MODELING THE DYNAMICS AND CONTROL OF CASSAVA MOSAIC DISEASE

Florence Magoyo

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ABSTRACT

Cassava mosaic disease (CMD) is caused by cassava mosaic virus (CMV) and is transmitted by the whitefly vector called Bemisia tabaci. In this study, the deterministic model for transmission dynamics of CMD is formulated by considering the whitefly vector, cassava resistant and susceptible breeds, and infected cassava. The basic reproduction number R_0 and sensitivity index for each parameter with respect to basic reproduction number R_0 are computed to determine which parameters are sensitive to the dynamics of cassava mosaic disease. Analysis shows that the death rate of whitefly vectors, infection rate for susceptible vectors, the number of vectors that can be supported and recruitment rate of whitefly are most sensitive parameters to the dynamics of cassava mosaic disease. The disease stability at cassava mosaic free equilibrium was investigated by using metzler matrix (box invariance). We found that disease free equilibrium is asymptotically stable when $R_0 < 1$. By using Lyapunovs direct method and LaSalles invariant principle, endemic equilibrium is asymptotically stable when $R_0 > 1$. Numerical simulation indicates that, cassava new infections increase as many whitefly vectors are recruited and acquire cassava mosaic disease. When controls are not considered, the susceptible breed and cassava resistant breed will be wiped out after five and ten months respectively. To control the disease interventions which target whitefly vectors, farmers are encouraged to apply control strategies such as spraying of insecticide, using of vector-resistant varieties, phytosanitation which involves the removal of infected cassava plants from the farm, crop hygiene and the use of free stem cutting method. Analysis shows that spraying of insecticide and the death of whitefly vector plays the most important role in the eradication of CMD. This study concludes that, spraying of insecticide is the possible way to get rid of both infected and susceptible vector as well as the removal of infected cassava plants from the farm will help to reduce the contact rate between plants and vectors.

DECLARATION

I, Florence D. Magoyo 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 degree award in any other institution.

Florence Magoyo (Candidate)	Date

The above declaration is confirmed

Prof. Dmitry Kuznetsov (Supervisor 1)	Date
 Dr. Jacob Ismail Irunde (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: Modeling the dynamics and control of cassava mosaic disease, in fulfillment of the requirements for the degree of Masters in Mathematical and Computer Sciences and Engineering of the Nelson Mandela African Institution of Science and Technology.

Prof. Dmitry Kuznetsov
(Supervisor 1)

Date: .....

Dr. Jacob Ismail Irunde
(Supervisor 2)

Date: .....

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DEDICATION

I dedicate this dissertation to my lovely husband Mr. Innocent Vitus Kitauka and to my beloved parents Mr. and Mrs. Damascus J. Magoyo.

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LIST OF ABBREVIATIONS

CBSD	Cassava Brown Streak Disease
CMD	Cassava Mosaic Disease
ACMV	African Cassava Mosaic Virus
EACMV	East African Cassava Mosaic Virus
ICMV	Indian cassava Mosaic Virus
SACMV	South African Cassava Mosaic Virus
COSTECH	Tanzania Commissions for Science and Technology
FAO	Food and Agriculture Organization

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CHAPTER ONE

INTRODUCTION

1.1 Background Information

Cassava (*Manihot esculenta*) is one of the crops which was firstly introduced in West Africa from Brazil at the end of 16^{th} Century by Portuguese and spread to other African countries (Fauquet and Fargette, 1990; Nweke, 2009). Cassava has become the major staple food in the world, particularly in developing countries like African countries, India and Sri Lanka. According to FAO, about 700 million people depend on cassava as their main food in Africa (Rogans and Rey, 2016).

Cassava is grown in semi-arid tropical and subtropical areas which experience low rainfall as the crop can survive in drought climate. The communities in tropics and sub-tropics regions have relied on this crop for food since their areas sometimes receive low rainfall and some of the areas have poor soil which cannot support other crops (Irungu, 2011; De Tafur *et al.*, 1997).

Production of cassava in Africa is becoming low due to a number of causes, notably pests and diseases (Hillocks, 1997). Cassava brown streak disease (CBSD) and cassava mosaic disease (CMD) are the most important biotic constraints which have led to decrease in yields. These diseases are the main threats to farmers who produce cassava (Kinene *et al.*, 2015; Calvert and Thresh, 2002). Cassava brown streak disease is caused by cassava brown streak virus, after the attack cassava plant can show the symptoms of CBSD, it attack the cassava tuber, leaves and stem. This depends on cassava variety since others cannot show the symptoms earlier, and also it depends on altitude, rain, plant age and virus species and sometimes the farmer confusing it with the cassava mosaic disease (Kinene *et al.*, 2015). While cassava mosaic disease is easy to be recognized by the farmers, it attacks the leaves of cassava by destroying chlorotic area which leads to the poor growth and poor health of a plant (Hillocks and Thresh, 2000). Both diseases (CBSD and CMD) are more destructive since some of the introduced resistant breed are easy to be affected by CBSD but highly susceptible to CMD while in contrast some are highly susceptible to CBSD but resistant to CMD (Ntawuruhunga and Legg, 2007).

1.1.1 Causes and Transmission of Cassava Mosaic Disease

Cassava mosaic virus is a virus in a family of begomoviruses (Zhou *et al.*, 1998). In this family there are three familiar species which include African cassava mosaic virus (ACMV), East African cassava mosaic virus (EACMV) and Indian cassava mosaic virus (ICMV) and lately

there is another virus known by the name South African cassava mosaic virus (SACMV) (Pita *et al.*, 2001). These diseases have been categorized in this way due to serological properties in tests with a panel of monoclonal antibodies (Thomas *et al.*, 1986; Zhou *et al.*, 1998).

African cassava mosaic virus (ACMV) contaminates the cassava leaves and is transmitted by the whitefly vector called Bemisia tabaci (Fauquet and Fargette, 1990). There are other 500 different plants including weeds and crops which are hosts to whitefly vector (Legg and Fauquet, 2004; Morales and Anderson, 2001). Cassava mosaic disease can also be transmitted through the use of infected stem cuttings as well as grafting infected bud wood onto uninfected cassava plants (Alabi *et al.*, 2011). Other factors, include the use of continuous infected plant materials (Kapinga *et al.*, 2005) as well as the use of CBSD resistant breed, which becomes vulnerable to the cassava mosaic disease (Thomas *et al.*, 1986; Zhou *et al.*, 1998).

The disease was first reported from East Africa in 1894 (Thresh and Cooter, 2005) and spread to the whole African continent, causing a great loss to cassava farmers. It attacks cassava plants and affect the cassava leaves and roots leading to poor harvest and hence food insecurity (Fauquet and Fargette, 1990). Other cassava disease that attack the cassava plants are cassava brown streak disease as it is explained above, cassava frog skin and green mite which is transmitted through grafts, cassava meal burg just mention the few of them (Calvert and Thresh, 2002).

1.1.2 Disease Symptoms

The cassava plant which has been infected with the disease shows some symptoms which can be recognized easily by the farmers (Thresh and Cooter, 2005). The infected cassava plant is characterized by leaf mosaic patterns and it can persist during the premature stage of cassava leaf development. The cassava leaves which are infected by the disease are warped, reduced in size and distorted with yellow color separating the ordinary green color which is the health part of the leaves. They then deteriorate and the new leaves bend (Hillocks and Thresh, 2000).

Figure 1: Symptoms of cassava mosaic disease on cassava plant.

The image (A) shows an infected cassava where the plant shows much severe stunting and leaves distortion, Image (B) is a healthy plant. It is not yet affected by the disease. Image (C) and image (D) the leaves of the plant becomes warped and abnormal leaflets mottling symptoms (Alabi *et al.*, 2011).

1.1.3 Impact of Cassava Mosaic Disease

Cassava mosaic diseasef poses food security problem in tropical and subtropical regions where cassava is a staple food and cause a great loss to the farmers. Since the production of cassava is interrupted with different factors the past two decades, in the year 2017 The world production of cassava is experience to decline to 278 million tonnes, around 1 million tonnes lower than the level of previous year (FAO, 2017). In Benin the cassava production has become lower by 9% from 2017 level and the reasons for the lower production is the outbreak of pest and disease (FAO, 2018). For the past years, the actual production of cassava in Africa was approximately to be 73 million tonnes but about 12-23 million tonnes are lost due to the disease (Thresh *et al.*, 1997). In Uganda almost four district have been rendered unproductive due to the incidence of cassava mosaic disease. It is estimated that the disease has brought loss of 60 000 hectares which were expected to produce 600 000 tonnes of tuberous roots. The same applies to other places in Rwanda and Western parts of Tanzania (Thresh and Cooter, 2005).

According to FAO, Tanzania is one of the main producer of cassava in the world (Legg and Hillocks, 2003). Cassava Mosaic Disease in Tanzania is one of the problem that is facing the production of cassava. The disease has been spread at a fast rate leading to food shortages (Uzokwe *et al.*, 2016). According to Tanzania Commission for Science and Technology

(COSTECH) production of cassava in Tanzania is only 8 t/ha which is lower compared to 20 t/ha that can be produced. The main causes of low production are pests and diseases.
Figure 2 shows the world production of cassava from 2011 to 2017.

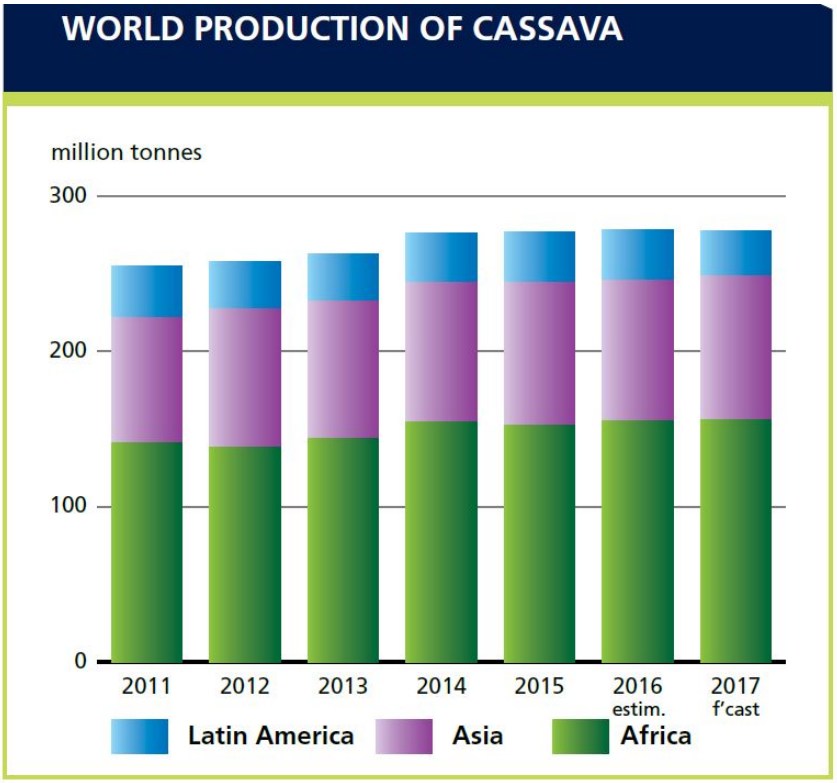


Figure 2: World production of cassava 2011-2017 (FAO, 2017).

1.2 Research Problem

Cassava mosaic disease is still a threat to the production of cassava in different parts of Africa (Kinene *et al.*, 2015). In Tanzania the rate of spread of cassava mosaic disease has increased dramatically, which has led to food shortage and cash due loss of harvest as cassava is a cash crop (Uzokwe *et al.*, 2016).

Different measures have been taken to control and combat the disease. Some of these measures include, the use of breeding which is resistant to diseases, CMD-free planting material, vector control by insecticides, and the sanitation method which involves the removal of infected cassava plants. Most of the measures which help in the control of transmission dynamics of disease (Thresh and Cooter, 2005) are biological. However, few studies have used mathematical models to investigate how control strategies can contain the disease. This work studies the dynamics of cassava mosaic disease by considering cassava resistant breed which only catch

cassava mosaic disease through unhealthy cutting and susceptible breed which catch mosaic disease through unhealthy cutting and contact from whitefly vector when the controls are not and are implemented.

1.3 Objectives

1.3.1 General Objective

To develop and analyze a mathematical model for cassava mosaic transmission dynamics with and without controls.

1.3.2 Specific Objectives

The specific objectives of this research were:

(i) To develop a mathematical model for the transmission dynamics of cassava mosaic disease.

(ii) To analyze the model equilibria states.

(iii) To perform sensitivity analysis, to determine which parameters are sensitive to the disease.

(iv) To determine the impact of cassava mosaic controls.

1.4 Research Questions

The study was guided by the following questions:

(i) How can a mathematical model for the transmission of cassava mosaic disease be formulated?

(ii) How can model equilibrium states be analyzed?

(iii) How can sensitivity analysis be performed?

(iv) Which parameters are sensitive to cassava mosaic disease?

(v) What impact do controls have on cassava mosaic dynamics?

1.5 Significance of the Study

The study will have the following advantageous to the farmers, researchers as well as to the agricultural stakeholders:

(i) To the farmers, researchers and other agricultural stakeholders, the study will improve current knowledge about the transmission dynamics of CMD.

(ii) To the agricultural stakeholders and farmers, this study will help them to decide on the suitable control strategies that will help them to reduce the persistence of CMD.

(iii) This study will act as a base for further research on CMD in Tanzania.

CHAPTER TWO

LITERATURE REVIEW

This chapter reviews some studies that have been conducted to investigate cassava mosaic disease (CMD) and its causes, disease symptoms, and impact, the possible solutions leading to its control. Also various models explained more about CMD. Some of these studies are briefly reviewed as follow.

Fargetie *et al.* (1994) presented analysis and modeling of the temporal spread of African cassava virus and its implications for disease control. The relationship between the ACMV epidemic in a given period of time which depends on the age of a crop and the planting date and the ACMV progress curve were presented. The simulation of the model shows that if the cutting is not selective by considering the healthy and better plants, the occurrence of a disease will increase sequentially to other plants in the field and ultimately will reach 100%. This can occur in any amount of a host resistance even if it is after a certain period, by contrast, with reversion and/or cutting selection, disease incidence may reach below 100% of equilibrium value. The effect of these methods balances the new transmission of virus by whitefly vectors. By exploring their ability to revert, it accentuates the possible way for the farmers to control the CMV, after infection, the farmers suffer low yield even after several crop rotations.

Fargette and Vie (1994), in their study title modelling and temporal primary spread of cassava mosaic virus into planting, shows that the rate of transmission of ACMV is dependent on the planting date p, and the age of a cassava plant at time t. The relationship between these two was expressed mathematically whereby the suitable functions were selected and the parameters were derived by using nonlinear regression. The disease progressive curves were obtained by using numerical integration of the differential equation. The fit between the model and the experimental curves shows the trend of the epidemic. After testing the model it was found that there was a good fit between observed and modelled disease progressive curves.

Fargette and Vié (1995) in their study on the Simulation of the effects of host resistance, reversion, and cutting selection on incidence of African cassava mosaic virus and yield losses in cassava, they discuss the model, which describes the epidemic of African cassava mosaic. They use the model to explain the effect of resistance and sanitation on epidemic severity and cassava productivity in successive annual cropping cycles. The parameters included in the model are host resistance, secondary spread within plantings, latent period, and yield losses. The resistance and sanitation were modelled in two different ways, firstly reversion which is percentage

of cutting the healthy plants from the infected plants and special cutting section which is the ratio of cutting from the healthy plants to the number of an infected plants. When these two methods are adopted, the disease incidence is increased during the first few annual crop cycles, but it reaches the equilibrium which is below 100%. At this step of equilibrium, new infection caused by whitefly vectors escapes, though the reversion and the yield losses become limited, as well as the yield losses are assessed due to the incidence of the disease by combined effects of host resistance reversion, and cutting selection.

Holt *et al.* (1997) in the study of an epidemiological model incorporating vector dynamics applied to African cassava mosaic virus disease formulated a model which specifies the healthy (susceptible) and infected cassava as well as the susceptible (non-infectious) and infectious vector (the model is SI-susceptible and infectious). The study show that the increase in the using of infected cutting have a little effect on the occurrences of a disease also the model shows that the elimination of infectious cassava plants had little impact on the occurrences of a disease.

In the study titled plant-vector-virus models with vector aggregation applied to cassava mosaic virus by (Hebert, 2014), three different differential equations as well as the corresponding Markov chain models were used to model the dynamics of cassava plants that can be infected by the virus from the vectors. These models included the effect of accretion of vectors implicitly through its transmission term. The basic reproduction numbers for these three models were computed, and the probability of eliminating the disease was computed by using the stochastic process models. The model was applied to cassava mosaic virus, the numerical and analytical results show that the vector aggregation is growing in intricacy for the vector movement and resistant crops as a well as the possibility of a disease to be recognized in host plant.

Lawrence and Wallace (2011) in their work named spatiotemporal dynamics of African Cassava mosaic disease, used the system of differential equation to find the equilibrium value of the whitefly vector and the cassava plants. The temporal ordinary differential equation system was modified to incorporate the spatial dynamics. The result was analyzed by using the finite difference method to assess the spatiotemporal spread of a disease. The PDE system was changed scientifically and this led to a solution which is in terms of the predicted relative cassava yields. The simulation of the model includes parameter sensitivity analysis, spatial modifications, analysis of the impact of a source term, and initial condition variance. Results obtained were compared to the field data and the implication of controlling the CMV practically were explored. The study concluded that using of ACMD resistant strains of cassava and windbreaks will have positive results to cassava yields.

The work on a general model of plant-virus disease infection incorporating vector aggregation, (Zhang *et al.*, 2000) They show that there is always a combination bilinear immunization rate which is direct proportional to susceptible plants and the profusion of infective vectors. Also the rate of acquiring is assumed to be directly proportional to uninfected vector (susceptible vector) and that of infectious plants and that of infectious plants. The incidence of CMD were examined, in which the combination of the new infection terms allowed the range of observed disease progressive curve types to be described. New evidence of a mutual interaction between the viruses and the whitefly vector, has shown that spatial aggregation of the vectors is an unavoidable result of infection, particularly with a severe virus strain or a sensitive host. Virus infection increases the fertility of a vector and the density of vectors on unhealthy plants. This assumes to increase the spread of the disease through an increased emigration rate of infective vectors to other crops. Paradoxically, within the infected crop, Vector aggregation reduces the effective contact rate between vector and plants and therefore the predicted disease incidence is less than when a bilinear contact rate is used.

Thresh and Cooter (2005) in their study titled strategies for controlling cassava mosaic virus disease in Africa. Plant pathology, explain the different possible control solution of the disease which can be taken. Among the possible control measures are phytosanitation. The study explains how the health of a cassava plant can be improved to eliminate the further spread of the disease. These include different features like crop hygiene which include, the removal of all affected cassava plants from the farm, also the use of free-CMD stem cutting and the removal of the diseased plants within the crop standing.

Generally, few of these studies considered the aspect of controlling the CMD mathematically. This work studies the dynamics of cassava mosaic disease by considering cassava resistant breed which only catch cassava mosaic disease through unhealthy cutting and susceptible breed which catch mosaic disease through unhealthy cutting and contact from whitefly vector when the controls are implemented.

CHAPTER THREE

MATERIALS AND METHODS

3.1 Methods on Formulation of the Basic Model

The study adopt an epidemiological process which is categorized into subpopulation. It includes cassava plants which consists of three compartments (resistant breeds, susceptible breed as well as infectious cassava) and the second subpopulation is vector population, which consists of two compartments of susceptible vector and infectious vector. The system of ordinary differential equation is formulated by considering the rate of change with time for each compartment. The basic properties of the model which includes positivity of the solution and invariant region were computed in order to prove if the model is mathematically and epidemiologically meaningfully. The disease free equilibrium was shown and the basic reproduction number R_0 was computed by using the next generation matrix. The disease stability at cassava mosaic free equilibrium was investigated by using Metzler matrix. Lyapunovs direct method and LaSalles invariant principle were used to determine stability of endemic equilibrium. The forward normalized sensitivity index was used to compute sensitivity index for each parameter. All methods are explained below:

3.2 Next Generation Method

Next generation method is used to compute the basic reproduction number R_0 and the effective basic reproduction number R_0^c, in this method we assume $f_i(\bar{x})$ is the rate of cassava and whitefly new infections and $V_i(\bar{x}) = V_i^-(\bar{x}) - V_i^+(\bar{x})$, where V_i^+, are the terms that are transferred into the compartment and V_i^- is the terms that are transferred out of the compartment. From this we obtain F and V by differentiating f_i and V_i respectively with respect to the infected classes.

$$F = \frac{\partial f_i(x_0)}{\partial(x_j)} \quad \text{and} \quad V = \frac{\partial V_i(x_0)}{\partial(x_j)}, \tag{3.1}$$

then the basic reproduction number R_0 is given as the maximum eigenvalue of FV^{-1} (Van den Driessche and Watmough, 2002).

