MODELING THE DYNAMICS OF BANANA XANTHOMONAS WILT DISEASE WITH MIXED-CROPPING CONTROL MEASURE

Tigist Asfaw Ashamo

A Thesis Submitted to the Department of Applied Mathematics
School of Applied Natural Science
Presented in Partial Fulfillment of the Requirement for the Degree of Master's of Science in Applied Mathematics (Mathematical Modeling)

Office of Graduate Studies
Adama Science and Technology University

June, 2024
Adama, Ethiopia

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Tigist Asfaw Ashamo

Major-Advisor: Dr. Shambel Tadesse Degefu

Co-Advisor: Dr. Mitiku Daba

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DECLARATION

I hereby declare that this Master Thesis entitled ***"Modeling the dynamics of banana xanthomonas wilt disease with mixed-cropping control measure"*** is my original work. That is, it has not been submitted for the award of any academic degree, diploma or certificate in any other university. All sources of materials that are used for this thesis have been duly acknowledged through citation.

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We, the advisors of this thesis, hereby certify that we have read the revised version of the thesis entitled ***"Modeling the dynamics of banana xanthomonas wilt disease with mixed-cropping control measure"*** prepared under our guidance by **Tigist Asfaw Ashamo** submitted in partial fulfillment of the requirements for the degree of Mater's of Science in Applied Mathematics (Mathematical Modeling). Therefore, we recommend the submission of revised version of the thesis to the department following the applicable procedures.

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APPROVAL PAGE

We, the advisors of the thesis entitled ***"Modeling the dynamics of banana xanthomonas wilt disease with mixed-cropping control measure"*** and developed by **Tigist Asfaw Ashamo**, hereby certify that the recommendation and suggestions made by the board of examiners are appropriately incorporated into the final version of the thesis.

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Co-advisor	Signature	Date

We, the undersigned, members of the Board of Examiners of the thesis by **Tigist Asfaw Ashamo** have read and evaluated the thesis entitled ***"Modeling the dynamics of banana xanthomonas wilt disease with mixed-cropping control measure"*** and examined the candidate during open defense. This is, therefore, to certify that the thesis is accepted for partial fulfillment of the requirement of the degree of Master of Science in Applied Mathematics (Mathematical Modeling).

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ABBREVIATION AND ACRONYMS

BXW	Banana Xanthomonas Wilt
DE	Differential Equation
DFEP	Disease Free Equilibrium Point
ECA	East and Central African
EEP	Endemic Equilibrium Point
FAO	Food and Agriculture Organization
ODE	Ordinary Differential Equation
SDSR	Single Diseased Stem Removal
SI	Susceptible Infectious
Xcm	Xanthomonas campestris pv.musacearum

ABSTRACT

Banana Xanthomonas Wilt (BXW) is a devastating disease of Banana plant caused by a bacterium called Xanthomonas campestris pv.musacearum (Xcm). This study focuses on modeling the dynamics of BXW with mixed-cropping control measure using a system of non-linear ordinary differential equations. Next-generation matrix is used to calculate the basic reproduction number. Then, the stability conditions of both the disease-free and endemic equilibria are studied. The qualitative analysis results shows that disease-free equilibrium is both locally and globally stable if the basic reproduction number is less than unity and endemic equilibrium point is globally stable if the basic reproduction number is greater than unity. Sensitivity analysis is carried out to determine the relative significance of parameters and the impacts of possible control method. It is shown that the parameter representing the rate at which contaminated soil causes infection (ω_2), total number of non-host plant in banana plant field (N), rate of Xcm bacteria losses from contaminated soil by planting non-host plant (a_4) are highly influential in determining the time evolution of the disease. That is, ω_2 increase has a positive role in increasing the number of new infections, where as an increase in a_4 and N show the suppression of a new infection of the disease. Finally, using MATLAB numerical simulations are performed for a brief illustration of the disease's dynamical behavior. The simulations results supports the analytic findings and suggest that applying the mixed-cropping cultural control method can reduces the occurrence of secondary infections thus it plays a crucial role in controlling BXW disease.

CHAPTER 1

INTRODUCTION

1.1 Background of the Study

One of the main problems that developing countries face is food security. In East and Central Africa (ECA), food crops such potatoes, rice, bananas, maize, and cassava are grown despite the issue of food security. FAO (FAOSTAT, 2018), provides statistics indicating that bananas hold great significance as a food crop in ECA, and ranked fourth in importance after cassava, maize, and sweet potatoes. Banana fruits are used by the region's farmers for a variety of purposes, including food consumption and business ventures, both of which are essential to their livelihoods. Commercial uses for bananas include the production of fiber, wine, beer, and, as ornamental plants. In 2021 the world produced 124,978,580 tonnes, where 22,941,960 tonnes of bananas were produced in Africa (FAO., 2022). In Africa bananas are largely grown in Tanzania, Kenya, Rwanda, Angola, Cameroon, Egypt and Ethiopia. In spite of these successes, banana production in East and Central Africa is threatened by diseases, pests and declining soil fertility. Banana Xanthomonas Wilt (BXW), Fusarium Wilt (Panama Disease), Black Sigatoka, Banana Bunchy Top Virus (BBTV) and Banana Corm Weevil (BCW) are among the diseases that may affect banana plants (Vazhacharickal et al., 2019). BXW is the main disease threatening banana growing in ECA. This affects farmers and others whose livelihoods depend on banana. (Petsakos et al., 2023).

Banana Xanthomonas Wilt (BXW) is a devastating disease of Banana and Enset caused by a bacterium called Xanthomonas campestris pv.musacearum (Xcm) has severely affected the production of banana and plantain in the East and Central African region. The initial reports of Xcm were documented in Ethiopia on enset (Ensete ventricosum) in 1964 and on banana (Musa spp.) in 1968 (Yirgou & Bradbury, 1974). Thirty years later, the bacteria and disease unexpectedly surfaced and rapidly spread across the Great Lakes region, beginning in central Uganda in 2001 (Carter et al., 2010).

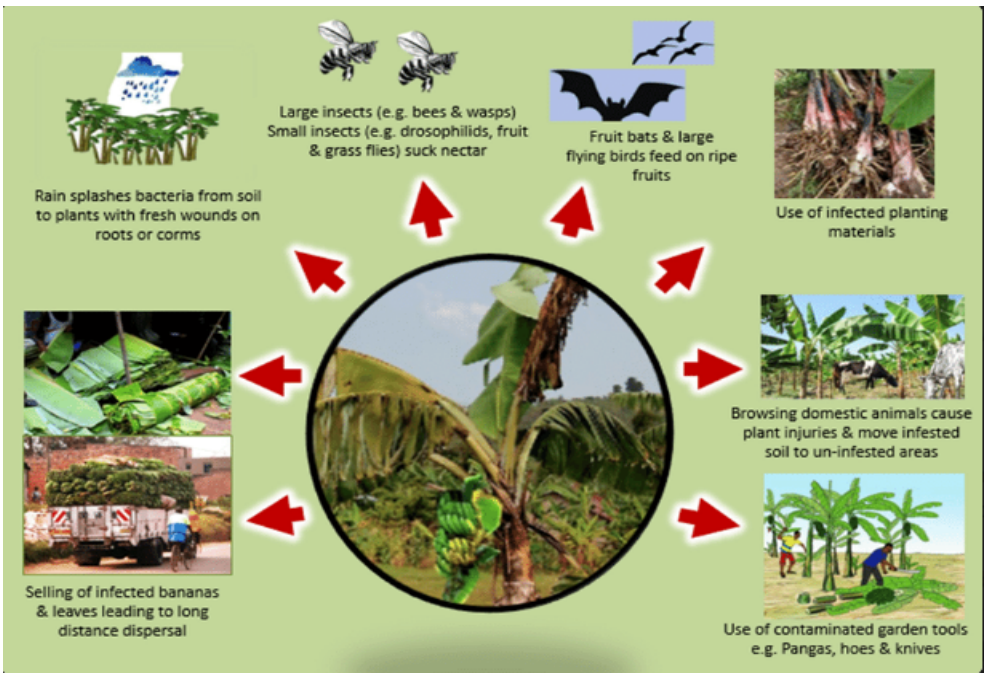

Figure 1.1: Cause and Transmission Ways of BXW Disease (Mulugo et al., 2022)

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Xcm bacterium spreads through contaminated agricultural equipment or soil ,insect vectors, infected planting materials, and transportation of diseased plants. Insects such as sting less bees, fruit flies, and grass flies are attracted to the infected nectar and ooze from male flowers. These insects can carry the bacteria from infected plants to healthy ones, causing the spread of the disease (Rutikanga, Tusiime, et al., 2016). Birds transmit the Xcm bacterium to susceptible plants' male buds by consuming ripe banana fruits from infected banana plants. Bats can also spread the disease by consuming nectar or ripe banana fruits from infected banana plants and passing them on to a healthy banana plant, and ants, such as Monomorium ants, can carry the pathogenic bacteria on their bodies and allowing for potential spread of Xcm bacteria (Ocimati et al., 2019).

(a) Yellowing of banana leaves

(b) Yellow ooze on the cross section cut of the pseudo stem

(c) Rotting of banana fruits

Figure 1.2: Signs seen in infected banana plant with BXW Disease (Mapinda et al., 2022).

Contaminated farming tools can infect susceptible plants during activities like removing male buds, weeding, pruning, removing suckers, and harvesting. The bacteria can contaminate soil for several months, which can lead to a susceptible banana plant can contract Xcm bacteria from polluted soil due to physical damage caused by agricultural activities and by organisms like nematodes and insects that depend on the roots of the plant (Sivirihauma et al., 2017). Additionally, a banana plant's lateral growth may become infected vertically from the mother plant that is infected (Ocimati et al., 2013).

Premature ripening and rotting of fruits; the inflorescence wilts and shrivels with bracts and male buds turning yellow and brown; wilting and yellowing of leaves; internal discoloration of fruits; a yellowish bacterial ooze is excreted from cut plant tissues; and finally, the death of the entire plant are among the usual signs of BXW (V. Nakato et al., 2018; Tripathi et al., 2009).

Figure 1.2 displays the symptoms of leaves, stems, and fruits of the banana plant affected by BXW disease in subplots (a), (b), and (c), respectively. There are no biological or chemical control measures for BXW. Intercropping, crop-rotation, de-budding, sterilization of the farming tools, roguing, timely removal of the male bud and burning of infected banana plants, participatory community education programs, clearance of Xcm bacteria in the soil, single diseased stem removal and control of vertical transmission in limiting the disease spread are some of the common measures used to control the BXW disease.

Intercropping is one of agricultural practice, involves planting non-host (barrier crop) plants alongside the bacterial host (in this study banana). The quality of the barrier plant in attracting vectors and clearance of contaminated soil determines the efficiency of the mixed-cropping method. Beans, sweet pepper, taro, pumpkins, potato, cassava, groundnuts, maize, sorghum, wheat and tomato, which are non-host plants of Xcm bacteria.

Mathematical models have performed and will continue to play a significant role in understanding the transmission dynamics of BXW, in order to determine the most effective mix of control measures to limit the disease's catastrophic impacts. A mathematical model is an abstract depiction of a phenomenon that is created using equations to produce views of the general behavior of an epidemic event. It also serves as a means of examining the impact of determinate factors over the spread of disease and offers an estimate of the general behavior of an epidemic as addressed by epidemic curves, allowing predictions about the duration and population magnitude of the epidemic as well as the evaluation of parameters affecting the dynamics of transmission along with the number of cases (Costa et al., 2021). In E. H. Kweyunga et al. (2018) a mathematical modeling of BXW was formulated and analyzed. The study incorporated infection forces from both asymptomatic and symptomatic banana plants to study the transmission of BXW. To effectively control the disease, the researchers recommended focusing on the monitoring and management of asymptomatic infected banana plants. However the researchers did not consider the impact of contaminated soil and vector transmission in the

dynamics of BXW disease, which is mixed-cropping control measure. Furthermore, Mapinda (2020) study the dynamics of BXW focused on the development and analysis of a deterministic mathematical model for BXW disease. The main goal of the study was to learn more about the dynamics of BXW disease propagation by taking polluted soil into account. Nevertheless, the study did not mention usefulness of mixed-cropping system to clear contaminated soil.

1.2 Statement of the Problem

Banana crop in East and Central Africa is under threat by a number of factors, the primary one being the BXW disease. It has the ability to cause up to 100 % yield loss, severely compromising food security and affect the livelihood of banana-based farming house holds and other individuals who make their living from bananas such as traders of banana fruits (Nakakawa et al., 2017). As a result this study developed mathematical modeling to assess transmission dynamics of BXW with control measure. Recently, the transmission dynamics have been studied through the development and analysis of mathematical models by (Mapinda, 2020; Mapinda et al., 2022). The researcher conducted simulations to assess the impact of different control strategies, including community education programs, soil bacteria clearance, removal of diseased stems, but mathematical models have rarely been used to define and investigate the protective properties of mixed-cropping systems against soil-borne and vector-borne plant bacterial infections that infect host. Therefore, the focus of this study is to create and analyze a mathematical model for studying the transmission dynamics of BXW with mixed-cropping as one of control measure. Consequently, this study endeavors the following fundamental research questions:

- How to determine the conditions for existence and stability of the equilibrium points?
- What is the most sensitive parameter for expansion of the BXW disease?
- How mixed-cropping systems influence the dynamics of BXW disease?

1.3 Objectives of the Study

1.3.1 General Objective of the Study

The general objective of the study is to develop and analyze a mathematical model for the transmission of BXW disease with incorporating mixed-cropping cultural control method as control measure.

1.3.2 Specific Objectives of the Study

The specific objectives of this study are to:

- Identify the conditions of the equilibrium points' existence and stability.
- identify most sensitive parameter for expansion of the disease.
- investigate how mixed-cropping control measure affect the dynamics of BXW disease transmission.

1.4 Significance of the Study

The finding of this study are helpful to:

- determine one cultural control method of BXW disease.
- give evidence-based information on controlling BXW disease in mixed-cropping systems, enhancing policy and decision-making, and farm-level practices to contain and possibly eliminate the disease.
- motivate additional research into discovering a cure to prevent the death of diseased banana plants.

1.5 Limitation of the Study

Conducting research on banana plant populations requires visiting banana fields to gather data using various methods, ensuring a comprehensive study. However, this was not feasible due to various constraints such as limited time, financial constraints, and others.

CHAPTER 2

LITERATURE REVIEW

In this chapter, we provide a brief overview of the literature that has already been written about intercropping method to control the spread of BXW disease. We also discuss the literature that has been written about mathematical modeling of the Banana Xanthomonas Wilt (BXW) disease, highlighting its importance and gaps that this study aims to fill.

