normalized forward sensitivity index method is an approach which is used to determine the sensitivity of the model parameters in the basic reproduction number Chitnis *et al.* (2006). The basic reproduction number R_0 is differentiable with respect to its parameter ς, then the sensitivity index of ς is given by 3.4.

$$\Upsilon_{\varsigma}^{R_0} = \frac{\partial R_0}{\partial \varsigma} \times \frac{\varsigma}{R_0}. \tag{3.4}$$

3.5 Lyapunov function

There are many ways of constructing lyapunov function. In this study, a Lyapunov function of the form

$$V = \omega^T V^{-1} x, \tag{3.5}$$

constructed by using matrix-theoretic method based on the Perron eigenvector Lazarus (2018) and Shuai and van den Driessche (2013) was used to prove the Global Asymptotically Stability (GAS) of the DFE X_{dfe}. Where ω^T is a left eigenvector of the matrix $V^{-1}F$ and the matrices V^{-1} and F as defined in equations 4.108 and 4.106 respectively as in Lazarus (2018) and Shuai and van den Driessche (2013).

Also, a Lyapunov function of the form

$$V = \sum c_j (X_i - X_i^* \ln X_i), \tag{3.6}$$

as defined in Korobeinikov (2004) was used determine the global stability of the endemic equilibrium point (Xee). Where c_j are carefully selected constants and X_i^* is the endemic equilibrium point.

3.6 Data and Numerical simulations

The parameter values are obtained from related literature and some are assumed in the interval (0,1). The values for the parameters d, α_p, r, μ_v and b_v, are adopted from Kweyunga (2011). The values for the parameters β_i, ω_1, ω_3 and a are from Nannyonga *et al.* (2015). The value for b_p is adopted from Nakakawa *et al.* (2016) and θ from Ocimati *et al.* (2013*a*). Table 1 shows the parameter value per day. In numerical analysis, the MATLAB Runge Kutta 4^{th} order solver was used to validate the results of the model.

Table 1: Values of the model parameters

Parameter	Value/Range	Parameter	Value/Range	Parameter	Value/Range
d	0.0167	ω_2	0.4	b_v	0.02
ϕ	0.89	β_i	0.1429	ω_1	0.2
α_p	0.0056	K	1000	μ_v	0.02
θ	0.0286	μ_h	0.01	ω_3	0.2
r	0.0105	q	0.3	η	0.0286
b_p	0.01667	a	0.2	β_e	0.3

CHAPTER FOUR
RESULTS AND DISCUSSION

4.1 Model formulation

4.1.1 Model description

In this study, the mathematical model on the transmission dynamics of BXW by Kweyunga *et al.* (2018) is modified to include the environment contaminated with Xcm bacteria, vector population and some control measures. Furthermore, replacing roguing with a method of single stem removal of a diseased plant is considered because it has been found that Xcm bacteria does not affect all the lateral shoots in the mat (Blomme *et al.*, 2017*b*; Ntamwira *et al.*, 2019*b*).

The model involves the Banana population and insect vector population. Depending on the infection status, the banana plant population is subdivided into three compartments: Susceptible banana plant (S_p); Latently infectious banana plants (E_p); and symptomatic infected banana plants (I_p). Susceptible are healthy banana plants which can be infected by BXW when they come into contact with the Xcm bacterium. It is assumed that susceptible banana plants have an equal chance of being infected when they come into contact with Xcm bacterium. Susceptible banana plants acquire BXW disease through vertical transmission, contaminated farming tools, soil contaminated with Xcm bacteria, planting of latently infected suckers or insect vector (Ocimati *et al.*, 2013*a*; Hashim, 2013; Buregyeya *et al.*, 2014).

The vectors such birds, bats and other flying insects such as bees are the carrying agent of the Xcm bacteria from an infected banana plant to the susceptible plant. Birds transmit the Xcm bacteria after feeding on ripe banana bunches of an infected banana plant to the male buds of a susceptible plant (Buregyeya *et al.*, 2014). Bats can transmit the disease through feeding on nectar or ripe banana fruits of an infected banana plant to the health banana plant (Buregyeya *et al.*, 2014). Also, other vectors such as bees transmit the disease to a susceptible banana plant when contaminated with Xcm bacteria from a male bud of an infected plant (Rutikanga *et al.*, 2016). Feeding on unsafe disposed remove banana plants and rarely from an inoculated farming tool. Generally, we assume that these vectors are contaminated with Xcm bacteria from an infected banana plant. In this model, the vector population is subdivided into susceptible vector (S_v) and vectors contaminated with Xcm bacteria (I_v). Therefore, the total population of the banana plant is given by $N_p = S_p + E_p + I_p$ and total vector population is $N_v = S_v + I_v$. An environment contaminated with Xcm bacteria is represented by A_h.

The model considers constant recruitment of banana plants by emerging of new healthy lateral shoots (suckers) from the banana plants and replanting at the rate of b_p. It is assumed that banana plant can be vertically infected from a diseased mother plant to the lateral shoots from infected banana plant and asymptomatic infected banana plant at the rate of θ and δ respectively (Ocimati *et al.*, 2013*a*). A susceptible plant can be infected through contaminated farming tool through farming activities such as weeding, pruning, removing access suckers, harvesting and male bud removal (Blomme *et al.*, 2014). A farming tool can be contaminated with Xcm bacteria from symptomatic infected banana plants or asymptomatic infected banana plants and transmit the disease at the rate β_i or β_e respectively. Also, susceptible banana plant can be infected by the Xcm bacteria found in the soil at a rate of ω_2. The average daily contact rate of an infected vector to a susceptible banana plant is given by a and ω_1 is the probability that the contact results to infection. Matured banana plants are harvested at the rate of α_p. Latently infected banana plant become an infected plant at the rate of q after showing BXW symptoms. An infected banana plant can be removed from the farm at the rate of r or die due to infection at a rate of d. It is assumed that the rate of vertical transmission is less than the sum of disease induced death rate and removal of infected banana plants ($\theta \leq d + r$).

Susceptible vector population has a constant recruitment rate of b_v, and it is assumed that both susceptible and contaminated vectors die naturally at the rate of μ_v. ω_3 is the probability that a susceptible vector gets contaminated with Xcm bacteria after coming into contact with an infected banana plant. It is assumed that the vector becomes infective right after been contaminated with the Xcm bacteria. According to Buregyeya *et al.* (2014), contaminated vectors retain Xcm bacteria viable for 3-5 days from the day of inoculation. This implies that, after 5 days contaminated vectors becomes susceptible again at the rate η. Furthermore, it is assumed that $\eta < \mu_v$.

Xcm bacteria released by dead banana plants due to infection, removed BXW diseased plant and other infected banana plant debris when not safely disposed and left in the farm result to Xcm soil inoculum at the rate of ϕ (Sivirihauma *et al.*, 2017). Sivirihauma *et al.* (2017) observed that Xcm soil inoculum has a role to play in the persistence of BXW disease in the field. Nakato *et al.* (2018) argued that, the survival of Xcm bacteria in the soil is highly affected by soil moisture. Also, Shimwela *et al.* (2016) revealed that there is positive correlation between rain and BXW disease transmission. This implies that, rain increase the soil moisture and hence fevers the survival of Xcm bacteria in the soil, Also through flow of rain, Xcm bacteria can be transported from one place to another. A susceptible banana plant can acquire Xcm bacteria from the contaminated soil through mechanical injuries caused by farming activities such as

weeding and organisms found in the soil such as nematodes and insects found in the lower parts of the plant such as roots (Mwebaze *et al.*, 2006; Hashim, 2013; Shehabu *et al.*, 2010; Sivirihauma *et al.*, 2017). The Xcm bacteria in the farm soil are cleared naturally at the rate μ_h due to lack of saprophytic or resting stage in soil (Mwebaze *et al.*, 2006).

The model is best described by the compartmental diagram in Fig. 4, where $\lambda_1 = a\omega_3 \frac{I_p}{N_p}$ and $\lambda_2 = (a\omega_1 \frac{I_v}{N_p} + \beta_e \frac{S_p E_p}{N_p} + \beta_i \frac{S_p I_p}{N_p} + \omega_2 \frac{A_h}{N_p(K+A_h)})$.

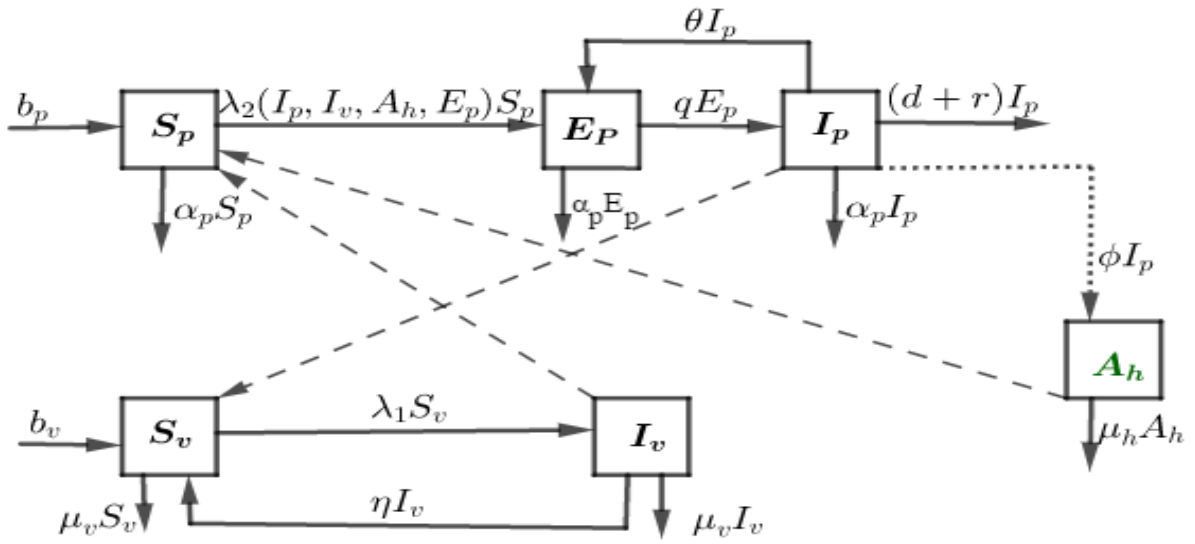


Figure 4: Basic compartmental diagram for the dynamics of BXW disease

From the compartmental diagram, sold lines represent a transition from one infection stage to another, recruitment, harvesting, natural death rate of vectors and clearance of Xcm bacteria from the soil. The dash lines represents normal interactions between different compartments and shedding of Xcm bacteria onto the environment is represented by dotted lines.

From the compartmental diagram we formulate a system of differential equations as follows:

$$\frac{dS_p}{dt} = b_p - a\omega_1 \frac{S_p I_v}{N_p} - \beta_e \frac{S_p E_p}{N_p} - \beta_i \frac{S_p I_p}{N_p} - \omega_2 \frac{S_p A_h}{N_p(K+A_h)} - \alpha_p S_p, \tag{4.1}$$

$$\frac{dE_p}{dt} = a\omega_1 \frac{S_p I_v}{N_p} + \beta_e \frac{S_p E_p}{N_p} + \beta_i \frac{S_p I_p}{N_p} + \omega_2 \frac{S_p A_h}{N_p(K+A_h)} + \theta I_p - \alpha_p E_p - qE_p, \tag{4.2}$$

$$\frac{dI_p}{dt} = qE_p - \alpha_p I_p - dI_p - rI_p, \tag{4.3}$$

$$\frac{dA_h}{dt} = \phi I_p - \mu_h A_h, \tag{4.4}$$

$$\frac{dS_v}{dt} = b_v + \eta I_v - a\omega_3 \frac{S_v I_p}{N_p} - \mu_v S_v, \tag{4.5}$$

$$\frac{dI_v}{dt} = a\omega_3 \frac{S_v I_p}{N_p} - \eta I_v - \mu_v I_v. \tag{4.6}$$

The equations of the total population of banana plants and total population of vectors are given by:

$$\frac{dN_p}{dt} = b_p - \alpha_p N_p + \theta I_p - (d+r) I_p, \tag{4.7}$$

$$\frac{dN_v}{dt} = b_v - \mu_v N_v. \tag{4.8}$$

Table 2: Variables' descriptions

Variable	Description
S_p	Susceptible banana plant
E_p	Latently infected banana plant
I_p	Infected banana plant
S_v	Susceptible vector
I_v	Vectors contaminated with Xcm bacteria
A_h	Concetration of Xcm bacteria in the soil

Table 3: Parameters' descriptions

Parameters	Description
b_p	Recruitment rate of susceptible suckers
b_v	Recruitment rate of susceptible vectors
α_p	Harvesting rate of Matured banana plants
θ	Rate of vertical transmission from an infected plant
r	Rate of removing infected banana plant from the farm
d	Disease induced death rate of an infected banana plant
β_e	Rate of infection by contaminated farming tools from asymptomatic infected banana plant
β_i	Rate of infection by contaminated farming tools from symptomatic infected banana plants
a	Contact rate of the vector with banana plant
ω_1	Probability that a contact results in transmission of Xcm bacteria from an infected vector to a susceptible banana plant
ω_2	Probability of transmission of Xcm bacteria from contaminated soil to a susceptible banana plant.
ω_3	Probability that a contact results in the transmission of Xcm bacteria from an infected banana plant to a susceptible vector
μ_v	Mortality rate of the vectors
η	Recovery rate of contaminated vectors
q	Rate of latently infected banana plant progress to infected state
ϕ	Shedding rate of Xcm bacteria from infected banana plant to the soil
μ_b	Natural clearance rate of bacteria in the Environment.
K	Half saturation constant of Xcm bacteria in the Environment

4.1.2 Invariant region and positivity of the solutions

In this section we are going to check whether the model system is epidemiologically and mathematically well posed. This is done by checking the invariant region of the model and positivity of the model solution to make sure that there is no negative solution to the model variables. From equation (4.7) we have

$$\frac{dN_p}{dt} \leq b_p - \alpha_p N_p, \tag{4.9}$$

$$N_p(t) \leq \frac{b_p}{\alpha_p} + \Big(N_p(0) - \frac{b_p}{\alpha_p}\Big)e^{-\alpha_p t}. \tag{4.10}$$

From equation (4.10), two cases are emerge. Case 1: When $N_p(0) \leq \frac{b_p}{\alpha_p}$, as $t \longrightarrow \infty$ the total number of banana plants $N_p(t)$ increases to $\frac{b_p}{\alpha_p}$. This implies that

$$N_p(0) \leq N_p(t) \leq \frac{b_p}{\alpha_p}, \forall_{t\geq 0} \tag{4.11}$$

Case 2: When $N_p(0) \geq \frac{b_p}{\alpha_p}$, $N_p(t)$ decreases to $\frac{b_p}{\alpha_p}$ as $t \longrightarrow \infty$. This implies that

$$N_p(t) \leq \frac{b_p}{\alpha_p} \leq N_p(0), \forall_{t\geq 0} \tag{4.12}$$

Generally, $D_1 = \{S_p(t), E_p(t), I_p(t) \in \mathbb{R}^3_+ : N_p(0) \leq N_p(t) \leq \frac{b_p}{\alpha_p}, \forall_{t\geq 0}\}$.
Again, from equation (4.8) we have

$$\frac{dN_v}{dt} \leq b_v - \mu_v N_v, \tag{4.13}$$

solving this, results into

$$N_v(t) \leq \frac{b_v}{\mu_p} + \Big(N_v(0) - \frac{b_v}{\mu_v}\Big)e^{-\mu_v t}. \tag{4.14}$$

From equation (4.14) it follows that, When $N_v(0) \geq \frac{b_v}{\mu_v}$, as $t \longrightarrow \infty$, The total number of vectors $N_v(t)$ reduces to $\frac{b_v}{\mu_v}$. This means that

$$N_v(t) \leq \frac{b_v}{\mu_v} \leq N_v(0), \forall_{t\geq 0} \tag{4.15}$$

Again, when $N_p(0) \leq \frac{b_p}{\alpha_p}$, as $t \longrightarrow \infty$ the number of vectors $N_p(t)$ approaches to $\frac{b_v}{\mu_v}$. This means that

$$N_v(0) \leq N_v(t) \leq \frac{b_v}{\mu_v}, \forall_{t\geq 0}. \tag{4.16}$$

Therefore,

$$D_2 = \{S_v(t), I_v(t) \in \mathbb{R}^2_+ : N_v(0) \leq N_v(t) \leq \frac{b_v}{\mu_v}, \forall_{t\geq 0}\}. \tag{4.17}$$

Furthermore, it is proved that

$$D_3 = \{A_h(t) \in \mathbb{R}^1_+, \forall_{t\geq 0}\}. \tag{4.18}$$

Considering the non-negative initial solutions of the model $S_p(0) > 0, E_p(0) \geq 0, I_p(0) \geq 0, A_h(0) \geq 0, S_v(0) \geq 0, I_v(0) \geq 0$, the model system (4.1-4.6) is positive invariant and attracting in the region

$$D = \{D_1 \times D_2 \times D_3 : D \in \mathbb{R}^6_+ \forall_{t\geq 0}\}. \tag{4.19}$$

Therefore, the model solutions remain positive and bounded in the region D, $\forall_{t\geq 0}$.