3.3 Linearization Method

Local stability of cassava mosaic free equilibrium is proved by using the linearization method, the ordinary differential equation formulates above was linearized and the Jacobean matrix was obtained by differentiating the model system with respect to $S_r, S_C, I_C, S_V), I_V)^T$. Cassava

mosaic free equilibrium is said to be locally asymptotically stable if Jacobean matrix has a negative eigenvalue (Routh, 1877) or negative and positive determinant.

3.4 Metzler Matrix Method

The global stability at cassava mosaic free equilibrium is established by approach used by (Castillo-Chavez *et al.*, 2002). When this approach is used, system (4.1a) - (4.1e) is written as follows:

$$\frac{dX_1}{dt} = H\left(X_1 - X_{F_0}\right) H_1 X_2, \tag{3.2}$$

$$\frac{dX_2}{dt} = GX_2, \tag{3.3}$$

Where x_1 presents the noninfectious classes and x_2 infectious class, $X_{(F_0)}$ present mosaic free equilibrium. Therefore mosaic free equilibrium is said to be globally asymptotically stable if eigenvalues of matrix H are negative and matrix G is a Metzler matrix.

3.5 Lyapunov Direct Method

Stability of cassava mosaic equilibrium is investigated by Lyapunov and LaSalles invariant principle (Kahuru *et al.*, 2017). The system of differential equation which is combined with Lyapunov equation. By using Lyapunov logarithmic function which is given by:

$$L = \sum G_i \left(P_i - P_i^* lnP_i\right), \tag{3.4}$$

where G_1, is a positive constant which is to be chosen carefully, P_i is a variable in a compartment i and P^* present a compartment variable at equilibrium point. At endemic equilibrium point is said to be globaly stable when the rate of change of Lyapunov logarithmic function L is negative for all value.

3.6 Sensitivity Analysis

In this part, we use the forward normalized sensitivity index to determine which parameters are sensitive to the disease, which is given by:

$$\Upsilon_f^{R_0} = \frac{dR_0}{df} \times \frac{f}{R_0}, \tag{3.5}$$

Where: R_0 is the basic reproduction number and f is a parameter in basic reproduction number R_0.

3.7 Data Collection and Model Simulation

The study has used different parameters values from the literature and assumed ones as they are summarized in Table 3. In numerical analysis, MATLAB R2014a software was used to in order to validate and study the model.

CHAPTER FOUR

RESULTS AND DISCUSSION

4.1 Model Formulation

The model is formulated by modifying the model which was developed by Holt *et al.* (1997) to include breed which catches cassava mosaic disease through unhealthy cutting and susceptible breed that catches mosaic disease through unhealthy cutting and through contact with whitefly vector. The model consists of two groups of population. The first group includes the cassava population (N_C) which is divided into resistant (S_r) and Susceptible (S_C) breeds, and infected cassava (I_C). Second group includes the whitefly vector population (N_V) which consists of susceptible vector (S_V) and infectious vector (I_V).

Resistant breed is replanted at a rate r_1 and is infected by cassava mosaic disease through unhealthy cutting at a rate β_1 and they are harvested at a rate ρ_1. The term k_1, represents the maximum plants for resistant breed which can be planted. Susceptible breed of cassava is replanted at a rate r_2, and is infected by cassava mosaic disease following contact with infected whitefly vector and unhealthy cutting at a rate β_2 while it is harvested at a rate ρ_2. The maximum plants of susceptible breed that can be planted is k_2. Infected cassava flourish following infection of resistant breed through unhealthy cutting at a rate β_1, and infection of susceptible breed through unhealthy cutting and contact with infected whitefly vector at a rate β_2 and they decrease due to the effect of cassava mosaic disease at a rate a and harvested at a rate ρ_3. Susceptible vector is recruited by birth at a rate b and catch infection following contact with infected cassava at a rate β_2. Also, k_3 is the maximum number of vectors that can be supported. Infected vector is recruited when susceptible vector catch infection following contact with infected cassava at a rate β_3 and γ is the death rate of whitefly vector.

4.1.1 Assumptions of the Model

The model has following assumptions:

(i) All whitefly vectors are born susceptible to cassava mosaic disease.

(ii) The replanted cassava for both breeds are susceptible to CMD.

(iii) The whitefly vector transmit cassava mosaic disease to resistant breed through unhealthy cutting.

(iv) Cassava susceptible breed gets cassava mosaic disease through contact with infected whitefly and through unhealthy cutting.

(v) Susceptible vectors can be infected when they come into contact with the infected cassava.

Interaction between cassava and vector population is shown in Fig. 3. Variables and parameters are described in Table 1 and 2; respectively.

Table 1: Variables' Description

Variables	**Description**
S_r	Resistant breed cassava at time t.
S_C	The susceptible population of cassava at time t.
I_C	The infectious population of cassava at time t.
S_V	The susceptible vectors population at time t.
I_V	The infectious vector population at time t.

Table 2: Parameters' Descriptions

Variables	**Description**
r_1	Rate of planting resistant breed.
ρ_1	The rate of harvesting resistant breed.
β_1	The rate of resistant breed become infected.
r_2	Rate of planting susceptible breed.
ρ_2	The rate of harvesting susceptible breed of cassava.
β_2	The rate of susceptible breed become infected.
ρ_3	The rate of harvesting infectious cassava.
a	The rate of loss of infected cassava due to disease.
b	Recruitment rate for whitefly vectors.
β_3	Vector infection rate .
γ	The death rate of whitefly vectors.
k_l	The maximum number of resistant breed that can be planted.
k_2	The maximum number of susceptible breed that can be planted.
k_3	Maximum number of vectors that can be supported.

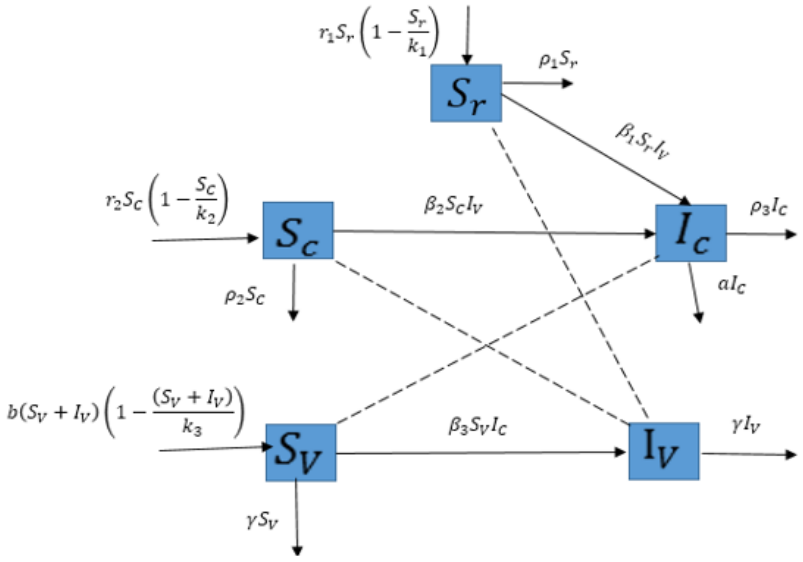


Figure 3: Compartmental Model for the transmission of Cassava Mosaic Disease.

4.1.2 Model Equations for the Two Groups

$$\frac{dS_r}{dt} = r_1 S_r\left(1-\frac{S_r}{k_1}\right) - \beta_1 S_r I_V - \rho_1 S_r, \tag{4.1a}$$

$$\frac{dS_C}{dt} = r_2 S_C\left(1-\frac{S_C}{k_2}\right) - \beta_2 S_C I_V - \rho_2 S_C, \tag{4.1b}$$

$$\frac{dI_C}{dt} = \beta_2 S_c I_v + \beta_1 S_r I_v - \rho_3 I_c - a I_c, \tag{4.1c}$$

$$\frac{dS_V}{dt} = b\left(S_V + I_V\right)\left(1-\frac{S_V+I_V}{k_3}\right) - \beta_3 S_V I_C - \gamma S_V, \tag{4.1d}$$

$$\frac{dI_V}{dt} = \beta_3 S_V I_C - \gamma I_V, \tag{4.1e}$$

Subject to $S_r > 0, S_C > 0, I_C \geq 0, S_V \geq 0, I_V \geq 0$.

The total population of cassava is given as $S_r + S_C + I_C = N_C$ and the total population of vector is given as $N_V = S_V + I_V$.

4.2 Basic Properties of the Model

4.2.1 Invariant Region

Metzer matrix is used to show the feasible region, in which the variables are positive $\forall t \geq 0$. To deduce the feasible region; the model system (4.1a) - (4.1e) can be written as:

$$\frac{dx}{dt} = Ax + F, \tag{4.2}$$

where $x = (S_r, S_C, I_C, S_V, I_V)^T$ and a constant term $F = (0,0,0,0,0)^T$ such that:

$$Ax = \begin{pmatrix} -q_1 & 0 & 0 & 0 & 0 \\ 0 & -q_2 & 0 & 0 & 0 \\ \beta_1 I_V & \beta_2 I_V & -q_3 & 0 & (\beta_2 S_C + \beta_1 S_r) \\ 0 & 0 & 0 & -q_4 & (b - 2\frac{(S_V+I_V)}{k_3}) \\ 0 & 0 & 0 & \beta_3 I_V & -\gamma \end{pmatrix}, \tag{4.3}$$

where;
$q_1 = \beta_1 I_V + \rho_1 - r_1\left(1 - 2\frac{S_r}{k_1}\right)$, $q_2 = \beta_2 I_V + \rho_2 - r_2\left(1 - 2\frac{S_C}{k_2}\right)$,
$q_3 = \rho_3 + a$, $q_4 = \gamma + \beta_3 I_C - b - 2\frac{(S_V+I_V)}{k_3}$.

In equation (4.3), A is a Metzler matrix $\forall x \in \mathbb{R}^5_+$ and due to the fact that $F \geq 0$, the model system (4.1a) - (4.1e) is positive invariant in $\mathbb{R}^5_+$ and F is Lipschitz continuous. Therefore the feasible region Ω is a set of $\Omega = \{S_r, S_C, I_C, S_V, I_V \in \mathbb{R}^5_+\}$ with initial condition $S_r > 0$, $S_C > 0$, $I_C \geq 0$, $S_V > 0$, $I_V \geq 0$.

4.2.2 Positivity of the Solutions

Let the initial condition be $S_r(0), S_C(0), I_C(0), S_V(0), I_V(0)$, the solution of model system (4.1a) - (4.1e) of set S_r, S_C, I_C, S_V, I_V is positive $\forall t > 0$. We show that, the solution of the model system (4.1a) - (4.1e) are positive by starting with equation (4.1a) that:

$$\frac{dS_r}{dt} \geq -(\beta_1 S_r I_V + \rho_1 S_r), \tag{4.4}$$

Separate the variables and integrate both sides of the equation,

$$\int \frac{1}{S_r} \mathrm{d}S_r \geq \int -(\beta_1 I_v + \rho_1)\, \mathrm{d}t, \tag{4.5}$$

$$\ln(S_r) \geq -(\beta_1 I_v + \rho_1)\, t + C. \tag{4.6}$$

This gives the values of S_r as:

$$S_r(t) \geq A e^{-(\beta_1 I_v + \rho_1)t}. \tag{4.7}$$

At initial condition time, $t = 0$, equation (4.7) above becomes

$$S_r(0) \geq A, \tag{4.8}$$

Therefore

$$S_r(t) \geq S_r(0)\, e^{-(\beta_1 I_v + \rho_1)t}, \tag{4.9}$$

Thus, $S_r(0) \geq 0, \forall t > 0$.

Apply the same procedure to the remaining equations (4.1a) - (4.1e).
We get:

$$S_C(t) \geq S_C(0)\, e^{-(\beta_2 I_v + \rho_2)t}. \tag{4.10}$$

$$I_C(t) \geq I_C(0)\, e^{-(\rho_3 + a)t}. \tag{4.11}$$

$$S_V(t) \geq S_V(0)\, e^{-(\beta_3 I_c + \gamma)t}. \tag{4.12}$$

$$I_v(0) \geq I_v(0)\, e^{-\gamma t}. \tag{4.13}$$

Here we conclude that, the requirement to study the dynamics of CMD is satisfied due to the fact that all the solutions of the model (4.1a) - (4.1e) are positive and bounded in the region:

$$\Omega = \{S_r(t), S_C(t), I_C(t), S_V(t), I_V(t)\}. \tag{4.14}$$

4.3 Model Analysis

4.3.1 Cassava Mosaic Free Equilibrium

The steady state when there is no cassava mosaic disease is called cassava mosaic free equilibrium. We compute cassava mosaic free equilibrium when $I_C = I_V = 0$. At this state the total cassava plants is the sum of susceptible and resistant breeds. However, the population of the vector at this state consists of susceptible whitefly vector. Cassava mosaic free equilibrium is given by:

$$F^0(S_r, S_C, I_C, S_V, I_V) = \left(\frac{(r_1 - \rho_1)\, k_1}{r_1}, \frac{(r_2 - \rho_2)\, k_2}{r_2}, 0, \frac{(b - \gamma)\, k_3}{b}, 0 \right). \tag{4.15}$$

4.3.2 Basic Reproduction Number R_0

The basic reproduction number is denoted by R_0, refers to an expected number of secondary infections from an infected whitefly when introduced into a susceptible population of cassava plants (Heffernan *et al.*, 2005). If $R_0 > 1$, the infectious whitefly can transmit the cassava mosaic disease to more than one cassava plants, and if $R_0 < 1$, an infectious whitefly transmits the cassava mosaic disease to less than one cassava plants, hence the disease is clearing out. The basic reproductive number R_0 will be determined by next generation matrix (Van den Driessche and Watmough, 2002).

Assume that, $f_i(\bar{x})$ is the rate of cassava and whitefly new infections and $V_i(\bar{x}) = V_i^-(\bar{x}) - V_i^+(\bar{x})$

transfer terms, where V_i^+ are the terms which are transferred into the compartment and $V_i^-(\bar{x})$ are the terms that are transferred out of the compartment such that: (Heffernan *et al.*, 2005).

$$F = \frac{\partial f_i(x_0)}{\partial(x_j)} \quad and \quad V = \frac{\partial V_i(x_0)}{\partial(x_j)}, \tag{4.16}$$

where $i, j = 1, 2, ..., m$ and x_0 indicates the cassava mosaic free equilibrium. From the model system (3.1), f_i and V_i are defined by:

$$f_i = \begin{pmatrix} \beta S_r I_V + \beta_2 S_C I_V \\ \beta_3 S_V I_C \end{pmatrix}, \tag{4.17}$$

and

$$V_i = \begin{pmatrix} \rho_3 + aI_C \\ \gamma I_V \end{pmatrix}. \tag{4.18}$$

F and V are obtained by differentiating equation (4.17) and (4.18) with respect to I_C and I_V so that:

$$F = \begin{pmatrix} 0 & \beta S_r + \beta_2 S_C \\ \beta_3 S_V & 0 \end{pmatrix}, \tag{4.19}$$

and

$$V = \begin{pmatrix} \rho_3 + aI_C & 0 \\ 0 & \gamma \end{pmatrix}. \tag{4.20}$$

The next generation matrix is given by,

$$FV^{-1} = \begin{pmatrix} 0 & \frac{\beta_2 S_C + \beta_1 S_r}{\gamma} \\ \frac{\beta_3 S_V}{\rho_3 + a} & 0 \end{pmatrix}. \tag{4.21}$$

The basic reproduction number R_0 for cassava plants and vector is a dominant eigenvalue of the next generation matrix FV^{-1} (Van den Driessche and Watmough, 2002). The basic reproduction number R_0 is therefore given by:

$$R_0 = \sqrt{\frac{\beta_3 (b-\gamma) k_3}{b(\rho_3 + a)\gamma} \left(\frac{(r_1 - \rho_1) k_1 \beta_1}{r_1} + \frac{(r_2 - \rho_2) k_2 \beta_2}{r_2} \right)}. \tag{4.22}$$

From equation (4.22), basic reproduction number R_0 is determined by all parameters from the model. The basic reproduction number R_0 increases in proportion to vector infection rate β_3,

recruitment rate for whitefly b, maximum number of vectors that can be supported k_3, the rate at which resistant breed becomes infected β_1, the rate at which susceptible breed becomes infected β_2, the maximum number of resistant breed that can be planted k_1, rate of planting resistant breed r_1, the maximum number of susceptible breed that can be planted k_2 and the rate at which susceptible breed of cassava is replanted r_2. It decreases as the death rate of whitefly vectors γ, the rate of harvesting infectious cassava ρ_3, the rate of harvesting susceptible breed of cassava ρ_2, the rate of loss of infected cassava due to disease a and the rate of harvesting resistant breed ρ_1 increase.

4.3.3 Local stability Analysis of Cassava Mosaic Free Equilibrium

To prove local stability of cassava mosaic free equilibrium, we use linearization method. In this case the system (4.1a) - (4.1e) is linearized at cassava mosaic free equilibrium to obtain a Jacobian matrix. If the Jacobian matrix has negative eigenvalues or negative real part, then cassava mosaic free equilibrium is said to be locally asymptotically stable. From system (4.1a) - (4.1e), Jacobian matrix at cassava mosaic free equilibrium is given by:

$$J(F^0) = \begin{pmatrix} -(r_1-\rho_1) & 0 & 0 & 0 & a_1 \\ 0 & -(r_2-\rho_2) & 0 & 0 & a_2 \\ 0 & 0 & -(\rho_3+a) & 0 & a_3 \\ 0 & 0 & -(\beta_3\frac{(b-\gamma)k_3}{b}) & -(b-\gamma) & a_4 \\ 0 & 0 & \beta_3\frac{(b-\gamma)k_3}{b} & 0 & -\gamma \end{pmatrix}, \tag{4.23}$$

where $a_1 = -\frac{\beta_1 k_1(r_1-\rho_1)}{r_1}, a_2 = -\frac{\beta_2 k_2(r_2-\rho_2)}{r_2}, a_3 = (\frac{\beta_1 k_1(r_1-\rho_1)}{r_1} + \frac{\beta_2 k_2(r_2-\rho_2)}{r_2})$,
$a_4 = b - \frac{2b(\frac{(b-\gamma)k_3}{b})}{k_3}$.

From matrix (4.23), it is clear from first, second and third columns that the eigenvalues are: $-(r_1-\rho_1)$, $-(r_2-\rho_2)$ and $-(b-\gamma)$ respectively. The matrix then reduces to:

$$J(F^0) = \begin{pmatrix} -(\rho_3+a) & (\frac{\beta_1 k_1(r_1-\rho_1)}{r_1} + \frac{\beta_2 k_2(r_2-\rho_2)}{r_2}) \\ \frac{\beta_3 k_3(b-\gamma)}{b} & -\gamma \end{pmatrix}. \tag{4.24}$$

The determinant and trace are then used to determine the sign of the remaining eigenvalues. From matrix (4.24):

$$TrJ^1(F^0) = -(\rho_3+a+\gamma), \tag{4.25}$$

and

$$DetJ^1(F^0) = \gamma(\rho_3+a)(1-(R_0)^2). \tag{4.26}$$

According to RouthHurwitz stability criterion the necessary and sufficient condition for a stability of any system, all the factors of characteristic polynomial of a system must be negative (Routh, 1877). Since the Eigenvalue of matrix above are negative hence at cassava mosaic free equilibrium it is asymptotically stable when basic reproduction number $R_0 < 1$ and it is unstable when $R_0 > 1$.