In study Rutikanga, Sivirihauma, et al. (2016), Xcm bacteria isolates were obtained from maize, beans, and sweet potato plant parts. This finding suggests a potential non-pathogenic relationship between Xcm isolates from these non-hosts and banana plants, highlighting the need for further research to understand the interactions between Xcm and different plant species, which could have implications for disease management strategies and intercropping practices in banana cultivation.

The study conducted by Hiddink et al. (2010), examined the presence of soilborne pathogens in three different types of farming systems: (i) growing a single crop continuously in monoculture, (ii) rotating crops, and (iii) mixed cropping (which involves cultivating multiple crops simultaneously in the same field). The study mainly focused on use fullness of mixed cropping method to suppress soilborne diseases.

According to Blomme et al. (2017), various crops such as maize, common beans, wheat, sweet pepper, cassava, taro, pumpkins, potato, groundnuts, sorghum and tomato are identified as non-host plants for Xcm bacteria.

Bekele (2021) revised intercropping systems and its advantages across the world. Some of the advantages of intercropping are better control of diseases, weeds, pests and increase productivity, greater stability of yield over different seasons, better use of growth resources, control erosion, fixation of nitrogen by the legume component and others.

A mathematical model by Degefa et al. (2022) investigated the impact of mixed-cropping systems on Potato virus Y (PVY) disease dynamics. The study found that controlling aphids with insecticides is ineffective, but mixed-cropping systems, which include potatoes and non-host crops, can be an important strategy to manage PVY. This study used to describe the advantage of mixed-cropping to control plant disease by using mathematical modeling.

Nannyonga et al. (2015) investigated the use of contaminated tools as a driver of banana Xanthomonas wilt outbreaks within plantations. The authors employ Runge-Kutta fourth-order algorithms to develop an optimal control model that examines the dynamics of the disease spread and the effectiveness of various intervention strategies. The study highlights the importance of proper tool sanitation and disinfection practices in mitigating the risk of disease

transmission and outbreaks within banana plantations. The findings provide valuable insights for agricultural practitioners and policymakers in developing effective disease management strategies to ensure the sustainability of banana production. However, the researcher did not recognize that intercropping banana with non-host plants could be a means of controlling vector transmission.

Horub et al. (2017) developed SI model of BXW disease in order to investigate the dynamics and regulation of vector transmission. The results of the investigation demonstrated that the BXW transmission dynamics were affected by minimizing the contact between the vector and the banana plant by promptly removing the male bud with a forked stick. The study also demonstrated that increasing rogueing and healthy sucker planting rates can reduce the dynamics of BXW transmission. Nevertheless, the study failed to account for the use of non-host plants in mixed-cropping as a means of reducing Xcm bacterial vector transmission.

In order to investigate the effectiveness of debudding and roguing in preventing the spread of BXW infection in a plantation with several cultivars, Nakakawa et al. (2017) created a mathematical model known as SI. Their study's findings showed that frequent debudding, tool disinfection, and roguing all of which serve as control measures significantly reduce the amount of diseased banana plants. The study also showed that the rate of infection increases quickly and finally reaches a stable and widespread infection condition if these control measures are stopped when the infection rate is less than 1%. Consequently, even in times when infection levels appear to be small, the researchers advised giving priority to ongoing monitoring.

E. H. Kweyunga et al. (2018) studied the transmission of BXW by developing a mathematical model that included infection pressures from both symptomatic and asymptomatic banana plants. The study looked at transmission mechanisms that are both vertical and horizontal. The results showed that the basic reproduction number, which indicates the disease's spread, was mostly determined by metrics associated with asymptomatic infected plants. The researchers suggested concentrating on the observation and care of infected banana plants that has no symptoms in order to effectively control the disease. However, it is important to note that the study did not consider the impact of contaminated soil and vector transmission in the dynamics of BXW disease which is control by mixed-cropping method.

The study conducted by Mapinda (2020) focused on the development and analysis of a deterministic mathematical model for BXW disease. The primary objective was to gain insights into the transmission dynamics of BXW disease by taking contaminated soil into consideration. The main tasks of the study are formulating and analyzing a basic model for BXW that incorporates contaminated soil and also formulating and analyzing a mathematical model that includes participatory community education programs, clearance of Xcm bacteria in the soil, removal of single diseased stems, and control measures for vertical transmission. The study aims to explored the impact of these factors on the transmission dynamics of

BXW disease and provide a better understanding of the disease's spread and potential control strategies. However, the study did not mention how to clear contaminated soil.

In a study by Mapinda et al. (2022), a mathematical model was developed and analyzed to gain insights into the dynamics and control of BXW disease. The researchers conducted simulations to assess the impact of different strategies, including community education programs, soil bacteria clearance, removal of diseased stems, and transmission control. The study recommended enhancing farmer education on disease detection and management strategies, in addition to the existing control measures of sterilizing farming tools, removing male buds promptly, and planting healthy suckers. Furthermore, it emphasized the need for more effective efforts in eliminating Xcm bacteria from the soil to prevent the persistence of the disease on farms. Despite this, in addition to other control strategies, mixed-cropping is an essential method for control BXW disease dynamics.

Petsakos et al. (2023) analyzed the potential impacts of Banana Xanthomonas Wilt (BXW) on banana production, demand, and food security in Sub-Saharan Africa. They combined a mathematical model of BXW spread with an economic model and analyzed three scenarios based on policy response and farmer adoption of management practices. The results showed that if left uncontrolled, BXW could decrease banana production , resulting in $25 billion in economic losses and 4.6% increase in the population at risk of hunger in banana-dependent countries. However, even a limited policy response can mitigate some of these consequences, and full adoption of management practices can almost completely negate the impacts. This highlights the need for coordinated policy frameworks and efforts to promote adoption of such practices, considering local conditions. Basing on the reviewed literature about Mathematical Models for BXW disease, there is no study that has consider mixed-cropping practice as a control measure of BXW.

Therefore, this study aims to develop a mathematical model which includes BXW disease dynamics in a mixed-cropping system by describing possible way by which a non-host plant can reduce the Xcm bacteria spread in the host plants. Mixed-cropping system was the main new idea included in the study to improve the understanding of the BXW disease dynamics.

CHAPTER 3

RESEARCH METHODOLOGY

This chapter describes an overview of study area, study period, source of data, data collection and analysis methodologies, mathematical procedures, and a detail description on modified model employed to fulfill the objectives of the study.

3.1 Description of Study Area

This study was conducted Adama, East Shewa, Oromia region, Ethiopia. Adama a city situated 100 kilometers from the capital city of Addis Ababa is located with in the East shewa zone of Oromia regon. The city sits between the Great Rift Valley to the east and the base of a mountain to the west.

3.2 Study Period

The study carried out from September, 2023 - June, 2024 under Adama Science and Technology University School of Applied Natural Science Department of Mathematics for partial fulfilment of the requirement for the Degree of Master's of Science in Applied Mathematics.

3.3 Source of Data

The applicable sources of information for this study are FAO, books, published journals, and related studies from the Internet are used.

3.4 Data Analysis

In the proposed model analysis, we focused on the mathematical and statical analysis of the datasets employing computational techniques and a numerical algorithm. We used Range Kutta method with in the MATLAB software to scrutinize the data. The ensuing results visually depicted in graphs to probe the influence of the model parameters on the prevalence of Xcm with in the banana population.

3.5 Mathematical Procedure

In this study, we constructed a mathematical model using a system of non-linear differential equations that describe the dynamics of Banana xanthomonas wilt disease. Firstly, we obtained the model's behavior in order to develop a better understanding the dynamics of the suggested model. Next, we shown that a model is well-posed within the region that is biologically feasible. The equilibrium points of the model were analyzed for both local and global stability using Jacobian matrix and Lyapunov function, respectively. Finally, we plot numerical simulations using an iterative forth-order Runge-Kutta integration scheme to support the analytical results by carefully selected the system parameters values with MATLAB software.

CHAPTER 4

MODEL FORMULATION AND ANALYSIS

4.1 Model Description and Formulation

Here, a new compartmental model for the transmission dynamics of BXW disease in banana crop population is considered by taking into account a inter-cropping control measure and Xcm contaminated soil. The model includes vector-mediated, contact and soil-born transmissions. The following assumptions are taken to formulate the model:
The banana plant (host) population is categorized into two segments: susceptible banana plants ($H(t)$), and infected banana plants ($I(t)$), therefore the total population of banana plant ($B(t)$) at time t is $B(t) = H(t) + I(t)$ per day time t scale. the vectors population ($V(t)$) is categorized into two segments: Susceptible vectors (S), and Infected vectors (Y) hence, the total vector population at time t is given by $V(t) = S(t) + Y(t)$ per day time t scale. Contaminated soil with Xcm bacteria is represented by $C(t)$. The bacteria does not affect the non-host plant N, and N remains constant throughout the growth cycle of banana crop, and distributed uniformly across the banana crop field per day time t scale. Susceptible and infected banana plants can be harvested with rate ψ. Infected banana plants can suffer disease-induced death at rate d. Also, infected plants can be removed from the crop field at a rate g due to roguing cultural control practice. The population of susceptible banana plant grows logistically in the direction of the environmental carrying capacity K via planting where initial carrying capacity at a banana plantation rate of Λ and from real world observation and (Rutikanga, Sivirihauma, et al., 2016), because of largeness of banana plant $3K/2$ number of non-host plants in the field to best control of disease transmission. Vectors are recruit in the host crop field at a rate b_v , vectors do not die from bacterial infection but after 3-5 days contaminated vectors clear naturally η and move away from the field with rate m. An infected vector may loss its infectivity due to contact with a non-host (mixed-cropped) plant with probability β_1 being attracted with a rate a_3. The soil can be contaminate with the rate θ and after a long period of time it can be clear naturally with the rate μ. Also, contaminated soil can be cleared by planting non-host plants with rate a_4. Based on the above assumptions, susceptible banana plant acquire Xcm bacteria by contact with infected vector and contaminated soil at rate λ_1, and susceptible vector acquire Xcm bacteria by contact with infected banana plant at rate λ_2.

Table 4.1: Notation and Description of the Model State Variables.

Variables	Description
$H(t)$)	Susceptible banana plants
$I(t)$	Infected banana plants
$C(t)$	Contaminated soil with Xcm bacteria
$S(t)$	Susceptible vectors
$Y(t)$	Infected vectors

Table 4.2: Notation and Description of the Model Parameters Per A day.

Parameters	Description
Λ	Recruitment rate of susceptible Banana suckers
b_v	Rate at which susceptible vectors are obtained
ψ	Harvesting rate of matured host plants
g	Rate of removing infected banana plant from the farm
d	Disease induced death rate of an infected banana plant
m	Death rate of the vectors
η	The rate at which contaminated vectors naturally recover
μ	The rate at which Xcm bacteria naturally disappear from the soil
ω_1	The rate that the spread of Xcm bacteria from Y to H
ω_2	The rate that Xcm bacteria spread from C to H
ω_3	The rate of a contact causing Xcm bacteria transfer from I to S
N	Total number of non-host plant in banana plant field
a_1	The ability of vectors to reach H effectively
a_2	The ability of vectors to reach I effectively
a_3	Proportion of vectors move towards N
a_4	Rate of Xcm bacteria losses from C by planting N
n_1	Time spent by Y searching for H due to the interference of N
n_2	Time spent by S searching for I due to the interference of N
β_1	The rate of Xcm bacteria losses from Y by planting N
θ	Rate of Xcm bacterial shedding from I to the soil
K	Field's carrying capacity for banana plants

Figure 4.1 shows the flow diagram, which summarizes the previous assumption. The black lines in the model's schematic diagram below denote the natural mortality rate of vectors, the recruitment and harvesting rates, the natural clearance rates of Xcm bacteria from contaminated soil, and the transitions between infection stages. Normal interactions between various compartments are represented by double dash dot (rounded) lines, while the line of dash shows the release of Xcm bacteria onto the soil (G. V. Nakato et al., 2019).
Where $\lambda_1 = \frac{a_1\omega_1 Y}{1+a_3 n_1 NY} + \frac{\omega_2 C}{1+a_4 NC}$ and $\lambda_2 = \frac{a_2\omega_3 I}{1+a_3 n_2 NS}$

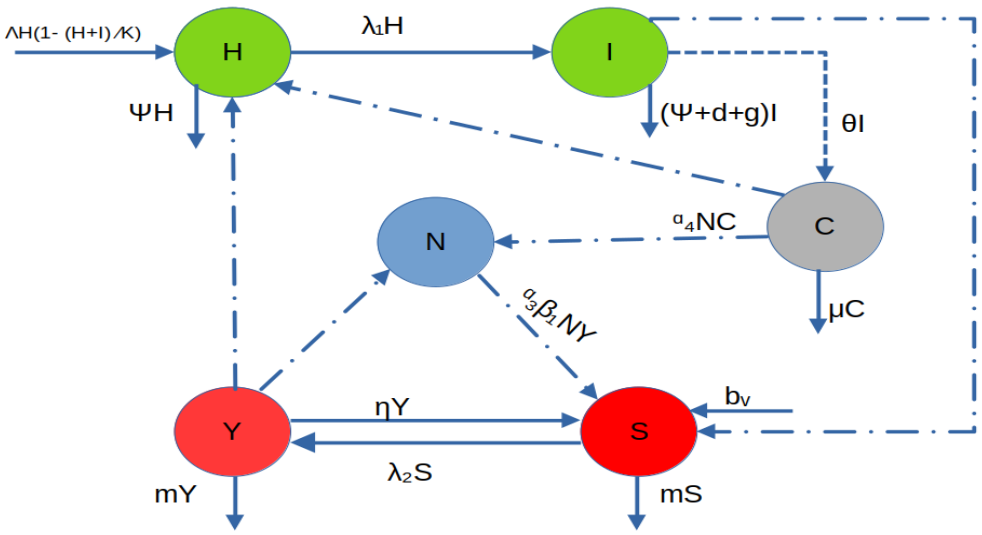


Figure 4.1: Schematic diagram of the BXW disease dynamics model.