4.2 Basic model analysis

4.2.1 Disease free equilibrium points

Disease free equilibrium (DFE) is the point at which there is no infection in the population. Thus, the populations comprise of susceptible banana plants and susceptible vectors only. In order to determine the DFE points $X_{dfe} = (S^0_p, E^0_p, I^0_p, S^0_v, I^0_v, A^0_h)$ from the system (4.1-4.6) we set the rate of change of each model variable to zero. Thus,

$$0 = b_p - a\omega_1 \frac{S_p I_v}{N_p} - \beta_e \frac{S_p E_p}{N_p} - \beta_i \frac{S_p I_p}{N_p} - \omega_2 \frac{S_p A_h}{N_p(K + A_h)} - \alpha_p S_p, \tag{4.20}$$

$$0 = a\omega_1 \frac{S_p I_v}{N_p} + \beta_e \frac{S_p E_p}{N_p} + \beta_i \frac{S_p I_p}{N_p} + \omega_2 \frac{S_p A_h}{N_p(K + A_h)} + \theta I_p - \alpha_p E_p - qE_p, \tag{4.21}$$

$$0 = qE_p - \alpha_p I_p - dI_p - rI_p, \tag{4.22}$$

$$0 = \phi I_p - \mu_h A_h \tag{4.23}$$

$$0 = b_v + \eta I_v - a\omega_3 \frac{S_v I_p}{N_p} - \mu_v S_v, \tag{4.24}$$

$$0 = a\omega_3 \frac{S_v I_p}{N_p} - \eta I_v - \mu_v I_v. \tag{4.25}$$

Now, from equation (4.22) we have,

$$E^0_p = \frac{(\alpha_p + d + r) I_p}{q}, \tag{4.26}$$

considering equation (4.23)

$$A_h^0 = \frac{\phi I_p}{\mu_h}. \tag{4.27}$$

Again solving equation 4.25 for I_v^0 results to

$$I_v^0 = \frac{a\omega_3 S_v I_p I_p}{(\eta+\mu_v)N_p}. \tag{4.28}$$

Substituting equation (4.26, 4.27, 4.28) in the equation (4.21), results to

$$\begin{aligned} 0 = \Bigg(& \frac{a\omega_1 S_p a\omega_3 S_v}{(\eta+\mu_v)N_p N_p} + \beta_i \frac{S_p}{N_p} + \beta_e \frac{S_p(\alpha_p+d+r)}{qN_p} + \frac{S_p\omega_2\phi}{\mu_h N_p(K+A_h)} \\ & - (\alpha_p+q)\frac{(\alpha_p+d+r)}{q} + \theta \Bigg) I_p. \end{aligned} \tag{4.29}$$

This shows that either

$$I_p^0 = 0, \tag{4.30}$$

or

$$M = 0, \tag{4.31}$$

where

$$\begin{aligned} M = & \frac{a\omega_1 S_p a\omega_3 S_v}{(\eta+\mu_v)N_p N_p} + \beta_i \frac{S_p}{N_p} + \beta_e \frac{S_p(\alpha_p+d+r)}{qN_p} + \frac{S_p\omega_2\phi}{\mu_h N_p(K+A_h)} \\ & - (\alpha_p+q)\frac{(\alpha_p+d+r)}{q} + \theta. \end{aligned} \tag{4.32}$$

Substituting equation 4.30 into equation (4.26, 4.27, 4.28) results to

$$E_p^0 = A_h^0 = I_v^0 = 0. \tag{4.33}$$

Again substituting equation (4.30 and 4.33) into equation 4.20 yields

$$S_p^0 = \frac{b_p}{\alpha_p}. \tag{4.34}$$

Finaly substituting equation (4.30 and 4.33) into equation (4.23) yields

$$S_v^0 = \frac{b_v}{\mu_v}. \tag{4.35}$$

Therefore, the disease free equilibrium point X_{dfe} of the system of equations 4.1-4.6 is given by (4.36)

$$X_{dfe} = (S_p^0, E_p^0, I_p^0, S_v^0, I_v^0, A_h^0) = (\frac{b_p}{\alpha_p}, 0, 0, \frac{b_v}{\mu_v}, 0, 0). \tag{4.36}$$

4.2.2 Basic reproduction number (R_0)

Basic reproduction number (R_0), is an average number of new infection caused by one infective individual in a population where all its members are susceptible. According to Van den Driessche and Watmough (2002) R_0 helps to understand the ability of the disease to invade the population. In this study we apply the next generation method as described by Van den Driessche and Watmough (2002), Diekmann *et al.* (2009) and applied by Nakakawa *et al.* (2016). from the model system of equations (4.1-4.6), consider the infected subsysetem

$$\begin{aligned}\frac{dE_p}{dt} &= a\omega_1\frac{S_pI_v}{N_p} + \beta_e\frac{S_pE_p}{N_p} + \beta_i\frac{S_pI_p}{N_p} + \omega_2\frac{S_pA_h}{N_p(K+A_h)} + \theta I_p - \alpha_pE_p \\ &\quad - qE_p, \qquad (4.37)\\ \frac{dI_v}{dt} &= a\omega_3\frac{S_vI_p}{N_p} - \eta I_v - \mu_vI_v, \qquad (4.38)\\ \frac{dI_p}{dt} &= qE_p - \alpha_pI_p - dI_p - rI_p, \qquad (4.39)\\ \frac{dA_h}{dt} &= \phi I_p - \mu_hA_h. \qquad (4.40)\end{aligned}$$

Let $x = (E_p, I_v, I_p, A_h)$ and $y = (S_p, S_v)$, where x and y are infected and susceptible compartments of the model, respectively. Separating the infected subsystem (4.37-4.40) into two parts, results into (4.41) and (4.42), Where $\mathscr{F}(x,y)$ is the transmission part which portray the generation of new infections and $\mathscr{V}$(x,y) is the transition part which involves change of states.

$$\mathscr{F}(x,y) = \begin{pmatrix} a\omega_1\frac{S_pI_v}{N_p} + \beta_e\frac{S_pE_p}{N_p} + \beta_i\frac{S_pI_p}{N_p} + \omega_2\frac{S_pA_h}{N_p(K+A_h)} + \theta I_p \\ a\omega_3\frac{S_vI_p}{N_p} \\ 0 \\ 0 \end{pmatrix}, \qquad (4.41)$$

$$\mathscr{V}(x,y) = \begin{pmatrix} -\alpha_pE_p - qE_p \\ -\eta I_p - \mu_vI_v \\ qE_p - \alpha_pI_p - dI_p - rI_p \\ \phi I_p - \mu_hA_h \end{pmatrix}. \qquad (4.42)$$

Let $F = \frac{\partial\mathscr{F}(x,y)}{\partial x_i}$ and $V = \frac{\partial\mathscr{V}(x,y)}{\partial x_i}$ where $x_i = (E_p, I_v, I_p, A_h)$ for $i = 1,2,3,4$. At the DFE every member of the population is susceptible, thus $S_p^0 = N_p(0)$. Differentiating and evaluating at

X_{dfe} results into (4.43) and (4.44),

$$F = \begin{pmatrix} \beta_e & a\omega_1 & \beta_i + \theta & \frac{\omega_2}{K} \\ 0 & 0 & \frac{a\omega_3 \alpha_p b_v}{\mu_v b_p} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}, \tag{4.43}$$

$$V = \begin{pmatrix} (\alpha_p + q) & 0 & 0 & 0 \\ 0 & (\eta + \mu_v) & 0 & 0 \\ -q & 0 & (\alpha_p + d + r) & 0 \\ 0 & 0 & -\phi & \mu_h \end{pmatrix}, \tag{4.44}$$

$$V^{-1} = \begin{pmatrix} \frac{1}{(\alpha_p+q)} & 0 & 0 & 0 \\ 0 & \frac{1}{(\eta+\mu_v)} & 0 & 0 \\ \frac{q}{(\alpha_p+q)(\alpha_p+d+r)} & 0 & \frac{1}{(\alpha_p+d+r)} & 0 \\ \frac{q\phi}{(\alpha_p+q)(\alpha_p+d+r)\mu_h} & 0 & \frac{\phi}{(\alpha_p+d+r)\mu_h} & \frac{1}{\mu_h} \end{pmatrix}. \tag{4.45}$$

Then it follows that

$$FV^{-1} = \begin{bmatrix} \frac{\beta_e}{\alpha_p+q} + \frac{(\beta_i+\theta)q}{(\alpha_p+q)(\alpha_p+d+r)} + \frac{\omega_2\phi\, q}{k(\alpha_p+d+r)(\alpha_p+q)\mu_h} & \frac{a\omega_1}{\eta+\mu_v} & \frac{\beta_i+\theta}{\alpha_p+d+r} + \frac{\omega_2\phi}{k(\alpha_p+d+r)\mu_h} & \frac{\omega_2}{k\mu_h} \\ \frac{a\omega_3\alpha_p b_v q}{\mu_v b_p(\alpha_p+q)(\alpha_p+d+r)} & 0 & \frac{a\omega_3\alpha_p b_v}{\mu_v b_p(\alpha_p+d+r)} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}. \tag{4.46}$$

Now, let the matrix $Q = FV^{-1}$, the basic reproduction number R_0 of the model is a dominant eigenvalue of the matrix Q. Therefore,

$$R_0 = \frac{1}{2}T_R + \frac{1}{2}\sqrt{(T_R)^2 + 4\,\frac{a^2\omega_1\omega_3\alpha\, b_v q}{(\eta+\mu_v)\,\mu_v b_p\,(\alpha+q)\,(\alpha+d+r)}}. \tag{4.47}$$

Where,

$$T_R = \frac{\beta_e}{\alpha_p+q} + \frac{(\beta_i+\theta)\,q}{(\alpha_p+q)\,(\alpha_p+d+r)} + \frac{\omega_2\phi\, q}{k\,(\alpha_p+d+r)\,(\alpha_p+q)\,\mu_h}. \tag{4.48}$$

From (4.47), $\frac{1}{\alpha_p+q}$ is the average time that a banana plant stays in an asymptomatic infected stage before proceeding to the symptomatic infected stage. In this duration β_e new infection are generated. $\frac{q}{\alpha_p+q}$ is the probability that an asymptomatic infected banana plant proceeds to

an infected compartment. $\frac{1}{\alpha_p+d+r}$ is the duration by which an infected banana plant stays in an infected group during its lifetime. $\beta_i+\theta$ is the expected number of new infections to the banana plant produced by a symptomatic infected banana plant before being harvested, removed from the farm or dying due to BXW disease infection. $\frac{\omega_2\phi}{k\mu_h}$ are the expected new infections caused by the contaminated soil.

4.2.3 Stability analysis of the disease free equilibrium (DFE) point

(i) **Local stability the disease free equilibrium (DFE)**

The disease free equilibrium point (DFE) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$. The stability of the DFE can be determined by checking the nature of the real parts of all the eigenvalues of the matrix $F-V$. With reference to Theorem 2 of Van den Driessche and Watmough (2002, p.33) we determine if DFE of the model is locally asymptotically stable. Since F is the non-negative matrix and V is a non-singular M-matrix, let

$$J = F - V, \tag{4.49}$$

then (4.50) has the Z pattern because all its off diagonal elements are less or equal to 0

$$-J = V - F. \tag{4.50}$$

In that case, $s(J) < 0 \Longleftrightarrow -J$ is a non-singular M-matrix. A matrix such that $T \in \mathbb{R}^{n\times n}$ is said to be Non-singular M-matrix if and only if it can be written in the form $T = sI - G$ where $G = (g_{ij})$ with $(g_{ij}) \geq 0$, $1 \leq i, j \leq n$ and $s > \rho(G)$. Thus, in order to find out if all the real parts of all eigenvalues of $-J$ are less than 0, we need to show that $-J$ is a non-singular M-matrix. Now, from (4.50), Multiplying by V^{-1} both sides results to (4.51).

$$-JV^{-1} = VV^{-1} - FV^{-1}, \tag{4.51}$$

Since $VV^{-1} = I$ where I is an identity matrix, substituting results to (4.52)

$$-JV^{-1} = I - FV^{-1}. \tag{4.52}$$

Applying lemma 5 of Van den Driessche and Watmough (2002), with $H = V$ and $B = -J$, then $-J$ is a non-singular M-matrix if and only if $I - FV^{-1}$ is a non-singular M-matrix in (4.52). Now, from (4.52), $s = 1$, which implies that $-J$ is a non-singular M-matrix if

and only if $1 > \rho(FV^{-1})$. But due to the fact in (4.53), $-J$ is a non-singular M-matrix if $R_0 < 1$.

$$\rho(FV^{-1}) = R_0, \tag{4.53}$$

Therefore, the spectral abscissa of J, $s(J) < 0 \Longleftrightarrow R_0 < 1$ This implies that the DFE point X_{dfe} of the model system (4.1-4.6) is locally asymptotically stable if $R_0 < 1$, but unstable if $R_0 > 1$.

(ii) **Global stability the disease free equilibrium (DFE)**

Global stability of the disease free equilibrium point means that the solutions of the system are attracted to the DFE point over indefinite time.

Theorem 4.1

If X_{dfe} is a DFE of the model given by (4.1–4.6), then X_{dfe} is globally asymptotically stable if $R_0 < 1$, and unstable if $R_0 > 1$.

Proof. Lyapunov function constructed using matrix-theoretic method based on the Perron eigenvector is applied to prove the Global Stability of the DFE X_{dfe} as done in Lazarus (2018) and Shuai and van den Driessche (2013).