4.3.4 The Global Stability Analysis of the Disease Free Equilibrium

The global stability of cassava mosaic free equilibrium is established by approach used by Castillo-Chavez *et al.* (2002). When this approach is used, system (4.1a) - (4.1e) is written as follows:

$$\frac{dX_1}{dt} = H\left(X_1 - X_{F_0}\right) H_1 X_2, \tag{4.27}$$

$$\frac{dX_2}{dt} = GX_2, \tag{4.28}$$

where X_1 presents the noninfectious classes and X_2 infectious classes. $X_{(F_0)}$ present mosaic free equilibrium. Mosaic free equilibrium is said to be globally asymptotically stable if eigenvalues of matrix H are negative and matrix G is a Metzler matrix (Irunde *et al.*, 2017). We thus define X_1, X_2 and X_{F_0} as follows:

$$X_1 = \begin{pmatrix} S_r \\ S_C \\ S_V \end{pmatrix}. \tag{4.29}$$

$$X_2 = \begin{pmatrix} I_C \\ I_V \end{pmatrix}. \tag{4.30}$$

$$X_{F_0} = \begin{pmatrix} \frac{(r_1-\rho_1)k_1}{r_1} \\ \frac{(r_2-\rho_2)k_2}{r_1} \\ 0 \\ \frac{(b-\gamma)k_3}{b} \\ 0 \end{pmatrix}. \tag{4.31}$$

Matrices H_1 and H are defined by:

$$H_1 = \begin{pmatrix} 0 & -\beta_1 S_r \\ 0 & -\beta_2 S_C \\ -\beta_3 S_V & b - \frac{2b(S_V+I_V)}{k_3} \end{pmatrix} \tag{4.32}$$

and

$$H = \begin{pmatrix} -q_1 & 0 & 0 \\ 0 & -q_2 & 0 \\ 0 & 0 & -q_3 \end{pmatrix}, \tag{4.33}$$

where
$q_1 = (r_1 + 2\frac{r_1 S_r}{k_1} + \beta_1 I_V + \rho_1)$, $q_2 = (r_2 + 2\frac{r_2 S_C}{k_2} + \beta_2 I_V + \rho_2)$, $q_3 = (b + \frac{2b(S_V + I_V)}{k_3} + \beta_3 I_C + \gamma)$.
Matrix G is also given by:

$$G = \frac{\partial (X_2)}{\partial (I_C, I_V)^T} = \begin{pmatrix} -(\rho_3 + a) & \beta_1 \frac{(r_1 - \rho_1)k_1}{r_1} + \beta_2 \frac{(r_2 - \rho_2)k_2}{r_2} \\ \beta_3 \frac{(b-\gamma)k_3}{b} & -\gamma \end{pmatrix}. \tag{4.34}$$

Matrix (4.32) has a real and non-positive eigenvalue and in matrix (4.34) all the diagonal elements are negative and the off diagonal elements are positive. Therefore, when the basic reproduction number R_0 of a disease, is less than one $(R_0 < 1)$ and greater than one $(R_0 > 1)$, then the disease free equilibrium point is said to be globally asymptotically stable and unstable respectively.

4.4 Cassava Mosaic Equilibrium

For the mosaic disease to continue to exist in the cassava population, $I_C(t) \neq 0$ and $I_V(t) \neq 0$, from that the equilibrium point of our model called endemic equilibrium point which is denoted by $F_1 = (S_r{}^*, S_C{}^*, I_C{}^*, S_V{}^*, I_V{}^*) \neq (0,0,0,0,0)$. For F_1 to exist, at least one infected class should not be zero. We obtain endemic equilibrium when the rate of change of each variable is equal to zero. To solve for endemic equilibrium, the system (4.1a)- (4.1e) is written as:

$$\begin{aligned} 0 &= r_1 S_r \left(1 - \frac{S_r}{k_1}\right) - \beta_1 S_r I_V - \rho_1 S_r, \\ 0 &= r_2 S_C \left(1 - \frac{S_C}{k_2}\right) - \beta_2 S_C I_V - \rho_2 S_C, \\ 0 &= \beta_2 S_c I_v + \beta_1 S_r I_v - \rho_3 I_c - a I_c, \\ 0 &= b(S_V + I_V)\left(1 - \frac{S_V + I_V}{k_3}\right) - \beta_3 S_V I_C - \gamma S_V, \\ 0 &= \beta_3 S_V I_C - \gamma I_V, \end{aligned} \tag{4.35}$$

Thus,

$$S_r{}^* = \frac{(a + \rho_3) I_C^* - S_C^* \beta_2 I_V^*}{\beta_1 I_V^*}. \tag{4.36}$$

$$S_C{}^* = \frac{(a+\rho_3)\, I_{C^*} - S_{r^*}\beta_1 I_V^*}{\beta_2 I_V^*}. \tag{4.37}$$

$$I_C{}^* = \frac{-bS_V{}^{2*} + ((-2I_V^* + k_3)\, b - \gamma k_3)\, S_V^* - bI_V^*\,(I_V^* - k_3)}{k_3\beta_3 S_V^*}. \tag{4.38}$$

$$S_V^* = 1/2\,\frac{\sqrt{k_3\left(\left(-\beta_3 I_C^* + b - \gamma\right)^2 k_3 + 4bI_V^*\left(\beta_3 I_C^* + \gamma\right)\right)} + \left(-\beta_3 I_C^* + b - \gamma\right) k_3 - 2bI_V^*}{b}. \tag{4.39}$$

$$I_V{}^* = 1/2\,\frac{\sqrt{\left(bk_3 - 4S_V^*\left(\beta_3 I_C^* + \gamma\right)\right) bk_3} + (-2S_V^* + k_3)\, b}{b}. \tag{4.40}$$

The equilibrium point is non-negative if:

$$(a+\rho_3)I_C > S_C\beta_2 I_V, \tag{4.41}$$

$$(a+\rho_3)I_C > S_r\beta_1 I_V, \tag{4.42}$$

$$(bS_V k_3 + bI_{Vk_3}) > (bS_V{}^2 + 2bS_V I_V + bI_V{}^2 + \gamma S_V k_3), \tag{4.43}$$

$$\sqrt{k_3\left((-\beta_3 I_C + b - \gamma)^2 k_3 + 4bI_V\left(\beta_3 I_C + \gamma\right)\right)} + k_3 b > -(\beta_3 I_C k_3 + 2bI_V + \gamma k_3) \tag{4.44}$$

and

$$\sqrt{\left(bk_3 - 4S_V^*\left(\beta_3 I_C^* + \gamma\right)\right) bk_3} + k_3 b > -2S_V \tag{4.45}$$

4.5 Global Stability of Cassava Mosaic Equilibrium

Stability of cassava mosaic equilibrium is investigated logarithmic Lyapunov function which is given by:

$$L = \sum G_i\,(P_i - P_i{}^* lnP_i)\,, \tag{4.46}$$

where G_1, is a positive constant which is to be chosen carefully, P_i is a variable in a compartment i and P^* present a compartment variable at equilibrium point. Using system (4.46) the Lyapunov function is defined by:

$$\begin{aligned} L(S_r S_C, I_C, S_V, I_V) =& G_1\,(S_r - S_r{}^* lnS_r) + G_2\,(S_C - S_C{}^* lnS_C) + G_3\,(I_C - I_C{}^* lnI_C) \\ &+ G_4\,(S_V - S_V{}^* lnS_V) + G_5\,(I_V - I_V{}^* lnI_V)\,. \end{aligned} \tag{4.47}$$

Differentiate the Lyapunov function (4.47)above with respect to time, we get:

$$\begin{aligned}\frac{dL}{dt} =& G_1\left(1-\frac{S_r^*}{S_r}\right)\frac{dS_r}{dt}+G_2\left(1-\frac{S_C^*}{S_C}\right)\frac{dS_C}{dt}+G_3\left(1-\frac{I_C^*}{I_C}\right)\frac{dI_C}{dt}\\ &+G_4\left(1-\frac{S_V^*}{S_V}\right)\frac{dS_V}{dt}+G_5\left(1-\frac{I_V^*}{I_V}\right)\frac{dI_V}{dt}.\end{aligned} \tag{4.48}$$

From equation 4.48 we have:

$$\begin{aligned}\frac{dL}{dt} =& G_1\left(1-\frac{S_r^*}{S_r}\right)(r_1S_r\left(1-\frac{S_r}{k_1}\right)-\beta_1S_rI_V-\rho_1S_r)\\ &+G_2\left(1-\frac{S_C^*}{S_C}\right)(r_2S_C\left(1-\frac{S_C}{k_2}\right)-\beta_2S_CI_V-\rho_2S_C)\\ &+G_3\left(1-\frac{I_C^*}{I_C}\right)(\beta_2S_CI_V+\beta_1S_rI_V-\rho_3I_C-aI_C)\\ &+G_4\left(1-\frac{S_V^*}{S_V}\right)(b\,(S_V+I_V)\left(1-\frac{S_V+I_V}{k_3}\right)-\beta_3S_VI_C-\gamma S_V)\\ &+G_5\left(1-\frac{I_V^*}{I_V}\right)(\beta_3\ S_VI_C-\gamma I_V).\end{aligned} \tag{4.49}$$

At cassava mosaic equilibrium, we have:

$$\begin{aligned}\frac{dL}{dt} =& G_1\left(1-\frac{S_r^*}{S_r}\right)((-r_1S_r^*\left(1-\frac{S_r^*}{k_1}\right)+\beta_1S_r^*I_V+\rho_1S_r^*)+(r_1S_r\left(1-\frac{S_r}{k_1}\right)\\ &-\beta_1S_rI_V-\rho_1S_r))+G_2\left(1-\frac{S_C^*}{S_C}\right)(((r_2S_C^*\left(1-\frac{S_C^*}{k_2}\right)\\ &-\beta_2S_C^*I_V-\rho_2S_C^*))+(r_2S_C\left(1-\frac{S_C}{k_2}\right)-\beta_2S_Ci_V-\rho_2S_C))\\ &+G_3\left(1-\frac{I_C^*}{I_C}\right)(((\beta_2S_CI_V^*+\beta_1S_rI_V^*-\rho_3I_C-aI_C^*))\\ &+(\beta_2S_CI_V+\beta_1S_rI_V-\rho_3I_C-aI_C))\\ &+G_4\left(1-\frac{S_V^*}{S_V}\right)(((b\,(S_V^*+I_V)\left(1-\frac{S_V^*+I_V}{k_3}\right)-\beta_3S_V^*I_C-\gamma S_V^*))\\ &+(b\,(S_V^*+I_V)\left(1-\frac{S_V^*+I_V}{k_3}\right)-\beta_3S_V^*I_C-\gamma S_V^*))\\ &+G_5\left(1-\frac{I_V^*}{I_V}\right)((\beta_3\ S_VI_C^*-\gamma I_V^*)+(\beta_3\ S_VI_C-\gamma I_V)).\end{aligned} \tag{4.50}$$

Manipulation of equation (4.50) gives:

$$\begin{aligned}\frac{dL}{dt} =& G_1\left(1-\frac{S_r^*}{S_r}\right)\left(-r_1S_r^*\left(1-\frac{S_r^*}{k_1}\right)+r_1S_r\left(1-\frac{S_r}{k_1}\right)+(\beta_1I_V+\rho_1)S_r^*\right.\\ &\left.-(\beta_1I_V+\rho_1)S_r\right)+G_2\left(1-\frac{S_C^*}{S_C}\right)\left(-r_2S_C^*\left(1-\frac{S_C^*}{k_2}\right)\right.\\ &\left.+r_2S_C\left(1-\frac{S_C}{k_2}\right)+(\beta_2I_V+\rho_2)S_C^*-(\beta_2I_V+\rho_2)S_C\right.\\ &\left.+G_3\left(1-\frac{i_C^*}{I_C}\right)\left((\rho_3+a)I_C^*-(\rho_3+a)I_C\right)+G_4\left(1-\frac{S_V^*}{S_V}\right)\right.\\ &\left.\left(-b\left(S_V^*+I_V\right)\left(1-\frac{S_V^*+I_V}{k_3}\right)+b\left(S_V+I_V\right)\left(1-\frac{S_V+I_V}{k_3}\right)\right.\right.\\ &\left.\left.+(\beta_3I_C+\gamma)S_V^*-(\beta_3I_C+\gamma)S_V\right)+G_5\left(1-\frac{I_V^*}{I_V}\right)(\gamma I_V^*-\gamma I_C).\end{aligned} \tag{4.51}$$

On simplifying, we get:

$$\begin{aligned}\frac{dL}{dt} =& G_1\left(1-\frac{S_r^*}{S_r}\right)\left(-r_1S_r^*\left(1-\frac{S_r^*}{k_1}\right)+r_1S_r\left(1-\frac{S_r}{k_1}\right)\right)-G_1(1-\frac{S_r^*}{S_r})^2\\ &+G_2\left(1-\frac{S_C^*}{S_C}\right)\left(-r_2S_C^*\left(1-\frac{S_C^*}{k_2}\right)\right)+r_2S_C\left(1-\frac{S_C}{k_2}\right)-G_2(1-\frac{S_C^*}{S_C})^2\\ &-G_3\left(1-\frac{I_C^*}{I_C}\right)^2+G_4\left(1-\frac{S_V^*}{S_V}\right)\left(-b\left(S_V^*+I_V\right)\left(1-\frac{S_V^*+I_V}{k_3}\right)\right.\\ &\left.+b\left(S_V+I_V\right)\left(1-\frac{S_V+I_V}{k_3}\right)-G_4(1-\frac{S_V^*}{S_V})^2-G_5\left(1-\frac{I_V^*}{I_V}\right)^2.\right.\end{aligned} \tag{4.52}$$

Now Arrange equation 4.52, we get

$$\begin{aligned}\frac{dL}{dt} =& -G_1\left(1-\frac{S_r^*}{S_r}\right)^2-G_2(1-\frac{S_C^*}{S_C})^2-G_3\left(1-\frac{I_C^*}{I_C}\right)^2-G_4(1-\frac{S_V^*}{S_V})^2\\ &-G_5\left(1-\frac{I_V^*}{I_V}\right)^2+G_1\left(1-\frac{S_r^*}{S_r}\right)\left(-r_1S_r^*\left(1-\frac{S_r^*}{k_1}\right)+r_1S_r\left(1-\frac{S_r}{k_1}\right)\right)\\ &+G_2\left(1-\frac{S_C^*}{S_C}\right)\left(-r_2S_C^*\left(1-\frac{S_C^*}{k_2}\right)+r_2S_C\left(1-\frac{S_C}{k_2}\right)\right)+G_4\left(1-\frac{S_V^*}{S_V}\right)\\ &-b\left(S_V^*+I_V\right)\left(1-\frac{S_V^*+I_V}{k_3}\right)+b\left(S_V+I_V\right)\left(1-\frac{S_V+I_V}{k_3}\right),\end{aligned} \tag{4.53}$$

which can be written as

$$\frac{dL}{dt} = -G_1\left(1-\frac{S_r^*}{S_r}\right)^2 - G_2(1-\frac{S_C^*}{S_C})^2 - G_3\left(1-\frac{I_C^*}{I_C}\right)^2 - G_4(1-\frac{S_V^*}{S_V})^2 - G_5\left(1-\frac{I_V^*}{I_V}\right)^2 + F(\Omega), \tag{4.54}$$

where

$$\begin{aligned} F(\Omega) =& G_1\left(1-\frac{S_r^*}{S_r}\right)\left(-r_1 S_r^*\left(1-\frac{S_r^*}{k_1}\right)+r_1 S_r\left(1-\frac{S_r}{k_1}\right)\right) \\ &+ G_2\left(1-\frac{S_C^*}{S_C}\right)\left(-r_2 S_C^*\left(1-\frac{S_C^*}{k_2}\right)+r_2 S_C\left(1-\frac{S_C}{k_2}\right)\right)+G_4\left(1-\frac{S_V^*}{S_V}\right) \\ &- b\left(S_V^*+I_V\right)\left(1-\frac{S_V^*+I_V}{k_3}\right)+b\left(S_V+I_V\right)\left(1-\frac{S_V+I_V}{k_3}\right), \end{aligned} \tag{4.55}$$

and $a \leq 0$ for $\forall S_r, S_C, I_C, S_V, I_V > 0$, for the $\frac{dL}{dt}$=0 if $S_r = S_r^*, S_C = S_C^*, I_C = I_C^*, S_V = S_V^*, I_V = I_V^* = 0$ Thus the singleton which is the endemic equilibrium point is the greatest invariant set by using the LaSalles invariant principle (LaSalle, 1976). We therefore conclude that if $R_0 > 1$, then endemic equilibrium is asymptotically stable and it is unstable if $R_0 < 1$.

4.6 Sensitivity Analysis

Sensitivity index of a parameter tells how a parameter is sensitive to the disease. In this section, sensitivity index of each parameter with respect to basic reproduction number R_0 is computed to determine how each parameters influences the disease. If f is a parameter in reproduction number R_0 then, sensitivity index of f with respect to R_0 is given by:

$$\Upsilon_f^{R_0} = \frac{dR_0}{df} \times \frac{f}{R_0}. \tag{4.56}$$

4.7 Parameters Adoption

Parameter values from the literature and assumed ones are used. Table 3 summarizes the parameter values, their range and the corresponding sources.

Table 3: Parameter Values.

Parameters	Value	Range	Source
r_1	$0.025 day^{-1}$		Assumed
ρ_1	$0.005 day^{-1}$		Assumed
β_1	$0.0012 vector^{-1} day^{-1}$		Assumed
r_2	$0.2 day^{-1}$	$0.025 - 0.2$	Lawrence and Wallace (2011)
ρ_2	$0.003 day^{-1}$	$0.002 - 0.004$	Holt *et al.* (1997)
β_2	$0.003 vector^{-1} day^{-1}$	$0.002 - 0.032$	Holt *et al.* (1997)
ρ_3	$0.003 day^{-1}$	$0.002 - 0.004$	Holt *et al.* (1997)
a	$0.033 day^{-1}$	$0 - 0.033$	Holt *et al.* (1997)
b	$0.5 vector^{-1} day^{-1}$	$0.1 - 1.0$	Sisterson and Stenger (2015)
β_3	$0.002 plant^{-1} day^{-1}$	$0.002 - 0.032$	Holt *et al.* (1997)
γ	$0.0782 day^{-1}$	$0.06 - 0.18$	Holt *et al.* (1997)
k_1	3000		Silva *et al.* (2013)
k_2	2000		Assumed
k_3	350	0-2500	Holt *et al.* (1997)

The forward normalized sensitivity index of the rate at which susceptible breed acquire infection β_2 with respect to basic reproduction number R_0 is derived as follows:

$$\Upsilon_{\beta_2}^{R_0} = \frac{dR_0}{d\beta_2} \times \frac{\beta_2}{R_0}. \tag{4.57}$$

$$\frac{dR_0}{d\beta_2} = 1/2 \frac{\beta_3 (b-\gamma) k_3 (r_2 - \rho_2) k_2}{r_2 b (\rho_3 + a) \gamma} \frac{1}{\sqrt{\frac{\beta_3 (b-\gamma) k_3}{b(\rho_3+a)\gamma} \left(\frac{(r_1-\rho_1) k_1 \beta_1}{r_1} + \frac{(r_2-\rho_2) k_2 \beta_2}{r_2} \right)}}. \tag{4.58}$$

Using equation (4.56), we have:

$$\Upsilon_{\beta_2}^{R_0} = +0.3362. \tag{4.59}$$

The solution above shows the sensitivity index for parameter β_2. We applied the same method to obtain sensitivity indices for other parameters. Table 4 summarizes sensitivity indices for other parameters with respect to basic reproduction number R_0.