Depending on the above model description and flow diagram, the governing mathematical model can be formulated as follows:

$$\frac{dH}{dt} = \Lambda H\left(1 - \frac{H+I}{K}\right) - \lambda_1 H - \psi H, \tag{4.1}$$

$$\frac{dI}{dt} = \lambda_1 H - (d+g+\psi)I, \tag{4.2}$$

$$\frac{dC}{dt} = \theta I - \mu C - a_4 NC, \tag{4.3}$$

$$\frac{dS}{dt} = b_v + \eta Y + a_3\beta_1 NY - \lambda_2 S - mS, \tag{4.4}$$

$$\frac{dY}{dt} = \lambda_2 S - \eta Y - mY - a_3\beta_1 NY. \tag{4.5}$$

With non-negative initial conditions, $H(0) > 0$, $I(0) \geq 0$, $C(0) \geq 0$, $S(0) \geq 0$, $Y(0) \geq 0$.
The equations of the total population of banana plants and total population of vectors respectively are given by:

$$\frac{dB}{dt} = \Lambda H\left(1 - \frac{B}{K}\right) - \psi B - (d+g)I, \tag{4.6}$$

$$\frac{dV}{dt} = b_v - mV. \tag{4.7}$$

4.2 Model Analysis

4.2.1 Invariant Region

The dynamical system of equations (4.1 - 4.5) is to be mathematically well-posed and epidemiological meaningful if the solution of the system with non negative initial data remain positive and bounded for all $t \geq 0$.

The total population of banana plant at time t given by:

$$B(t) = H(t) + I(t),$$

Then, differentiating B with respect to time we obtain equation (4.6)

$$\frac{dB}{dt} = \Lambda H\left(1 - \frac{B}{K}\right) - \psi B - (d+g)I \leq \Lambda H\left(1 - \frac{B}{K}\right) \leq \Lambda B\left(1 - \frac{B}{K}\right),$$

from this

$$\frac{dB}{dt} \leq \Lambda B\left(1 - \frac{B}{K}\right),$$

integrating both sides of this differential inequality from 0 to t,

$$B(t) \leq K,$$

as t $\rightarrow \infty$, it is clear that the total population of banana plants B(t) is bounded, i.e., the sub populations H(t), I(t) are bounded.
Next, the total population of vectors at time t given by:

$$V(t) = S(t) + Y(t),$$

Then, differentiating V with respect to time we obtain equation (4.7). From equation (4.7) it follows that,

$$\frac{dV}{dt} + mV \leq b_v,$$

then integrate by using integrating factor from 0 to t.

$$V(t) \leq V(0)e^{-mt} - \frac{b_v}{m}e^{-mt} + \frac{b_v}{m},$$

from this we see that as $t \rightarrow \infty$

$$V(t) \leq \frac{b_v}{m},$$

this shows that the total population of vector V (t) is bounded, that means the sub populations S(t), Y(t) are bounded.

Furthermore,

$$\frac{dC}{dt} \leq \theta I \leq \theta K.$$

Integrating both sides of equation, then we have as $t \to \infty$, $C(t) < \infty$. it is proved that C(t) is bounded. Hence, let Ω be a positive region in $\mathbb{R}^5_+$;

$$\begin{aligned}\Omega = \Big\{ & (H, I, C, S, Y) \in \mathbb{R}^5_+; \;\; H(0) > 0, I(0) \geq 0, C(0) \geq 0, S(0) \geq 0, Y(0) \geq 0, \\ & \Big| \; 0 < H \leq K, \; 0 \leq I \leq K, \;\; 0 \leq S, Y \leq \frac{b_\nu}{m}, C < \infty \Big\},\end{aligned} \tag{4.8}$$

then the solution H(t), I(t), C(t), S (t), Y(t) of the non-linear system of differential equations (4.1 - 4.5) above will enter and remain in Ω.

4.2.2 Non-negativity of the Solution

Theorem 4.2.1 (Non-negativity of the Solution)**.** *If all initial conditions are non-negative i.e, $H(0) > 0$, $I(0) \geq 0$, $C(0) \geq 0$, $S(0) \geq 0$ and $Y(0) \geq 0$, then the solutions H(t), I(t), C(t), S(t) and Y(t) of the system (4.1-4.5) are non-negative for all $t \geq 0$.*

Proof. First, let us take equation (4.1) from system of differential equation (4.1-4.5)

$$\frac{dH}{dt} = \Lambda H \left(1 - \frac{H+I}{K}\right) - \lambda_1 H - \psi H.$$

From the first equation (4.1) we see that

$$\frac{dH}{dt} \geq -\psi H,$$

then integrating both sides of this differential inequality from 0 to t, we have

$$H(t) \geq H(0) e^{-\psi t},$$

which implies that $H(t) \geq 0$ for all $t \geq 0$. From the second equation (4.2) we see that

$$\frac{dI}{dt} \geq -(d + g + \psi) I,$$

then integrating both sides of this differential inequality from 0 to t, we have
I(t) $\geq$ I(0) $e^{-(d+g+\psi)t}$, which implies that $I(t) \geq 0$ for all $t \geq 0$.
Since

$$\frac{dC(t)}{C(t)} \geq -\mu d(t), \quad \frac{dS(t)}{S(t)} \geq -m d(t), \quad \frac{dY(t)}{Y(t)} \geq -\eta d(t),$$

using a similar technique,

$$C(t) \geq C(0)e^{-\mu t}, S(t) \geq S(0)e^{-mt}, Y(t) \geq Y(0)e^{-\eta t},$$

show that, $C(t) \geq 0$, $S(t) \geq 0$ and $Y(t) \geq 0$ for all time $t \geq 0$. □

The solution of the system with non-negative initial data remain positive and bounded for all $t \geq 0$. There fore, the model system (4.1-4.5) is epidemiological meaningful and mathematically well-posed in Ω. Hence, it is sufficient to study the dynamic properties of the system (4.1-4.5) in Ω.

4.2.3 Disease Free Equilibrium Point

The point at which the dynamical system (4.1-4.5) equal zero are referred to as equilibrium points or fixed points or stationary points or steady state solutions.
Disease free equilibrium point (DFEP) is the steady state solutions where there is no BXW disease i.e $I = C = Y = 0$. Thus the disease free equilibrium point E_0 for dynamical system (4.1-4.5) is given by:

$$E_0 = \left(\frac{bK}{\Lambda}, 0, 0, \frac{b_v}{m}, 0\right)$$

When $b = \Lambda - \psi$ is always positive over maximum age of banana tree (Bhende & Kurien, 2016; Kanchana et al., 2021).

4.2.4 Basic Reproduction Number

The basic reproduction number ($\mathscr{R}_0$) is defined as the expected number of secondary cases produced by a single primary case in a completely susceptible population. $\mathscr{R}_0$ is an important tool to determine whether the disease persist in the population or not. To compute $\mathscr{R}_0$, we use the method of next generation matrix (Van den Driessche & Watmough, 2002).
The infected compartments are

$$\frac{dI}{dt} = \lambda_1 H - (d + g + \psi)I, \tag{4.9}$$

$$\frac{dC}{dt} = \theta I - \mu C - a_4 NC, \tag{4.10}$$

$$\frac{dY}{dt} = \lambda_2 S - \eta Y - mY - a_3\beta_1 NY. \tag{4.11}$$

The next generation matrix is constructed by partitioning the system of differential equations describing disease transmission into terms representing the production of new infections ($\mathscr{F}$) and the transitions between compartments ($\mathscr{V}$).

Now the system equation (4.9-4.11) is given by $\mathscr{F}$ - $\mathscr{V}$, where

$$\mathscr{F}(x,y)=\begin{bmatrix}\lambda_1 H\\ 0\\ \lambda_2 S\end{bmatrix},\quad \mathscr{V}(x,y)=\begin{bmatrix}(d+g)I+\psi I\\ \mu C+a_4CN-\theta I\\ \eta Y+mY+a_3\beta_1 NY\end{bmatrix}.$$

The Jacobian matrices of $\mathscr{F}(x)$ and $\mathscr{V}(x)$ at the disease-free equilibrium (E_0) are

$$D\mathscr{F}(E_0)=F=\begin{bmatrix}0 & \frac{\omega_2 bK}{\Lambda} & \frac{a_1\omega_1 bK}{\Lambda}\\ 0 & 0 & 0\\ \frac{a_2\omega_3 b_v}{m+a_3n_2Nb_v} & 0 & 0\end{bmatrix},\quad D\mathscr{V}(E_0)=V=\begin{bmatrix}d+g+\psi & 0 & 0\\ -\theta & \mu+a_4N & 0\\ 0 & 0 & \eta+m+a_3\beta_1N\end{bmatrix},$$

$$\text{from this } V^{-1}=\begin{bmatrix}\frac{1}{d+g+\psi} & 0 & 0\\ \frac{\phi}{(d+g+\psi)(\mu+a_4N)} & \frac{1}{\mu+a_4N} & 0\\ 0 & 0 & \frac{1}{\eta+m+a_3\beta_1N}\end{bmatrix}.$$

The basic reproduction number, $\mathscr{R}_0=\rho(FV^{-1})$, is the maximum spectral radius of the product FV^{-1}, where

$$FV^{-1}=\begin{bmatrix}\frac{\theta\omega_2 bK}{\Lambda(d+g+\psi)(\mu+a_4N)} & \frac{\omega_2 bK}{\Lambda(\mu+a_4N)} & \frac{a_1\omega_1 bK}{\Lambda(\eta+m+a_3\beta_1N)}\\ 0 & 0 & 0\\ \frac{a_2\omega_3 b_v}{(m+a_3n_2Nb_v)(d+g+\psi)} & 0 & 0\end{bmatrix}.$$

Then the basic reproduction number of the model system (4.1-4.5) at E_0 is

$$\mathscr{R}_0=R_1+\sqrt{R_1^2+R_2}, \tag{4.12}$$

$$where\qquad R_1=\frac{\theta\omega_2 Kb}{2\Lambda(d+g+\psi)(\mu+a_4N)},\ R_2=\frac{a_1\omega_1 a_2\omega_3 b_v Kb}{\Lambda(m+a_3n_2Nb_v)(d+g+\psi)(\eta+m+a_3\beta_1N)},$$

- R_1: Average number of secondary infection from contaminated soil.
- R_2: Average number of secondary infection from contaminated vectors.

Without intercropping system ($N=0$), the basic reproduction number will be $\mathscr{R}_{01}>\mathscr{R}_0$. The fact that $\mathscr{R}_{01}>\mathscr{R}_0$ is interpreted as the role of planting non-host plant (N) as interference with the Xcm bacteria transmission process.

4.2.5 Local Stability of Disease Free Equilibrium

Theorem 4.2.2. *The DFEP E_0 of the system (4.1-4.5) is locally asymptotically stable if $\mathscr{R}_0 < 1$. Otherwise it is unstable if $\mathscr{R}_0 > 1$.*

Proof. The Jacobian matrix associated to these state variables of model system (4.1 - 4.5) is

$$J = \begin{bmatrix} \Lambda - \frac{\Lambda(2H+I)}{K} - \lambda_1 - \psi & \frac{-\Lambda H}{K} & \frac{-\omega_2 H}{1+a_4 NC} & 0 & \frac{-a_1\omega_1 H}{1+a_3 n_1 NY} \\ \lambda_1 & -(d+g+\psi) & \frac{\omega_2 H}{1+a_4 NC} & 0 & \frac{a_1\omega_1 H}{1+a_3 n_1 NY} \\ 0 & \theta & -\mu - a_4 N & 0 & 0 \\ 0 & \frac{-a_2\omega_3 S}{1+a_3 n_2 NS} & 0 & -m & \eta + a_3\beta_1 N \\ 0 & \frac{a_2\omega_3 S}{1+a_3 n_2 NS} & 0 & 0 & -\eta - m - a_3\beta_1 N \end{bmatrix}.$$

Then associated Jacobain matrix at disease free equilibrium point E_0 is

$$J(E_0) = \begin{bmatrix} -b & -b & \frac{-\omega_2 Kb}{\Lambda} & 0 & \frac{-a_1\omega_1 Kb}{\Lambda} \\ 0 & -(d+g+\psi) & \frac{\omega_2 Kb}{\Lambda} & 0 & \frac{a_1\omega_1 Kb}{\Lambda} \\ 0 & \theta & -\mu - a_4 N & 0 & 0 \\ 0 & \frac{-a_2\omega_3 b_v}{m+a_3 n_2 Nb_v} & 0 & -m & \eta + a_3\beta_1 N \\ 0 & \frac{a_2\omega_3 b_v}{m+a_3 n_2 Nb_v} & 0 & 0 & -\eta - m - a_3\beta_1 N \end{bmatrix}.$$

The eigenvalues of the matrix $J(E_0)$ are the roots of the characteristic polynomial $det(J(E_0) - \lambda I)$. It is easy to see that $-b$ and $-m$ are negative eigenvalues. The remaining three eigenvalues are the eigenvalues of the 3 x 3 matrix

$$\begin{bmatrix} -(d+g+\psi) - \lambda & \frac{\omega_2 Kb}{\Lambda} & \frac{a_1\omega_1 Kb}{\Lambda} \\ \theta & -\mu - a_4 N - \lambda & 0 \\ \frac{a_2\omega_3 b_v}{m+a_3 n_2 Nb_v} & 0 & -\eta - m - a_3\beta_1 N - \lambda \end{bmatrix}.$$

The characteristic equation takes the form

$$\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0, \tag{4.13}$$

where

$$
\begin{aligned}
a_1 =& (d+g+\psi)+(\eta+m+a_3\beta_1 N)+(\mu+a_4 N),\\
a_2 =& (\mu+a_4 N)(d+g+\psi)+(\eta+m+a_3\beta_1 N)(\mu+a_4 N)+(\eta+m+a_3\beta_1 N)(d+g+\psi)\\
&-\frac{a_1\omega_1 a_2\omega_3 b_v bK}{\Lambda(m+a_3 n_2 N b_v)}-\frac{\theta\omega_2 bK}{\Lambda},\\
a_3 =& (\mu+a_4 N)(d+g+\psi)(\eta+m+a_3\beta_1 N)-(\mu+a_4 N)\frac{a_1\omega_1 a_2\omega_3 b_v bK}{\Lambda(m+a_3 n_2 N b_v)}\\
&+(\mu+a_4 N)(d+g+\psi)(\eta+m+a_3\beta_1 N)-\frac{\theta\omega_2 bK}{\Lambda}(\eta+m+a_3\beta_1 N).
\end{aligned}
$$

To determine the sign of the remaining three eigen values we apply Routh Hurwitz criteria (Merkin, 2012) for the characteristic polynomial equation (4.13) with n = 3. Thus, the roots of the (4.13) will have negative real parts if we have

- $a_1 > 0$; Moreover
- $a_2 = (\eta+m+a_3\beta_1 N)(\mu+a_4 N)+(\mu+a_4 N)(d+g+\psi)(1-2R_1)+(\eta+m+a_3\beta_1 N)(d+g+\psi)(1-R_2) \Longrightarrow a_2 > 0$ holds if $R_1 < \frac{1}{2}$, $R_2 < 1$, and $R_2 < 1-2R_1$ generally if $\mathscr{R}_0 < 1$.
- $a_3 = (\mu+a_4 N)(d+g+\psi)(\eta+m+a_3\beta_1 N)(1-R_2)+(\mu+a_4 N)(d+g+\psi)(\eta+m+a_3\beta_1 N)(1-2R_1) \Longrightarrow a_3 > 0$ holds if $R_2 < 1$, $R_1 < \frac{1}{2}$, and $R_2 < 1-2R_1$ generally if $\mathscr{R}_0 < 1$.
- For the condition, $a_1 a_2 > a_3 \Longrightarrow a_1 a_2 - a_3 > 0$.
  For more expression: where $Q_1 = d+g+\psi$, $Q_2 = \eta+m+a_3\beta_1 N$, $Q_3 = \mu+a_4 N$,
  $a_1 a_2 > a_3 \Longrightarrow \big(Q_1+Q_2+Q_3\big)\big(Q_2Q_3+Q_1Q_3(1-2R_1)+Q_1Q_2(1-R_2)\big)$
  $= Q_2Q_3(Q_1+Q_2+Q_3)+Q_1Q_2Q_3(1-2R_1)+Q_1Q_3(Q_1+Q_3)(1-2R_1)+Q_1Q_2Q_3(1-R_2)+Q_1Q_2(Q_1+Q_2)(1-R_2) > Q_1Q_2Q_3(1-R_2)+Q_1Q_2Q_3(1-2R_1)$
  $\Longrightarrow Q_2Q_3(Q_1+Q_2+Q_3)+Q_1Q_3(Q_1+Q_3)(1-2R_1)+Q_1Q_2(Q_1+Q_2)(1-R_2) > 0$
  $\Longrightarrow a_1 a_2 - a_3 > 0$ holds if $R_2 < 1$, $R_1 < \frac{1}{2}$, and $R_2 < 1-2R_1$ generally if $\mathscr{R}_0 < 1$.