Now, let $x = (E_p, I_v, I_p, A_h)$ and $y = (S_p, S_v)$. From a subsystem (4.37– 4.40), the function $f(x,y)$ and x' can be written as in (4.54) and (4.55) respectively.

$$f(x,y) = (F - V)x - \mathscr{F}(x,y) + \mathscr{V}(x,y), \tag{4.54}$$

and

$$x' = (F - V)x - f(x,y), \tag{4.55}$$

Solving for $f(x,y)$ results to (4.56)

$$f(x,y) = \begin{pmatrix} \frac{\beta_e(S_p^0 - S_p)E_p}{N_p} + \frac{a\omega_1(S_p^0 - S_p)I_p}{N_p} + \frac{\beta_i(S_p^0 - S_p)I_p}{N_p} + \frac{\omega_2(S_p^0 - S_p)A_h}{N_p} \\ \frac{a\omega_3(S_p^0 - S_p)I_p}{\mu_v N_p} \\ 0 \\ 0 \end{pmatrix}. \tag{4.56}$$

Referring to Theorem 2.1 of Shuai and van den Driessche (2013). Since from (4.43) $F \geq 0$, in (4.45) $V^{-1} \geq 0$ and from (4.56) $f(x,y) \geq 0$ then (4.57) is a Lyapunov function of the model (4.37 - 4.40) where ϑ^T is the left eigenvector of the matrix $V^{-1}F$ corresponding to its spectral radius R_0.

$$Q = \vartheta^T V^{-1} x. \tag{4.57}$$

Reducing the matrix $V^{-1}F$ to its row echelon form, the left eigenvector is $\vartheta^T = (1,0,0,0)$.

$$Q = \frac{E_p}{\alpha_p + q}. \tag{4.58}$$

Since $\vartheta^T > 0$ and the matrix $V^{-1}F$ is irreducible and non negative, then Theorem 2.2 of Shuai and van den Driessche (2013) can be applied. Differentiating (4.57) results to (4.59).

$$Q' = \vartheta^T V^{-1} x'. \tag{4.59}$$

Substituting (4.55) into (4.59) gives (4.60)

$$Q' = \vartheta^T V^{-1}\big((F-V)x - f(x,y)\big), \tag{4.60}$$

$$= \vartheta^T V^{-1}(F-V)x - \vartheta^T V^{-1} f(x,y), \tag{4.61}$$

$$= (R_0 - 1)\vartheta^T x - \vartheta^T V^{-1} f(x,y). \tag{4.62}$$

Substituting the required equations in (4.62) yields (4.63).

$$Q' = (R_0-1)E_p - \frac{1}{\alpha_p + q}\Big(\frac{\beta_e(S_p^0 - S_p)E_p}{N_p} + \frac{a\omega_1(S_p^0 - S_p)I_p}{N_p} + \frac{\beta_i(S_p^0 - S_p)I_p}{N_p} + \frac{\omega_2(S_p^0 - S_p)A_h}{N_p}\Big). \tag{4.63}$$

From (4.63) it can be observed that $Q' \leq 0$ if $R_0 \leq 1$. But if $R_0 = 1$, $Q' = 0 \Longleftrightarrow S_p^0 = S_p$ or $E_p = I_p = I_v = A_h = 0$. Thus, by Theorem 2.2 of Shuai and van den Driessche (2013) , X_{dfe} is Global Asymptotically Stable in D when $R_0 \leq 1$ and unstable when $R_0 > 1$.

4.2.4 Sensitivity analysis

Sensitivity analysis is the process of determining the influence of each model parameter in the basic reproduction number (R_0). This guides the selection of the disease control measures, where the most sensitive parameters are highly considered. We applied the Normalized forward sensitivity index to determine the sensitivity of the model parameters as in Chitnis *et al.* (2006). If the R_0 is differentiable with respect to its parameter u, then the sensitivity index of u is given by (4.64).

$$\Upsilon_u^{R_0} = \frac{\partial R_0}{\partial u} \times \frac{u}{R_0}. \tag{4.64}$$

Given,

$$R_0 = \frac{1}{2}T_R + \frac{1}{2}\sqrt{(T_R)^2 + 4\frac{a^2\omega_1\omega_3\alpha b_v q}{(\eta+\mu_v)\mu_v b_p(\alpha+q)(\alpha+d+r)}}. \tag{4.65}$$

Where,

$$TR = \frac{\beta_e}{\alpha_p + q} + \frac{(\beta_i + \theta)\, q}{(\alpha_p + q)(\alpha_p + d + r)} + \frac{\omega_2 \phi\, q}{k(\alpha_p + d + r)(\alpha_p + q)\mu_h}. \tag{4.66}$$

Therefore,

$$\frac{\partial R_0}{\partial \beta_i} = \frac{1}{2}\left(\frac{q}{(\alpha+q)(\alpha+d+r)} + \frac{TR\left(\frac{q}{(\alpha+q)(\alpha+d+r)}\right)}{\sqrt{(TR)^2 + 4\frac{a^2\omega_1\omega_3\alpha b_v q}{(\eta+\mu_v)\mu_v b_p(\alpha+q)(\alpha+d+r)}}} \right). \tag{4.67}$$

Putting (4.67) in (4.64) and replacing u with the parameter β_i gives

$$\Upsilon_{\beta_i}^{R_0} = \frac{1}{2}\left(\frac{q}{(\alpha+q)(\alpha+d+r)} + \frac{TR\left(\frac{q}{(\alpha+q)(\alpha+d+r)}\right)}{\sqrt{(TR)^2 + 4\frac{a^2\omega_1\omega_3\alpha b_v q}{(\eta+\mu_v)\mu_v b_p(\alpha+q)(\alpha+d+r)}}} \right) \times \frac{\beta_i}{R_0}. \tag{4.68}$$

Substituting the value of parameters in (4.68) results to sensitivity index in (4.69).

$$\Upsilon_{\beta_i}^{R_0} = 0.6024153. \tag{4.69}$$

Since the R_0 in (4.47) is differentiable to all its parameters, now we apply (4.64) to calculate the sensitivity indices of the model parameter using the values in table (1). This results to sensitivity indices as indicated in Fig. 5.

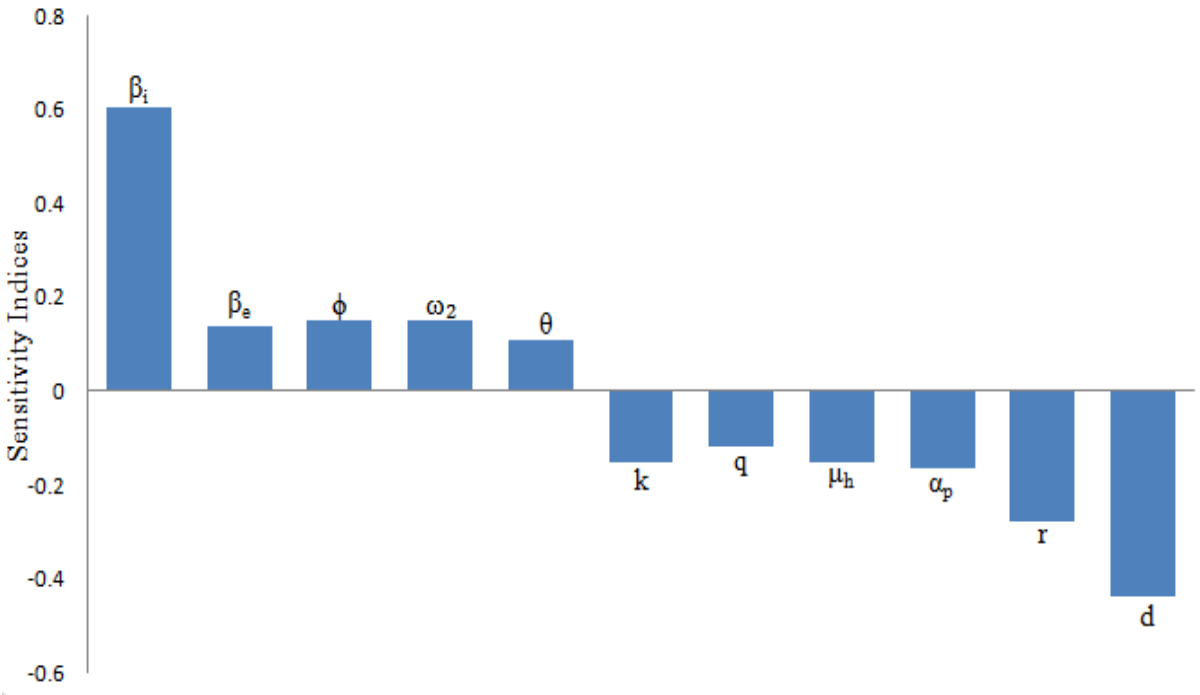


Figure 5: Sensitivity indices for the basic model parameters

Positive indices implies the direct proportionality of the basic reproduction number with the corresponding parameter. A negative index means that the parameter is inversely proportional to the basic reproduction number. Increasing the R_0 implies increase in the BXW disease endemicity while decreasing R_0 to less than one lowers the endemicity of the BXW disease. From Fig. 5, the parameters ω_2, ϕ, θ and β_T have positive indices, which sends the message that increasing (or decreasing) any of these parameters keeping other parameters constant, results into the increase (or decrease) of the basic reproduction number (R_0). For instance, $\beta_i = 0.6024153$ means that increasing (or decreasing) the value of the parameter β_i by 10% increases(or decreases) the R_0 by 6.024153% . Thus, decreasing the rate of infection by farming tools, vertical transmission, rate of shedding Xcm bacteria in the soil and reducing the rate of infection through contaminated soil reduces the value of the R_0 and hence helps to contain the disease. Conversely, the parameters with negative indices are r, d, α_p, q, K and μ_h which means that, increasing (or decreasing) any of these parameters results to decrease (or increase) of the R_0. In order to best control the disease these parameters with negative indices should be increased so as to reduce the value of the R_0.

According to Ocimati *et al.* (2013*a*) Xcm bacterium is systemic in nature, it can invade the whole plant from the point of infection to its lateral shoots if the diseased plant is not properly removed on time. Now, leaving the diseased plant to die in the farm gives a chance of the Xcm bacteria to spread wider and hence spread of the disease. Therefore, the parameter d should be carefully considered during the selection of control measures. Other parameters whose indices are more close to zero are considered to be less sensitive to the R_0, hence they can be tolerated.

4.2.5 Existence and stability of endemic equilibrium point

Endemic equilibrium point is the point where the infected compartments of the model are non-zero. In this study the existence and Global stability of the endemic equilibrium point of the model (4.1- 4.6) is discussed. To check for existence of the endemic equilibrium point, Let $\lambda_p = \frac{a\omega_1 I_v}{N_p} + \beta_e \frac{S_p E_p}{N_p} + \beta_i \frac{S_p I_p}{N_p} + \frac{\omega_2 A_h}{N_p(K+A_h)}$ be the general force of infection at which Susceptible Banana plant gets infected by contaminated vector, contaminated farming tools from symptomatic and asymptomatic infected banana plants also contaminated soil and $\lambda_v = \frac{a\omega_3 I_p}{N_p}$ be the rate at which Susceptible vector becomes infected by an infected banana plant. Substituting these forces of

infections in the model system (4.1-4.6) and setting it to zero we obtain the following system.

$$0 = b_p - \lambda_p S_p - \alpha_p S_p, \tag{4.70}$$

$$0 = \lambda_p S_p + \theta I_p - (\alpha_p + q) E_p, \tag{4.71}$$

$$0 = q E_p - (\alpha_p + d + r) I_p, \tag{4.72}$$

$$0 = \phi I_p - \mu_h A_h, \tag{4.73}$$

$$0 = b_v + \eta I_v - \lambda_v S_v - \mu_v S_v, \tag{4.74}$$

$$0 = \lambda_v S_v - (\eta + \mu_v) I_v. \tag{4.75}$$

Solving the system (4.70-4.75) for endemic equilibrium point, results to the point $X_{ee} = (S_p^*, E_p^*, I_p^*, S_v^*, I_v^*, A_h^*)$ where,

$$S_p^* = \frac{b_p}{\alpha_p + \lambda_p}, \tag{4.76}$$

$$E_p^* = \frac{(\alpha_p + d + r)\, b_p \lambda_p}{(\alpha_p + \lambda_p)\,(dq + d\alpha_p + qr - q\theta + q\alpha_p + r\alpha_p + {\alpha_p}^2)}, \tag{4.77}$$

$$I_p^* = \frac{q b_p \lambda_p}{(\alpha_p + \lambda_p)\,(dq + d\alpha_p + qr - q\theta + q\alpha_p + r\alpha_p + {\alpha_p}^2)}, \tag{4.78}$$

$$A_h^* = \frac{\phi\, q b_p \lambda_p}{(dq + d\alpha_p + qr - q\theta + q\alpha_p + r\alpha_p + {\alpha_p}^2)\,(\alpha_p + \lambda_p)\,\mu_h}, \tag{4.79}$$

$$S_v^* = \frac{(\eta + \mu_v)\, b_v}{\mu_v\,(\eta + \lambda_v + \mu_v)}, \tag{4.80}$$

$$I_v^* = \frac{b_v \lambda_v}{\mu_v\,(\eta + \lambda_v + \mu_v)}. \tag{4.81}$$

In this study we determine the global stability of the endemic equilibrium point (Xee) using lyapunov function as described by Korobeinikov (2004). The Lyapunov function is constructed using the formula in

$$V = \sum c_j (X_i - X_i^* \ln X_i). \tag{4.82}$$

Where c_j are carefully selected constants and X_i^* is the endemic equilibrium point. Now, in this study the lyapunov function is given by (4.83)

$$\begin{aligned} V = {} & c_1 (S_p - S_p^* \ln S_p) + c_2 (E_p - E_p^* \ln E_p) + c_3 (I_p - I_p^* \ln I_p) + c_4 (A_h - A_h^* \ln A_h) \\ & + c_5 (S_v - S_v^* \ln S_v) + c_6 (I_v - I_v^* \ln I_v). \end{aligned} \tag{4.83}$$

Differentiating (4.83) with respect to time, gives

$$\frac{dV}{dt} = c_1(1-\frac{S_p^*}{S_p})\frac{dS_p}{dt} + c_2(E_p-\frac{E_p^*}{E_p})\frac{dE_p}{dt} + c_3(I_p-\frac{I_p^*}{I_p})\frac{dI_p}{dt} + c_4(A_h-\frac{A_h^*}{A_h})\frac{dA_h}{dt} + c_5(S_v-\frac{S_v^*}{S_v})\frac{dS_v}{dt} + c_6(I_v-\frac{I_v^*}{I_v})\frac{dI_v}{dt}. \tag{4.84}$$