Table 4: Sensitivity Indices

Parameters	Sensitivity index	Parameters	Sensitivity index
β_3	$+0.5000$	r_2	$+0.0051$
β_1	$+0.1638$	γ	-0.5927
β_2	$+0.3362$	ρ_3	-0.0417
k_3	$+0.5000$	ρ_2	-0.0051
b	$+0.0927$	ρ_1	-0.0410
k_1	$+0.1638$	a	-0.4583
r_1	$+0.0410$	k_2	$+0.3362$

From the Table 4, the rate at which susceptible breed becomes infeced β_2, the vector infection rate β_3, the rate at which resistant breed becomes infected β_1, the maximum number of resistant breed that can be planted k_1, the maximum number of susceptible breed that can be planted k_2, maximum number of vector that can be supported k_3, recruitment for whitefly b, rate of planting resistant breed r_1, rate of replanting susceptible breed r_2 have positive indices showing that the basic reproduction number R_0 increase as the rate of susceptible breed becomes infeced β_2, the vector infection rate β_3, the rate at which resistant breed becomes infected β_1, the maximum number of resistant breed that can be planted k_1, the maximum number of susceptible breed that can be planted k_2, maximum number of vector that can be supported k_3, recruitment for whitefly b, rate of planting resistant breed r_1, rate of replanting susceptible breed r_2 increase. The most sensitive parameters are the vector infection rate β_3, maximum number of vector that can be supported k_3 and recruitment for whitefly b. Parameters such as the rate of loss of infected cassava due to disease a, the rate of harvesting resistant breed ρ_1, the rate of harvesting susceptible breed of cassava ρ_2, the rate of harvesting infectious cassava ρ_3 and the death rate of whitefly vectors γ have negative indices. This shows that the basic reproduction number will decrease when a, the rate of harvesting resistant breed ρ_1, the rate of harvesting susceptible breed of cassava ρ_2, the rate of harvesting infectious cassava ρ_3 and the death rate of whitefly vectors γ increase.

4.8 The Model with Controls for Cassava Mosaic disease

Different methods are used to control cassava mosaic disease (CMD) so as to minimize the effect of disease to plants and hence improve yields for farmers. To control the disease, farmers are encouraged to apply control strategies such as spraying of insecticide, and to use vector resistant varieties. Other methods include the phytosanitation method, which improves plant healthy and it improves crop hygiene, and removes unhealthy cassava from the farm, CMD-free stem cuttings method, as well as the removal roguing of diseased plants from within the plant standing (Thresh *et al.*, 1998).

Few authors have studied different control strategies which can be applied to eliminate cassava mosaic virus. These strategies include, the use of insecticide to kill the vector among the plants, using of vector resistant varieties: These methods decrease the number of white fly vector and transmission of cassava mosaic disease (Thresh and Cooter, 2005).

4.8.1 The Model with controls for Cassava Mosaic disease

In this model, cassava resistant breed are planted at a rate r_1, they are infected by cassava mosaic disease through unhealthy cutting at a rate $\beta_1(1-\varepsilon)$ and harvested at a rate ρ_1. Parameter ε measures the effectiveness of cleaning tools which are used to cut cassava during planting. The term k_1 represents the maximum plants for cassava resistant breed which can be planted. Cassava susceptible breed are replanted at a rate r_2, they are infected by cassava mosaic disease following contact with infected whitefly vector and unhealthy cutting at a rate $\beta_2(1-\varepsilon)$ and harvested at a rate ρ_2. The maximum plants of cassava susceptible breed that can be planted are represented by k_2.

Infected cassava flourish following infection of resistant breed through unhealthy cutting at a rate $\beta_1(1-\varepsilon)$, and infection of susceptible breed through unhealthy cutting and contact with infected whitefly vectors at a rate $\beta_2(1-\varepsilon)$. However, they decrease due to cassava mosaic disease at a rate a and harvested at a rate ρ_3. The parameter σ_2 is the rate of removal and burning of infected cassava plants.

Susceptible vector is recruited by birth at a rate b and catch infection following contact with infected cassava at a rate β_3. Also, k_3 is the maximum number of vectors that can be supported. Infected vector is recruited when susceptible vector catch infection following contact with infected cassava at a rate β_3 and γ is the natural mortality rate for whitefly vectors. The insecticides kill white fly vector both susceptible and infected at a rate σ_1.

$$
\begin{aligned}
\frac{dS_r}{dt} &= r_1 S_r\left(1-\frac{S_r}{k_1}\right) - \beta_1(1-\varepsilon)S_r I_V - \rho_1 S_r,\\
\frac{dS_c}{dt} &= r_2 S_c\left(1-\frac{S_c}{k_2}\right) - \beta_2(1-\varepsilon)S_c I_V - \rho_2 S_c,\\
\frac{dI_c}{dt} &= \beta_2(1-\varepsilon)S_c I_v + \beta_1(1-\varepsilon)S_r I_v - \rho_3 I_c - aI_c - \sigma_2 I_c,\\
\frac{dS_V}{dt} &= b\,(S_V+I_V)\left(1-\frac{S_V+I_V}{k_3}\right) - \beta_3 S_V I_c - \gamma S_V - \sigma_1 S_v,\\
\frac{dI_v}{dt} &= \beta_3 S_V I_c - \gamma I_V - \sigma_1 I_v.
\end{aligned}
\tag{4.60}
$$

Subject to initial condition $S_r > 0, S_C > 0, I_C \geq 0, S_V \geq 0, I_V \geq 0$.
The total population of cassava is given as $S_r + S_C + I_C = N_C$ and the total population of vector is given as $N_V = S_V + I_V$.

4.8.2 Cassava Mosaic Free Equilibrium When Controls are Applied.

The steady state when there is no disease is given by:

$$F^0(S_r, S_c, I_c, S_V, I_V) = \left(\frac{(r_1 - \rho_1)\, k_1}{r_1}, \frac{(r_2 - \rho_2)\, k_2}{r_2}, 0, \frac{(b - \gamma - \sigma_1)\, k_3}{b}, 0 \right). \tag{4.61}$$

4.8.3 Effective Reproduction Number R_0^c

The effective reproduction number R_0^c is used to assess the effect of control strategies. The control strategies are effective if on their administration R_0^c and ineffective if $R_0^c > 1$. Using next generation matrix operator (Van den Driessche and Watmough, 2002), the effective reproduction number R_0^c is computed by identifying new infections $\mathscr{F}_i$ and transfer terms $\mathscr{V}_i$ such that:

$$R_0^c = \rho \left(\mathscr{F} \mathscr{V}^{-1} \right). \tag{4.62}$$

From the model (4.60), new infections and transfer terms are given by:

$$\mathscr{F}_i = \begin{pmatrix} \beta_1 (1 - \varepsilon)\, S_r I_V + \beta_2 (1 - \varepsilon) S_c I_V \\ \beta_3 S_V I_c \\ \end{pmatrix} \tag{4.63}$$

and

$$\mathscr{V}_i = \begin{pmatrix} \rho_3 I_c + a I_c + \sigma_2 I_c \\ \gamma i_V + \sigma_1 I_v \end{pmatrix}. \tag{4.64}$$

The matrices $\mathscr{F}$ and $\mathscr{V}$ are:

$$\mathscr{F} = \begin{pmatrix} 0 & \beta\, S_r + \beta_2 S_c \\ \beta_3 S_V & 0 \end{pmatrix} \tag{4.65}$$

and

$$\mathscr{V} = \begin{pmatrix} \rho_3 + a + \sigma_2 & 0 \\ 0 & \gamma + \sigma_1 \end{pmatrix}. \tag{4.66}$$

From equation (4.62), the effective reproduction number R_0^c is given by:

$$R_0^c = \sqrt{\frac{\beta_3 k_3 (b-\gamma-\sigma_1)}{b(\rho_3+a+\sigma_2)(\gamma+\sigma_1)}\left(k_1\beta_1(1-\varepsilon)\frac{(r_1-\rho_1)}{r_1}+\frac{k_2\beta_2(1-\varepsilon)(r_2-\rho_2)}{r_2}\right)}. \quad (4.67)$$

The control parameters which includes the rate of spraying insecticides is to kill whitefly vector both susceptible and infected σ_1, the rate of removing and burning of infected cassava plants σ_2 and the rate of of measures effectiveness of cleaning tools which are used to cut cassava during planting ε.

The effective reproduction number R_0^c decreases as the rates of spraying insecticides is to kill white fly vectors σ_1, removal and burning of infected cassava plants σ_2 and cleaning tools which are used cut cassava during planting increase. Control strategies will eradicate the disease if when they are administered, the effective reproduction number R_0^c becomes less than unity. In the absence of control strategies that is when $\sigma_1 = \sigma_2 = \varepsilon = 0$, the effective reproduction becomes basic reproduction number R_0 which is:

$$R_0^c = \sqrt{\frac{\gamma\beta_3 k_3 (b-\gamma)}{b(\rho_3+a)}\left(k_1\beta_1(1-\varepsilon)\frac{(r_1-\rho_1)}{r_1}+\frac{k_2\beta_2(1-\varepsilon)(r_2-\rho_2)}{r_2}\right)}. \quad (4.68)$$

4.8.4 Parameters Adoption

Table 5 shows the adopted parameters and their source.

Table 5: Parameter Values.

Parameters	Value	Range	Source
r_1	$0.025 day^{-1}$		Assumed
ρ_1	$0.005 day^{-1}$		Assumed
β_1	$0.0012 vector^{-1} day^{-1}$		Assumed
r_2	$0.2 day^{-1}$	$0.025 - 0.2$	Lawrence and Wallace (2011)
ρ_2	$0.003 day^{-1}$	$0.002 - 0.004$	Holt *et al.* (1997)
β_2	$0.003 vector^{-1} day^{-1}$	$0.002 - 0.032$	Holt *et al.* (1997)
ρ_3	$0.003 day^{-1}$	$0.002 - 0.004$	Holt *et al.* (1997)
a	$0.033 day^{-1}$	$0 - 0.033$	Holt *et al.* (1997)
b	$0.5 vector^{-1} day^{-1}$	0.1-1.0	Sisterson and Stenger (2015)
β_3	$0.002 plant^{-1} day^{-1}$	$0.002 - 0.032$	Holt *et al.* (1997)
γ	$0.0782 day^{-1}$	$0.06 - 0.18$	Holt *et al.* (1997)
k_1	3000		Silva *et al.* (2013)
k_2	2000		Assumed
k_3	350	0-2500	Holt *et al.* (1997)
σ_1	0.4		Assumed
σ_2	0.7		Assumed
ε	0.5		Assumed

4.9 Numerical Simulation by Considering the Basic Model

We simulate dynamics of cassava mosaic disease when controls are not included. The result shows that susceptible whitefly vectors will catch infection within first two months which results to more mosaic disease in cassava as shown in Figs. 4, 5a and 5b.

Figure 4 demonstrate the dynamics of cassava mosaic disease. the graph shows that susceptible vectors contact the CMD before three months. This cause the number of susceptible vectors to decrease exponentially and the number of infected vector to increase. This cause the number of susceptible breed and resistant breed to decrease. As the number of susceptible cassava decrease due to CMD, it leads the increase of infected cassava. Fig. 5a and 5b demonstrate dynamics of cassava and vector population respectively.

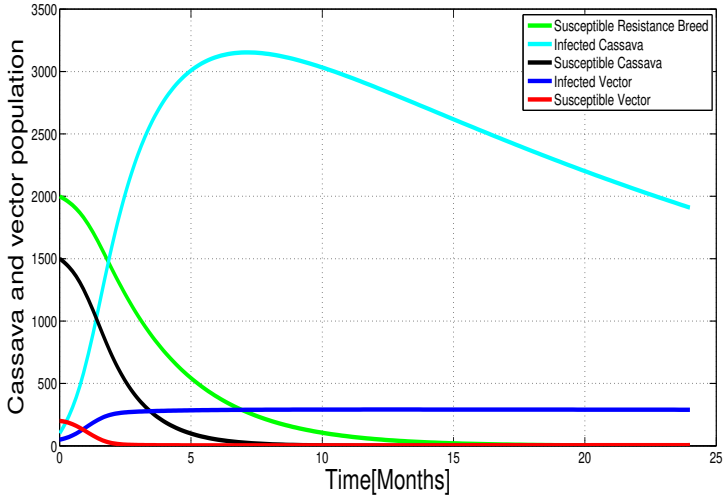
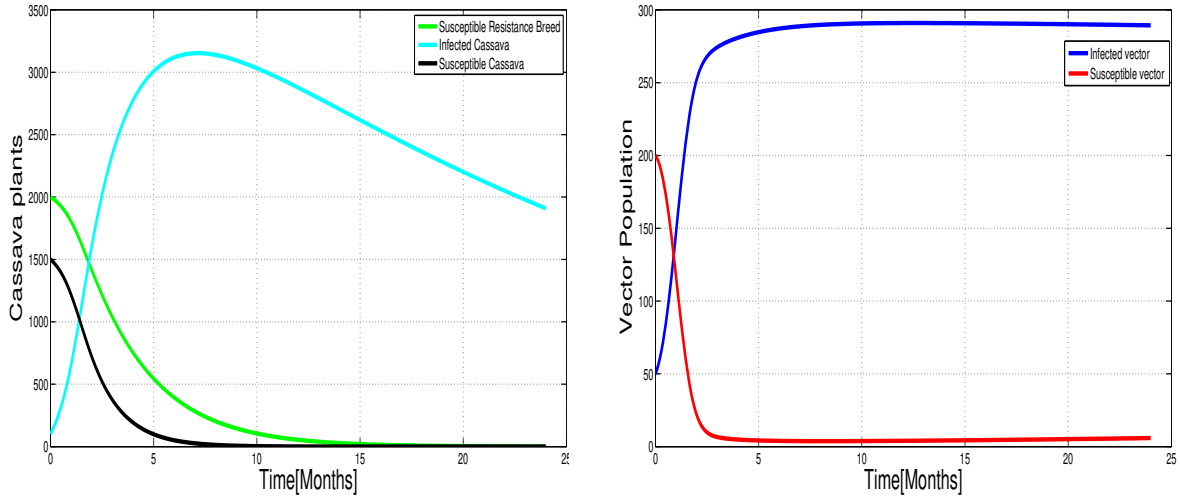


Figure 4: Dynamics of cassava mosaic disease in cassava plants and whitefly vectors.



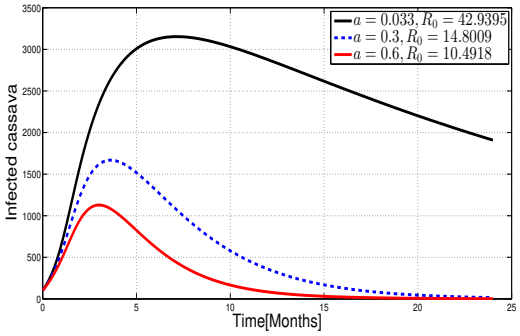
(a) Dynamics of cassava mosaic disease in cassava plants.

(b) Dynamics of cassava mosaic disease in whitefly vectors.

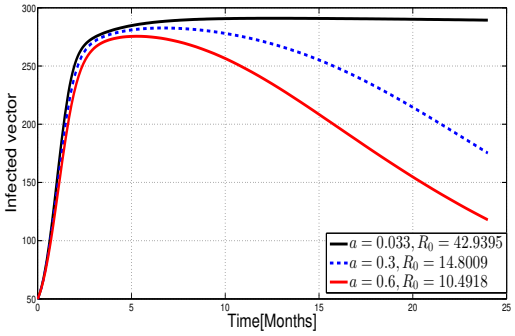
Figure 5: Total dynamics of CMD.

4.10 Numerical Simulation of Sensitive Parameters

Figure 6 demonstrates the variation of the rate of loss of infected cassava to the infected classes. It shows the behavior of infected cassava and infected vectors when the parameter a vary, the increase of a lead to the decrease of infected cassava and the decrease of infected vector.



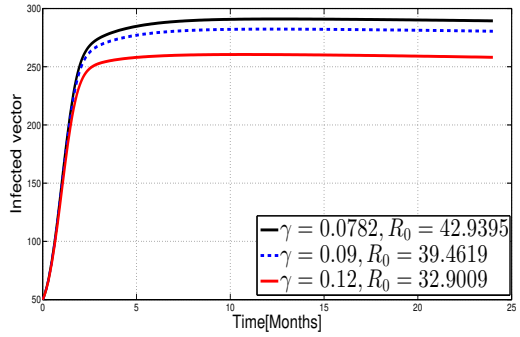
(a) Infected cassava.



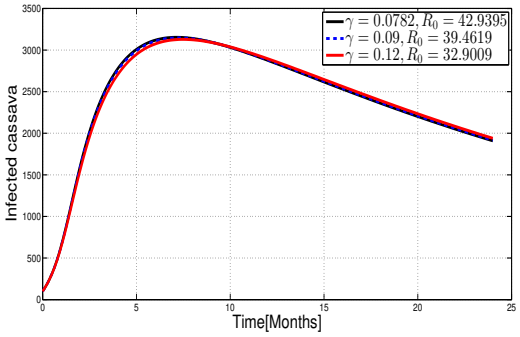
(b) infected vector.

Figure 6: Variation of loss of infected cassava rate in infected class.

Figure 7, shows the variation of vector mortality rate γ to the infectious vector and infected cassava class, if the rate of vector mortality increase the number of infected vector and infected cassava decreases.



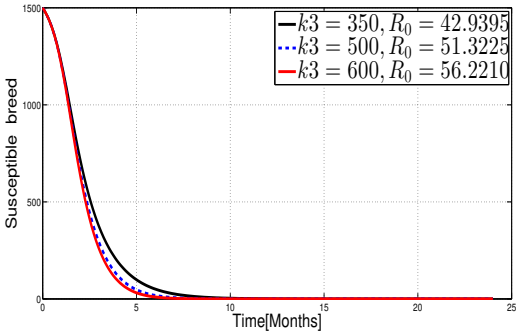
(a) Infected vector.



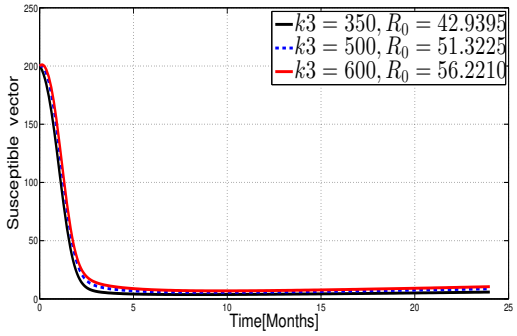
(b) infected cassava.

Figure 7: Variation of vector mortality rate in infectious class.

From Fig. 8 the graphs demonstrate the variation of vector carrying capacity k_3 to the susceptible class of cassava and susceptible class of vector. The graphs show as the carrying capacity of whitefly vectors increase the number of susceptible cassava breed decrease, the number of susceptible vector increase.



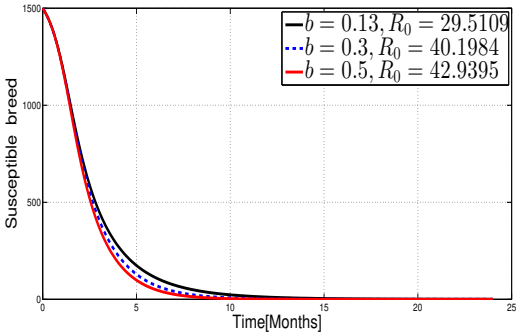
(a) Susceptible breed.



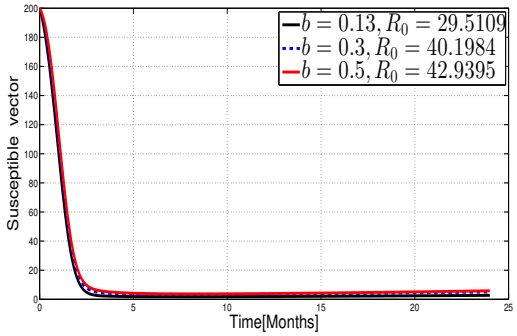
(b) Susceptible vector.

Figure 8: Variation of vector carrying capacity to the susceptible class.

Figure 9 shows the impact of variation in the recruitment of susceptible vector to the susceptible class of cassava and susceptible class of vector. It shows that as the recruitment rate of whitefly vector increases the number of susceptible cassava and vectors decreases.



(a) Susceptible breed.



(b) Susceptible vector.

Figure 9: Variation of susceptible vector recruitment rate to the susceptible class.

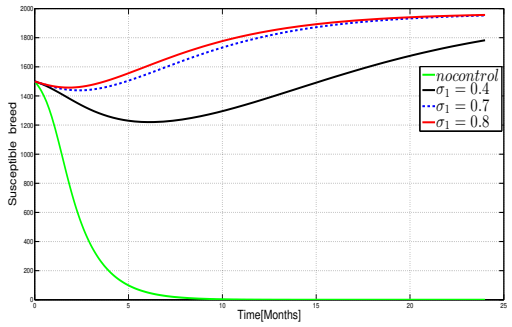
4.11 Numerical Simulation of a Control Model

This part is showing the numerical simulation of a control model. It comprises of a numerical simulation when the control parameters varies as well as before control strategies have been applied to the model. The figures shown below, demonstrate the impact of applying control strategies to the cassava and vector population at large.