For $\mathscr{R}_0 < 1$, this suggests that the remaining three eigenvalues are also distinct negative real numbers. Thus, when $\mathscr{R}_0 < 1$, the disease free equilibrium point E_0 of the ordinary differential equation system (4.1-4.5) is locally asymptotically stable. □

4.2.6 Global Stability of Disease Free Equilibrium

To examine the global asymptotic stability of E_0, we apply the approach of (Castillo-Chavez, 2002). First, rewrite the system as follows:

$$
\begin{aligned}
\frac{dX}{dt} &= F(X,Z), \\
\frac{dZ}{dt} &= G(X,Z), \qquad G(X,0) = 0.
\end{aligned}
\tag{4.14}
$$

where X stands for the uninfected population, that is $X = (H, S)$, and Z stands for the infected population, that is $Z = (I, C, Y)$. Let disease-free equilibrium point of the model is denoted by $E_0 = (X^*, 0)$.

For the point $E_0 = (X^*, 0)$ to be globally asymptotically stable equilibrium for the system (4.14) provided satisfies the following conditions **(H1)** and **(H2)** below:

H1: X^* is globally asymptotically stable for $\frac{dX}{dt} = F(X, 0)$,

H2: $G(X, Z) = AZ - \widehat{G}(X, Z), \widehat{G}(X, Z) \geq 0$ for $(X, Z) \in \Omega$,

where A derivative of $G(X, Z)$ in the direction of Z at $(X^*, 0)$ is an Metzler matrix and Ω is the region where the model makes biological sense. If system (4.1-4.5) satisfies the above two conditions then the following theorem holds:

Theorem 4.2.3. *If $\mathscr{R}_0 < 1$ and conditions (**H1**) and (**H2**) are satisfied, then equilibrium point $E_0 = (X^*, 0)$ of system (4.14) is globally asymptotically stable.*

Proof. From (4.1 - 4.5) we have,

$$F(X, Z) = \begin{bmatrix} \Lambda H\left(1 - \frac{H+I}{K}\right) - \lambda_1 H - \psi H \\ b_v + \eta Y + a_3\beta_1 NY - \lambda_2 S - mS \end{bmatrix},$$

$$G(X, Z) = \begin{bmatrix} \lambda_1 H - (d + g + \psi) I \\ \theta I - \mu C - a_4 NC \\ \lambda_2 S - \eta Y - mY - a_3\beta_1 NY \end{bmatrix}.$$

Consider the reduced system $\frac{dX}{dt} = F(X, 0)$, it is clear that

$$\frac{dX}{dt} = \begin{bmatrix} \Lambda H(1 - \frac{H}{K}) - \psi H \\ b_v - mS \end{bmatrix}.$$

Which implies that H(t)= $\frac{-K(\Lambda-\psi)}{e^{\psi(t+H_0K)}e^{-\Lambda(t+H_0K)}-\Lambda}$ as $t \to \infty$ independent of initial conditions. Therefore, $H(t) \to \frac{bK}{\Lambda}$ globally in Ω and $S(t) = \frac{b_v}{m} + (S(0) - \frac{b_v}{m})e^{-mt}$. As $t \to \infty$, the solution $S(t) \to \frac{b_v}{m}$, implying the global convergence of $F(X, 0)$ in Ω. Next, we need to show that $\widehat{G} \geq 0$ for all $(X, Z) \in \Omega$. Then,

$$G(X, Z) = AZ - \widehat{G}(X, Z),$$

where

$$A(X^*, 0) = \begin{bmatrix} -(d + g + \psi) & \frac{\omega_2 Kb}{\Lambda} & \frac{a_1\omega_1 Kb}{\Lambda} \\ \theta & -\mu - a_4 N & 0 \\ \frac{a_2\omega_3 b_v}{m + a_3 n_2 N b_v} & 0 & -\eta - m - a_3\beta_1 N \end{bmatrix},$$

it can written as,

$$\widehat{G} = \begin{bmatrix} \frac{\omega_2 Cb}{\Lambda}(K - \frac{H}{\nu_1}) + \frac{\omega_1 a_1 Y b}{\Lambda}(K - \frac{H}{\nu_2}) \\ 0 \\ \nu_3(\frac{b_\nu}{m} - S) \end{bmatrix},$$

where, $\nu_1 = \frac{b(1+a_4 NC)}{\Lambda}$, $\nu_2 = \frac{b(1+a_3 n_1 NY)}{\Lambda}$ and $\nu_3 = \frac{a_3 \omega_2 I}{(1+a_3 n_2 NS)(1+a_3 n_2 N \frac{b_\nu}{m})}$.

Since $K \geq H$, $\frac{b_\nu}{m} \geq S$, we get $\widehat{G}(X, Z) \geq 0$ for all $(X, Z) \in \Omega$. And also the matrix A is an M-matrix as all of its off-diagonal elements are non-negative. Which means the system (4.14) satisfies the conditions **(H1)** and **(H2)**. Therefore, equilibrium point $E_0 = (X^*, 0)$ of system (4.14) is globally asymptotically stable. □

4.2.7 Endemic Equilibrium Point

An endemic equilibrium E^* of system (4.1-4.5) represents the steady state solution that occurs when the disease continues to exist. That means,

$E^* = (H^*, I^*, C^*, S^*, Y^*)$; $H^* > 0, I^* > 0, C^* > 0, S^* > 0, Y^* > 0$. It is obtained by equating the right-hand sides of every equation in (4.1-4.5) to zero and then solving the resulting equations.

$$\begin{aligned} 0 &= \Lambda H\left(1 - \frac{H+I}{K}\right) - \lambda_b H - \psi H, \\ 0 &= \lambda_b H - (d + g + \psi) I, \\ 0 &= \theta I - \mu C - a_1 NC, \\ 0 &= b_\nu + \eta Y + a_3 \beta_1 NY - \lambda_\nu S - mS, \\ 0 &= \lambda_\nu S - \eta Y - mY - a_3 \beta_1 NY. \end{aligned} \tag{4.15}$$

Solving the system (4.15) for endemic equilibrium point, $E^* = (H^*, I^*, C^*, S^*, Y^*)$, where

$$\begin{aligned} H^* &= \frac{K(d+g+\psi)\left(\Lambda - \psi - {\lambda_b}^*\right)}{\Lambda\left({\lambda_b}^* + d + g + \psi\right)}, \\ I^* &= \frac{{\lambda_b}^* K\left(\Lambda - \psi - {\lambda_b}^*\right)}{\Lambda\left({\lambda_b}^* + d + g + \psi\right)}, \\ C^* &= \frac{\theta K\left(\Lambda - \psi - {\lambda_b}^*\right){\lambda_b}^*}{\Lambda\left(\mu + Na_4\right)\left({\lambda_b}^* + d + g + \psi\right)}, \\ S^* &= \frac{b_\nu\left(m + \eta + Na_3\beta_1\right)}{m\left(m + \eta + Na_3\beta_1 + {\lambda_\nu}^*\right)}, \\ Y^* &= \frac{b_\nu {\lambda_\nu}^*}{m\left(m + \eta + Na_3\beta_1 + {\lambda_\nu}^*\right).} \end{aligned} \tag{4.16}$$

$provided\ that\ b > {\lambda_b}^*$.

As a result, system (4.1-4.5) has a unique endemic equilibrium whenever $R_0 > 1$.

4.2.8 Global Stability Endemic Equilibrium

In this study to determine global stability of the endemic equilibrium point (E^*) we use Lyapunov function (4.17), this function is well-known by the ecologists as the first integral and Lyapunov function for the Lotka-Volterra prey-predator system (Goh, 2012; Takeuchi, 1996) , and it has also been successfully applied to epidemiological models (Korobeinikov & Maini, 2004; Korobeinikov & Wake, 2002). Which constructed using the formula in,

$$V = \sum_{i=1}^{n} (x_i - x_i^* \ln x_i). \tag{4.17}$$

Where x_i^* are the endemic equilibrium points, x_i are state variables, and n is number of state variables. Hence in this study the lyapunov function is given by:

$$\begin{aligned} V =& (H - H^* \ln H) + (I - I^* \ln I) + (C - C^* \ln C) + (S - S^* \ln S) \\ &+ (Y - Y^* \ln Y). \end{aligned} \tag{4.18}$$

Differentiating (4.18) with respect to time, gives

$$\begin{aligned} \frac{dV}{dt} =& \left(1 - \frac{H^*}{H}\right)\frac{dH}{dt} + \left(1 - \frac{I^*}{I}\right)\frac{dI}{dt} + \left(1 - \frac{C^*}{C}\right)\frac{dC}{dt} + \left(1 - \frac{S^*}{S}\right)\frac{dS}{dt} \\ &+ \left(1 - \frac{Y^*}{Y}\right)\frac{dY}{dt}, \end{aligned} \tag{4.19}$$

substituting the expression for the derivatives values from the system (4.1-4.5) in (4.19), then results to

$$\begin{aligned} \frac{dV}{dt} =& \left(1 - \frac{H^*}{H}\right)\left[\Lambda H\left(1 - \frac{H+I}{K}\right) - \frac{a_1\omega_1 YH}{1 + a_3 n_1 NY} - \frac{\omega_2 CH}{1 + a_4 NC} - \psi H\right] \\ &+ \left(1 - \frac{I^*}{I}\right)\left[\frac{a_1\omega_1 YH}{1 + a_3 n_1 NY} + \frac{\omega_2 CH}{1 + a_4 NC} - (d + g + \psi)I\right] \\ &+ \left(1 - \frac{C^*}{C}\right)\left[\theta I - (\mu + a_4 N)C\right] \\ &+ \left(1 - \frac{S^*}{S}\right)\left[b_v + \eta Y + a_3\beta_1 NY - \frac{a_2\omega_3 IS}{1 + a_3 n_2 NS} - mS\right] \\ &+ \left(1 - \frac{Y^*}{Y}\right)\left[\frac{a_2\omega_3 IS}{1 + a_3 n_2 NS} - (\eta + m + a_3\beta_1 N)Y\right], \end{aligned} \tag{4.20}$$

at endemic equilibrium point

$$\begin{aligned}\frac{dV}{dt} =&\left(1-\frac{H^*}{H}\right)\left[\frac{a_1\omega_1 Y^* H^*}{1+a_3n_1NY}+\frac{\omega_2 C^* H^*}{1+a_1NC}+\psi H^*-\frac{a_1\omega_1 YH}{1+a_3n_1NY}-\frac{\omega_2 CH}{1+a_4NC}-\psi H\right]\\&+\left(1-\frac{I^*}{I}\right)\left[(d+g+\psi)I^*-(d+g+\psi)I\right]\\&+\left(1-\frac{C^*}{C}\right)\left[(\mu+a_4N)C^*-(\mu+a_4N)C\right]\\&+\left(1-\frac{S^*}{S}\right)\left[\frac{a_2\omega_3 I^* S^*}{1+a_3n_2NS}+mS^*-\frac{a_2\omega_3 IS}{1+a_3n_2NS}-mS\right]\\&+\left(1-\frac{Y^*}{Y}\right)\left[(\eta+m+a_3\beta_1N)Y^*-(\eta+m+a_3\beta_1N)Y\right],\end{aligned}\tag{4.21}$$

which can be simplified to

$$\begin{aligned}\frac{dV}{dt} =&\left(\frac{H-H^*}{H}\right)\left[\frac{a_1\omega_1}{1+a_3n_1NY}(Y^*H^*-YH)+\frac{\omega_2}{1+a_4NC}(C^*H^*-CH)+\psi(H^*-H)\right]\\&+\left(\frac{I-I^*}{I}\right)\left[(d+g+\psi)(I^*-I)\right]\\&+\left(\frac{C-C^*}{C}\right)\left[(\mu+a_4N)(C^*-C)\right]\\&+\left(\frac{S-S^*}{S}\right)\left[\frac{a_2\omega_3}{1+a_3n_2NS}(I^*S^*-IS)+m(S^*-S)\right]\\&+\left(\frac{Y-Y^*}{Y}\right)\left[(\eta+m+a_3\beta_1N)(Y^*-Y)\right],\end{aligned}\tag{4.22}$$

by collecting the like terms

$$\begin{aligned}\frac{dV}{dt} =&-\left(\frac{H-H^*}{H}\right)\left[\psi(H-H^*)\right]-\left(\frac{I-I^*}{I}\right)\left[(d+g+\psi)(I-I^*)\right]\\&-\left(\frac{C-C^*}{C}\right)\left[(\mu+a_4N)(C-C^*)\right]-\left(\frac{S-S^*}{S}\right)\left[m(S-S^*)\right]\\&-\left(\frac{Y-Y^*}{Y}\right)\left[(\eta+m+a_3\beta_1N)(Y-Y^*)\right]\\&-\left(\frac{H-H^*}{H}\right)\left[\frac{a_1\omega_1}{1+a_3n_1NY}(YH-Y^*H^*)+\frac{\omega_2}{1+a_4NC}(CH-C^*H^*)\right]\\&-\left(\frac{S-S^*}{S}\right)\left[\frac{a_2\omega_3}{1+a_3n_2NS}(IS-I^*S^*)\right],\end{aligned}\tag{4.23}$$

finally

$$\begin{aligned}\frac{dV}{dt} =&-\left(\frac{(H-H^*)^2}{H}\right)\psi-\left(\frac{(I-I^*)^2}{I}\right)(d+g+\psi)-\left(\frac{(C-C^*)^2}{C}\right)(\mu+a_4N)\\&-\left(\frac{(S-S^*)^2}{S}\right)m-\left(\frac{(Y-Y^*)^2}{Y}\right)(\eta+m+a_3\beta_1N)+P(W),\end{aligned}\tag{4.24}$$

where

$$P(W) = -\Big(\frac{H-H^*}{H}\Big)\Big[\frac{a_1\omega_1}{1+a_3n_1NY}(YH-Y^*H^*)+\frac{\omega_2}{1+a_4NC}(CH-C^*H^*)\Big]$$
$$-\Big(\frac{S-S^*}{S}\Big)\Big[\frac{a_2\omega_3}{1+a_3n_2NS}(IS-I^*S^*)\Big] \leq 0,$$

and $W = (H, I, C, S, Y) > 0$ for all elements in W.
Therefore it is clear that $\frac{dV}{dt} \leq 0$ with equality holding only if $W = W^*$. Implying that the largest invariant set in W when $\frac{dV}{dt} = 0$ is singleton (unique) W^* which is the endemic equilibrium point. Therefore, by the LaSalle's invariance principle described in (La Salle, 1976), it means that endemic equilibrium point W^* is asymptotically stable in Ω (4.8) when $\mathscr{R}_0 > 1$ and otherwise unstable.