Substituting values from the system (4.1-4.6) in (4.84) results to,

$$\begin{aligned}\frac{dV}{dt} &= c_1(1-\frac{S_p^*}{S_p})[b_p - a\omega_1\frac{S_pI_v}{N_p} - \beta_e\frac{S_pE_p}{N_p} - \beta_i\frac{S_pI_p}{N_p} - \omega_2\frac{S_pA_h}{N_p(K+A_h)} - \alpha_pS_p] \\ &+ c_2(E_p-\frac{E_p^*}{E_p})[a\omega_1\frac{S_pI_v}{N_p} + \beta_e\frac{S_pE_p}{N_p} + \beta_i\frac{S_pI_p}{N_p} + \omega_2\frac{S_pA_h}{N_p(K+A_h)} + \theta I_p - \alpha_pE_p - qE_p] \\ &+ c_3(I_p-\frac{I_p^*}{I_p})[qE_p - \alpha_pI_p - dI_p - rI_p] + c_4(A_h-\frac{A_h^*}{A_h})[\phi I_p - \mu_hA_h] \\ &+ c_5(S_v-\frac{S_v^*}{S_v})[b_v + \eta I_v - a\omega_3\frac{S_vI_p}{N_p} - \mu_vS_v] + a_6(I_v-\frac{I_v^*}{I_v})[a\omega_3\frac{S_vI_p}{N_p} - \eta I_v - \mu_vI_v]. \end{aligned}\tag{4.85}$$

At endemic equilibrium point

$$\begin{aligned}\frac{dV}{dt} &= c_1(1-\frac{S_p^*}{S_p})[a\omega_1\frac{S_p^*I_v^*}{N_p} + \beta_e\frac{S_p^*E_p^*}{N_p} + \beta_i\frac{S_p^*I_p^*}{N_p} + \omega_2\frac{S_p^*A_h^*}{N_p(K+A_h)} + \alpha_pS_p^* - a\omega_1\frac{S_pI_v}{N_p} \\ &- \beta_e\frac{S_pE_p}{N_p} - \beta_i\frac{S_pI_p}{N_p} - \omega_2\frac{S_pA_h}{N_p(K+A_h)} - \alpha_pS_p] + c_2(1-\frac{E_p^*}{E_p})[(\alpha_p+q)E_p^* - (\alpha_p+q)E_p] \\ &+ c_3(1-\frac{I_p^*}{I_p})[(\alpha_p+d+r)I_p^* - (\alpha_p+d+r)I_p] + c_4(1-\frac{A_h^*}{A_h})[\mu_hA_h^* - \mu_hA_h] \\ &+ c_5(1-\frac{S_v^*}{S_v})[(a\omega_3\frac{I_p}{N_p} + \mu_v)S_v^* - (a\omega_3\frac{I_p}{N_p} + \mu_v)S_v] \\ &+ c_6(1-\frac{I_v^*}{I_v})[(\eta+\mu_v)I_v^* - (\eta+\mu_v)I_v^*]. \end{aligned}\tag{4.86}$$

Further simplification

$$\begin{aligned}\frac{dV}{dt} &= c_1\Big(\frac{S_p-S_p^*}{S_p}\Big)\Big[\frac{a\omega_1}{N_p}(S_p^*I_v^* - S_pI_v) + \frac{\beta_i}{N_p}(S_p^*I_p^* - S_pI_p) + \frac{\beta_e}{N_p}(S_p^*E_p^* - S_pE_p) \\ &+ \frac{\omega_2}{N_p(K+A_h)}(S_p^*A_h^* - S_pA_h) + \alpha_pS_p^* + \alpha_p(S_p^*-S_p)\Big] + c_2\Big(\frac{E_p-E_p^*}{E_p}\Big)\Big[(\alpha_p+q)(E_p^*-E_p)\Big] \\ &+ c_3\Big(\frac{I_p-I_p^*}{I_p}\Big)\Big[(\alpha_p+d+r)(I_p^*-I_p)\Big] + c_4\Big(\frac{A_h-A_h^*}{A_h}\Big)\Big[\mu_h(A_h^*-A_h)\Big] \\ &+ c_5\Big(\frac{S_v-S_v^*}{S_v}\Big)\Big[\frac{a\omega_3}{N_p}(I_p^*-I_p) - \mu_v(S_v^*-S_v)\Big] + c_6\Big(\frac{I_v-I_v^*}{I_v}\Big)\Big[(\eta+\mu_v)(I_v^*-I_v)\Big]. \end{aligned}\tag{4.87}$$

Collecting the like terms

$$\begin{aligned}\frac{dV}{dt} &= -c_1\Big(\frac{S_p-S_p^*}{S_p}\Big)\Big[\alpha_p(S_p^*-S_p)\Big] - c_2\Big(\frac{E_p-E_p^*}{E_p}\Big)\Big[(\alpha_p+q)(E_p^*-E_p)\Big] \\ &\quad - c_3\Big(\frac{I_p-I_p^*}{I_p}\Big)\Big[(\alpha_p+d+r)(I_p^*-I_p)\Big] - c_4\Big(\frac{A_h-A_h^*}{A_h}\Big)\Big[\mu_h(A_h^*-A_h)\Big] \\ &\quad - c_5\Big(\frac{S_v-S_v^*}{S_v}\Big)\Big[\mu_v(S_v^*-S_v)\Big] - c_6\Big(\frac{I_v-I_v^*}{I_v}\Big)\Big[(\eta+\mu_v)(I_v^*-I_v)\Big] \\ &\quad - c_1\Big(\frac{S_p-S_p^*}{S_p}\Big)\Big[\frac{a\omega_1}{N_p}(S_p^*I_v^*-S_pI_v) + \frac{\beta_i}{N_p}(S_p^*I_p^*-S_pI_p) + \frac{\beta_e}{N_p}(S_p^*E_p^*-S_pE_p) \\ &\quad + \frac{\omega_2}{N_p(K+A_h)}(S_p^*A_h^*-S_pA_h) + \alpha_pS_p^* - c_5\Big(\frac{S_v-S_v^*}{S_v}\Big)\Big[\frac{a\omega_3}{N_p}(I_p^*-I_p)\Big]. \end{aligned} \tag{4.88}$$

Finally

$$\begin{aligned}\frac{dV}{dt} &= -c_1\Big(\frac{(S_p-S_p^*)^2}{S_p}\Big)\alpha_p - c_2\Big(\frac{(E_p-E_p^*)^2}{E_p}\Big)(\alpha_p+q) - c_3\Big(\frac{(I_p-I_p^*)^2}{I_p}\Big)(\alpha_p+d+r) \\ &\quad - c_4\Big(\frac{(A_h-A_h^*)^2}{A_h}\Big)\mu_h - c_5\Big(\frac{(S_v-S_v^*)^2}{S_v}\Big)\mu_v - c_6\Big(\frac{(I_v-I_v^*)^2}{I_v}\Big)(\eta+\mu_v) + F(D). \end{aligned} \tag{4.89}$$

Where

$$D = (S_p, E_p, I_p, A_h, S_v, I_v) > 0.$$

From (4.89), $F(D) \leq 0$ for all elements in D and this implies that $\frac{dV}{dt} \leq 0$ in D. It can be seen that $\frac{dV}{dt} = 0$ in D only when $D = D^*$, implying that the largest invariant set in D when $\frac{dV}{dt} = 0$ is singleton D which is the endemic equilibrium point. Therefore, by LaSalle's invariance principle described in LaSalle (1976), it means that endemic equilibrium point D is asymptotically stable in D when $R_0 > 1$ and unstable otherwise.

4.3 Model with control measures

In this section, the basic model is modified to include the control measures. The existing mathematical models (Nannyonga *et al.*, 2015; Nakakawa *et al.*, 2016, 2017; Horub and Julius, 2017; Kweyunga *et al.*, 2018) have examined the effectiveness of the control measures such as adopting the following: timely removal of the male bud, cleaning of the farming tools, and cutting or uprooting the whole mat from where the diseased plant arose. This study examined the impact of applying the following control measures: Single Diseased Stem Removal (SDSR) approach; Control of a vertical disease transmission; Clearance of Xcm bacteria in the soil to reduce/remove soil inoculum and hence eliminate infections emanating from contaminated soil; and Community participatory education programmes.

According to Ocimati *et al.* (2013*b*), cutting the infected banana plant at the ground level is one of the means used to control further transmission of the BXW disease from infected mother plant to its suckers. This approach is effective only when the infected banana plant in question is only inflorescence infected. The implementation of this control measure is possible since infected banana plant shows symptoms before Xcm bacteria get to its lower parts. This study suggested and examined the impact of finding a way/technology to control vertical infections even if the infections starts from the lower parts of the banana plant. The introduction of the technology for clearing the Xcm bacteria in the soil aimed to speed up the rate of clearance of Xcm bacteria in the soil to reduce soil inoculum. This can be done by fumigating the whole farm soil with anti-Xcm herbicides in such a way that it kills all the Xcm bacteria in the soil without disturbing the ecosystem. Single diseased stem removal (SDSR) control strategy involves timely identifying and cutting pseudo stem of the infected banana plants at the ground level. This approach can be effectively applied when farmers are aware of the early symptoms of an infected banana plants. In community participatory farming education programmes, all the farmers are practically involved in the farming training programmes. This helps farmers to understand the transmission dynamics of the BXW disease and correctly implement the suggested control measures of the disease.

4.3.1 Formulation of the model to include the control measures

In this section the basic model in chapter 4, section 4.1 is modified to include the control measures. The control parameters included in the basic model includes: Participatory community education programmes (ξ); Clearance of Xcm bacteria in the soil (ψ); Vertical transmission control (δ); and Single diseased stem removal (r) whose values ranges from 0 to 1. For instance ξ is an education parameter which ranges from 0 to 1, where 1 represents effective community participation in farming education programmes which ensures no new BXW disease transmission and 0 represent poor community participation in farming education programmes which lead lack of disease information to the farmers and hence high BXW disease transmission. $(1-\delta)$ is a control parameter to control vertical infection from mother plant to its suckers. ψ is an artificial clearance rate of Xcm bacteria in the soil and r represent timely identification and removal of the diseased plant only and not the whole mat.

Figure 6 is a schematic diagram that best describes model for the dynamics of BXW disease with control measure.

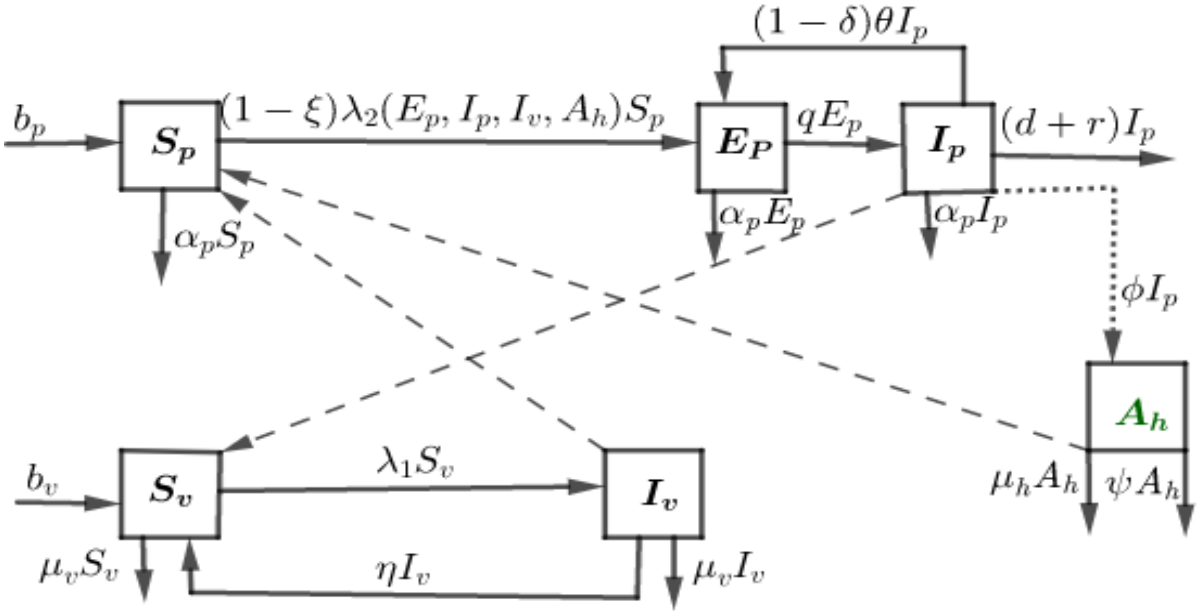


Figure 6: Compartmental diagram for the dynamics of BXW disease with control.

In the compartmental diagram, solid lines represent a transition from one infection stage to another, recruitment, harvesting, natural death rate of vectors and clearance of Xcm bacteria from the soil. The dashed lines represents normal interactions between different compartments while shedding of Xcm bacteria into the environment is represented by dotted lines. From the compartmental diagram we formulate a system of differential equations as follows:

$$\frac{dS_p}{dt} = b_p - (1-\xi)\Big(a\omega_1\frac{S_pI_v}{N_p} - \beta_e\frac{S_pE_p}{N_p} - \beta_i\frac{S_pI_p}{N_p} - \omega_2\frac{S_pA_h}{N_p(K+A_h)}\Big) - \alpha_pS_p, \tag{4.90}$$

$$\frac{dE_p}{dt} = (1-\xi)\Big(a\omega_1\frac{S_pI_v}{N_p} + \beta_e\frac{S_pE_p}{N_p} + \beta_i\frac{S_pI_p}{N_p} + \omega_2\frac{S_pA_h}{N_p(K+A_h)}\Big) + (1-\delta)\theta I_p - \alpha_pE_p - qE_p, \tag{4.91}$$

$$\frac{dI_p}{dt} = qE_p - \alpha_pI_p - dI_p - rI_p, \tag{4.92}$$

$$\frac{dA_h}{dt} = \phi I_p - \mu_hA_h - \psi A_h, \tag{4.93}$$

$$\frac{dS_v}{dt} = b_v + \eta I_v - a\omega_3\frac{S_vI_p}{N_p} - \mu_vS_v, \tag{4.94}$$

$$\frac{dI_v}{dt} = a\omega_3\frac{S_vI_p}{N_p} - \eta I_v - \mu_vI_v. \tag{4.95}$$

The equations of the total population of banana plants and total population of vectors are given by:

$$\frac{dN_p}{dt} = b_p - \alpha_pN_p + (1-\delta)\theta I_p - (d+r)I_p, \tag{4.96}$$

$$\frac{dN_v}{dt} = b_v - \mu_v N_v. \tag{4.97}$$

4.3.2 Basic properties of the model

Taking into consideration the non-negative initial solutions of the model $S_p(0) > 0, E_p(0) \geq 0, I_p(0) \geq 0, A_h(0) \geq 0, S_v(0) \geq 0, I_v(0) \geq 0$, the solutions of the modified model system (4.1-4.6) remain positive invariant and attracting in the region

$$D = \{D_1 \times D_2 \times D_3 : D \in \mathbb{R}^6_+, \forall_{t \geq 0}\}. \tag{4.98}$$

Proof: The proof is similar to the one section 4.1.2 of this study.