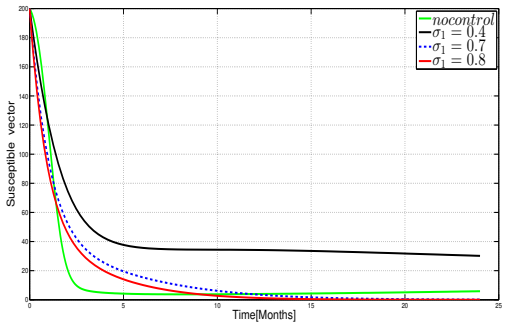
4.11.1 Impact of Applying Insecticides σ_1

Figure 10 demonstrates the impact of applying insecticide to the susceptible cassava and vector. After applying the insecticide there is an increase of harvesting susceptible breed and resistant breed in 24 months from 150 plants to about 1300 out of 2000 plants in the farm before 10

months. As the control parameter vary the number of susceptible cassava increase. It also shows the decrease of susceptible vector before four months as the control parameter vary.



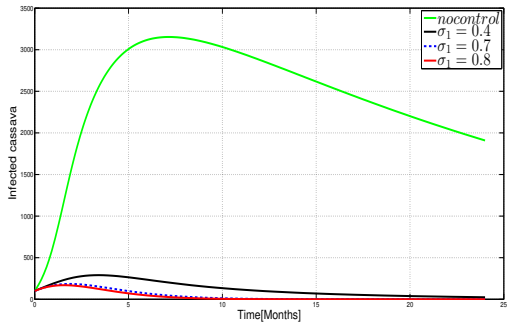
(a) Susceptible breed.



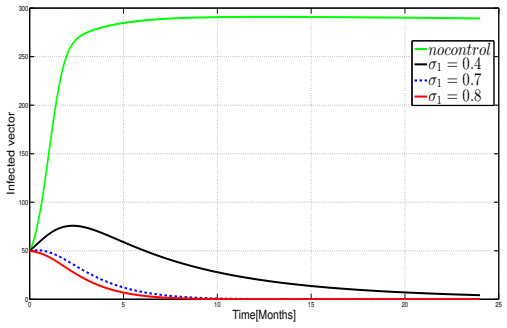
(b) Susceptible vector.

Figure 10: Effect of applying insecticides to the susceptible class.

Figure 11 shows the impact of applying insectcides to the infectious class of cassava and vector Fig. 9a demonstrate that there is a decrease of infected cassava from 3100 plants within 7 months to 700 plants before 5 Months. As we increase the control the infected cassava is decreasing fast. The number of infected vector is decreases as the control is applied, graph 9b shows that the infected vector is decreaded from 225 to 55 vectors before 2 months.



(a) Infected cassava.



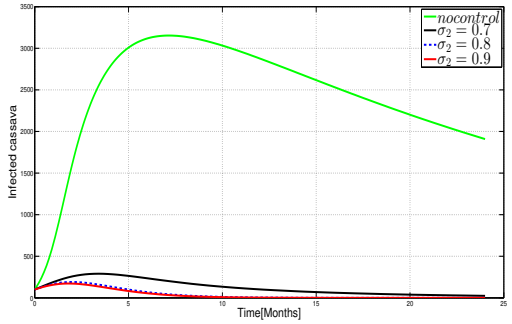
(b) Infected vector.

Figure 11: Effect of applying insecticides to the Infected class.

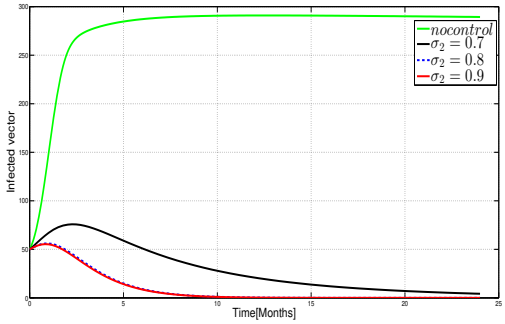
4.11.2 Impact of Removing Infected Cassava from the Farm σ_2

Figure 12 shows the impact of removing the infected cassava σ_2 from the farm to the infectious class. Its impact has been shown to both infected cassava and infected vector. The graphs shows that Fig. 12 demonstrates how the number of infected cassava change as the parameter varies.

The number of infected cassava decreases from 3000 plants before 10 months to 600 before 3 months, also the number of infected vector is decreasing as the control parameter increase, hence they have an inverse relationship.



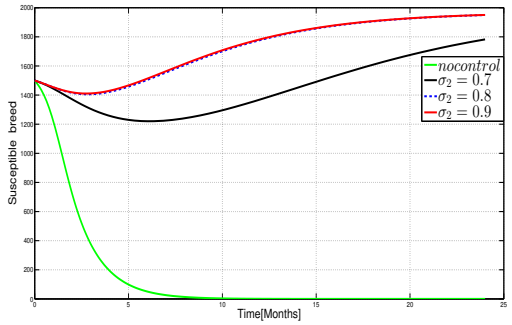
(a) Infected cassava.



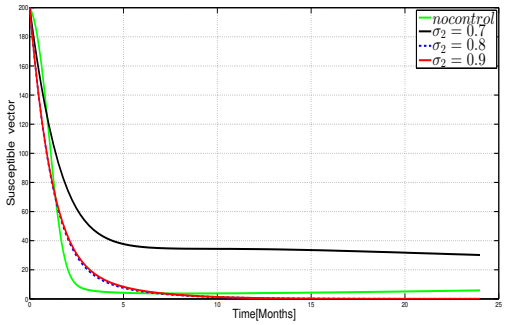
(b) Infected vector.

Figure 12: Impact of removing infected cassava to the Infected class.

Figure 13 shows the impact or removing infected cassava to the susceptible class and until the harvesting time, there is an increase of susceptible breed from less than 100 plants to 1000 plants, and as the parameter increase the number of susceptible breed of cassava is increasing. Since the infected cassava is removed from the farm. The impact have also seen in susceptible vector, graph 13b shows that as we continue to apply it, the number of susceptible vector is decreasing.



(a) Susceptible breed.

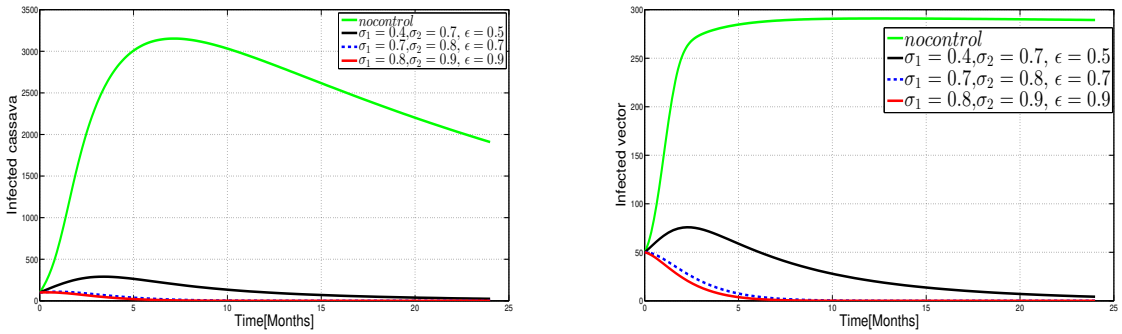


(b) Susceptible vector.

Figure 13: Impact of removing infected cassava to the Susceptible class.

4.11.3 The Impact of Control Strategies When Both Parameters Vary

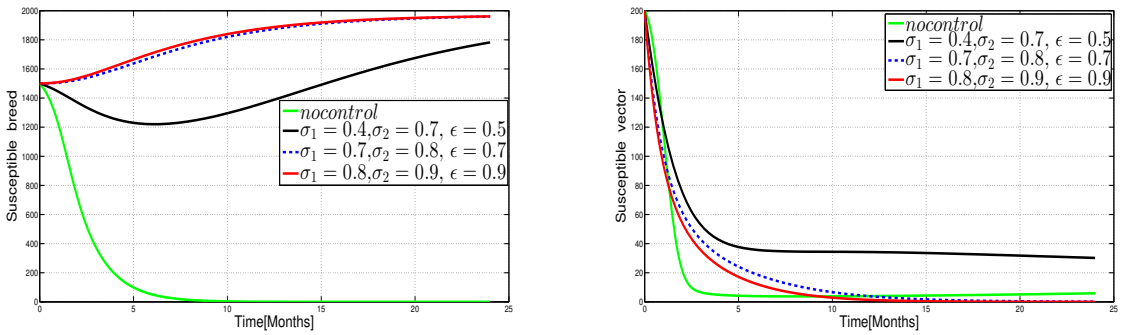
Figure 14 shows the Impact of control strategies when both parameters vary to the Infected class. Its impact has been shown to both infected cassava in Fig. 12a and infected vector in Fig. 12b. The number of infected cassava decreases from 3000 plants before 10 months to 400 before 3 months, also the number of infected vector is decreasing as the control parameters increase.



(a) Infected cassava. **(b)** Infected vector.

Figure 14: Impact of control strategies to the Infected class.

Figure 15 demonstrates the impact of applying control strategies to the susceptible class when these parameters vary. In Fig. 13a, after applying control there is an increase in harvesting of susceptible breed from less than 100 plants to 1000 out of 2000 plants in the farm before 10 months and keep in increasing as we increase the control strategies. It also shows the decrease of susceptible vector before four months as the control parameter vary as it is shown in Fig. 13b.



(a) Susceptible cassava. **(b)** Susceptible vector.

Figure 15: Impact of control strategies to the Susceptible class.

Figure 16 demonstrates the impact of control strategies to the resistant breed. There is an

increase of harvesting resistant breed from 150 plants to about 1500 out of 2000 plants in the farm before five months. As the control parameter vary the number of resistant breed increase to 1900 plants during the harvesting time of cassava.

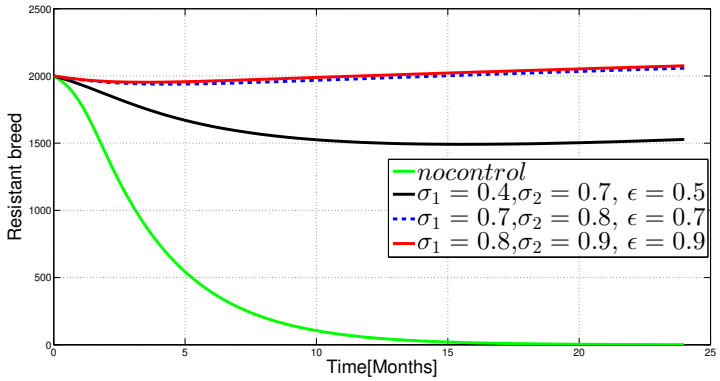


Figure 16: Impact of control strategies to the resistant breed

CHAPTER FIVE

CONCLUSION AND RECOMMENDATIONS

In this work we used mathematical model to study transmission dynamics of cassava mosaic disease. The aim was to formulate the model and compute reproduction number R_0 to determine which parameters are sensitive to the dynamics and propose controls to eradicate the disease. Equilibrium states were derived and their stability investigated. After identifying sensitive parameters two control strategies were proposed to eradicate the disease and improve cassava yields. By using appropriate assumptions, models with and without controls were formulated with the aid of differential equations and it was tested whether it is meaningful or not, basic and effective reproduction numbers were computed by next generation matrix operator and stability analysis for equilibrium states was established by linearization method, Metzler matrix and Lyapunov function. Sensitivity index for each parameter was computed by forward normalized sensitivity index.

5.1 Conclusion

The deterministic model for transmission dynamics of CMD which includes cassava plants and whitefly vector is presented and analyzed. The basic reproduction number R_0 and sensitivity index for each parameter with respect to basic reproduction number R_0 are computed to determine which parameters are sensitive to the dynamics of cassava mosaic disease.

Sensitivity analysis was performed to identify sensitive parameters. Analysis shows that the rates at which vectors are recruited and acquire the disease play the important role in the transmission dynamics of cassava mosaic disease. New infections will increase as the rates of recruitment and infection of vectors increase. Other parameters which are sensitive to disease includes vector infection rate, maximum number of vectors that can be supported, the rate of susceptible breed become infected , the rate at which resistant breed becomes infected, the maximum number of resistant breed that can be planted, rate of planting resistant breed, the rate at which susceptible breed of cassava is replanted and the maximum number of susceptible breed that can be planted.

The disease stability at cassava mosaic free equilibrium was investigated by using Metzler matrix (box invariance), It found that cassava mosaic free equilibrium is locally and globally asymptotically stable when $R_0 < 1$. Using logarithmic Lyapunov function and LaSalles invariant principle, we found that cassava mosaic equilibrium is globally asymptotically stable when $R_0 > 1$ and unstable when $R_0 < 1$. To improve cassava productivity, campaigns to eradicated

cassava mosaic disease should focus on strategies which reduce vectors' population. These strategies include spraying insecticide, use of vector-resistant varieties, phytosanitation which involve the removal of infected cassava plants from the place that will be used for the new plantings, crop hygiene and the use of free stem cutting method. The Control model developed here, use spraying of insecticide and the removal of infected cassava from the farm as effective control strategies that can be applied. Applying of insecticide and the removal of infected cassava plants gives the positive results. Analysis shows that after applied the control strategies effective reproduction number $R_0^c < 1$, analysis further shows that, spraying of insecticide that will cause the death of susceptible and infected whitefly vectors which transmit CMD.

Generally spraying of insecticide is the possible way to get rid of both infected and susceptible vector, as well as the removal of infected cassava plants form the farm will help to reduce the contact rate between plants and vectors.

5.2 Recommendations

In this study, we recommend that, in order to control the CMD and to reduce the number of whitefly vectors, the mentioned control parameters in the model as well as the parameters which have negative impact to the basic reproduction number must be applied by the farmers. The study also encourage the farmers to use the effective cleaned tools to all kinds of cassava breed. The implementation of these intervention strategies will help to get rid of CMD.

Government and other agriculture stakeholders should use different modes of communication like seminars, social media, mobile phone applications, newspaper, and door to door seminar in order to provide education to the farmers on the general transmission dynamics of Cassava mosaic disease.

The study study can be extended more as follows:

(i) More scientific investigation and further research are still needed on the dynamics and transmission of CMD.

(ii) The study can be extended by performing cost effective analysis in controlling cassava mosaic disease.

(iii) The study can be extended to a stochastic model or Markov chain to show more about the dynamics of CMD.

(iv) Another class can be added to the model to show more on how the CMD can be eradicated

or transmitted, example the environmental factors can be included in the model.

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APPENDICES

Appendix 1: Matlab Scripts and Functions Used in Simulations

Codes for the Total Dynamics of CMD

```
%\linespread{1.0}
Function dy=EACMD(~,y)
dy= zeros(size(y));
%Declaration of Parameters
r1 =0.025;rho1=0.005;beta1=0.0012;
r2=0.2;beta2=0.003;rho2=0.003;
rho3=0.003;a=0.033;
b=0.13;beta3=0.002;
gamma=0.0782;k1=3000;k2=2000;k3=350;
%Declaration of variables
Sr=y(1); Sc=y(2); Ic=y(3); Sv=y(4); Iv=y(5);
%Equation of the model
dy(1)=r1*Sr*(1-(Sr)/k1)-beta1*Sr*Iv-rho1*Sr;
dy(2)=r2*Sc*(1-(Sc)/k2)-beta2*Sc*Iv-rho2*Sc;
dy(3)=beta1*Sr*Iv+beta2*Sc*Iv-rho3*Ic-a*Ic;
dy(4)=b*(Sv+Iv)*(1-((Sv+Iv)/k3))-beta3*Sv*Ic-gamma*Sv;
dy(5)=beta3*Sv*Ic-gamma*Iv;
%Basicreproduction number formula written in Matlab codes
A= beta3*k3*(b-gamma)./b;
B=beta1*k1*(r1-rho1)./r1;
C= beta2*k2*(r2- rho2)./r2;
D=gamma*(rho3+ a);
R0 = sqrt(A*(B+C)./D)
clear all
clc
%Runge Kuta forth order approach
tspan =[0 24];%Time in Months
y0 =[2000 1500 100 200 50];
[t,y]= ode45(@EACMD,tspan,y0);
figure(6)
set(gca,'FontSize', 14')
set(legend,'FontSize', 10')
plot(t,y(:,1),'g',t,y(:,3),'c',t,y(:,2),'k',t,y(:,5),'b',t,y
    (:,4),'r','linewidth',4)
xlabel('Time[Months]','Fontsize',25)
ylabel('Cassava and vector population','Fontsize',25)
% title('TOTAL DYNAMICS VS TIME','Fontsize',25)
legend('Susceptible Resistance Breed','Infected Cassava','
    Susceptible Cassava','Infected Vector','Susceptible Vector','
    Interpreter', 'Latex','FontSize',50)
grid on
```

```
hold off%Stop operation
hold on
figure(7)
set(gca,'FontSize', 14')
set(legend,'FontSize', 20')
plot(t,y(:,1),'g',t,y(:,3),'c',t,y(:,2),'k','linewidth',4)
xlabel('Time[Months]','Fontsize',25)
ylabel('Cassava plants','Fontsize',25)
% title('CASSAVA POPULATION VS TIME','Fontsize',25)
legend('Susceptible Resistance Breed','Infected Cassava','
   Susceptible Cassava','Interpreter', 'Latex','FontSize',50)
grid on
hold off%Stop operation
hold on
figure(8)
set(gca,'FontSize', 14')
set(legend,'FontSize', 20')
plot(t,y(:,5),'b',t,y(:,4),'r','linewidth',4)
xlabel('Time[Months]','Fontsize',25)
ylabel('Vector Population','Fontsize',25)
% title('VECTOR POPULATION VS TIME','Fontsize',25)
legend('Infected vector','Susceptible vector','Interpreter', '
   Latex','FontSize',50)
grid on
hold off%Stop operation
hold on
```