4.3 Sensitivity Analysis

The importance of each parameter in the transmission dynamics must be determined in order to develop potential disease management methods. The significance of each parameter for the transmission of disease can be determined by the sensitivity analysis for the basic reproduction number $\mathscr{R}_0$. We use the normalized forward sensitivity index of $\mathscr{R}_0$ Rodrigues et al. (2013) to perform this analysis and ascertain the model parameter's sensitivity. When the $\mathscr{R}_0$ be differentiable about its parameter ζ, the sensitivity index of ζ can be obtained as follows:

$$\Upsilon_{\zeta}^{\mathscr{R}_0} = \frac{\partial \mathscr{R}_0}{\partial \zeta} \times \frac{\zeta}{\mathscr{R}_0}. \tag{4.25}$$

$$\mathscr{R}_0 = \frac{1}{2}\Big(\frac{\theta\omega_2 Kb}{\Lambda(d+g+\psi)(\mu+a_4N)} + \sqrt{\Big(\frac{\theta\omega_2 Kb}{\Lambda(d+g+\psi)(\mu+a_4N)}\Big)^2 + 4x_r}\Big),$$

$$where \qquad x_r = \frac{a_1\omega_1 a_2\omega_3 b_v Kb}{\Lambda(m+a_3n_2Nb_v)(d+g+\psi)(\eta+m+a_3\beta_1N)},$$

therefore for parameter ω_2 gives,

$$\frac{\partial \mathscr{R}_0}{\partial \omega_2} = \frac{\theta Kb}{2\Lambda(d+g+\psi)(\mu+a_4N)} + \frac{\theta Kb}{\Lambda(d+g+\psi)(\mu+a_4N)} \frac{\frac{\theta\omega_2 Kb}{\Lambda(d+g+\psi)(\mu+a_4N)}}{\sqrt{(\frac{\theta\omega_2 Kb}{\Lambda(d+g+\psi)(\mu+a_4N)})^2 + 4x_r}},$$

then

$$\Upsilon_{\omega_2}^{\mathscr{R}_0} = \frac{1}{2}\frac{\theta K b}{\Lambda(d+g+\psi)(\mu+a_4N)} + \frac{\theta K b}{\Lambda(d+g+\psi)(\mu+a_4N)}\frac{\frac{\theta\omega_2 K b}{\Lambda(d+g+\psi)(\mu+a_4N)}}{\sqrt{(\frac{\theta\omega_2 K b}{\Lambda(d+g+\psi)(\mu+a_4N)})^2+4x_r}} \times \frac{\omega_2}{\mathscr{R}_0}. \tag{4.26}$$

When the sensitivity index in (4.26) is calculated using the parameter values from table (4.4),

$$\Upsilon_{\omega_2}^{\mathscr{R}_0} = 0.977544$$

We now apply (4.25) to get the sensitivity indices of the model parameter using the values in table (4.4), as the $\mathscr{R}_0$ in (4.12) is differentiable to its parameters. Sensitivity indices are the result, as shown in figure (4.2).

Table 4.3: The Normalized Forward Sensitivity Indices of $\mathscr{R}_0$ to Model Parameters.

Parameters	Description	Sensitivity indices
ω_2	The rate that Xcm bacteria spread from C to H	+0.9775
θ	Rate of Xcm bacterial shedding from I to the soil	+0.8375
Λ	Recruitment rate of susceptible banana suckers	+0.5001
K	Field's carrying capacity for banana plants	+0.0989
ω_1	The rate that the spread of Xcm bacteria from Y to H	+0.0112
ω_3	The rate of a contact causing Xcm bacteria transfer from I to S	+0.0112
a_1	The ability of vectors to reach H effectively	+0.0112
a_2	The ability of vectors to reach I effectively	+0.0112
b_v	Recruitment rate of susceptible vectors	+0.00109
μ	The rate at which Xcm bacteria naturally disappear from the soil	-8.1414×10^{-6}
η	The rate at which contaminated vectors naturally recover	-3.566×10^{-6}
m	Death rate of the vectors	−0.00109
n_2	Time spent by S searching for I due to the interference of N	−0.0101
β_1	The rate of Xcm bacteria losses from Y by planting N	−0.0112
a_3	Proportion of vectors move towards N	−0.0213
g	Rate of removing infected banana plant from the farm	−0.3165
d	Disease induced death rate of an infected banana plant	−0.5034
ψ	Harvesting rate of matured host plants	−0.611
a_4	Rate of Xcm bacteria losses from C by planting N	−0.9008
N	Total number of non host plant in banana plant field	−0.9119

Positive sensitivity index implies the direct proportionality of the corresponding parameter with basic reproduction number ($\mathscr{R}_0$). A negative sensitivity index means that the parameter is inversely proportional to the basic reproduction number ($\mathscr{R}_0$). Increasing the $\mathscr{R}_0$ implies increase in the BXW disease endemicity while decreasing $\mathscr{R}_0$ to less than one decrease the endemicity of the BXW disease.

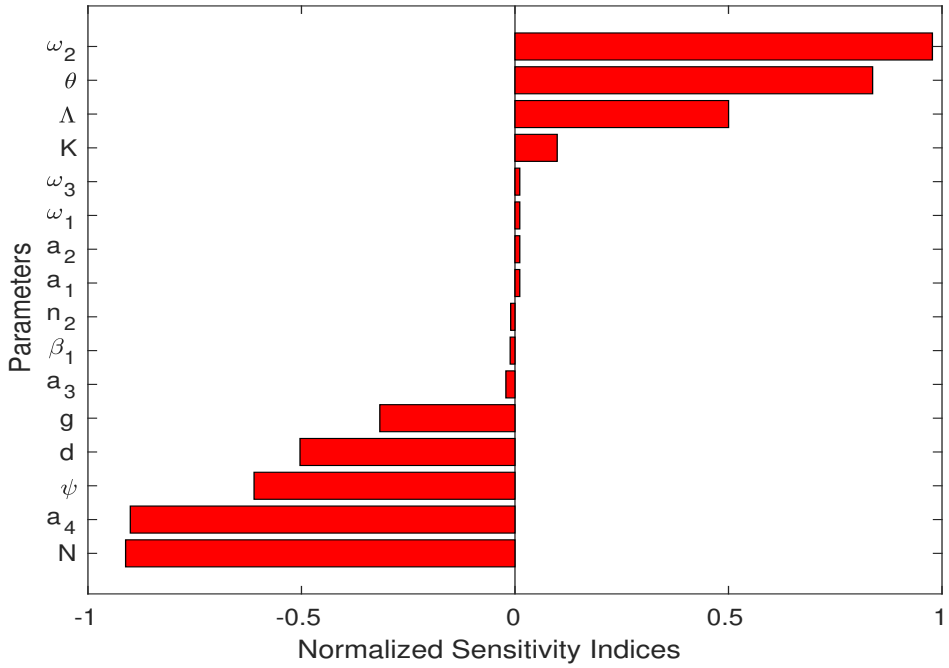


Figure 4.2: Normalized sensitivity indices of $\mathscr{R}_0$ with respect to most sensitive parameters of the model.

Figure (4.2) shows that most sensitive parameters when the parameters with positive indices ω_2, θ and Λ are increased (or decreased) keeping other parameters constant they increase (or decrease) the value of $\mathscr{R}_0$ respectively. Figure (4.2) further shows that the probability of Xcm bacteria being transmitted from contaminated soil to susceptible banana plant is most sensitive to the basic reproduction number compared to another parameters.

For instance, $\Upsilon_{\omega_2}^{\mathscr{R}_0} = 0.977544$ which means that increasing (or decreasing) the value of the parameter ω_2 by 10% increases (or decreases) the $\mathscr{R}_0$ by 9.8%.
While the parameters with negative indices are ψ, a_4, N decrease (or increase) the value of $\mathscr{R}_0$ when they increase (or decrease) respectively, while keeping the other parameters constant, implying that they decrease the endemicity of the disease. Therefore, in order to best control the disease these parameters with negative indices should be increased so as to reduce the value of the $\mathscr{R}_0$ or to reduce secondary infection.

4.4 Numerical Simulations

In this section we present numerically the behavior of the solutions of the system (4.1-4.5) to support the theoretical analysis given in the previous sections.

Table 4.4: The Values of Parameters Used in Numerical Simulations.

Parameters	Value	Range	Source
Λ	0.01667	0-1	(Nakakawa et al., 2016)
b_v	0.02	0.005-0.5	(E. Kweyunga, 2011)
N	3000	450-6000	Calculated
g	0.0105	0.000371-0.946	(E. Kweyunga, 2011)
θ	0.89	0.0375-0.943	Assumed
d	0.0167	0.00368-0.238	(E. Kweyunga, 2011)
ω_1	0.2	0.0042-0.478	(Nannyonga et al., 2015)
ω_2	0.1	0.053-0.5	Assumed
ω_3	0.2	0.0042-0.478	(Nannyonga et al., 2015)
a_1	0.3	0.00145-0.473	Assumed
a_2	0.2	0.00457-0.469	Assumed
ψ	0.0056	0-1	(E. Kweyunga, 2011)
a_3	0.1	0.0056-0.457	Assumed
a_4	0.4	0.00367-0.45	Assumed
m	0.02	0.0134-0.0467	(E. Kweyunga, 2011)
n_1	0.037	0.0071-0.045	Assumed
n_2	0.031	0.0071-0.045	Assumed
η	0.0286	0.00235-0.035	(Mapinda, 2020)
β_1	0.3	0.01-0.4391	Assumed
K	2000	300-4000	Assumed
μ	0.01	0.002-0.05	(Mapinda, 2020)

From Fig.4.3, it is noticed that when a plant becomes infected with BXW disease, its number of susceptible plants drops off dramatically. In the initial months after infection, there is a rise in the number of infected banana plants. It is also clear that, in the absence of control measures, all banana plants will get infected a few months after the bacterium first occurs. This implies according to Nakakawa et al. (2017) with out any control method BXW can cause up to 100% yield losses.

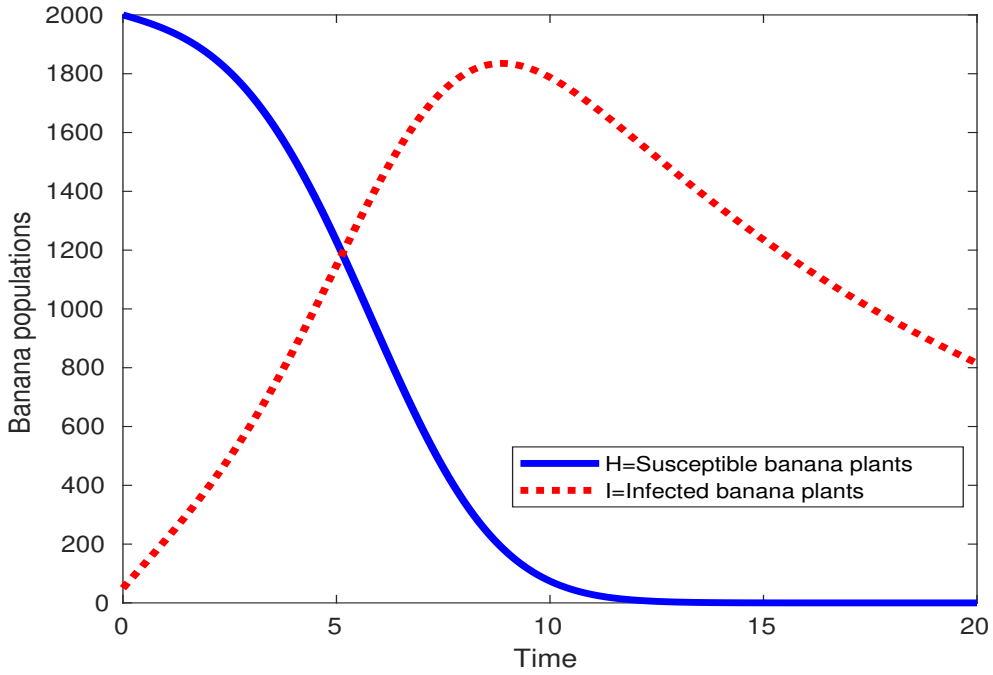
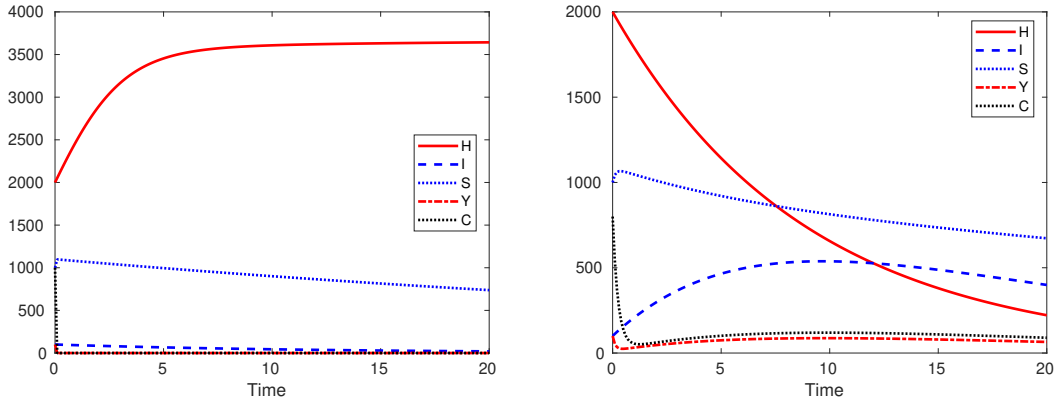


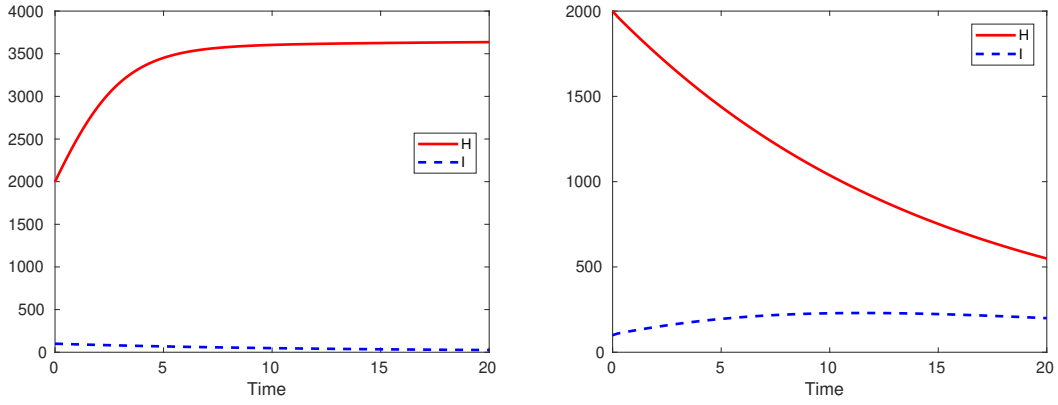
Figure 4.3: Banana population dynamics with out control measures.