4.3.3 Disease free equilibrium point

The disease free equilibrium point (X_0) of the system of equations (4.90-4.95) when there is no BXW disease in the field is given by (4.99):

$$X_0 = (S_p^0, E_p^0, I_p^0, S_v^0, I_v^0, A_h^0) = (\frac{b_p}{\alpha_p}, 0, 0, \frac{b_v}{\mu_v}, 0, 0). \tag{4.99}$$

4.3.4 Effective reproduction number (R_e)

Effective reproduction number (R_e), is used to assess the effect of control measures. The control measures are effective if on their adoption $(R_e < 1)$ 0 and ineffective if $(R_e > 1)$. It helps to understand the ability of the disease to spread over the whole population when control measures are applied. This study applied the next generation method to compute the effective reproduction number (R_e) as described by Van den Driessche and Watmough (2002) and Diekmann *et al.* (2009) and applied by Nakakawa *et al.* (2016). From the model system of equations (4.1-4.6), consider the infected subsystem

$$\frac{dE_p}{dt} = (1-\xi)\Big(a\omega_1 \frac{S_p I_v}{N_p} + \beta_e \frac{S_p E_p}{N_p} + \beta_i \frac{S_p I_p}{N_p} + \omega_2 \frac{S_p A_h}{N_p(K + A_h)}\Big) + (1-\delta)\theta I_p - \alpha_p E_p - qE_p, \tag{4.100}$$

$$\frac{dI_p}{dt} = qE_p - \alpha_p I_p - dI_p - rI_p, \tag{4.101}$$

$$\frac{dA_h}{dt} = \phi I_p - \mu_h A_h - \psi A_h, \tag{4.102}$$

$$\frac{dI_v}{dt} = a\omega_3 \frac{S_v I_p}{N_p} - \eta I_v - \mu_v I_v. \tag{4.103}$$

Let $x_1 = (E_p, I_v, I_p, A_h)$ and $y_1 = (S_p, S_v)$, where x_1 and y_1 are infected and susceptible compartments of the model respectively. Separating the infected subsystem (4.100-4.103) into two parts, $\mathscr{F}(x,y)$ is the transmission part which portrays the production of new infections and $\mathscr{V}$(x,y) is the transition part which involves change of states:

$$\mathscr{F}(x,y) = \begin{pmatrix} (1-\xi)\left(a\omega_1 \frac{S_p I_v}{N_p} + \beta_e \frac{S_p E_p}{N_p} + \beta_i \frac{S_p I_p}{N_p} + \omega_2 \frac{S_p A_h}{N_p(K+A_h)}\right) + (1-\delta)\theta I_p \\ a\omega_3 \frac{S_v I_p}{N_p} \\ 0 \\ 0 \end{pmatrix}, \tag{4.104}$$

$$\mathscr{V}(x,y) = \begin{pmatrix} -\alpha_p E_p - qE_p \\ -\eta I_p - \mu_v I_v \\ qE_p - \alpha_p I_p - dI_p - rI_p \\ \phi I_p - \mu_h A_h - \psi A_h \end{pmatrix}. \tag{4.105}$$

Let $F = \frac{\partial \mathscr{F}(x,y)}{\partial x_i}$ and $V = \frac{\partial \mathscr{V}(x,y)}{\partial x_i}$ where $x_i = (E_p, I_v, I_p, A_h)$ for $i = 1,2,3,4$. At the DFE every member of the population is susceptible, thus $S_p^0 = N_p(0)$. Differentiating and evaluating at X_0 results into (4.106) and (4.107),

$$F = \begin{pmatrix} (1-\xi)\beta_e & (1-\xi)a\omega_1 & (1-\xi)\beta_i + (1-\delta)\theta & (1-\xi)\frac{\omega_2}{K} \\ 0 & 0 & \frac{a\omega_3 \alpha_p b_v}{\mu_v b_p} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}, \tag{4.106}$$

$$V = \begin{pmatrix} (\alpha_p + q) & 0 & 0 & 0 \\ 0 & (\eta + \mu_v) & 0 & 0 \\ -q & 0 & (\alpha_p + d + r) & 0 \\ 0 & 0 & -\phi & (\mu_h + \psi) \end{pmatrix}, \tag{4.107}$$

$$V^{-1} = \begin{pmatrix} \frac{1}{(\alpha_p+q)} & 0 & 0 & 0 \\ 0 & \frac{1}{(\eta+\mu_v)} & 0 & 0 \\ \frac{q}{(\alpha_p+q)(\alpha_p+d+r)} & 0 & \frac{1}{(\alpha_p+d+r)} & 0 \\ \frac{q\phi}{(\alpha_p+q)(\alpha_p+d+r)(\mu_h+\psi)} & 0 & \frac{\phi}{(\alpha_p+d+r)(\mu_h+\psi)} & \frac{1}{\mu_h+\psi} \end{pmatrix}. \tag{4.108}$$

Then it follows that FV^{-1} is

$$FV^{-1} = \begin{pmatrix} T_R & \frac{(1-\xi)a\omega_1}{\eta+\mu_v} & T_C & \frac{(1-\xi)\omega_2}{k(\mu_h++\psi)} \\ \frac{a\omega_3\alpha_p b_v q}{\mu_v b_p(\alpha_p+q)(\alpha_p+d+r)} & 0 & \frac{a\omega_3\alpha_p b_v}{\mu_v b_p(\alpha_p+d+r)} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}. \tag{4.109}$$

Now, denote the matrix $Q = FV^{-1}$ in (4.109), the effective reproduction number R_e of the model is a largest eigenvalue of the matrix Q. Therefore,

$$R_e = \frac{1}{2}T_R + \frac{1}{2}\sqrt{(T_R)^2 + 4\frac{(1-\xi)a^2\omega_1\omega_3\alpha b_v q}{(\eta+\mu_v)\mu_v b_p(\alpha+q)(\alpha+d+r)}}. \tag{4.110}$$

Where,

$$T_R = \frac{(1-\xi)\beta_e}{\alpha_p+q} + \frac{((1-\xi)\beta_i+(1-\delta)\theta)q}{(\alpha_p+q)(\alpha_p+d+r)} + \frac{(1-\xi)\omega_2\phi q}{k(\alpha_p+d+r)(\alpha_p+q)(\mu_h+\psi)}, \tag{4.111}$$

and

$$T_C = \frac{(1-\xi)\beta_i+(1-\delta)\theta}{\alpha_p+d+r} + \frac{(1-\xi)\omega_2\phi}{k(\alpha_p+d+r)(\mu_h+\psi)}. \tag{4.112}$$

From the effective reproduction number (R_e) in (4.110), it shows that increasing farmers participatory in farming education programmes (ξ) decreases the average number of new infections (R_e). Clearing of Xcm bacteria in the soil (ψ) reduces the life span of Xcm bacteria in the soil and hence reduces the ability of the disease to transmit through soil. Timely removal of diseased plants (r) reduces the average time at which an infected banana plant stay in a symptomatic infected stage which reduces further spread of the BXW disease in the field. Furthermore, increase in the control to avoid vertical transmission (δ) reduces further spread of the BXW disease from the mother plant to its suckers and hence reduces the effective reproduction number.

4.4 Numerical simulation

4.4.1 Numerical simulation of the basic model

In this section we simulate the basic model to study the dynamics of BXW disease when control measures are not included. Although these results seem to be the expected behaviour, however this study has established the optimal rate that will reduce the new infections to the lowest possible level.

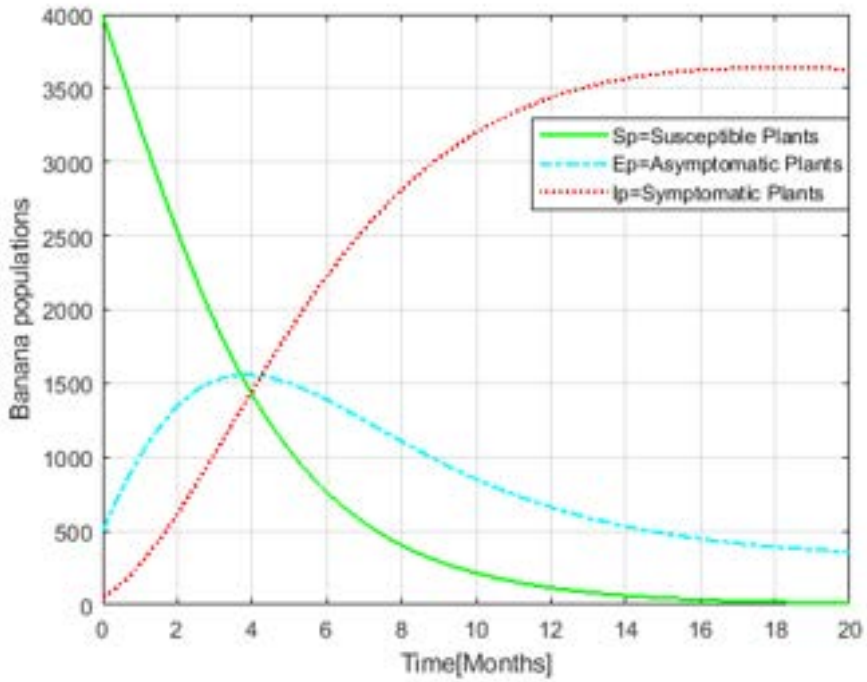
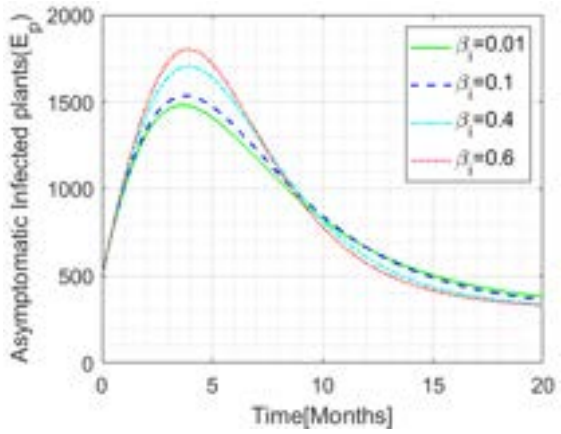
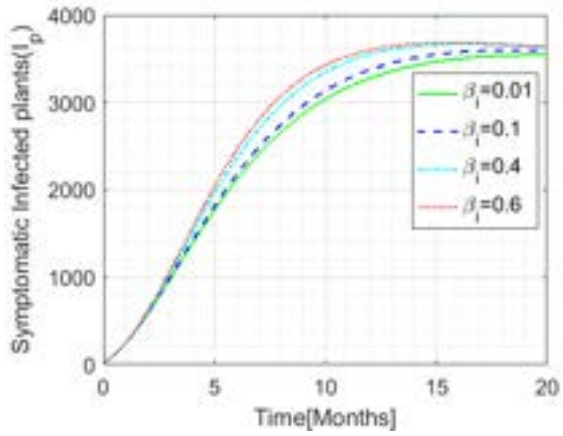


Figure 7: Banana population dynamics

From Fig. 7, it is observed that the number of susceptible plants decreases exponentially due to infection by BXW disease. The number of asymptomatic banana plants increases during the first four months since infection. After four months the number of asymptomatic plants starts to decrease while the number of symptomatic plants continue to increase. This is because most of the banana cultivars start showing symptoms after 3 months and hence reduce the number of asymptomatic plants. It can also be observed that without control measures, after 18 months since onset of the infection, all the banana plants will be infected.



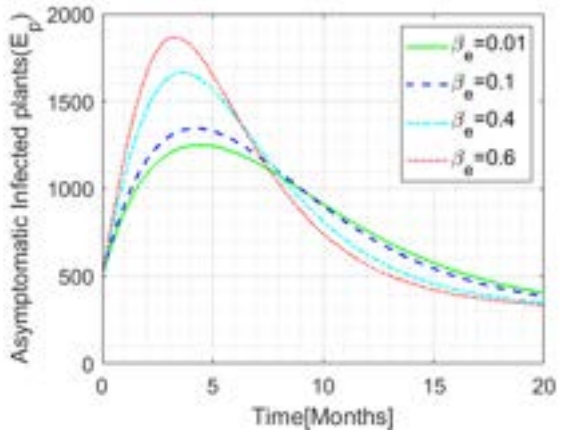
(a) Effect on E_p



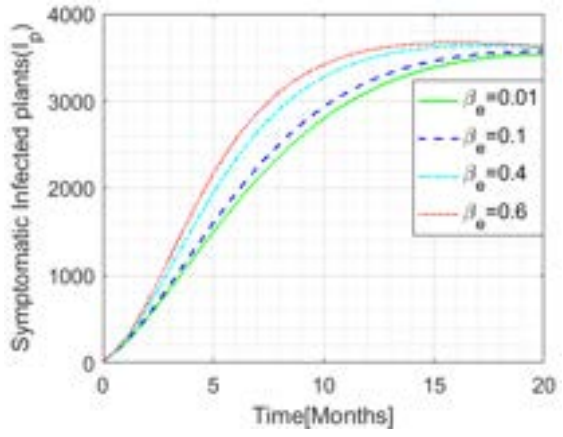
(b) Effect on I_p

Figure 8: Variations in the rate of infection from symptomatic infected banana

Figure 8 shows that as the rate of infection through farming tools contaminated with Xcm bacteria from symptomatic infected banana plants (β_i) increases, dramatically increases the number of asymptomatic and symptomatic infected banana plants.



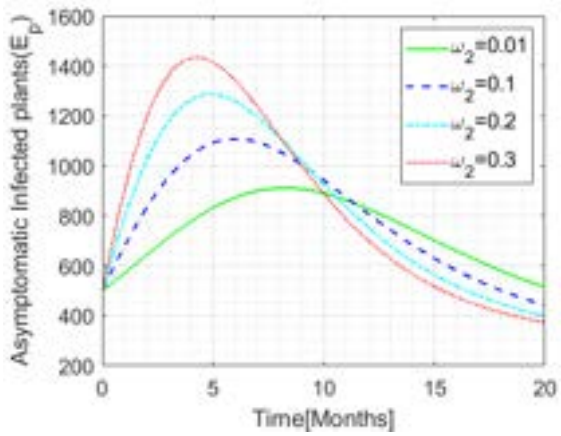
(a) Effect on E_p



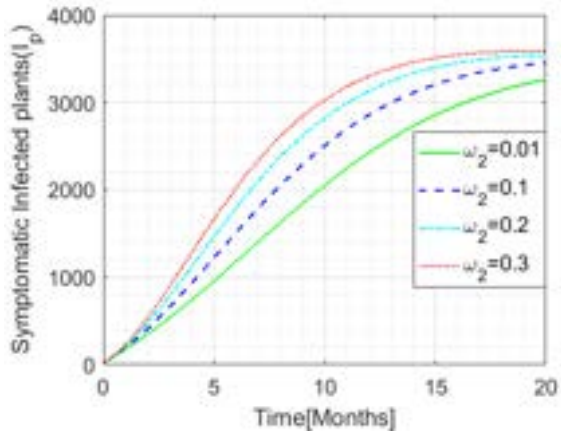
(b) Effect on I_p

Figure 9: Variations in the rate of infection from asymptomatic infected banana

Figure 9 shows that increasing the rate of infection through farming tools contaminated with Xcm bacteria by an asymptomatic infected banana plant (β_e) also increases the number of asymptomatic and symptomatic infected banana plants. This implies that when performing farming activities, farmers may transmit the BXW disease unknowingly through asymptomatic infected banana plants thinking that they are health plants.