MATLAB Script for the sensitive parameter of a basic model

```
 %\linespread{1.0}
dy= zeros(size(y));
%Declaration of Parameters
r1 =0.025;rho1=0.005;beta1=0.0012;
r2=0.2;beta2=0.003;rho2=0.003;
rho3=0.003;a=0.3;
b=0.13;beta3=0.002;
gamma=0.0782;k1=3000;k2=2000;k3=350;
%Declaration of variables
Sr=y(1); Sc=y(2); Ic=y(3); Sv=y(4); Iv=y(5);
%Equation of the model
dy(1)=r1*Sr*(1-(Sr)/k1)-beta1*Sr*Iv-rho1*Sr;
dy(2)=r2*Sc*(1-(Sc)/k2)-beta2*Sc*Iv-rho2*Sc;
dy(3)=beta1*Sr*Iv+beta2*Sc*Iv-rho3*Ic-a*Ic;
dy(4)=b*(Sv+Iv)*(1-((Sv+Iv)/k3))-beta3*Sv*Ic-gamma*Sv;
dy(5)=beta3*Sv*Ic-gamma*Iv;
%Basicreproduction number formula written in Matlab codes
```

```
A= beta3*k3*(b-gamma)./b;
B=beta1*k1*(r1-rho1)./r1;
C= beta2*k2*(r2- rho2)./r2;
D=gamma*(rho3+ a);
R0 = sqrt(A*(B+C)./D)
function $dy=a_2(~,y)$
dy= zeros(size(y));
%Declaration of Parameters
r1 =0.025;rho1=0.005;beta1=0.0012;
r2=0.2;beta2=0.003;rho2=0.003;
rho3=0.003;a=0.6;
b=0.13;beta3=0.002;
gamma=0.0782;k1=3000;k2=2000;k3=350;
%Declaration of variables
Sr=y(1); Sc=y(2); Ic=y(3); Sv=y(4); Iv=y(5);
%Equation of the model
dy(1)=r1*Sr*(1-(Sr)/k1)-beta1*Sr*Iv-rho1*Sr;
dy(2)=r2*Sc*(1-(Sc)/k2)-beta2*Sc*Iv-rho2*Sc;
dy(3)=beta1*Sr*Iv+beta2*Sc*Iv-rho3*Ic-a*Ic;
dy(4)=b*(Sv+Iv)*(1-((Sv+Iv)/k3))-beta3*Sv*Ic-gamma*Sv;
dy(5)=beta3*Sv*Ic-gamma*Iv;
%Basicreproduction number formula written in Matlab codes
A= beta3*k3*(b-gamma)./b;
B=beta1*k1*(r1-rho1)./r1;
C= beta2*k2*(r2- rho2)./r2;
D=gamma*(rho3+ a);
R0 = sqrt(A*(B+C)./D)
clear all
clc
%Runge Kuta forth order approach
tspan =[0 24];%Time in Months
y0 =[2000 1500 100 200 50];
[t,y]= ode45(@EACMD,tspan,y0);
[t1,y1]= ode45(@a_1,tspan,y0);
[t2,y2]= ode45(@a_2,tspan,y0);
figure(1)
set(gca,'FontSize', 14')
set(legend,'FontSize', 10')
plot(t,y(:,3),'k',t1,y1(:,3),'b--',t2,y2(:,3),'r','linewidth',4)
xlabel('Time[Months]','Fontsize',25)
ylabel('Infected cassava','Fontsize',25)
% title('TOTAL DYNAMICS VS TIME','Fontsize',25)
legend({'$a=0.033,R_0=29.5109$','$a=0.3,R_0=10.1721$','$a=0.6,
   R_0=7.2107$'},'Interpreter', 'Latex','FontSize',25)
grid on
hold off%Stop operation
```

```
hold on
figure(2)
set(gca,'FontSize', 14')
set(legend,'FontSize', 10')
plot(t,y(:,5),'k',t1,y1(:,5),'b—',t2,y2(:,5),'r','linewidth',4)
xlabel('Time[Months]','Fontsize',25)
ylabel('Infected vector','Fontsize',25)
% title('TOTAL DYNAMICS VS TIME','Fontsize',25)
legend({'$a=0.033,R_0=29.5109$','$a=0.3,R_0=10.1721$','$a=0.6,
   R_0=7.2107$'},'Interpreter', 'Latex','FontSize',35)
grid on
hold off%Stop operation
hold on
when $\gamma$ vary
function dy=gamma1(~,y)
dy= zeros(size(y));
%Declaration of Parameters
r1 =0.025;rho1=0.005;beta1=0.0012;
r2=0.2;beta2=0.003;rho2=0.003;
rho3=0.003;a=0.033;
b=0.13;beta3=0.002;
gamma=0.09;k1=3000;k2=2000;k3=350;
%Declaration of variables
Sr=y(1); Sc=y(2); Ic=y(3); Sv=y(4); Iv=y(5);
%Equation of the model
dy(1)=r1*Sr*(1-(Sr)/k1)-beta1*Sr*Iv-rho1*Sr;
dy(2)=r2*Sc*(1-(Sc)/k2)-beta2*Sc*Iv-rho2*Sc;
dy(3)=beta1*Sr*Iv+beta2*Sc*Iv-rho3*Ic-a*Ic;
dy(4)=b*(Sv+Iv)*(1-((Sv+Iv)/k3))-beta3*Sv*Ic-gamma*Sv;
dy(5)=beta3*Sv*Ic-gamma*Iv;
%Basicreproduction number formula written in Matlab codes
A= beta3*k3*(b-gamma)./b;
B=beta1*k1*(r1-rho1)./r1;
C= beta2*k2*(r2- rho2)./r2;
D=gamma*(rho3+ a);
R0 = sqrt(A*(B+C)./D)
function dy=gamma2(~,y)
dy= zeros(size(y));
%Declaration of Parameters
r1 =0.025;rho1=0.005;beta1=0.0012;
r2=0.2;beta2=0.003;rho2=0.003;
rho3=0.003;a=0.033;
b=0.13;beta3=0.002;
gamma=0.12;k1=3000;k2=2000;k3=350;
%Declaration of variables
Sr=y(1); Sc=y(2); Ic=y(3); Sv=y(4); Iv=y(5);
```

```
%Equation of the model
dy(1)=r1*Sr*(1-(Sr)/k1)-beta1*Sr*Iv-rho1*Sr;
dy(2)=r2*Sc*(1-(Sc)/k2)-beta2*Sc*Iv-rho2*Sc;
dy(3)=beta1*Sr*Iv+beta2*Sc*Iv-rho3*Ic-a*Ic;
dy(4)=b*(Sv+Iv)*(1-((Sv+Iv)/k3))-beta3*Sv*Ic-gamma*Sv;
dy(5)=beta3*Sv*Ic-gamma*Iv;
%Basicreproduction number formula written in Matlab codes
A= beta3*k3*(b-gamma)./b;
B=beta1*k1*(r1-rho1)./r1;
C= beta2*k2*(r2- rho2)./r2;
D=gamma*(rho3+ a);
R0 = sqrt(A*(B+C)./D)
clear all
clc
%Runge Kuta forth order approach
tspan =[0 24];%Time in Months
y0 =[2000 1500 100 200 50];
[t,y]= ode45(@EACMD,tspan,y0);
[t1,y1]= ode45(@gamma1,tspan,y0);
[t2,y2]= ode45(@gamma2,tspan,y0);
figure(1)
set(gca,'FontSize', 14')
set(legend,'FontSize', 10')
plot(t,y(:,3),'k',t1,y1(:,3),'b--',t2,y2(:,3),'r','linewidth',4)
xlabel('Time[Months]','Fontsize',25)
ylabel('Infected cassava','Fontsize',25)
% title('TOTAL DYNAMICS VS TIME','Fontsize',25)
legend({'$\gamma=0.0782,R_0= 29.5109$','$\gamma=0.09,R_0
    =24.1729$','$\gamma=0.12,R_0=10.4672$'},'Interpreter', 'Latex
    ','FontSize',35)
grid on
hold off%Stop operation
hold on
figure(2)
set(gca,'FontSize', 14')
set(legend,'FontSize', 10')
plot(t,y(:,5),'k',t1,y1(:,5),'b--',t2,y2(:,5),'r','linewidth',4)
xlabel('Time[Months]','Fontsize',25)
ylabel('Infected vector','Fontsize',25)
% title('TOTAL DYNAMICS VS TIME','Fontsize',25)
legend({'$\gamma=0.0782,R_0= 29.5109$','$\gamma=0.09,R_0
    =24.1729$','$\gamma=0.12,R_0=10.4672$'},'Interpreter', 'Latex
    ','FontSize',35)
grid on
hold off%Stop operation
hold on
```

```
when k3 vary
function dy=k3_1(~,y)
dy= zeros(size(y));
%Declaration of Parameters
r1 =0.025;rho1=0.005;beta1=0.0012;
r2=0.2;beta2=0.003;rho2=0.003;
rho3=0.003;a=0.033;
b=0.13;beta3=0.002;
gamma=0.0782;k1=3000;k2=2000;k3=500;
%Declaration of variables
Sr=y(1); Sc=y(2); Ic=y(3); Sv=y(4); Iv=y(5);
%Equation of the model
dy(1)=r1*Sr*(1-(Sr)/k1)-beta1*Sr*Iv-rho1*Sr;
dy(2)=r2*Sc*(1-(Sc)/k2)-beta2*Sc*Iv-rho2*Sc;
dy(3)=beta1*Sr*Iv+beta2*Sc*Iv-rho3*Ic-a*Ic;
dy(4)=b*(Sv+Iv)*(1-((Sv+Iv)/k3))-beta3*Sv*Ic-gamma*Sv;
dy(5)=beta3*Sv*Ic-gamma*Iv;
%Basicreproduction number formula written in Matlab codes
A= beta3*k3*(b-gamma)./b;
B=beta1*k1*(r1-rho1)./r1;
C= beta2*k2*(r2- rho2)./r2;
D=gamma*(rho3+ a);
R0 = sqrt(A*(B+C)./D)
function dy=k3_2(~,y)
dy= zeros(size(y));
%Declaration of Parameters
r1 =0.025;rho1=0.005;beta1=0.0012;
r2=0.2;beta2=0.003;rho2=0.003;
rho3=0.003;a=0.033;
b=0.13;beta3=0.002;
gamma=0.0782;k1=3000;k2=2000;k3=600;
%Declaration of variables
Sr=y(1); Sc=y(2); Ic=y(3); Sv=y(4); Iv=y(5);
%Equation of the model
dy(1)=r1*Sr*(1-(Sr)/k1)-beta1*Sr*Iv-rho1*Sr;
dy(2)=r2*Sc*(1-(Sc)/k2)-beta2*Sc*Iv-rho2*Sc;
dy(3)=beta1*Sr*Iv+beta2*Sc*Iv-rho3*Ic-a*Ic;
dy(4)=b*(Sv+Iv)*(1-((Sv+Iv)/k3))-beta3*Sv*Ic-gamma*Sv;
dy(5)=beta3*Sv*Ic-gamma*Iv;
%Basicreproduction number formula written in Matlab codes
A= beta3*k3*(b-gamma)./b;
B=beta1*k1*(r1-rho1)./r1;
C= beta2*k2*(r2- rho2)./r2;
D=gamma*(rho3+ a);
R0 = sqrt(A*(B+C)./D)
clear all
```

```
clc
%Runge Kuta forth order approach
tspan =[0 24];%Time in Months
y0 =[2000 1500 100 200 50];
[t,y]= ode45(@EACMD,tspan,y0);
[t1,y1]= ode45(@k3_1,tspan,y0);
[t2,y2]= ode45(@k3_2,tspan,y0);
figure(1)
set(gca,'FontSize', 14')
set(legend,'FontSize', 10')
plot(t,y(:,2),'k',t1,y1(:,2),'b—',t2,y2(:,2),'r','linewidth',4)
xlabel('Time[Months]','Fontsize',25)
ylabel('Susceptible breed','Fontsize',25)
% title('TOTAL DYNAMICS VS TIME','Fontsize',25)
legend({'$k3=350,R_0= 29.5109$','$k3=500,R_0=35.2722$','$k3=600,
    R_0=38.6388$'},'Interpreter', 'Latex','FontSize',35)
grid on
hold off%Stop operation
hold on
figure(2)
set(gca,'FontSize', 14')
set(legend,'FontSize', 10')
plot(t,y(:,4),'k',t1,y1(:,4),'b—',t2,y2(:,4),'r','linewidth',4)
xlabel('Time[Months]','Fontsize',25)
ylabel('Susceptible vector','Fontsize',25)
% title('TOTAL DYNAMICS VS TIME','Fontsize',25)
legend({'$k3=350,R_0= 29.5109$','$k3=500,R_0=35.2722$','$k3=600,
    R_0=38.6388$'},'Interpreter', 'Latex','FontSize',35)
grid on
hold off%Stop operation
hold on
When$ b$ vary
function dy=b_1(~,y)
dy= zeros(size(y));
%Declaration of Parameters
r1 =0.025;rho1=0.005;beta1=0.0012;
r2=0.2;beta2=0.003;rho2=0.003;
rho3=0.003;a=0.033;
b=0.3;beta3=0.002;
gamma=0.0782;k1=3000;k2=2000;k3=350;
%Declaration of variables
Sr=y(1); Sc=y(2); Ic=y(3); Sv=y(4); Iv=y(5);
%Equation of the model
dy(1)=r1*Sr*(1-(Sr)/k1)-beta1*Sr*Iv-rho1*Sr;
dy(2)=r2*Sc*(1-(Sc)/k2)-beta2*Sc*Iv-rho2*Sc;
dy(3)=beta1*Sr*Iv+beta2*Sc*Iv-rho3*Ic-a*Ic;
```

```
dy(4)=b*(Sv+Iv)*(1-((Sv+Iv)/k3))-beta3*Sv*Ic-gamma*Sv;
dy(5)=beta3*Sv*Ic-gamma*Iv;
%Basicreproduction number formula written in Matlab codes
A= beta3*k3*(b-gamma)./b;
B=beta1*k1*(r1-rho1)./r1;
C= beta2*k2*(r2- rho2)./r2;
D=gamma*(rho3+ a);
R0 = sqrt(A*(B+C)./D)
function dy=b_2(~,y)
dy= zeros(size(y));
%Declaration of Parameters
r1 =0.025;rho1=0.005;beta1=0.0012;
r2=0.2;beta2=0.003;rho2=0.003;
rho3=0.003;a=0.033;
b=0.5;beta3=0.002;
gamma=0.0782;k1=3000;k2=2000;k3=350;
%Declaration of variables
Sr=y(1); Sc=y(2); Ic=y(3); Sv=y(4); Iv=y(5);
%Equation of the model
dy(1)=r1*Sr*(1-(Sr)/k1)-beta1*Sr*Iv-rho1*Sr;
dy(2)=r2*Sc*(1-(Sc)/k2)-beta2*Sc*Iv-rho2*Sc;
dy(3)=beta1*Sr*Iv+beta2*Sc*Iv-rho3*Ic-a*Ic;
dy(4)=b*(Sv+Iv)*(1-((Sv+Iv)/k3))-beta3*Sv*Ic-gamma*Sv;
dy(5)=beta3*Sv*Ic-gamma*Iv;
%Basicreproduction number formula written in Matlab codes
A= beta3*k3*(b-gamma)./b;
B=beta1*k1*(r1-rho1)./r1;
C= beta2*k2*(r2- rho2)./r2;
D=gamma*(rho3+ a);
R0 = sqrt(A*(B+C)./D)
clear all
clc
%Runge Kuta forth order approach
tspan =[0 24];%Time in Months
y0 =[2000 1500 100 200 50];
[t,y]= ode45(@EACMD,tspan,y0);
[t1,y1]= ode45(@b_1,tspan,y0);
[t2,y2]= ode45(@b_2,tspan,y0);
figure(1)
set(gca,'FontSize', 14')
set(legend,'FontSize', 10')
plot(t,y(:,2),'k',t1,y1(:,2),'b--',t2,y2(:,2),'r','linewidth',4)
xlabel('Time[Months]','Fontsize',25)
ylabel('Susceptible breed','Fontsize',25)
% title('TOTAL DYNAMICS VS TIME','Fontsize',25)
legend({'$b=0.13,R_0= 29.5109$','$b=0.3,R_0= 40.1984$','$b=0.5,
```

```
   R_0= 42.9395$'},'Interpreter', 'Latex','FontSize',35)
grid on
hold off%Stop operation
hold on
figure(2)
set(gca,'FontSize', 14')
set(legend,'FontSize', 10')
plot(t,y(:,4),'k',t1,y1(:,4),'b—',t2,y2(:,4),'r','linewidth',4)
xlabel('Time[Months]','Fontsize',25)
ylabel('Susceptible vector','Fontsize',25)
% title('TOTAL DYNAMICS VS TIME','Fontsize',25)
legend({'$b=0.13,R_0= 29.5109$','$b=0.3,R_0= 40.1984$','$b=0.5,
   R_0= 42.9395$'},'Interpreter', 'Latex','FontSize',35)
grid on
hold off%Stop operation
hold on
```