Figure 4.4 depicted the global stability of the disease-free and endemic equilibrium points of the BXW disease model under different parameter values. (a) The disease-free equilibrium point is globally asymptotically stable when: $\theta = 0.89,\ K = 4000,\ \Lambda = 0.01667,\ d = 0.0167,\ g = 0.0105,\ \psi = 0.0056,\ \mu = 0.01,\ N = 6000,\ \omega_1 = 0.2,\ \omega_2 = 0.02,\ \omega_3 = 0.2,\ a_1 = 0.2,\ a_2 = 0.1,\ a_3 = 0.35,\ a_4 = 0.4,\ b_v = 0.02,\ m = 0.02,\ \eta = 0.0286,\ \beta_1 = 0.4,\ n_1 = 0.037,\ n_2 = 0.031$. Under these conditions, the basic reproduction number $\mathscr{R}_0 = 0.56875 < 1$, indicating that the disease will die out, and the disease-free equilibrium is globally stable. (b) The endemic equilibrium point is globally asymptotically stable when: $\theta = 0.89,\ K = 4000,\ \Lambda = 0.0166,\ d = 0.0167,\ g = 0.0105,\ \psi = 0.0056,\ \mu = 0.01, N = 2500,\ \omega_1 = 0.2,\ \omega_2 = 0.05,\ \omega_3 = 0.2,\ a_1 = 0.2,\ a_2 = 0.1,\ a_3 = 0.1,\ a_4 = 0.2,\ b_v = 0.02,\ m = 0.02,\ \eta = 0.0286,\ \beta_1 = 0.4,\ n_1 = 0.037,\ n_2 = 0.031$. Under these conditions, the basic reproduction number $\mathscr{R}_0 = 2.8425 > 1$, indicating that the disease will persist, and the endemic equilibrium is globally stable. The figure demonstrated that when $\mathscr{R}_0 < 1$, the density of healthy host plants (H) increases, while the density of infected plants (I) decreases and eventually vanishes as time approaches infinity. Conversely, when $\mathscr{R}_0 > 1$, the density of healthy plants decreases, while the density of infected plants persists.



(a) (b)

Figure 4.4: (a) The disease free equilibrium point is globally asymptotically stable when $\mathscr{R}_0 = 0.56875 < 1$. (b) The endemic equilibrium point is globally asymptotically stable when $\mathscr{R}_0 = 2.8425 > 1$.



(a) (b)

Figure 4.5: (a) Density of healthy host plants H increases whereas I decrease and vanishes eventually as $t \to \infty$ when $\mathscr{R}_0 < 1$. (b) For $\mathscr{R}_0 > 1$ healthy plant decreases whereas infectious plant persists.

Figure 4.5 (a) showed the disease-free equilibrium E_0 is globally asymptotically stable if $\mathscr{R}_0 < 1$ which means that the disease will be eliminated from the host crop population eventually. (b) When $\mathscr{R}_0 > 1$, E_0 becomes unstable and a locally asymptotically stable endemic equilibrium E^* exists.

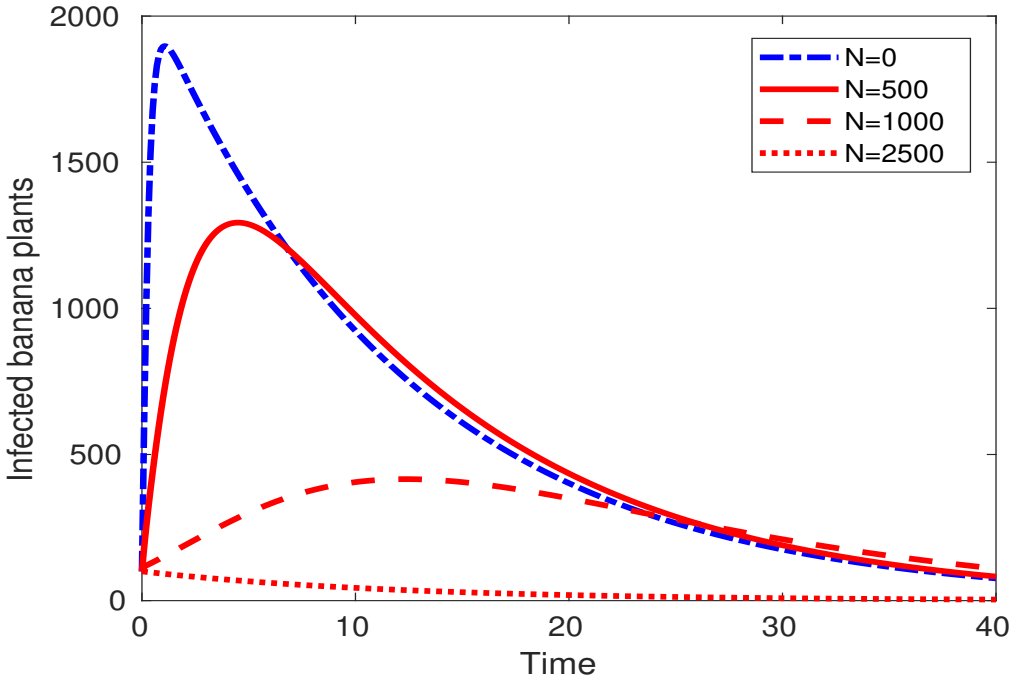


Figure 4.6: Simulation shows that the impact of the number of non-host plants varies.

Figure 4.6 observed the stability of the endemic equilibrium when the number of non-host plants varies, and without any control strategy, infected banana plants rise from 50 to more than 1500 within 2 months, then decrease because of disease-induced death. With a mixed-cropping control measure, the number of infected plants continuously decreases with time and approaches zero (0) after 15 months. This also indicates that when a banana tree becomes large when it is mature, it will use an increasing number of non-host plants in order to best control BXW disease with mixed-cropping method.

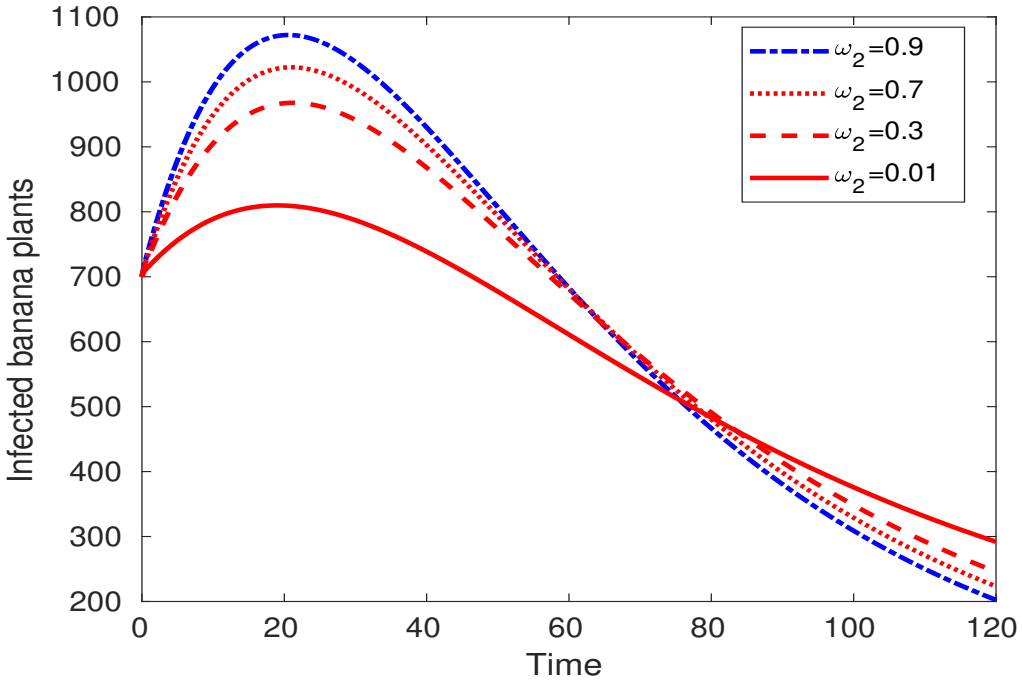
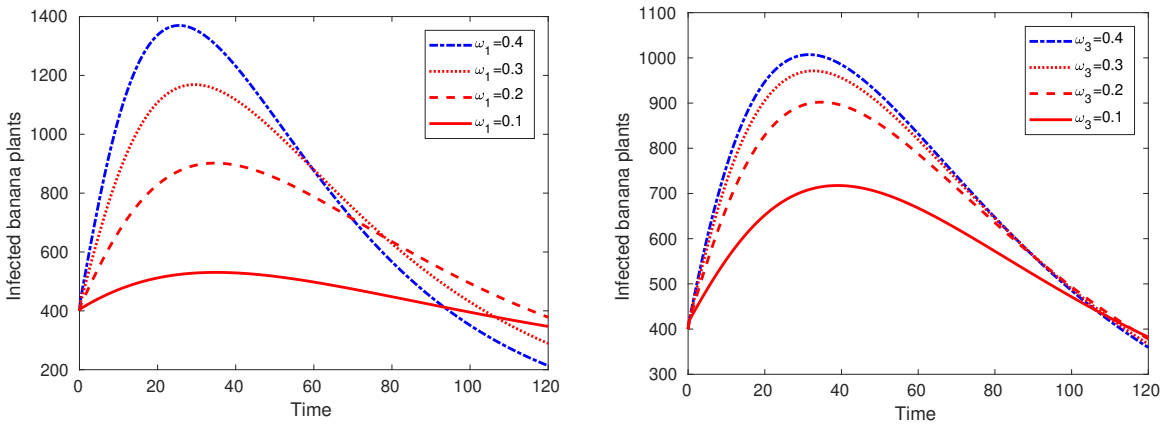


Figure 4.7: Changes in the rate at which contaminated soil causes infection.

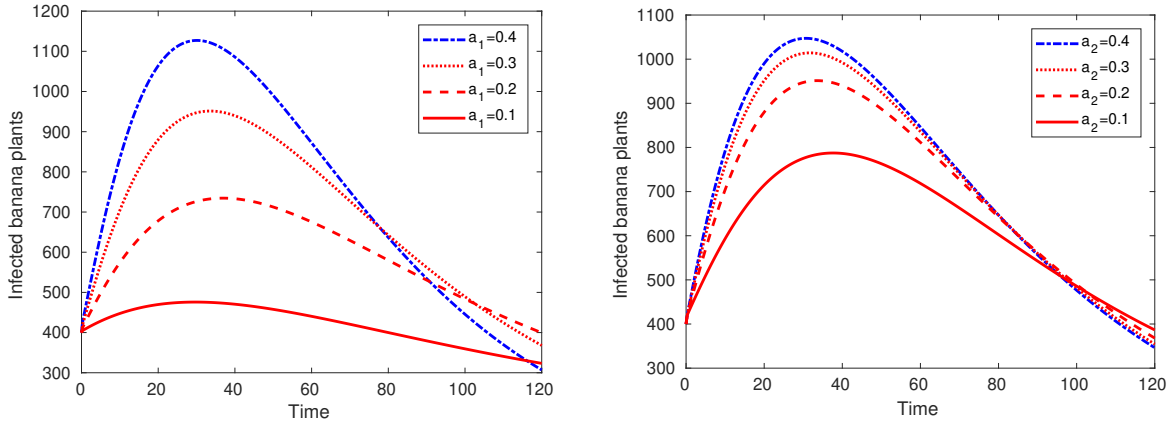
From the numerical result depicted in Figure 4.7 above, we observed that the number of infected banana plants grows as the probability of infection through contaminated soil (ω_2) increases. Figure 4.8 (a) shows that the number of infected banana plants grows as the probability that Xcm bacteria are transmitted from contaminated vector to susceptible banana as a result of a contact (ω_1) increases. Figure 4.8 (b) shows that the number of infected banana plants grows as the probability that a contact will cause Xcm to be transferred from infected banana to susceptible vector (ω_3) increases.



(a) (b)

Figure 4.8: (a) Changes in the rate at which contaminated vector causes infection (ω_1). (b) Changes in the rate at which a contact will cause Xcm to be transferred from infected banana to susceptible vector (ω_3).

Figure 4.9 (a) illustrates that as the rate of effective reach of an infected vector to susceptible bananas (a_1) decreases, the infected population also decreases. Figure 4.9 (b) illustrates that as the effective reach of the susceptible vector to the infected banana population (a_2) decreases, the number of infected bananas also decreases. Therefore, one possible approach is to use a mixed-cropping strategy to reduce the effective reach of vectors.



(a) (b)

Figure 4.9: (a) Changes in the rate at which effective reach of an infected vector to susceptible bananas. (b) Changes in the rate at which effective reach of the susceptible vector to the infected banana population.