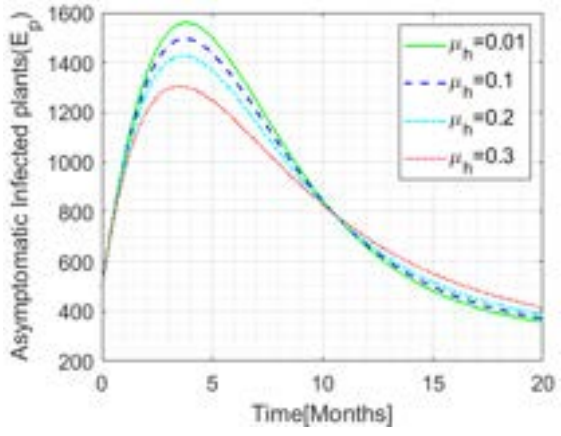
(a) Effect on E_p



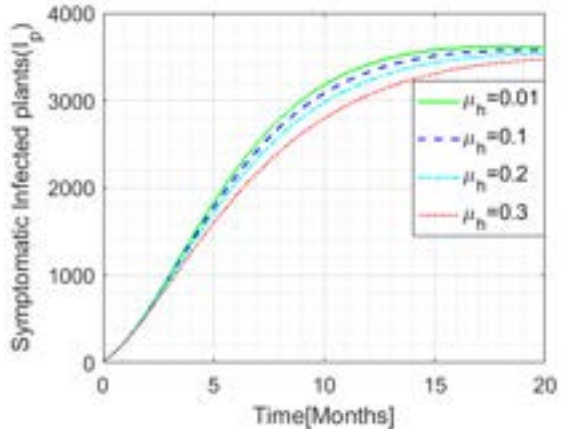
(b) Effect on I_p

Figure 10: Variations in the rate of infection through contaminated soil

In Fig. 10 it can be observed that increasing the rate of infection through contaminated soil (ω_2) increases the number of asymptomatic and symptomatic infected banana plants. With reference to Fig. 11, the natural clearance rate of Xcm bacteria in the soil (μ_h) has an impact of on the dynamics of BXW disease. Increasing μ_h reduces the number of new infections generated through contaminated soil. Therefore increasing this parameter decreases the number of Xcm bacterium in the soil and hence reduces the number of secondary infection through contaminated soil.



(a) Effect on E_p



(b) Effect on I_p

Figure 11: Variations in the natural mortality rate of Xcm bacteria in the soil

Furthermore, Fig. 12 shows that timely removal of infected symptomatic plants reduces the number of new infections generated by a symptomatic infected banana plant.

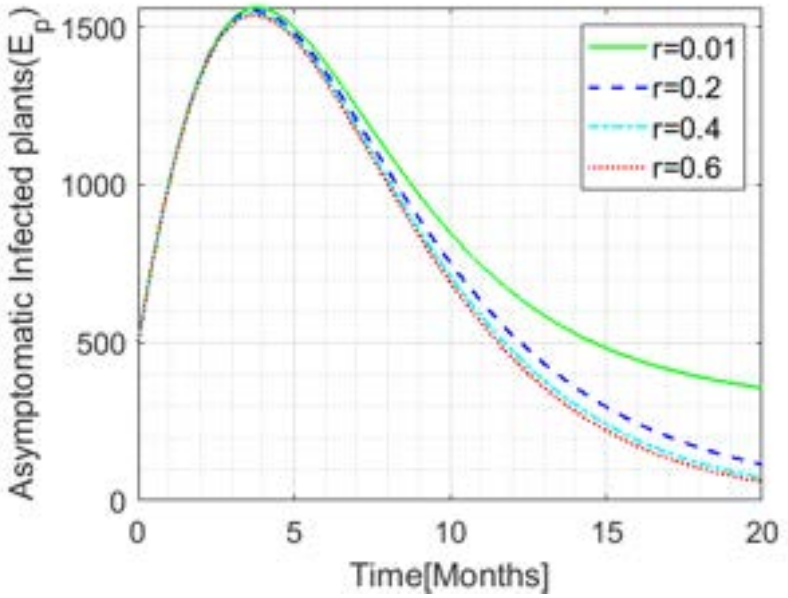


Figure 12: Variations in the rate of removing I_p from the farm

Figures 13-16 illustrate the effect of the most sensitive and moderate parameters to the basic reproduction number. Both parameters with positive indices and parameters with negative indices are included. Figure 13 illustrate the effect of removing symptomatic infected banana plants in the field, It shows that increasing the rate of removing infected plants in the field exponentially decreases the basic reproduction number. This results is also in agreement with biological studies conducted by Blomme *et al.* (2017*b*) and Ntamwira *et al.* (2019*b*).

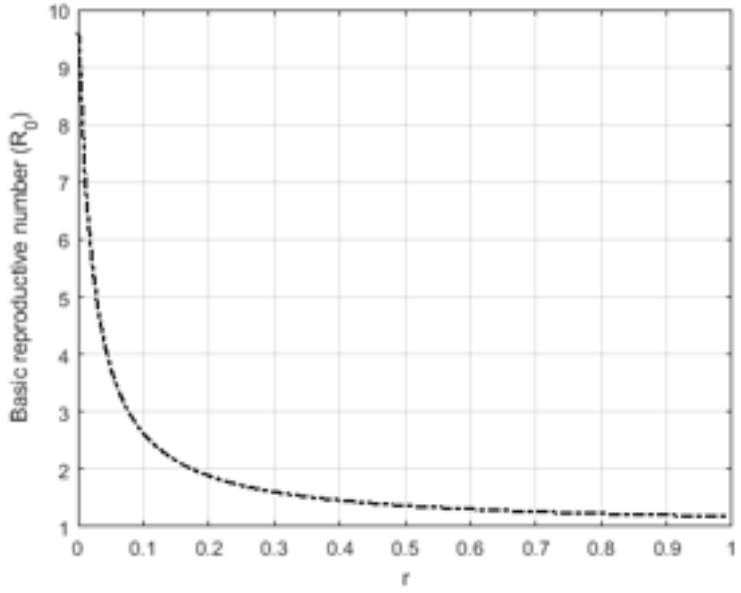


Figure 13: The effect of removing Symptomatic infected plants in the field on the R_0

Figure 14 shows that the increase in the natural clearance rate of Xcm bacteria in the soil (μ_h) leads to the decrease in the basic reproduction number. Furthermore, it shows that μ_h alone cannot make $R_0 < 1$. Nevertheless, this results can not undermine the need for a technology to speed up the clearance rate of Xcm bacteria in the soil to avoid secondary infections resulting from soil inoculum.

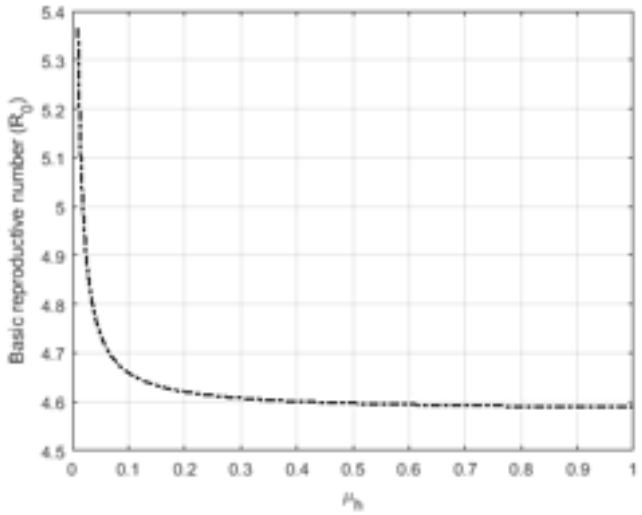
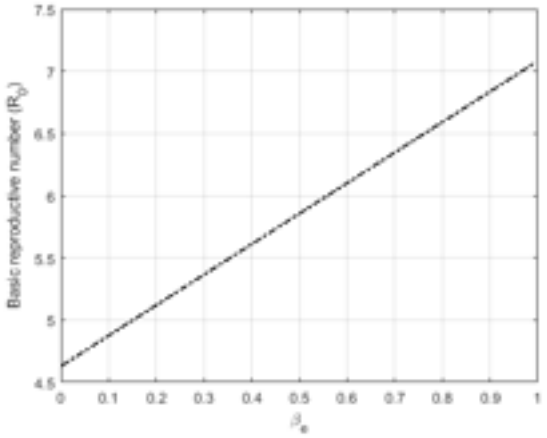
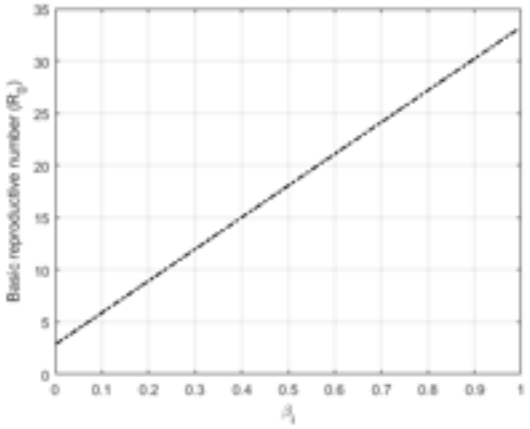


Figure 14: The effect of Xcm bacteria clearance on the R_0



(a) Infections from asymptomatic infected plants



(b) Infections from symptomatic infected plants

Figure 15: The effect of contaminated farming tools infections on the R_0

Figure 15 shows that infections rates through contaminated tools emanating from asymptomatic and symptomatic infected plants, both have a direct proportional relations to the basic reproduction number. Which means increasing any of these infection rates results to the increase in the basic reproduction number. Figure 15 further shows that infection rate from the contaminated tools emanating from symptomatic infected plants is more sensitive to the basic reproduction number compared to that emanating from asymptomatic infected plants. This might be due to the fact that in symptomatic infected plants the Xcm bacteria inoculum is very high.

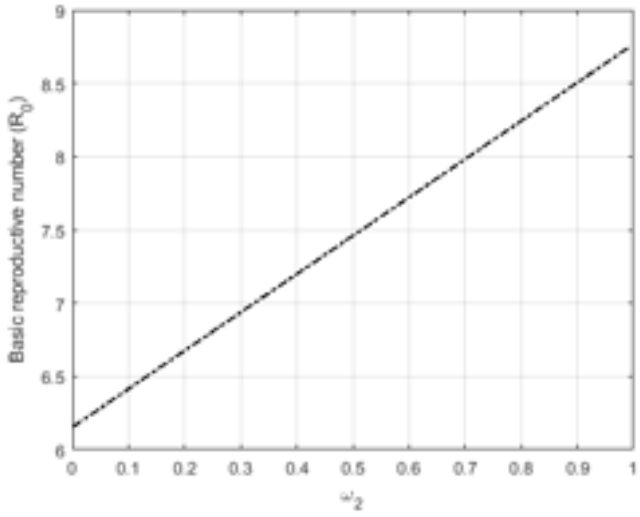


Figure 16: The effect of infections through contaminated soil on the R_0

Figure 16 reveals that as the rate of infection through contaminated soil increases, it increases the basic reproduction number. The possible solution to this kind of infections is to clear out all Xcm bacteria found in the soil.

4.4.2 Simulation of the model with control measures

This section shows the numerical simulation of the model with control measures. I comprises of the simulations of the relationship between effective reproduction number (R_e) to the control measures such as participatory community education programmes (ξ), Clearance of Xcm bacteria in the soil (ψ), Single diseased stem removal (r), and vertical transmission control (δ). Also this section presents the effect of control measures adoption to the control of the disease. Figure 17 shows that, participatory education programmes causes a significant reduction on the effective reproduction number and hence control of the disease. According to Kubiriba *et al.* (2012) and Ochola *et al.* (2015), this education programmes helps farmers to be aware of different modes of BXW disease transmission and proper application the suggested control measures.

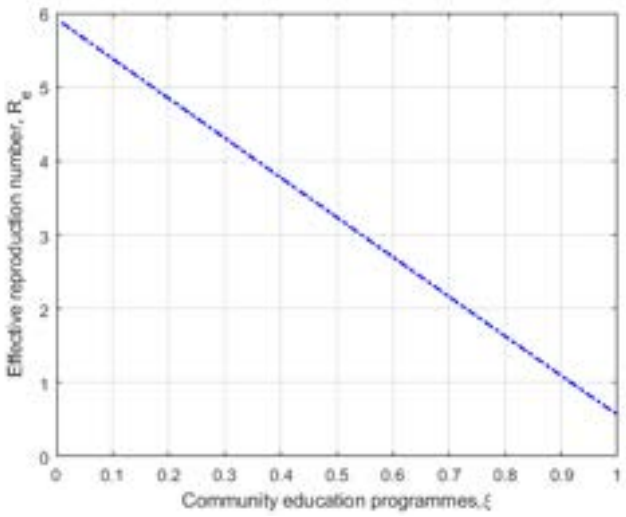
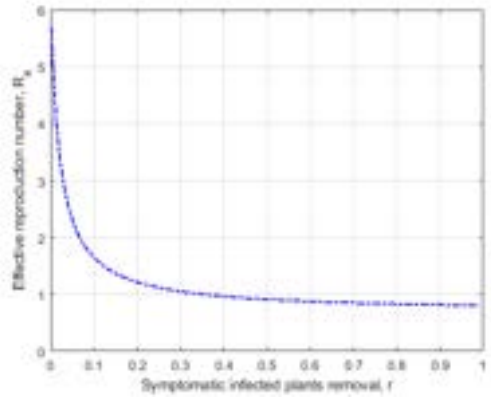


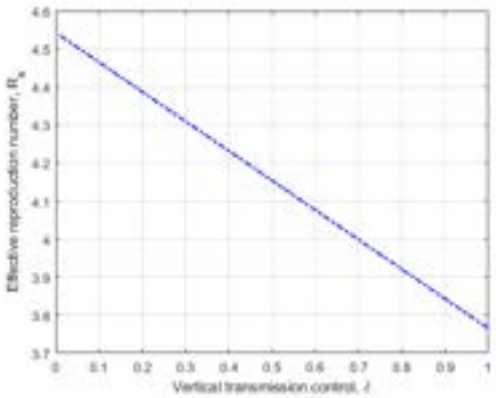
Figure 17: The effect of community participatory education programmes on the R_e

Figure 18 Shows that timely removal of symptomatic infected plants from the field and control of vertical transmission are negatively related to the effective reproduction number. But removal of symptomatic infected plants is more sensitive compared to control of mother to child infections. r dramatically reduce the effective reproduction number to less than a unit where the disease can be controlled. These results are in agreement with Ntamwira *et al.* (2019*b*) and

Blomme *et al.* (2017*b*) who established that Single diseased stem removal (r) approach play a great role in avoiding further spread of the BXW disease. Ocimati *et al.* (2015) established that it is possible to find susceptible suckers in the mat where infected plant rose, this is due to incomplete systemic movement of the Xcm bacteria.



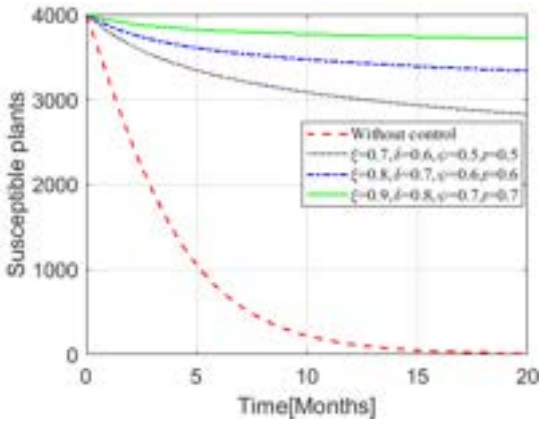
(a) Removal of symptomatic infected banana plants



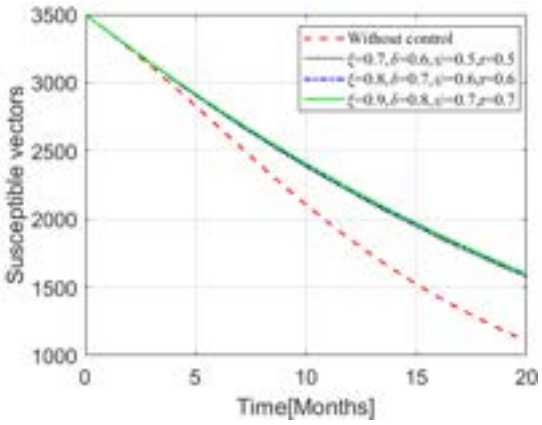
(b) Vertical transmission control

Figure 18: The effect of SDSR and vertical transmission control on the R_e

This argument have been supported by Blomme *et al.* (2017*b*) who found out that proper timely removal of an infected banana plant helps to prevent further spread of the disease to its lateral shoots. Therefore, while finding an effective technology to control vertical transmission, Timely removal of infected banana plant can serve that purpose.