MATLAB script for the simulation of a Control model

```
%Before and after control when \sigma1 vary
function dy=EACMDCONTROL(~,y)
dy= zeros(size(y));
%Declaration of Parameters
r1 =0.025;rho1=0.005;beta1=0.0012;
r2=0.2;beta2=0.003;rho2=0.003;
rho3=0.003;a=0.033;
b=0.13;beta3=0.002;
gamma=0.0782;k1=3000;k2=2000;k3=350;sigma1=0.5;sigma2=0.1;
%Declaration of variables
Sr=y(1); Sc=y(2); Ic=y(3); Sv=y(4); Iv=y(5);
%Equation of the model
dy(1)=r1*Sr*(1-(Sr)/k1)-beta1*Sr*Iv-rho1*Sr;
dy(2)=r2*Sc*(1-(Sc)/k2)-beta2*Sc*Iv-rho2*Sc;
dy(3)=beta1*Sr*Iv+beta2*Sc*Iv-rho3*Ic-a*Ic-sigma2*Ic;
dy(4)=b*(Sv+Iv)*(1-((Sv+Iv)/k3))-beta3*Sv*Ic-gamma*Sv-sigma1*Sv;
dy(5)=beta3*Sv*Ic-gamma*Iv-sigma1*Iv;
%Effective Basic reproduction number formula written in Matlab
   codes
A= beta3*k3*(b-gamma-sigma1)./b;
B=beta1*k1*(r1-rho1)./r1;
C= beta2*k2*(r2- rho2)./r2;
D=(gamma+sigma1)*(rho3+ a+sigma2);
Re = sqrt(A*(B+C)./D)
% %Basicreproduction number formula written in Matlab codes
% A= beta3*k3*(b-gamma)./b;
% B=beta1*k1*(r1-rho1)./r1;
```

```
% C= beta2*k2*(r2- rho2)./r2;
% D=gamma*(rho3+ a);
% R0 = sqrt(A*(B+C)./D)
function dy=control1(~,y)
dy= zeros(size(y));
%Declaration of Parameters
r1 =0.025;rho1=0.005;beta1=0.0012;
r2=0.2;beta2=0.003;rho2=0.003;
rho3=0.003;a=0.033;
b=0.13;beta3=0.002;
gamma=0.0782;k1=3000;k2=2000;k3=350;sigma1=0.7;sigma2=0.1;
%Declaration of variables
Sr=y(1); Sc=y(2); Ic=y(3); Sv=y(4); Iv=y(5);
%Equation of the model
dy(1)=r1*Sr*(1-(Sr)/k1)-beta1*Sr*Iv-rho1*Sr;
dy(2)=r2*Sc*(1-(Sc)/k2)-beta2*Sc*Iv-rho2*Sc;
dy(3)=beta1*Sr*Iv+beta2*Sc*Iv-rho3*Ic-a*Ic-sigma2*Ic;
dy(4)=b*(Sv+Iv)*(1-((Sv+Iv)/k3))-beta3*Sv*Ic-gamma*Sv-sigma1*Sv;
dy(5)=beta3*Sv*Ic-gamma*Iv-sigma1*Iv;
%Basicreproduction number formula written in Matlab codes
A= beta3*k3*(b-gamma-sigma1)./b;
B=beta1*k1*(r1-rho1)./r1;
C= beta2*k2*(r2- rho2)./r2;
D=(gamma+sigma1)*(rho3+ a+sigma2);
R0 = sqrt(A*(B+C)./D)
function dy=control2(~,y)
dy= zeros(size(y));
%Declaration of Parameters
r1 =0.025;rho1=0.005;beta1=0.0012;
r2=0.2;beta2=0.003;rho2=0.003;
rho3=0.003;a=0.033;
b=0.13;beta3=0.002;
gamma=0.0782;k1=3000;k2=2000;k3=350;sigma1=0.8;sigma2=0.1;
%Declaration of variables
Sr=y(1); Sc=y(2); Ic=y(3); Sv=y(4); Iv=y(5);
%Equation of the model
dy(1)=r1*Sr*(1-(Sr)/k1)-beta1*Sr*Iv-rho1*Sr;
dy(2)=r2*Sc*(1-(Sc)/k2)-beta2*Sc*Iv-rho2*Sc;
dy(3)=beta1*Sr*Iv+beta2*Sc*Iv-rho3*Ic-a*Ic-sigma2*Ic;
dy(4)=b*(Sv+Iv)*(1-((Sv+Iv)/k3))-beta3*Sv*Ic-gamma*Sv-sigma1*Sv;
dy(5)=beta3*Sv*Ic-gamma*Iv-sigma1*Iv;
%Basicreproduction number formula written in Matlab codes
A= beta3*k3*(b-gamma-sigma1)./b;
B=beta1*k1*(r1-rho1)./r1;
C= beta2*k2*(r2- rho2)./r2;
D=(gamma+sigma1)*(rho3+ a+sigma2);
```

```
R0 = sqrt(A*(B+C)./D)
clear all
clc
%Runge Kuta forth order approach
tspan =[0 24];%Time in Months
y0 =[2000 1500 100 200 50];
[t,y]= ode45(@EACMD,tspan,y0);
[t1,y1]= ode45(@EACMDCONTROL,tspan,y0);
[t2,y2]= ode45(@control1,tspan,y0);
[t3,y3]= ode45(@control2,tspan,y0);
%ploting of graphs on susceptible casava resistant breed
   population
figure(1)
set(gca,'FontSize', 14')
set(legend,'FontSize', 10')
plot(t,y(:,1),'g',t1,y1(:,1),'k',t2,y2(:,1),'b--',t3,y3(:,1),'r'
   ,'linewidth',4)
xlabel('Time[Months]','Fontsize',25)
ylabel('Resistant breed','Fontsize',25)
% title('TOTAL DYNAMICS VS TIME','Fontsize',25)
legend({'$no$ $control$','$\sigma_1=0.5$','$\sigma_1=0.7$','$\
   sigma_1=0.8$'},'Interpreter', 'Latex','FontSize',35)
grid on
hold off%Stop operation
hold on
figure(2)
set(gca,'FontSize', 14')
set(legend,'FontSize', 10')
plot(t,y(:,2),'g',t1,y1(:,2),'k',t2,y2(:,2),'b--',t3,y3(:,2),'r'
   ,'linewidth',4)
xlabel('Time[Months]','Fontsize',25)
ylabel('Susceptible breed','Fontsize',25)
% title('TOTAL DYNAMICS VS TIME','Fontsize',25)
legend({'$no$ $control$','$\sigma_1=0.5$','$\sigma_1=0.7$','$\
   sigma_1=0.8$'},'Interpreter', 'Latex','FontSize',35)
grid on
hold off%Stop operation
hold on
figure(3)
set(gca,'FontSize', 14')
set(legend,'FontSize', 10')
plot(t,y(:,3),'g',t1,y1(:,3),'k',t2,y2(:,3),'b--',t3,y3(:,3),'r'
   ,'linewidth',4)
xlabel('Time[Months]','Fontsize',25)
ylabel('Infected cassava','Fontsize',25)
% title('TOTAL DYNAMICS VS TIME','Fontsize',25)
```

```
113 legend({'$no$ $control$','$\sigma_1=0.5$','$\sigma_1=0.7$','$\
    sigma_1=0.8$'},'Interpreter', 'Latex','FontSize',35)
114 grid on
115 hold off%Stop operation
116 hold on
117 figure(4)
118 set(gca,'FontSize', 14')
119 set(legend,'FontSize', 10')
120 plot(t,y(:,4),'g',t1,y1(:,4),'k',t2,y2(:,4),'b—',t3,y3(:,4),'r'
    ,'linewidth',4)
121 xlabel('Time[Months]','Fontsize',25)
122 ylabel('Susceptible vector','Fontsize',25)
123 % title('TOTAL DYNAMICS VS TIME','Fontsize',25)
124 legend({'$no$ $control$','$\sigma_1=0.5$','$\sigma_1=0.7$','$\
    sigma_1=0.8$'},'Interpreter', 'Latex','FontSize',35)
125 grid on
126 hold off%Stop operation
127 hold on
128 figure(5)
129 set(gca,'FontSize', 14')
130 set(legend,'FontSize', 10')
131 plot(t,y(:,5),'g',t1,y1(:,5),'k',t2,y2(:,5),'b—',t3,y3(:,5),'r'
    ,'linewidth',4)
132 xlabel('Time[Months]','Fontsize',25)
133 ylabel('Infected vector','Fontsize',25)
134 % title('TOTAL DYNAMICS VS TIME','Fontsize',25)
135 legend({'$no$ $control$','$\sigma_1=0.5$','$\sigma_1=0.7$','$\
    sigma_1=0.8$'},'Interpreter', 'Latex','FontSize',35)
136 grid on
137 hold off%Stop operation
138 hold on
139 When sigma2 vary
140 function dy=control3(~,y)
141 dy= zeros(size(y));
142 %Declaration of Parameters
143 r1 =0.025;rho1=0.005;beta1=0.0012;
144 r2=0.2;beta2=0.003;rho2=0.003;
145 rho3=0.003;a=0.033;
146 b=0.13;beta3=0.002;
147 gamma=0.0782;k1=3000;k2=2000;k3=350;sigma1=0.5;sigma2=0.3;
148 %Declaration of variables
149 Sr=y(1); Sc=y(2); Ic=y(3); Sv=y(4); Iv=y(5);
150 %Equation of the model
151 dy(1)=r1*Sr*(1-(Sr)/k1)-beta1*Sr*Iv-rho1*Sr;
152 dy(2)=r2*Sc*(1-(Sc)/k2)-beta2*Sc*Iv-rho2*Sc;
153 dy(3)=beta1*Sr*Iv+beta2*Sc*Iv-rho3*Ic-a*Ic-sigma2*Ic;
```

```
dy(4)=b*(Sv+Iv)*(1-((Sv+Iv)/k3))-beta3*Sv*Ic-gamma*Sv-sigma1*Sv;
dy(5)=beta3*Sv*Ic-gamma*Iv-sigma1*Iv;
%Basicreproduction number formula written in Matlab codes
A= beta3*k3*(b-gamma-sigma1)./b;
B=beta1*k1*(r1-rho1)./r1;
C= beta2*k2*(r2- rho2)./r2;
D=(gamma+sigma1)*(rho3+ a+sigma2);
R0 = sqrt(A*(B+C)./D)
function dy=control4(~,y)
dy= zeros(size(y));
%Declaration of Parameters
r1 =0.025;rho1=0.005;beta1=0.0012;
r2=0.2;beta2=0.003;rho2=0.003;
rho3=0.003;a=0.033;
b=0.13;beta3=0.002;
gamma=0.0782;k1=3000;k2=2000;k3=350;sigma1=0.5;sigma2=0.5;
%Declaration of variables
Sr=y(1); Sc=y(2); Ic=y(3); Sv=y(4); Iv=y(5);
%Equation of the model
dy(1)=r1*Sr*(1-(Sr)/k1)-beta1*Sr*Iv-rho1*Sr;
dy(2)=r2*Sc*(1-(Sc)/k2)-beta2*Sc*Iv-rho2*Sc;
dy(3)=beta1*Sr*Iv+beta2*Sc*Iv-rho3*Ic-a*Ic-sigma2*Ic;
dy(4)=b*(Sv+Iv)*(1-((Sv+Iv)/k3))-beta3*Sv*Ic-gamma*Sv-sigma1*Sv;
dy(5)=beta3*Sv*Ic-gamma*Iv-sigma1*Iv;
clear all
clc
%Runge Kuta forth order approach
tspan =[0 24];%Time in Months
y0 =[2000 1500 100 200 50];
[t,y]= ode45(@EACMD,tspan,y0);
[t1,y1]= ode45(@EACMDCONTROL,tspan,y0);
[t2,y2]= ode45(@control3,tspan,y0);
[t3,y3]= ode45(@control4,tspan,y0);
%%ploting of graphs on susceptible casava resistant breed
   population
figure(1)
set(gca,'FontSize', 14')
set(legend,'FontSize', 10')
plot(t,y(:,1),'g',t1,y1(:,1),'k',t2,y2(:,1),'b--',t3,y3(:,1),'r'
   ,'linewidth',4)
xlabel('Time[Months]','Fontsize',25)
ylabel('resistant breed','Fontsize',25)
% title('TOTAL DYNAMICS VS TIME','Fontsize',25)
legend({'$no$ $control$','$\sigma_2=0.1$','$\sigma_2=0.3$','$\
   sigma_2=0.5$'},'Interpreter', 'Latex','FontSize',35)
grid on
```

```
hold off%Stop operation
hold on
figure(2)
set(gca,'FontSize', 14')
set(legend,'FontSize', 10')
plot(t,y(:,2),'g',t1,y1(:,2),'k',t2,y2(:,2),'b—',t3,y3(:,2),'r'
    ,'linewidth',4)
xlabel('Time[Months]','Fontsize',25)
ylabel('susceptible breed','Fontsize',25)
% title('TOTAL DYNAMICS VS TIME','Fontsize',25)
legend({'$no$ $control$','$\sigma_2=0.1$','$\sigma_2=0.3$','$\
   sigma_2=0.5$'},'Interpreter', 'Latex','FontSize',35)
grid on
hold off%Stop operation
hold on
figure(3)
set(gca,'FontSize', 14')
set(legend,'FontSize', 10')
plot(t,y(:,3),'g',t1,y1(:,3),'k',t2,y2(:,3),'b—',t3,y3(:,3),'r'
    ,'linewidth',4)
xlabel('Time[Months]','Fontsize',25)
ylabel('infected cassava','Fontsize',25)
% title('TOTAL DYNAMICS VS TIME','Fontsize',25)
legend({'$no$ $control$','$\sigma_2=0.1$','$\sigma_2=0.3$','$\
   sigma_2=0.5$'},'Interpreter', 'Latex','FontSize',25)
grid on
hold off%Stop operation
hold on
figure(4)
set(gca,'FontSize', 14')
set(legend,'FontSize', 10')
plot(t,y(:,4),'g',t1,y1(:,4),'k',t2,y2(:,4),'b—',t3,y3(:,4),'r'
    ,'linewidth',4)
xlabel('Time[Months]','Fontsize',25)
ylabel('susceptible vector','Fontsize',25)
% title('TOTAL DYNAMICS VS TIME','Fontsize',25)
legend({'$no$ $control$','$\sigma_2=0.1$','$\sigma_2=0.3$','$\
   sigma_2=0.5$'},'Interpreter', 'Latex','FontSize',35)
grid on
hold off%Stop operation
hold on
figure(5)
set(gca,'FontSize', 14')
set(legend,'FontSize', 10')
plot(t,y(:,5),'g',t1,y1(:,5),'k',t2,y2(:,5),'b—',t3,y3(:,5),'r'
    ,'linewidth',4)
```

```
xlabel('Time[Months]','Fontsize',25)
ylabel('infected vector','Fontsize',25)
% title('TOTAL DYNAMICS VS TIME','Fontsize',25)
legend({'$no$ $control$','$\sigma_2=0.1$','$\sigma_2=0.3$','$\
    sigma_2=0.5$'},'Interpreter', 'Latex','FontSize',35)
grid on
hold off%Stop operation
hold on
```

Effect of varying both control strategies

```
function dy=control5(~,y)
dy= zeros(size(y));
%Declaration of Parameters
r1 =0.025;rho1=0.005;beta1=0.0012;
r2=0.2;beta2=0.003;rho2=0.003;
rho3=0.003;a=0.033;
b=0.13;beta3=0.002;
gamma=0.0782;k1=3000;k2=2000;k3=350;sigma1=0.7;sigma2=0.3;
%Declaration of variables
Sr=y(1); Sc=y(2); Ic=y(3); Sv=y(4); Iv=y(5);
%Equation of the model
dy(1)=r1*Sr*(1-(Sr)/k1)-beta1*Sr*Iv-rho1*Sr;
dy(2)=r2*Sc*(1-(Sc)/k2)-beta2*Sc*Iv-rho2*Sc;
dy(3)=beta1*Sr*Iv+beta2*Sc*Iv-rho3*Ic-a*Ic-sigma2*Ic;
dy(4)=b*(Sv+Iv)*(1-((Sv+Iv)/k3))-beta3*Sv*Ic-gamma*Sv-sigma1*Sv;
dy(5)=beta3*Sv*Ic-gamma*Iv-sigma1*Iv;
function dy=control6(~,y)
dy= zeros(size(y));
%Declaration of Parameters
r1 =0.025;rho1=0.005;beta1=0.0012;
r2=0.2;beta2=0.003;rho2=0.003;
rho3=0.003;a=0.033;
b=0.13;beta3=0.002;
gamma=0.0782;k1=3000;k2=2000;k3=350;sigma1=0.8;sigma2=0.5;
%Declaration of variables
Sr=y(1); Sc=y(2); Ic=y(3); Sv=y(4); Iv=y(5);
%Equation of the model
dy(1)=r1*Sr*(1-(Sr)/k1)-beta1*Sr*Iv-rho1*Sr;
dy(2)=r2*Sc*(1-(Sc)/k2)-beta2*Sc*Iv-rho2*Sc;
dy(3)=beta1*Sr*Iv+beta2*Sc*Iv-rho3*Ic-a*Ic-sigma2*Ic;
dy(4)=b*(Sv+Iv)*(1-((Sv+Iv)/k3))-beta3*Sv*Ic-gamma*Sv-sigma1*Sv;
dy(5)=beta3*Sv*Ic-gamma*Iv-sigma1*Iv;
clear all
clc
%Runge Kuta forth order approach
```

```
tspan =[0 24];%Time in Months
y0 =[2000 1500 100 200 50];
[t,y]= ode45(@EACMD,tspan,y0);
[t1,y1]= ode45(@EACMDCONTROL,tspan,y0);
[t2,y2]= ode45(@control5,tspan,y0);
[t3,y3]= ode45(@control6,tspan,y0);
figure(1)
set(gca,'FontSize', 14')
set(legend,'FontSize', 10')
plot(t,y(:,1),'g',t1,y1(:,1),'k',t2,y2(:,1),'b--',t3,y3(:,1),'r'
    ,'linewidth',4)
xlabel('Time[Months]','Fontsize',25)
ylabel('Resistant breed','Fontsize',25)
% title('TOTAL DYNAMICS VS TIME','Fontsize',25)
legend({'$no$ $control$','$\sigma_1=0.5,\sigma_2=0.1$','$\
   sigma_1=0.7,\sigma_2=0.3$','$\sigma_1=0.8,\sigma_2=0.5$'},'
   Interpreter', 'Latex','FontSize',35)
grid on
hold off%Stop operation
hold on

figure(2)
set(gca,'FontSize', 14')
set(legend,'FontSize', 10')
plot(t,y(:,2),'g',t1,y1(:,2),'k',t2,y2(:,2),'b--',t3,y3(:,2),'r'
    ,'linewidth',4)
xlabel('Time[Months]','Fontsize',25)
ylabel('Susceptible  breed','Fontsize',25)
% title('TOTAL DYNAMICS VS TIME','Fontsize',25)
legend({'$no$ $control$','$\sigma_1=0.5,\sigma_2=0.1$','$\
   sigma_1=0.7,\sigma_2=0.3$','$\sigma_1=0.8,\sigma_2=0.5$'},'
   Interpreter', 'Latex','FontSize',35)
grid on
hold off%Stop operation
hold on

figure(3)
set(gca,'FontSize', 14')
set(legend,'FontSize', 10')
plot(t,y(:,3),'g',t1,y1(:,3),'k',t2,y2(:,3),'b--',t3,y3(:,3),'r'
    ,'linewidth',4)
xlabel('Time[Months]','Fontsize',25)
ylabel('Infected cassava','Fontsize',25)
% title('TOTAL DYNAMICS VS TIME','Fontsize',25)
legend({'$no$ $control$','$\sigma_1=0.5,\sigma_2=0.1$','$\
   sigma_1=0.7,\sigma_2=0.3$','$\sigma_1=0.8,\sigma_2=0.5$'},'
```

```
    Interpreter', 'Latex','FontSize',25)
grid on
hold off%Stop operation
hold on

figure(4)
set(gca,'FontSize', 14')
set(legend,'FontSize', 10')
plot(t,y(:,4),'g',t1,y1(:,4),'k',t2,y2(:,4),'b--',t3,y3(:,4),'r'
    ,'linewidth',4)
xlabel('Time[Months]','Fontsize',25)
ylabel('Susceptible  vector','Fontsize',25)
% title('TOTAL DYNAMICS VS TIME','Fontsize',25)
legend({'$no$ $control$','$\sigma_1=0.5,\sigma_2=0.1$','$\
    sigma_1=0.7,\sigma_2=0.3$','$\sigma_1=0.8,\sigma_2=0.5$'},'
    Interpreter', 'Latex','FontSize',35)
grid on
hold off%Stop operation
hold on

figure(5)
set(gca,'FontSize', 14')
set(legend,'FontSize', 10')
plot(t,y(:,5),'g',t1,y1(:,5),'k',t2,y2(:,5),'b--',t3,y3(:,5),'r'
    ,'linewidth',4)
xlabel('Time[Months]','Fontsize',25)
ylabel('Infected vector','Fontsize',25)
% title('TOTAL DYNAMICS VS TIME','Fontsize',25)
legend({'$no$ $control$','$\sigma_1=0.5,\sigma_2=0.1$','$\
    sigma_1=0.7,\sigma_2=0.3$','$\sigma_1=0.8,\sigma_2=0.5$'},'
    Interpreter', 'Latex','FontSize',25)
grid on
hold off%Stop operation
hold on
```

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MODELING THE DYNAMICS AND TRANSMISSION OF CASSAVA MOSAIC DISEASE IN TANZANIA

FLORENCE MAGOYO[1,*], JACOB ISMAIL IRUNDE[2], DMITRY KUZNETSOV[1]

[1]The Nelson Mandela African Institution of Science and Technology (NM-AIST), P.O Box 447, Arusha - Tanzania

[2]Department of Mathematics, Mkwawa University College of Education, P.O. Box 2513 Iringa-Tanzania

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Abstract. Cassava mosaic disease (CMD) is caused by cassava mosaic virus (CMV) and is transmitted by the whitefly vector called Bemisia tabaci. In this paper, the deterministic model for transmission dynamics of CMD is formulated by considering the whitefly vector, cassava resistant and susceptible breeds, and infected cassava. The basic reproduction number R_0 and sensitivity index for each parameter with respect to basic reproduction number R_0 are computed to determine which parameters are sensitive to the dynamics of cassava mosaic disease. Analysis shows that the death rate of whitefly vectors, the infection rate for susceptible vectors, the number of vectors that can be supported and the rate of loss of infected cassava due to disease are the most sensitive parameters to the dynamics of cassava mosaic disease. Numerical simulation indicates that, cassava new infections increase as the number of vectors that can be supported increase and acquire cassava mosaic disease. It shows that if control measures are not considered, then the susceptible breed and cassava resistant breed will be wiped out after five and ten months respectively. To control the disease, farmers are encouraged to apply control strategies such as spraying of insecticide, using of vector-resistant varieties, phytosanitation which involve the removal of infected cassava plants from the farm, crop hygiene and the use of free stem cutting method.

Keywords: cassava mosaic; dynamics; transmission; basic reproduction number; Tanzania; whitefly vector.

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*Corresponding author
E-mail addresses: floredamasi@gmail.com
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1. Introduction

Cassava (Manihot esculenta) is one of the crops which was firstly introduced in West Africa from Brazil at the end of 16^{th} Century by Portuguese and spread to other African countries [3, 19]. Cassava is grown in tropical and subtropical areas which experience low rainfall as the crop survives in drought climate [10], and this makes cassava a major staple food in the world. According to FAO, about 700 million people depend on cassava as their main food in Africa [20]. Production of cassava in Africa is becoming low due to a number of causes, notably pests and diseases [6]. Cassava Brown Streak Disease (CBSD) and Cassava Mosaic Disease (CMD) are the most important biotic constraints which have led to decrease in yields [13, 1]. Cassava mosaic virus (CMV) contaminates the cassava leaves and is transmitted by the whitefly vector called Bemisia tabaci [3]. There are other 500 different plants including weeds and crops which are host to whitefly vector [15, 17]. Different causes for transmission of cassava mosaic disease have been reported, this includes the use of infected cassava stem, the use of infected plant materials by the farmers [12] as well as the use of CBSD resistant breed which later becomes vulnerable to cassava mosaic disease [22, 25].

The infected cassava plant is characterized by leaf mosaic patterns and it can persist during the premature stage of cassava leaf development. The cassava leaves which are infected by the disease are warped, reduced in size and distorted with yellow color separating the ordinary green color which is the health part of the leaves. They then deteriorate and the new leaves bend [7]. Tanzania is among the countries that face this problem and the disease has been spreading at a fast rate leading to food shortages [24]. According to Tanzania Commissions for Science and Technology (COSTECH) production of cassava in Tanzania is only 8t/ha which is lower compared to 20t/ha that can be produced, the main causes of lower production are pests and diseases.

Studies have been conducted to analyze the transmission dynamics of cassava mosaic disease and the impact of different control strategies. Holt et al: [8] studied the model with susceptible and infected cassava, and susceptible and infectious vectors. The study show that using infected cutting tools and elimination of infectious cassava have a little effect on the occurrence of the disease. Hebert M.P [4], use the Markov chain models to find the probability of eliminating the

disease by using the stochastic process models.The model was applied to CMV, the numerical and analytical results show that the vector aggregation is growing in intricacy as a well as the possibility of a disease to be recognized in host plant. Lawrence et al: [14] use the system of differential equation to find the equilibrium value of the whitefly vector and the cassava plants. The result was analyzed using the finite difference method to assess the spatiotemporal spread of the disease. Results obtained were compared to the field data and the implication of controlling the CMV through the practical were explored. The study concluded that using of ACMD resistant strains of cassava and windbreaks will have positive results on cassava yields. This paper studies the dynamics of cassava mosaic disease by considering cassava resistant breed which only catch cassava mosaic disease through unhealthy cutting and susceptible breed which catch mosaic disease through unhealthy cutting and contact with whitefly vectors before implementing controls.