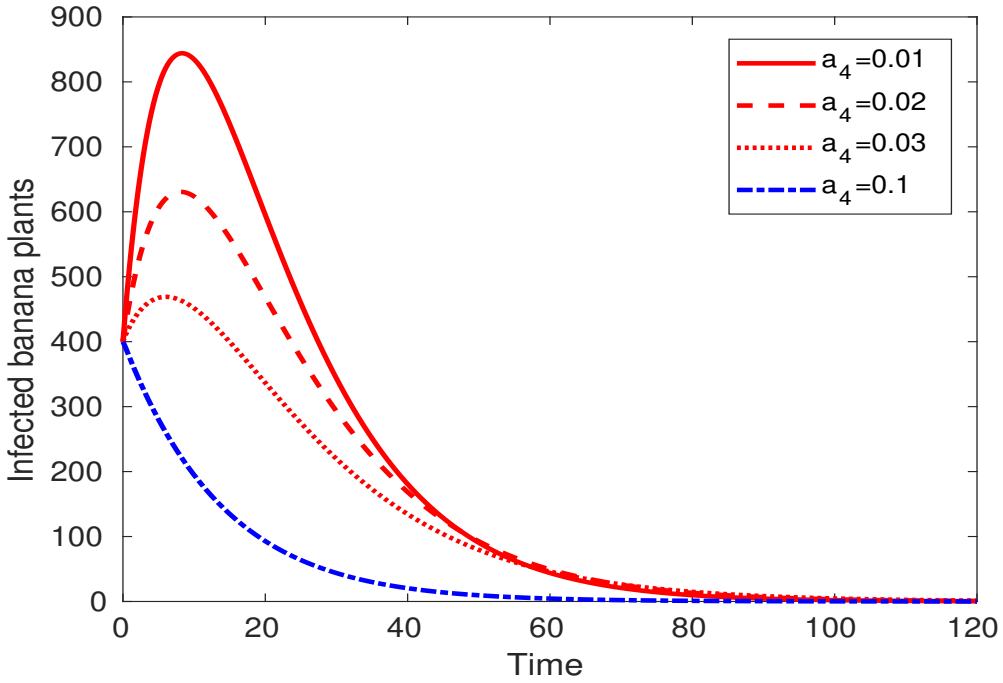
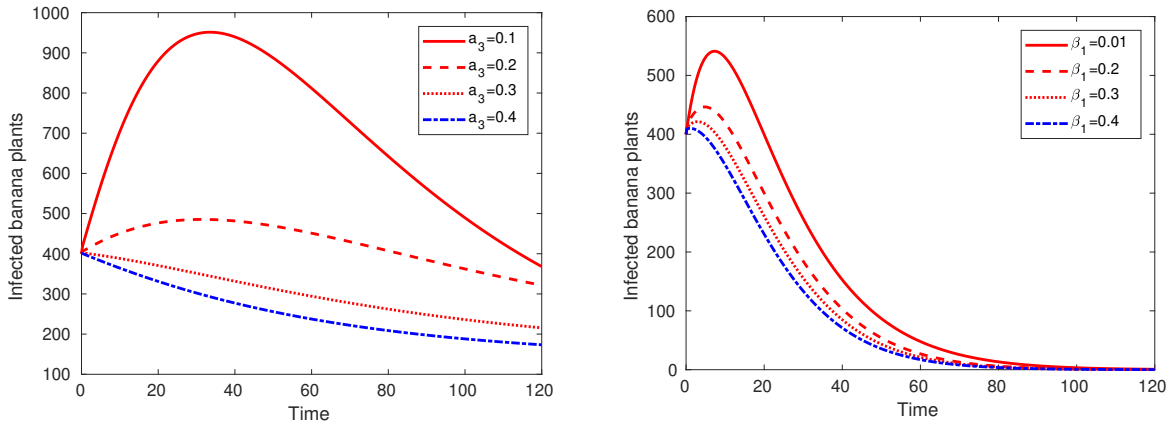


Figure 4.10: Changes in the rate of Xcm bacteria losses from contaminated soil by planting non-host plant.

From the numerical result depicted in Figure 4.10 above, we observed that the rate of Xcm bacteria losses from contaminated soil by planting non host plants (a_4) increases, causing a decrease in the number of infected host plants. Figure 4.11 (a) shows that the proportion of vectors moving towards non-host plants (a_3) increases, causing a decrease in the number of infected host plants; (b) shows that the rate of Xcm bacteria losses from contaminated vectors by planting non-host plants (β_1) increases, causing a decrease in the number of infected bananas. These results suggest that growing a lot of non-host plants alongside bananas can help prevent the spread of the BXW disease by blocking vectors' movement and clearance of contaminated vectors.



(a) (b)

Figure 4.11: (a) Changes in the rate at which effective reach of an infected vector to susceptible bananas. (b) Changes in the rate at which effective reach of the susceptible vector to the infected banana population.

Figure 4.12 shows that the basic reproduction number exponentially increase with the rate of infection through contaminated soil. Eliminating all Xcm bacteria from the soil, for example by mixed cropping, is one potential treatment for reduces the number of secondary infection through contaminated soil.

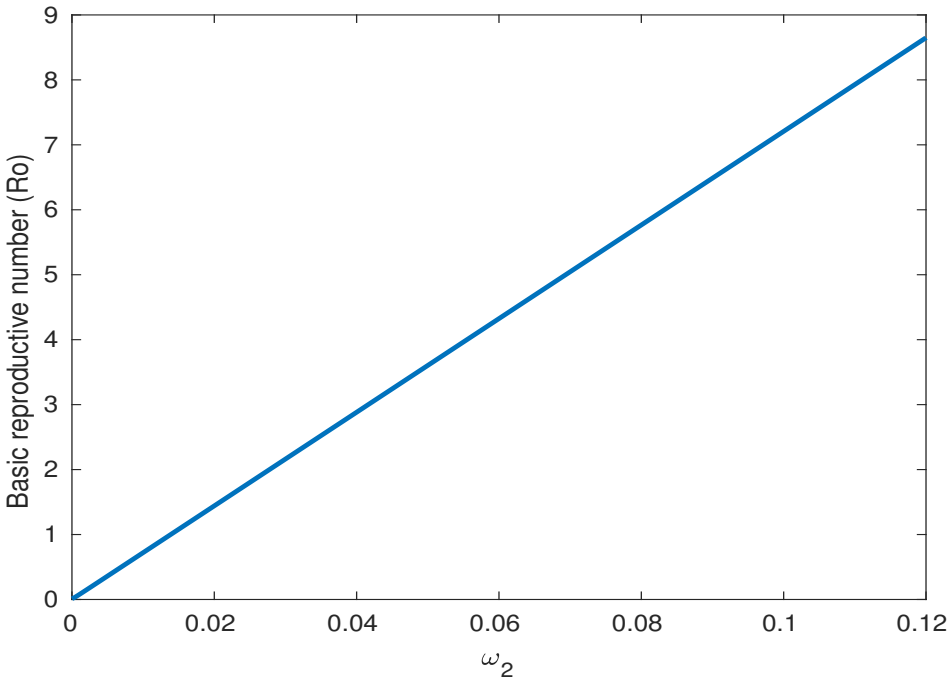
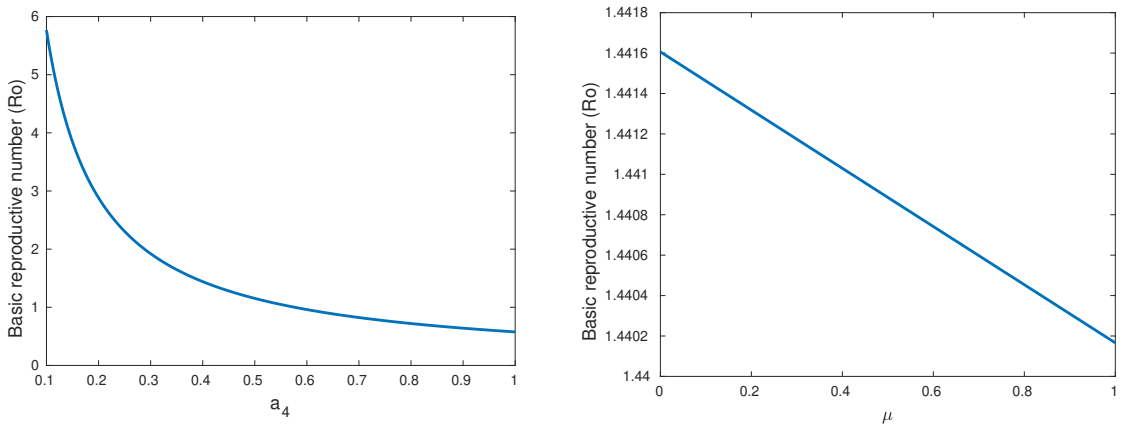


Figure 4.12: The impact of probability of Xcm bacteria being transmitted from C to H on the $\mathscr{R}_0$.

Figure 4.13 shows that rate of Xcm bacteria losses from C by planting N and naturally clearance rate of Xcm bacteria from the soil are negatively related to the basic reproduction number. But rate of Xcm bacteria losses from C by planting N is more sensitive compared to naturally clearance rate of Xcm bacteria from the soil. (a) implies a_4 dramatically reduce the basic reproduction number to less than one where the disease can be controlled. Moreover, (b) indicates that $\mathscr{R}_0 < 1$ cannot be achieved by μ alone. However, these findings still support the importance of developing a mixed-cropping method to accelerate the removal of Xcm bacteria from the soil in order to avoid secondary infections resulting from soil inoculum.



(a) (b)

Figure 4.13: (a) The impact of rate of Xcm bacteria losses from C by planting N on the $\mathscr{R}_0$. (b) The impact of naturally clearance rate of Xcm bacteria from the soil on the $\mathscr{R}_0$.

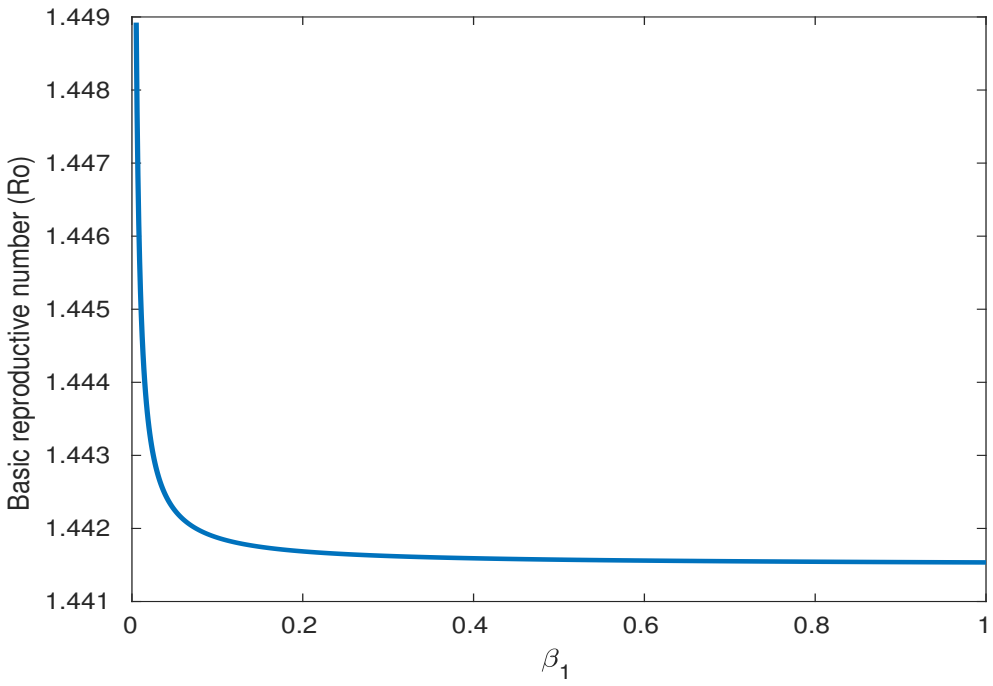


Figure 4.14: The impact of the rate of Xcm bacteria losses from Y by planting N on the $\mathscr{R}_0$.

Figure 4.14 shows that the increase in the rate of Xcm bacteria losses from infected vector by planting non host plants (β_1) leads to the decrease in the basic reproduction number. Furthermore, it shows that β_1 alone cannot make $\mathscr{R}_0 < 1$. According to (Ocimati et al., 2013), mixed cropping alone is not an effective strategy for controlling the vector-borne transmission of BXW. The study highlights the need for a more integrated approach to BXW management that goes beyond just mixed cropping practices.

CONCLUSION AND RECOMMENDATIONS

Conclusion

In this thesis work, we have developed and analyzed a mathematical model for the Banana Xanthomonas Wilt (BXW) disease. The primary goals of this study is to develop and evaluate a mathematical model that incorporates the dynamics of BXW disease in banana crop population planted in a mixed- cropping system as control measure.

After developing the basic model, the basic reproduction number as defined by Van den Driessche and Watmough was calculated using the next generation method. The stability of the model's equilibrium points was studied. The findings demonstrated the existence of the disease-free equilibrium, which is unstable when $\mathscr{R}_0 > 1$ and locally and globally asymptotically stable when $\mathscr{R}_0 < 1$. Similarly, the model endemic equilibrium exists and if and only if $\mathscr{R}_0 > 1$, the model's endemic equilibrium is globally asymptotically stable.

The sensitivity analysis suggests that the following are the model's most sensitive parameters: the rate of infection through contaminated soil (ω_2), the rate at which infected banana plants release Xcm bacteria into the soil (θ),the rate of infection through contaminated vector (ω_1), the rate of Xcm bacteria losses from contaminated soil by planting non host plant (a_4), total number of non host plant in banana plant field (N) and the rate of removing infected banana plant from the farm (g). Additionally, a numerical simulation was performed to support the findings with: figures (4.4-4.6) stability analysis of equilibrium points and figures (4.12-4.14) sensitivity analysis of basic reproduction number.

Therefore, if the recommended control measures such as de-budding and sterilization of farming tools, roguing, timely removal of the male bud using a forked stick, burning of infected banana plants, clearing Xcm bacteria from the soil, removing single diseased stems, controlling vertical transmission, and planting of healthy suckers are implemented along with the current control measure (mixed-cropping system), the disease could be eliminated.

Recommendations

Based on the study's findings, we recommend the following actions to contain banana xanthomonas wilt disease and prevent new infections. Farmers should be educated about the benefits of mixed-cropping method and trained in its implementation. There fore, the government should strengthen extension services, develop policies supporting intercropping disease control method, allocate funding for further research to disseminate effective BXW disease management methods.