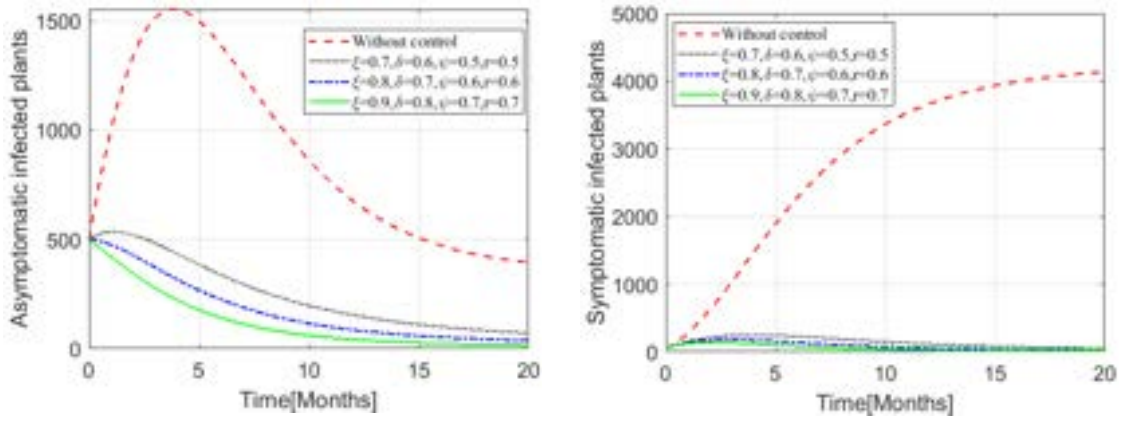
(a) Susceptible banana plants



(b) Susceptible vectors

Figure 19: Impact of control measures to the Susceptible banana plants and vectors

Figure 19 shows that without control measures Susceptible banana plants decreases dramatically to 0 in 18 months. This is because of the high number of secondary infections. Figure 19 further shows that as farmers adopt and practice the suggested control measures the number of susceptible banana plants decreases slowly due to harvesting activities and uprooting for replanting to other fields. Figure 19 also shows that with control measures the rate of susceptible vectors been contaminated with Xcm bacteria decreases hence decrease disease transmission through vectors.



(a) Effect on E_p

(b) Effect on I_p

Figure 20: Impact of control measures to infected plants

Figure 20 shows that without control measures asymptomatic infected banana plants raise from 500 to 1500 in 5 months while with control measures the number of asymptomatic infected plants continuously decreases with time and approaches zero (0) after 15 months.

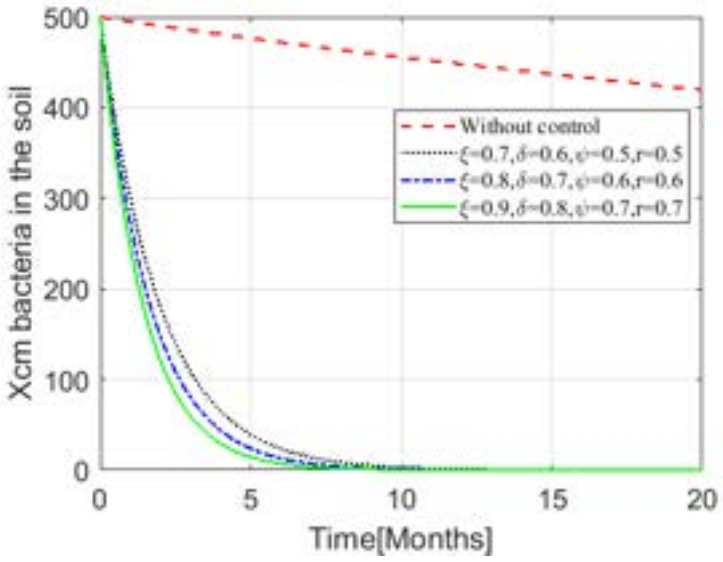


Figure 21: The effect of Xcm bacteria clearance on the R_e

On the other hand when no control applied in the field, symptomatic infected banana plants increases up to 4000 after 20 months while with control measures symptomatic infected banana plants increases in a very small number from 2 to 6 months and decreases to zero (0) in 15 months. Figure 21 shows that without control measures the number of Xcm bacteria in the soil decreases slowly depending on natural clearance. But when control measures are correctly adopted and applied including artificial Xcm bacteria clearance in the soil. the number of Xcm bacteria in the soil decreases rapidly approaching 0 in 8 months.

CHAPTER FIVE

CONCLUSION AND RECOMMENDATIONS

5.1 Conclusion

In this study a deterministic mathematical model for Banana Xanthomonas wilt (BXW) disease is formulated and analysed. The aim was to get an understanding of the transmission dynamics of BXW disease when contaminated soil is taken into consideration. The main tasks in this study includes (a) to formulate and analyse a basic model for BXW which includes contaminated soil (b) to formulate and analyse the mathematical model with participatory community education programmes, Clearance of Xcm bacteria in the soil, Single diseased stem removal and vertical transmission control measures.

The basic model was formulated and using next generation method, the basic reproduction number as described by Van den Driessche and Watmough was derived. Stability analysis of the model equilibria points was carried out. The results showed that the disease free equilibrium exists and is locally and globally asymptotically stable when $R_0 < 1$ and unstable when $R_0 > 1$. Similarly, the model endemic equilibrium exists and is globally asymptotically stable if and only if $R_0 > 1$.

From the sensitivity analysis, it is observed that the most sensitive parameters of the model are: the rate of infection through farming tools contaminated by Xcm bacteria from symptomatic infected plants (β_i), the rate of infection through farming tools contaminated by Xcm bacteria from asymptomatic infected plants (β_e), the rate of infection through contaminated soil (ω_2), rate of removing infected banana plant from the farm (r), Clearance rate of bacteria in the environment (μ_h), vertical transmission (θ), and disease induced death rate of an infected banana plant (d). Furthermore, numerical simulation wa conducted to validate the results. Results from the parameter ω_2 and μ_h show that contaminated soil contributes to BXW disease transmission and persistence. Thus ignoring this component of the model may lead to underestimation of BXW disease transmission.

The basic model was then extended to include participatory community education programmes, Clearance of Xcm bacteria in the soil, Single diseased stem removal and vertical transmission control measures. The results showed that when participatory community education programmes, Clearance of Xcm bacteria in the soil, Single diseased stem removal and vertical transmission control are applied they dramatically reduces further spread of the BXW disease.

Therefore, if the suggested control measures are applied inline with the current control strategies such as timely removal of the male bud using a forked stick, sterilization of farming tools, and planting of healthy suckers to avoid further introduction new infections in the field, it is possible to contain the disease in less than 20 months.

5.2 Recommendations

Therefore, in order to best contain the disease, along with the current control measures such as timely removal of the male bud using a forked stick, sterilization of farming tools, and planting of healthy suckers to avoid further introduction new infections in the field we propose the following recommendations to scientists and technologist, farmers and government:

(i) To carry out studies that will find a way to speed up the clearance rate of Xcm bacteria in the soil without disturbing the ecosystem so as to avoid soil inoculum which is the source of soil borne infections and persistence of the disease in farm.

(ii) To carry out studies that will find a way infection from infected mother plant to its suckers can be reduced or completely stopped.

(iii) Furthermore, the government through its respective organs should facilitate participatory community education programmes so as to raise farmers awareness on the BXW disease transmission dynamics and its control strategies. Moreover, the government also should should find the way that the technology for early detection of infected plants should be brought down to the local farmers at affordable costs, this will help stakeholders to detect and remove the infected plants from the farm on time.

(iv) Finally, farmers when performing farming activities such as harvesting, pruning, weeding and removing of the infected symptomatic banana plants should sterilize their farming tools before moving to another banana plant. moreover, farmers should avoid weeding near infected banana plants as can causes wounds in the roots of the healthy banana plant and give a room for new infections from the soil inoculum.

(v) This study has not covered everything on the transmission dynamics of BXW disease, therefore further studies are required to improve the understanding on the dynamics of BXW disease. Areas where this study can be extended includes but not limited to:

   (a) Seasonal variations (temperature and humidity),

(b) Resistant breed,

(c) stochastic model or Markov chain,

(d) Social factors that hinder farmers from the adoption of suggested control strategies.

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APPENDICES

Appendix I: MATLAB CODES

A.1 MATLAB codes for Figure 7

```
%Defining a functions 'Mycontrol_model0.m, Mycontrol_model.m, Mycontrol_model1.m Mycontrol_model2.m' and their corresponding equations as follows:
function dy=Mycontrol_model0(~,y)
dy=zeros(size(y));
%Declaration of parameters
edu=0;delta=0;psi=0;delta=0.3;psi=0.2;beta2=0.3;beta1=0.1429;bp=0.1667;theta=0.0256;muh=0.01;d=0.0167;k=1000;r=0;q=0.3;eta=0.0286;phi=0.86;alpha=0.0056;a=0.2;bv=0.03;muv=0.05;omega2=0.4;omega1=0.2;omega3=0.2;
%Variables declaration
Sp=y(1);Ep=y(2);Ip=y(3);Sv=y(4);Iv=y(5);Ah=y(6);
%Equations of the model
dy(1)=bp-(1-edu)*((a*omega1*Iv)/(Sp+Ep+Ip)+(beta2*Ep/(Sp+Ep+Ip)+(beta1*Ip)/(Sp+Ep+Ip)+(omega2*Ah)/(k+Ah)+alpha)*Sp;
dy(2)=(1-edu)*((a*omega1*Iv)/(Sv+Iv)+(beta2*Ep)/(Sp+Ep+Ip)+(beta1*Ip)/(Sp+Ep+Ip)+(omega2*Ah)/(k+Ah))*Sp+(1-delta)*theta*Ip-(alpha+q)*Ep;
dy(3)=q*Ep-(alpha+d+r)*Ip;
dy(4)=bv+eta*Iv-((a*omega3*Ip)/(Sp+Ep+Ip)+muv)*Sv;
dy(5)=((a*omega3*Sv*Ip)/(Sp+Ep+Ip))-(eta+muv)*Iv;
dy(6)=phi*Ip/(Sp+Ep+Ip)-(muh+psi)*Ah;
++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
function dy=Mycontrol_model(~,y)
dy=zeros(size(y));
%Declaration of parameters
edu=0.7;delta=0.6;psi=0.5;delta=0.3;psi=0.2;beta2=0.3;beta1=0.1429;bp=0.1667;theta=0.0256;muh=0.01;d=0.0167;k=1000;r=5;q=0.3;eta=0.0286;phi=0.86;alpha=0.0056;a=0.2;bv=0.03;muv=0.05;omega2=0.4;omega1=0.2;omega3=0.2;
%Variables declaration
Sp=y(1);Ep=y(2);Ip=y(3);Sv=y(4);Iv=y(5);Ah=y(6);
%Equations of the model
dy(1)=bp-(1-edu)*((a*omega1*Iv)/(Sp+Ep+Ip)+(beta2*Ep/(Sp+Ep+Ip)+(beta1*Ip)/(Sp+Ep+Ip)+(omega2*Ah)/(k+Ah)+alpha)*Sp;
dy(2)=(1-edu)*((a*omega1*Iv)/(Sv+Iv)+(beta2*Ep)/(Sp+Ep+Ip)+(beta1*Ip)/(Sp+Ep+Ip)+(omega2*Ah)/(k+Ah))*Sp+(1-delta)*theta*Ip-(alpha+q)*Ep;
dy(3)=q*Ep-(alpha+d+r)*Ip;
dy(4)=bv+eta*Iv-((a*omega3*Ip)/(Sp+Ep+Ip)+muv)*Sv;
dy(5)=((a*omega3*Sv*Ip)/(Sp+Ep+Ip))-(eta+muv)*Iv;
```

```
dy(6)=phi*Ip/(Sp+Ep+Ip)-(muh+psi)*Ah;
++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
function dy=Mycontrol_model1(~,y)
dy=zeros(size(y));
%Declaration of parameters
edu=0.8;delta=0.7;psi=0.6;delta=0.3;psi=0.2;beta2=0.3;beta1
    =0.1429;bp=0.1667;theta=0.0256;muh=0.01;d=0.0167;k=1000;r=6;
    q=0.3;eta=0.0286;phi=0.86;alpha=0.0056;a=0.2;bv=0.03;muv
    =0.05;omega2=0.4;omega1=0.2;omega3=0.2;
%Variables declaration
Sp=y(1);Ep=y(2);Ip=y(3);Sv=y(4);Iv=y(5);Ah=y(6);
%Equations of the model
dy(1)=bp-(1-edu)*((a*omega1*Iv)/(Sp+Ep+Ip)+(beta2*Ep/(Sp+Ep+Ip)
    +(beta1*Ip)/(Sp+Ep+Ip)+(omega2*Ah)/(k+Ah)+alpha)*Sp;
dy(2)=(1-edu)*((a*omega1*Iv)/(Sv+Iv)+(beta2*Ep)/(Sp+Ep+Ip)+(
    beta1*Ip)/(Sp+Ep+Ip)+(omega2*Ah)/(k+Ah))*Sp+(1-delta)*theta*
    Ip-(alpha+q)*Ep;
dy(3)=q*Ep-(alpha+d+r)*Ip;
dy(4)=bv+eta*Iv-((a*omega3*Ip)/(Sp+Ep+Ip)+muv)*Sv;
dy(5)=((a*omega3*Sv*Ip)/(Sp+Ep+Ip))-(eta+muv)*Iv;
dy(6)=phi*Ip/(Sp+Ep+Ip)-(muh+psi)*Ah;
++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
function dy=Mycontrol_model2(~,y)
dy=zeros(size(y));
%Declaration of parameters
edu=0.9;delta=0.8;psi=0.7;delta=0.3;psi=0.2;beta2=0.3;beta1
    =0.1429;bp=0.1667;theta=0.0256;muh=0.01;d=0.0167;k=1000;r
    =0.7;q=0.3;eta=0.0286;phi=0.86;alpha=0.0056;a=0.2;bv=0.03;
    muv=0.05;omega2=0.4;omega1=0.2;omega3=0.2;
%Variables declaration
Sp=y(1);Ep=y(2);Ip=y(3);Sv=y(4);Iv=y(5);Ah=y(6);
%Equations of the model
dy(1)=bp-(1-edu)*((a*omega1*Iv)/(Sp+Ep+Ip)+(beta2*Ep/(Sp+Ep+Ip)
    +(beta1*Ip)/(Sp+Ep+Ip)+(omega2*Ah)/(k+Ah)+alpha)*Sp;
dy(2)=(1-edu)*((a*omega1*Iv)/(Sv+Iv)+(beta2*Ep)/(Sp+Ep+Ip)+(
    beta1*Ip)/(Sp+Ep+Ip)+(omega2*Ah)/(k+Ah))*Sp+(1-delta)*theta*
    Ip-(alpha+q)*Ep;
dy(3)=q*Ep-(alpha+d+r)*Ip;
dy(4)=bv+eta*Iv-((a*omega3*Ip)/(Sp+Ep+Ip)+muv)*Sv;
dy(5)=((a*omega3*Sv*Ip)/(Sp+Ep+Ip))-(eta+muv)*Iv;
dy(6)=phi*Ip/(Sp+Ep+Ip)-(muh+psi)*Ah;
++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
%%RUNNING FILE
clear all
%Runge kuta fourth order approach
tspan=[0:0.0001:20]; %Time in Months, the first day banana is
```

```
    planted to the Harvesting Month.
y0=[4000 500 50 3500 2000 500]; % Population size for Sp, Ep,
    Ip, Sv, Iv and Ah
[t01,y01]=ode45(@Mycontrol_model0,tspan, y0); %Runge kuta 4th
    order function.
[t,y]=ode45(@Mycontrol_model,tspan, y0);
[t1,y1]=ode45(@Mycontrol_model1,tspan, y0);
[t2,y2]=ode45(@Mycontrol_model2,tspan, y0);
%plot
figure(1)
set(gca,'Fontsize',15)
set(legend,'Fontsize',15)
plot(t01,y01(:,2),'r--',t,y(:,2),'k:',t1,y1(:,2),'b-',t2,y2
    (:,2),'g-','LineWidth', 1.5)
legend('Without control','\xi=0.7,\delta=0.6,\psi=0.5,r=0.5','\
    xi=0.8,\delta=0.7,\psi=0.6,r=0.6','\xi=0.9,\delta=0.8,\psi
    =0.7,r=0.7')
xlabel('Time[Months]')
ylabel('Symptomatic infected plants')
hold on
```