Future Works

In the future work, this study will extend to a mathematical model with optimal control by conduct field trials to validate the mathematical model's predictions. Further more, this work has not addressed all aspects of the transmission dynamics, it is possible to extend this study in the following areas: seasonal changes in humidity and temperature, crop-rotation, breed that is resistant, social variables that prevent farmers from implementing recommended control method.

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APPENDIX

Mathematical Preliminary

In this topic, we state some theorems and the definitions of terminologies that provide basic mathematical definitions and results, which are crucial for a better understanding of this thesis work.

Mathematical Modeling

A mathematical model is an abstract description of a concrete system using mathematical concepts and language. The process of developing a mathematical model is called mathematical modeling. Mathematical models are used in various fields such as physics, biology, engineering, economics, and social sciences to help explain systems, study the effects of different components, and make predictions (Meerschaert, 2013).

Epidemiology is the subject that studies the patterns of health and illness and associated factors at the population level (Martcheva, 2015).
Epidemiological modeling can also contribute to the design and analysis of epidemiological surveys, suggest crucial data that should be collected, identify trends, make general forecasts, and estimate the uncertainty in forecasts. The epidemiological modeling of infection disease contributes a lot of achievements in the control of different diseases in the world (Hethcote, 1989).

System of Ordinary Differential Equations

Anggreini (2016), consider a system of ordinary differential equation, which is non autonomous:

$$\begin{aligned}
\frac{dx_1}{dt} &= f_1(x_1, x_2, ...x_n, t), \\
\frac{dx_2}{dt} &= f_2(x_1, x_2, ...x_n, t), \\
&\vdots \\
\frac{dx_n}{dt} &= f_n(x_1, x_2, ...x_n, t),
\end{aligned} \tag{27}$$

with f_i is a general function of $x_i, i = 1, 2, ..., n$ and time t. If the system (27) can be further simplified into a system that do not depend on time, then the system is called autonomous

and can be written as:

$$\begin{aligned}
\frac{dx_1}{dt} &= f_1(x_1, x_2, ...x_n), \\
\frac{dx_2}{dt} &= f_2(x_1, x_2, ...x_n), \\
&\vdots \\
\frac{dx_n}{dt} &= f_n(x_1, x_2, ...x_n),
\end{aligned} \tag{28}$$

with f_i is a general function of x_i, $i = 1, 2, ..., n$ that do not depend explicitly on the time t. The compact form of system (28) can be written as;

$$\dot{x} = f(x), \quad x(0) = x_0. \tag{29}$$

Let $E \subset \mathbb{R}^n$ and $f : E \to \mathbb{R}^n$ be continuous function on E, then the system (28) is said to be linear if $f_1, f_2, ..., f_n$ each linear with respect to $x_1, x_2, ..., x_n$. So the system (28) can be written in the form,

$$\begin{aligned}
\frac{dx_1}{dt} &= a_{11}x_1 + a_{12}x_2 + ... + a_{1n}x_n, \\
\frac{dx_2}{dt} &= a_{21}x_1 + a_{22}x_2 + ... + a_{2n}x_n, \\
&\vdots \\
\frac{dx_n}{dt} &= a_{n1}x_1 + a_{n2}x_2 + ... + a_{nn}x_n.
\end{aligned} \tag{30}$$

The system (28) can be expressed in the form,

$$\dot{x} = Ax, \tag{31}$$

where $x \in E, \dot{x} = (\frac{dx_1}{dt}, \frac{dx_2}{dt}, ..., \frac{dx_n}{dt})$ and A is an $n \times n$ matrix. The system (28) is said to be nonlinear if there is i such that f_i is nonlinear. The long term behavior of the solution around the equilibrium point of nonlinear systems (28) can be determined through linearization around the equilibrium point of the system.

Equilibrium Point and Stability Analysis

Definition .0.1. *(Layek et al., 2015) A point $x^* \in \mathbb{R}^n$ is said to be an **equilibrium point** (fixed point) of the system (29) if*

$$f(x^*) = 0, \qquad forall \ \ t \geq 0.$$

In the context of differential equations, an equilibrium point is a constant solution where the system can stay unchanged over time.

Definition .0.2. *An equilibrium point x^* of $\dot{x} = f(x)$ is a hyperbolic equilibrium point if $f'(x^*) \neq 0$*

Definition .0.3. *(Hale & Koçak, 2012)*

- *An equilibrium point $x^* \in \mathbb{R}^n$ of a dynamical system $\dot{x} = f(t,x)$ is called locally stable if for every neighborhood U of x^*, there exists $V \subset U$ of x^* such that every solution $x(t)$ with $x(t_0) \in V$.*
- *An equilibrium point x^* is globally stable if every solution $x(t)$ of the system satisfies $\lim_{t\to\infty} x(t) = x^*$.*

Linearization

Around the equilibrium point x^*, the non-linear system (29) is linearized as the following. If $f = (f_1, f_2, ..., f_n)$ and $x = (x_1, x_2, ..., x_n)$, then the first order partial derivative matrix:

$$D(f) = J = \begin{bmatrix} \frac{\partial f_1}{\partial x_1} & \frac{\partial f_1}{\partial x_2} & \cdots & \frac{\partial f_1}{\partial x_n} \\ \frac{\partial f_2}{\partial x_1} & \frac{\partial f_2}{\partial x_2} & \cdots & \frac{\partial f_2}{\partial x_n} \\ \vdots & \vdots & \ddots & \vdots \\ \frac{\partial f_n}{\partial x_1} & \frac{\partial f_n}{\partial x_2} & \cdots & \frac{\partial f_n}{\partial x_n} \end{bmatrix}.$$

J is called a Jacobian matrix of f in point x^*. By using the Jacobian matrix $J(x^*)$ we study the local stability properties of hyperbolic equilibrium point x^*.

Definition .0.4. *A point x^* is said to be stable if for a given $\epsilon > 0$, there exist $\delta = \delta(\epsilon) > 0$ such that, for any other solution, $y(t)$ of (29) satisfying $|x^*(t_0) - y(t_0)| < \delta \Rightarrow |x^*(t) - y(t)| < \epsilon, \ \forall t > t_0, \ t_0 \in \mathbb{R}$ (Layek et al., 2015).*

Definition .0.5. *(Layek et al., 2015), let A be a constant matrix in the system $\frac{dx}{dt} = Ax$ with eigenvalues λ_i, for $i = 1, 2, ..., n$:*
(i) If the system is stable, then $Re(\lambda_i) \leq 0$ for $i = 1, 2, ..., n$.
(ii) If either $Re(\lambda_i) < 0$; or if $Re(\lambda_i) \leq 0$ for $i = 1, 2, ..., n$ and there is no zero repeated eigenvalue, then the system is uniformly stable.
(iii) The system is asymptotically stable if and only if $Re(\lambda_i) < 0$ for $i = 1, 2, ..., n$;note that it is also uniformly stable by (ii).
(iv) If $Re(\lambda_i) > 0$ for any $i = 1, 2, ..., n$ then, the solution is unstable.

The Routh-Hurwitz Criteria

The Routh-Hurwitz Criteria is a method used to determine the stability of a linear system by analyzing the coefficients of the characteristic equation.

For a system of differential equations (31) the characteristic polynomial of matrix A is given by $p(\lambda) = \lambda_n + a_1\lambda_n - 1 + ... + a_{n-1}\lambda + a_n$ where the coefficients a_i are real constants, $i = 1,2,3...n$ define the n Hurwitz matrices using the coefficients of the characteristic polynomial. Here the matrices M_k , $k = 1,2,3,...,n$, are given by

$$M_1 = a_1, M_2 = \begin{bmatrix} a_1 & 1 \\ a_3 & a_2 \end{bmatrix}, \tag{32}$$

$$M_3 = \begin{bmatrix} a_1 & 1 & 0 \\ a_3 & a_2 & a_1 \\ a_5 & a_4 & a_3 \end{bmatrix}, M_4 = \begin{bmatrix} a_1 & 1 & 0 & 0 \\ a_3 & a_2 & a_1 & 1 \\ a_5 & a_4 & a_3 & a_2 \\ a_7 & a_6 & a_5 & a_4 \end{bmatrix}, \tag{33}$$

$$M_n = \begin{bmatrix} a_1 & 1 & 0 & \cdots & 0 \\ a_3 & a_2 & a_1 & \cdots & 0 \\ a_5 & a_4 & a_3 & \cdots & 0 \\ \vdots & \vdots & \vdots & \ddots & \vdots \\ 0 & 0 & 0 & 0 & a_n \end{bmatrix}, \tag{34}$$

where $a_j = 0$ for $j > n$.
All the roots of polynomial $p(\lambda)$ have negative real parts if and only if the following conditions hold:

- $a_1, a_2, ..., a_n > 0$
- The determinants of the Hurwitz matrices $M_1, M_2, ..., M_n$ are all positive.

Definition .0.6. *(Plemmons, 1977) An $n \times n$ matrix A that can be expressed in the form $A = sI - B$, where $B = b_{ij}$ with $b_{ij} \geq 0$, $1 \leq i, j \leq n$, and $s \geq \rho(B)$, the maximum of the moduli of the eigenvalues of B, is called an M-matrix.*

Lyapunov Function

Definition .0.7. *A function $L : \mathscr{D} \subset \mathbb{R}^n \to \mathbb{R}$ a continuously differentiable, real-valued function is said to be positive definite if*

- $L(x) > 0$ *, for all* $x \neq 0$,

- $L(x) = 0$, *for all* $x = 0$,
- $L(x) \to 0$, *for all* $x \to \infty$, *Any function* L *is called* ***a Lyapunov function****, if it satisfies the conditions of positive definiteness and* $\frac{dL}{dt} \leq 0$ *for all* $x \in \mathscr{D} \setminus x_0$ *(Layek et al., 2015).*

Theorem .0.1. *Let* x^* *be an equilibrium point of the system (29) and consider Lyapunov function* $L : \mathscr{D} \subset \mathbb{R}^n \to \mathbb{R}$ *be continuously differentiable, real valued function with the following properties:*

- $L(x) > 0$ *for all* $x \neq x^*$, *and* $L(x^*) = 0$. *(We say that* L *is positive definite.)*
- $\dot{L}(x) < 0$ *for all* $x \neq x^*$. *(All trajectories flow "downhill" toward* x^*.*)*

Then x^* *is globally asymptotically stable: for all initial conditions,* $x(t) \to x^*$ *as* $t \to \infty$. *In particular the system has no closed orbits. (For a proof, see (Smith & Jordan, 1987).)*

Lasalle's Invariance Principle

Theorem .0.2. *Suppose that* $L : U \subset \mathbb{R}^n \to \mathbb{R}$ *is a Lyapunov function for the system* $\dot{x} = f(x)$. *If the set* $x \in U | \dot{L}(x) = 0$ *contains only one fixed point* x^*, *then* x^* *is asymptotically stable.*

Next Generation Method

The average number of new infections produced by the introduction of one infected person into a community of persons who are all completely susceptible is known as the basic reproduction number. The basic reproduction number is determined using Van den Driessche & Watmough (2002)'s next generation approach. By using this technique the infected population model system $f_i(x)$ is split into two categories, thus

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

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. 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^* is the disease free equilibrium point of the model system, then

$$F = \frac{\partial \mathscr{F}(x)}{\partial x_i} \text{ and } V = \frac{\partial \mathscr{V}(x)}{\partial x_i},$$

where the infected populations are denoted by x_i. Consequently, determining the highest eigenvalue of the matrix FV^{-1} yields the basic reproduction number, thus

$$\mathscr{R}_0 = \rho(FV^{-1}).$$

Numerical Methods for System of ODEs

The following is a summary of numerical techniques taken from (Faragó, 2014), to compute the approximate solution of an ODE system that contains equations that cannot be solved analytically, numerical techniques are applied. For instance, initial value problems (IVP) for ODE can be solved using fourth order Runge-Kutta methods. Which is given by

$$\dot{x} = \frac{dx}{dt} = f(t,x), x(t_0) = x_0, \tag{35}$$

on the interval $t_0 \leq t \leq T$ into N small segments of constant length h, which is called the step size given by

$$h = \frac{T - t_0}{N}. \tag{36}$$

Systems of equations can be easily solved using the numerical techniques that were first proposed for a single equation. For instance, take the ordinary differential equation system with initial condition,

$$\frac{dx}{dt} = f(t,x,y), \quad x(t_0) = x_0, \tag{37}$$

$$\frac{dy}{dt} = g(t,x,y), \quad y(t_0) = y_0. \tag{38}$$

Specifically, we are looking for a numerical solution on the interval $t_0 < t \leq T$. The interval is initially discretized by determining the time steps provided by

$$t_n = t_0 + nh, \quad n = 0, ..., N, \tag{39}$$

where the number of steps is N. Again, the approximate values of $x(t,n)$ and $y(t,n)$ are indicated by the notations x_n and y_n, respectively. The numerical procedures can be summarized as follow:

Runge-Kutta Fourth Order

The Runge-Kutta technique of order four ($RK4$) produces a sequence of approximation points $(t,x) \approx (t,x(t))$ and $(t,y) \approx (t,y(t))$ to the exact solution of an ODE by to the exact solution of

ODE by $t_{n+1} = t_n + h$ to get the following values of eight slope:

$$
\begin{aligned}
k_{11} &= f(t_n, x_n, y_n), \\
k_{21} &= g(t_n, x_n, y_n), \\
k_{12} &= f(t_n + \frac{h}{2}, x_n + \frac{h}{2}k_{11}, y_n + \frac{h}{2}k_{21}), \\
k_{22} &= g(t_n + \frac{h}{2}, x_n + \frac{h}{2}k_{11}, y_n + \frac{h}{2}k_{21}), \\
k_{13} &= f(t_n + \frac{h}{2}, x_n + \frac{h}{2}k_{12}, y_n + \frac{h}{2}k_{22}), \\
k_{23} &= g(t_n + \frac{h}{2}, x_n + \frac{h}{2}k_{12}, y_n + \frac{h}{2}k_{22}), \\
k_{14} &= f(t_n + h, x_n + hk_{13}, y_n + hk_{23}), \\
k_{24} &= g(t_n + h, x_n + hk_{13}, y_n + hk_{23}).
\end{aligned}
$$

Next, we use the weighted averages of the previous slopes to construct the next approximation.

$$
\begin{aligned}
x_{n+1} &= x_n + \frac{h}{6}(k_{11} + 2(k_{12} + k_{13}) + k_{14}), \\
y_{n+1} &= y_n + \frac{h}{6}(k_{21} + 2(k_{22} + k_{23}) + k_{24}).
\end{aligned}
$$

Using this approach, the discrete time model that may be approximated through computer simulation replaces the continuous time model.