A.2 MATLAB codes for Figure 8-12

```
%Defining a functions 'Mycontrol_model0.m,Mycontrol_model.m,
    Mycontrol_model1.m Mycontrol_model2.m' and their
    corresponding equations as follows:
function dy=Mycontrol_model0(~,y)
dy=zeros(size(y));
%Declaration of parameters
edu=0;delta=0;psi=0;delta=0.3;psi=0.2;beta2=0.3;beta1=0.1429;bp
    =0.1667;theta=0.0256;muh=0.01;d=0.0167;k=1000;r=0;q=0.3;eta
    =0.0286;phi=0.86;alpha=0.0056;a=0.2;bv=0.03;muv=0.05;omega2
    =0.4;omega1=0.2;omega3=0.2;
%Variables declaration
Sp=y(1);Ep=y(2);Ip=y(3);Sv=y(4);Iv=y(5);Ah=y(6);
%Equations of the model
dy(1)=bp-(1-edu)*((a*omega1*Iv)/(Sp+Ep+Ip)+(beta2*Ep/(Sp+Ep+Ip)
    +(beta1*Ip)/(Sp+Ep+Ip)+(omega2*Ah)/(k+Ah)+alpha)*Sp;
dy(2)=(1-edu)*((a*omega1*Iv)/(Sv+Iv)+(beta2*Ep)/(Sp+Ep+Ip)+(
    beta1*Ip)/(Sp+Ep+Ip)+(omega2*Ah)/(k+Ah))*Sp+(1-delta)*theta*
    Ip-(alpha+q)*Ep;
dy(3)=q*Ep-(alpha+d+r)*Ip;
dy(4)=bv+eta*Iv-((a*omega3*Ip)/(Sp+Ep+Ip)+muv)*Sv;
dy(5)=((a*omega3*Sv*Ip)/(Sp+Ep+Ip))-(eta+muv)*Iv;
dy(6)=phi*Ip/(Sp+Ep+Ip)-(muh+psi)*Ah;
++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
```

```
function dy=Mycontrol_model(~,y)
dy=zeros(size(y));
%Declaration of parameters
edu=0.7;delta=0.6;psi=0.5;delta=0.3;psi=0.2;beta2=0.3;beta1
    =0.1429;bp=0.1667;theta=0.0256;muh=0.01;d=0.0167;k=1000;r=5;
    q=0.3;eta=0.0286;phi=0.86;alpha=0.0056;a=0.2;bv=0.03;muv
    =0.05;omega2=0.4;omega1=0.2;omega3=0.2;
%Variables declaration
Sp=y(1);Ep=y(2);Ip=y(3);Sv=y(4);Iv=y(5);Ah=y(6);
%Equations of the model
dy(1)=bp-(1-edu)*((a*omega1*Iv)/(Sp+Ep+Ip)+(beta2*Ep/(Sp+Ep+Ip)
    +(beta1*Ip)/(Sp+Ep+Ip)+(omega2*Ah)/(k+Ah)+alpha)*Sp;
dy(2)=(1-edu)*((a*omega1*Iv)/(Sv+Iv)+(beta2*Ep)/(Sp+Ep+Ip)+(
    beta1*Ip)/(Sp+Ep+Ip)+(omega2*Ah)/(k+Ah))*Sp+(1-delta)*theta*
    Ip-(alpha+q)*Ep;
dy(3)=q*Ep-(alpha+d+r)*Ip;
dy(4)=bv+eta*Iv-((a*omega3*Ip)/(Sp+Ep+Ip)+muv)*Sv;
dy(5)=((a*omega3*Sv*Ip)/(Sp+Ep+Ip))-(eta+muv)*Iv;
dy(6)=phi*Ip/(Sp+Ep+Ip)-(muh+psi)*Ah;
++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
function dy=Mycontrol_model1(~,y)
dy=zeros(size(y));
%Declaration of parameters
edu=0.8;delta=0.7;psi=0.6;delta=0.3;psi=0.2;beta2=0.3;beta1
    =0.1429;bp=0.1667;theta=0.0256;muh=0.01;d=0.0167;k=1000;r=6;
    q=0.3;eta=0.0286;phi=0.86;alpha=0.0056;a=0.2;bv=0.03;muv
    =0.05;omega2=0.4;omega1=0.2;omega3=0.2;
%Variables declaration
Sp=y(1);Ep=y(2);Ip=y(3);Sv=y(4);Iv=y(5);Ah=y(6);
%Equations of the model
dy(1)=bp-(1-edu)*((a*omega1*Iv)/(Sp+Ep+Ip)+(beta2*Ep/(Sp+Ep+Ip)
    +(beta1*Ip)/(Sp+Ep+Ip)+(omega2*Ah)/(k+Ah)+alpha)*Sp;
dy(2)=(1-edu)*((a*omega1*Iv)/(Sv+Iv)+(beta2*Ep)/(Sp+Ep+Ip)+(
    beta1*Ip)/(Sp+Ep+Ip)+(omega2*Ah)/(k+Ah))*Sp+(1-delta)*theta*
    Ip-(alpha+q)*Ep;
dy(3)=q*Ep-(alpha+d+r)*Ip;
dy(4)=bv+eta*Iv-((a*omega3*Ip)/(Sp+Ep+Ip)+muv)*Sv;
dy(5)=((a*omega3*Sv*Ip)/(Sp+Ep+Ip))-(eta+muv)*Iv;
dy(6)=phi*Ip/(Sp+Ep+Ip)-(muh+psi)*Ah;
++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
function dy=Mycontrol_model2(~,y)
dy=zeros(size(y));
%Declaration of parameters
edu=0.9;delta=0.8;psi=0.7;delta=0.3;psi=0.2;beta2=0.3;beta1
    =0.1429;bp=0.1667;theta=0.0256;muh=0.01;d=0.0167;k=1000;r
    =0.7;q=0.3;eta=0.0286;phi=0.86;alpha=0.0056;a=0.2;bv=0.03;
```

```
    +(beta1*Ip)/(Sp+Ep+Ip)+(omega2*Ah)/(k+Ah)+alpha)*Sp;
dy(2)=(1-edu)*((a*omega1*Iv)/(Sv+Iv)+(beta2*Ep)/(Sp+Ep+Ip)+(
    beta1*Ip)/(Sp+Ep+Ip)+(omega2*Ah)/(k+Ah))*Sp+(1-delta)*theta*
    Ip-(alpha+q)*Ep;
dy(3)=q*Ep-(alpha+d+r)*Ip;
dy(4)=bv+eta*Iv-((a*omega3*Ip)/(Sp+Ep+Ip)+muv)*Sv;
dy(5)=((a*omega3*Sv*Ip)/(Sp+Ep+Ip))-(eta+muv)*Iv;
dy(6)=phi*Ip/(Sp+Ep+Ip)-(muh+psi)*Ah;
++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
function dy=Mycontrol_model(~,y)
dy=zeros(size(y));
%Declaration of parameters
edu=0.7;delta=0.6;psi=0.5;delta=0.3;psi=0.2;beta2=0.3;beta1
    =0.1429;bp=0.1667;theta=0.0256;muh=0.01;d=0.0167;k=1000;r=5;
    q=0.3;eta=0.0286;phi=0.86;alpha=0.0056;a=0.2;bv=0.03;muv
    =0.05;omega2=0.4;omega1=0.2;omega3=0.2;
%Variables declaration
Sp=y(1);Ep=y(2);Ip=y(3);Sv=y(4);Iv=y(5);Ah=y(6);
%Equations of the model
dy(1)=bp-(1-edu)*((a*omega1*Iv)/(Sp+Ep+Ip)+(beta2*Ep/(Sp+Ep+Ip)
    +(beta1*Ip)/(Sp+Ep+Ip)+(omega2*Ah)/(k+Ah)+alpha)*Sp;
dy(2)=(1-edu)*((a*omega1*Iv)/(Sv+Iv)+(beta2*Ep)/(Sp+Ep+Ip)+(
    beta1*Ip)/(Sp+Ep+Ip)+(omega2*Ah)/(k+Ah))*Sp+(1-delta)*theta*
    Ip-(alpha+q)*Ep;
dy(3)=q*Ep-(alpha+d+r)*Ip;
dy(4)=bv+eta*Iv-((a*omega3*Ip)/(Sp+Ep+Ip)+muv)*Sv;
dy(5)=((a*omega3*Sv*Ip)/(Sp+Ep+Ip))-(eta+muv)*Iv;
dy(6)=phi*Ip/(Sp+Ep+Ip)-(muh+psi)*Ah;
++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
function dy=Mycontrol_model1(~,y)
dy=zeros(size(y));
%Declaration of parameters
edu=0.8;delta=0.7;psi=0.6;delta=0.3;psi=0.2;beta2=0.3;beta1
    =0.1429;bp=0.1667;theta=0.0256;muh=0.01;d=0.0167;k=1000;r=6;
    q=0.3;eta=0.0286;phi=0.86;alpha=0.0056;a=0.2;bv=0.03;muv
    =0.05;omega2=0.4;omega1=0.2;omega3=0.2;
%Variables declaration
Sp=y(1);Ep=y(2);Ip=y(3);Sv=y(4);Iv=y(5);Ah=y(6);
%Equations of the model
dy(1)=bp-(1-edu)*((a*omega1*Iv)/(Sp+Ep+Ip)+(beta2*Ep/(Sp+Ep+Ip)
    +(beta1*Ip)/(Sp+Ep+Ip)+(omega2*Ah)/(k+Ah)+alpha)*Sp;
dy(2)=(1-edu)*((a*omega1*Iv)/(Sv+Iv)+(beta2*Ep)/(Sp+Ep+Ip)+(
    beta1*Ip)/(Sp+Ep+Ip)+(omega2*Ah)/(k+Ah))*Sp+(1-delta)*theta*
    Ip-(alpha+q)*Ep;
dy(3)=q*Ep-(alpha+d+r)*Ip;
dy(4)=bv+eta*Iv-((a*omega3*Ip)/(Sp+Ep+Ip)+muv)*Sv;
```

```
dy(5)=((a*omega3*Sv*Ip)/(Sp+Ep+Ip))-(eta+muv)*Iv;
dy(6)=phi*Ip/(Sp+Ep+Ip)-(muh+psi)*Ah;
++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
function dy=Mycontrol_model2(~,y)
dy=zeros(size(y));
%Declaration of parameters
edu=0.9;delta=0.8;psi=0.7;delta=0.3;psi=0.2;beta2=0.3;beta1
    =0.1429;bp=0.1667;theta=0.0256;muh=0.01;d=0.0167;k=1000;r
    =0.7;q=0.3;eta=0.0286;phi=0.86;alpha=0.0056;a=0.2;bv=0.03;
    muv=0.05;omega2=0.4;omega1=0.2;omega3=0.2;
%Variables declaration
Sp=y(1);Ep=y(2);Ip=y(3);Sv=y(4);Iv=y(5);Ah=y(6);
%Equations of the model
dy(1)=bp-(1-edu)*((a*omega1*Iv)/(Sp+Ep+Ip)+(beta2*Ep/(Sp+Ep+Ip)
    +(beta1*Ip)/(Sp+Ep+Ip)+(omega2*Ah)/(k+Ah)+alpha)*Sp;
dy(2)=(1-edu)*((a*omega1*Iv)/(Sv+Iv)+(beta2*Ep)/(Sp+Ep+Ip)+(
    beta1*Ip)/(Sp+Ep+Ip)+(omega2*Ah)/(k+Ah))*Sp+(1-delta)*theta*
    Ip-(alpha+q)*Ep;
dy(3)=q*Ep-(alpha+d+r)*Ip;
dy(4)=bv+eta*Iv-((a*omega3*Ip)/(Sp+Ep+Ip)+muv)*Sv;
dy(5)=((a*omega3*Sv*Ip)/(Sp+Ep+Ip))-(eta+muv)*Iv;
dy(6)=phi*Ip/(Sp+Ep+Ip)-(muh+psi)*Ah;
++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
%%RUNNING FILE
clear all
%Runge kuta fourth order approach
tspan=[0:0.0001:20]; %Time in Months, the first day banana is
    planted to the Harvesting Month.
y0=[4000 500 50 3500 2000 500]; % Population size for Sp, Ep,
    Ip, Sv, Iv and Ah
[t01,y01]=ode45(@Mycontrol_model0,tspan, y0); %Runge kuta 4th
    order function.
[t,y]=ode45(@Mycontrol_model,tspan, y0);
[t1,y1]=ode45(@Mycontrol_model1,tspan, y0);
[t2,y2]=ode45(@Mycontrol_model2,tspan, y0);
%plot
figure(1)
set(gca,'Fontsize',15)
set(legend,'Fontsize',15)
plot(t01,y01(:,2),'r--',t,y(:,2),'k:',t1,y1(:,2),'b-',t2,y2
    (:,2),'g-','LineWidth', 1.5)
legend('Without control','\xi=0.7,\delta=0.6,\psi=0.5,r=0.5','\
    xi=0.8,\delta=0.7,\psi=0.6,r=0.6','\xi=0.9,\delta=0.8,\psi
    =0.7,r=0.7')
xlabel('Time[Months]')
ylabel('Symptomatic infected plants')
```

```
75 hold on
```