2. Materials and Methods

2.1. Model Development

The model is formulated by modifying the model which was developed by Holt *et al*. [8] to include breed which catches cassava mosaic disease through unhealthy cutting and susceptible breed that catches mosaic disease through unhealthy cutting and through contact with whitefly vector. The model consists of two groups of population. The first group includes the cassava population (N_C) which is divided into resistant (S_r) and Susceptible (S_C) breeds, and infected cassava (I_C). Second group includes the whitefly vector population (N_V) which consists of susceptible vector (S_V) and infectious vector (I_V).

Cassava resistant breed is replanted at a rate r_1 and is infected by cassava mosaic disease through unhealthy cutting at a rate β_1 and they are harvested at a rate ρ_1. The term k_1, represents the maximum plants for cassava resistant breed which can be planted. Cassava susceptible breed is replanted at a rate r_2, and is infected by cassava mosaic disease following contact with infected whitefly vector and unhealthy cutting at a rate β_2 while it is harvested at a rate ρ_2. The maximum plants of cassava susceptible breed that can be planted is k_2. Infected cassava

flourish following infection of cassava resistant breed through unhealthy cutting at a rate β_1, and infection of cassava susceptible breed through unhealthy cutting and contact with infected whitefly vector at a rate β_2 and they decrease due to the effect of cassava mosaic disease at a rate a and harvested at a rate ρ_3. Susceptible vector is recruited by birth at a rate b and catch infection following contact with infected cassava at a rate β_2. Also, k_3 is the maximum number of vectors that can be supported. Infected vector is recruited when susceptible vector catch infection following contact with infected cassava at a rate β_3 and γ is the death rate of whitefly vector.

2.2. Assumptions of the Model

The model assumes that, all whitefly vectors are born susceptible to cassava mosaic disease. The replanted cassava for both breeds are susceptible to CMD. The whitefly vector cannot transmit cassava mosaic disease to cassava resistant breed except through unhealthy cutting. Cassava susceptible breed gets cassava mosaic disease through contact with infected whitefly and through unhealthy cutting. Susceptible vectors can be infected when they come into contact with the infected cassava. The interaction between cassava and vector population is shown in Figure 1. Variables and parameters are described in Table 1 and 2 respectively.

TABLE 1. Variables' Descriptions

Variables	**Description**
S_r	Cassava resistant breed at time t.
S_C	Cassava susceptible breed at time t.
I_C	Infected cassava at time t.
S_V	Susceptible vectors at time t.
I_V	Infectious vectors at time t.

TABLE 2. Parameters' Descriptions

Parameters	Description
r_1	The rate of planting cassava resistant breed
ρ_1	The rate of harvesting cassava resistant breed
β_1	The rate of infection for cassava resistant breed.
r_2	The rate at which cassava susceptible breed is replanted.
ρ_2	The rate at which cassava susceptible breed is harvested
β_2	The rate at which cassava susceptible breed is infected
ρ_3	The rate at which infected cassava is harvested
a	The rate of loss of infected cassava due to disease
b	Recruitment rate for whitefly.
β_3	Vector infection rate
γ	The death rate of whitefly vectors
k_l	The maximum number of resistant breed that can be planted.
k_2	The maximum number of susceptible breed that can be planted
k_3	Maximum number of vectors that can be supported

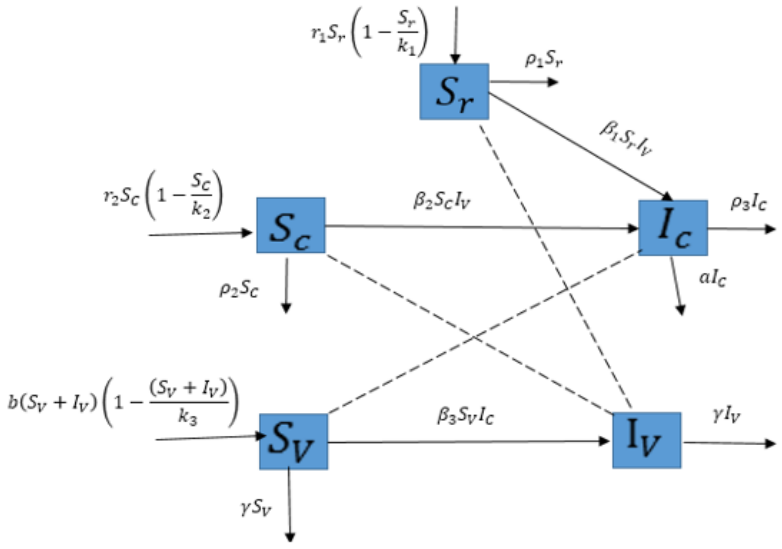


FIGURE 1. Compartmental Model for the transmission dynamics of Cassava Mosaic Disease

2.3. Model equations for the two groups

$$\frac{dS_r}{dt} = r_1 S_r \left(1 - \frac{S_r}{k_1}\right) - \beta_1 S_r I_V - \rho_1 S_r, \tag{1a}$$

$$\frac{dS_C}{dt} = r_2 S_C \left(1 - \frac{S_C}{k_2}\right) - \beta_2 S_C I_V - \rho_2 S_C, \tag{1b}$$

$$\frac{dI_C}{dt} = \beta_2 S_c I_v + \beta_1 S_r I_v - \rho_3 I_c - a I_c, \tag{1c}$$

$$\frac{dS_V}{dt} = b\left(S_V + I_V\right)\left(1 - \frac{S_V + I_V}{k_3}\right) - \beta_3 S_V I_C - \gamma S_V, \tag{1d}$$

$$\frac{dI_V}{dt} = \beta_3 S_V I_C - \gamma I_V, \tag{1e}$$

Subject to $S_r > 0, S_C > 0, I_C \geq 0, S_V \geq 0, I_V \geq 0$.

The total population of cassava is given as $S_r + S_C + I_C = N_C$ and the total population of vector is given as $N_V = S_V + I_V$.

2.4. Basic Properties of the Model

Invariant Region: Metzer matrix is used to show the feasible region, in which the variables are positive $\forall t \geq 0$. To deduce the feasible region; the model system (1a)-(1e) can be written as:

$$\frac{dx}{dt} = Ax + F, \tag{2}$$

where $x = (S_r, S_C, I_C, S_V, I_V)^T$ and a constant term $F = (0,0,0,0,0)^T$ such that:

$$Ax = \begin{pmatrix} -q_1 & 0 & 0 & 0 & 0 \\ 0 & -q_2 & 0 & 0 & 0 \\ \beta_1 I_V & \beta_2 I_V & -q_3 & 0 & (\beta_2 S_C + \beta_1 S_r) \\ 0 & 0 & 0 & -q_4 & (b - 2\frac{(S_V + I_V)}{k_3}) \\ 0 & 0 & 0 & \beta_3 I_V & -\gamma \end{pmatrix}, \tag{3}$$

for;

$q_1 = \beta_1 I_V + \rho_1 - r_1\left(1 - 2\frac{S_r}{k_1}\right)$, $q_2 = \beta_2 I_V + \rho_2 - r_2\left(1 - 2\frac{S_C}{k_2}\right)$,

$q_3 = \rho_3 + a$, $q_4 = \gamma + \beta_3 I_C - b - 2\frac{(S_V+I_V)}{k_3}$.

In equation (3), A is a Metzler matrix $\forall x \in \mathbb{R}^5$ and due to the fact that $F \geq 0$, the model system (1a) - (1e) is positive invariant in $\mathbb{R}^5$ and F is Lipschitz continuous. Therefore the feasible region Ω is a set of $\Omega = \{S_r, S_C, I_C, S_V, I_V \in \mathbb{R}^5\}$ with initial condition $S_r > 0$, $S_C > 0$, $I_C \geq 0$, $S_V > 0$, $I_V \geq 0$.

Positivity of the solutions:

Let the initial condition be $S_r(0), S_C(0), I_C(0), S_V(0), I_V(0)$, the solutions S_r, S_c, I_c, S_v, I_v of the model system (1a) - (1e) are positive $\forall t > 0$. We show that, the solution of the model system (1a) - (1e) are positive by starting with equation (1a) that:

$$\frac{dS_r}{dt} \geq -(\beta_1 S_r I_V + \rho_1 S_r). \tag{4}$$

Separate the variables and integrate both sides of the equation,

$$\int \frac{1}{S_r} \mathrm{d}S_r \geq \int -(\beta_1 I_v + \rho_1)\, \mathrm{d}t, \tag{5}$$

$$\ln(S_r) \geq -(\beta_1 I_v + \rho_1)\, t + C. \tag{6}$$

This give the values of S_r as:

$$S_r(t) \geq A e^{-(\beta_1 I_v + \rho_1)t}. \tag{7}$$

At initial condition time, $t = 0$, equation (7) above becomes

$$S_r(0) \geq A, \tag{8}$$

Therefore

$$S_r(t) \geq S_r(0)\, e^{-(\beta_1 I_v + \rho_1)t}. \tag{9}$$

Thus, $S_r(0) \geq 0$, $\forall t > 0$.

Apply the same procedure to the remaining equations (1b), (1c), (1d) and (1e):
We get

$$S_C(t) \geq S_C(0)\, e^{-(\beta_2 I_v + \rho_2)t}. \tag{10}$$

$$I_C(t) \geq I_C(0)\, e^{-(\rho_3 + a)t}. \tag{11}$$

$$S_V(t) \geq S_V(0)\, e^{-(\beta_3 I_c + \gamma)t}. \tag{12}$$

$$I_v(0) \geq I_v(0)\, e^{-\gamma t}. \tag{13}$$

Here we conclude that, the requirement to study the dynamics of CMD is satisfied considering that, all the solutions of the model (1a) - (1e) are positive and bounded in the region:

$$\Omega = \{S_r(t), S_C(t), I_C(t), S_V(t), I_V(t)\}. \tag{14}$$

2.5. Cassava Mosaic Free Equilibrium

The steady state when there is no cassava mosaic disease is called cassava mosaic free equilibrium. We compute cassava mosaic free equilibrium when $I_c = I_v = 0$. At this state, the total cassava plants is the sum of susceptible and resistant breeds. However, the population of the vector at this state consists of susceptible whitefly vector. Cassava mosaic free equilibrium is given by:

$$F^0 = (S_r, S_C, I_C, S_V, I_V) = \left(\frac{(r_1 - \rho_1)\, k_1}{r_1}, \frac{(r_2 - \rho_2)\, k_2}{r_2}, 0, \frac{(b - \gamma)\, k_3}{b}, 0 \right). \tag{15}$$

2.6. Basic Reproduction Number R_0

The basic reproduction number is denoted by R_0. It refers to an expected number of secondary infections from an infected whitefly when introduced into a susceptible population of cassava plants [5]. If $R_0 > 1$, the infectious whitefly can transmit the cassava mosaic disease to more

than one cassava plants, and if $R_0 < 1$, an infectious whitefly transmits the cassava mosaic disease to less than one cassava plants, hence the disease is dying out. The basic reproductive number will be determined by next generation matrix [11] as follows:

Assume that, $f_i(\bar{x})$ is the rate of cassava and whitefly new infections and $V_i(\bar{x}) = V_i^-(\bar{x}) - V_i^+(\bar{x})$ where $V_i^+(\bar{x})$ are the terms that are transferred into the compartment and $V_i^-(\bar{x})$ are the terms that are transferred out of the compartment such that: [5].

$$F = \frac{\partial f_i(x_0)}{\partial(x_j)} \quad and \quad V = \frac{\partial V_i(x_0)}{\partial(x_j)}, \tag{16}$$

where $i, j = 1, 2, ..., m$ and x_0 indicates the cassava mosaic free equilibrium. From the model system (1a)- (1e), f_i and V_i are defined by:

$$f_i = \begin{pmatrix} \beta S_r I_V + \beta_2 S_C I_V \\ \beta_3 S_V I_C \\ \end{pmatrix} \tag{17}$$

and

$$V_i = \begin{pmatrix} \rho_3 + aI_C \\ \gamma I_V \\ \end{pmatrix}. \tag{18}$$

Matrices F and V are obtained by differentiating equation (17) and (18) respectively, with respect to I_c and I_v so that:

$$F = \begin{pmatrix} 0 & \beta S_r + \beta_2 S_C \\ \beta_3 S_V & 0 \\ \end{pmatrix} \tag{19}$$

and

$$V = \begin{pmatrix} \rho_3 + aI_C & 0 \\ 0 & \gamma \\ \end{pmatrix}. \tag{20}$$

The next generation matrix is given by:

$$FV^{-1} = \begin{pmatrix} 0 & \frac{\beta_2 S_C + \beta_1 S_r}{\gamma} \\ \frac{\beta_3 S_V}{\rho_3 + a} & 0 \end{pmatrix}. \tag{21}$$

The basic reproduction number R_0 for cassava plants and vector is a dominant eigenvalue of the next generation matrix FV^{-1} [18]. The basic reproduction number R_0 is therefore given by:

$$R_0 = \sqrt{\frac{\beta_3 (b-\gamma) k_3}{b(\rho_3 + a)\gamma} \left(\frac{(r_1 - \rho_1) k_1 \beta_1}{r_1} + \frac{(r_2 - \rho_2) k_2 \beta_2}{r_2} \right)}. \tag{22}$$

From equation (22), basic reproduction number R_0 is determined by all parameters from the model. The basic reproduction number R_0 increases in proportion to $\beta_3, b, k_3, \beta_1, \beta_2, k_1, r_1, k_2$ and r_2, and decreases as $\gamma, \rho_3, \rho_2, a$ and ρ_1 increase.

3 . Sensitivity analysis

Sensitivity index of a parameter tells how a parameter is sensitive to the disease. In this section, sensitivity index of each parameter with respect to basic reproduction number R_0 is derived to determine how each parameter influences the disease. If f is a parameter in reproduction number R_0 then, sensitivity index of f with respect to R_0 is given by:

$$\Upsilon_f^{R_0} = \frac{dR_0}{df} \times \frac{f}{R_0}. \tag{23}$$

3.1. Parameters Adoption

Parameter values from the literature and assumed ones are used. Table 3 summarizes the parameter values, range and their sources.

TABLE 3. Parameter Values.

Parameters	**Value**	**Range**	**Source**
r_1	$0.025 day^{-1}$		Assumed
ρ_1	$0.005 day^{-1}$		Assumed
β_1	$0.0012 vector^{-1} day^{-1}$		Assumed
r_2	$0.2 day^{-1}$	$0.025-0.2$	[14]
ρ_2	$0.003 day^{-1}$	$0.002-0.004$	[8]
β_2	$0.003 vector^{-1} day^{-1}$	$0.002-0.032$	[8]
ρ_3	$0.003 day^{-1}$	$0.002-0.004$	[8]
a	$0.033 day^{-1}$	$0-0.033$	[8]
b	$0.5 vector^{-1} day^{-1}$	0.1-1.0	[26]
β_3	$0.002 plant^{-1} day^{-1}$	$0.002-0.032$	[8]
γ	$0.0782 day^{-1}$	$0.06-0.18$	[8]
k_1	3000		[21]
k_2	2000		Assumed
k_3	350	0-2500	[8]

Using forward normalized sensitivity index for each parameter with respect to basic reproduction number R_0, sensitivity index for β_2 is derived as follows:

$$\Upsilon_{\beta_2}^{R_0} = \frac{dR_0}{d\beta_2} \times \frac{\beta_2}{R_0}, \tag{24}$$

$$\frac{dR_0}{d\beta_2} = 1/2\,\frac{\beta_3\,(b-\gamma)\,k_3\,(r_2-\rho_2)\,k_2}{r_2 b\,(\rho_3+a)\,\gamma}\,\frac{1}{\sqrt{\frac{\beta_3(b-\gamma)k_3}{b(\rho_3+a)\gamma}\left(\frac{(r_1-\rho_1)k_1\beta_1}{r_1}+\frac{(r_2-\rho_2)k_2\beta_2}{r_2}\right)}}. \tag{25}$$

Full computation gives:

$$\Upsilon_{\beta_2}^{R_0} = +0.3362. \tag{26}$$

We apply the same method to obtain sensitivity indices for other parameters. Table 4 summarizes sensitivity indices for all parameters with respect to basic reproduction number R_0.

TABLE 4. Sensitivity Indices.

Parameters	**Sensitivity index**	**Parameters**	**Sensitivity index**
β_3	+0.5000	r_2	$+0.0051$
β_1	$+0.1638$	γ	-0.5927
β_2	$+0.3362$	ρ_3	-0.0417
k_3	$+0.5000$	ρ_2	-0.0051
b	$+0.0927$	ρ_1	-0.0410
k_1	$+0.1638$	a	-0.4583
r_1	$+0.0410$	k_2	$+0.3362$

From the Table 4 parameters $\beta_2, \beta_3, \beta_1, k_1, k_2, k_3, b, r_1, r_2$ have positive indices, this means that the basic reproduction number R_0 increase in proportion to these parameters. Parameters a, ρ_1, ρ_2, ρ_3 and γ have negative indices. This means that the basic reproduction number R_0 decrease when a, ρ_1, ρ_2, ρ_3 and γ increase. The most sensitive parameter is the death rate of whitefly vectors γ, the increase of this parameter decrease the basic reproduction number R_0.

4 . Global Stability of Cassava Mosaic Free Equilibrium

The global stability of cassava mosaic free equilibrium is established by approach used by Castillo-Chavez [2]. When this approach is used, system (1a) - (1e) is written as follows:

$$\frac{dX_1}{dt} = H(X_1 - X_{F_0}) + H_1 X_2, \tag{27}$$

$$\frac{dX_2}{dt} = GX_2, \tag{28}$$

where X_1 presents the noninfectious classes and X_2 infectious classes. $X_{(F_0)}$ present mosaic free equilibrium. Mosaic free equilibrium is said to be globally asymptotically stable if eigenvalues of matrix H are negative and matrix G is a Metzler matrix [9]. We thus define X_1, X_2 and

X_{F_0} by:

$$X_1 = \begin{pmatrix} S_r \\ S_C \\ S_V \end{pmatrix}. \tag{29}$$

$$X_2 = \begin{pmatrix} I_C \\ I_V \end{pmatrix}. \tag{30}$$

$$X_{F_0} = \begin{pmatrix} \frac{(r_1-\rho_1)k_1}{r_1} \\ \frac{(r_2-\rho_2)k_2}{r_1} \\ 0 \\ \frac{(b-\gamma)k_3}{b} \\ 0 \end{pmatrix}. \tag{31}$$

Matrices H_1 and H are defined by:

$$H_1 = \begin{pmatrix} 0 & -\beta_1 S_r \\ 0 & -\beta_2 S_C \\ -\beta_3 S_V & b - \frac{2b(S_V+I_V)}{k_3} \end{pmatrix} \tag{32}$$

and

$$H = \begin{pmatrix} -q_1 & 0 & 0 \\ 0 & -q_2 & 0 \\ 0 & 0 & -q_3 \end{pmatrix}, \tag{33}$$

where

$q_1 = (r_1 + 2\,\frac{r_1 S_r}{k_1} + \beta_1 I_V + \rho_1)$, $q_2 = (r_2 + 2\,\frac{r_2 S_C}{k_2} + \beta_2 I_V + \rho_2)$, $q_3 = (b + \frac{2b(S_V+I_V)}{k_3} + \beta_3 I_C + \gamma)$.

Matrix H has negative eigenvalues and matrix G is Metlzer matrix since elements in the main diagonal are negative and the off diagonal elements are positive provided the rate of planting cassava is greater than the rate at which they are harvested and the recruitment rate of whitefly vectors is greater than their death rate. Therefore, when the basic reproduction number R_0,

is less than one $(R_0 < 1)$ and greater than one $(R_0 > 1)$, then the disease free equilibrium is globally asymptotically stable and unstable respectively.

5 . Global Stability of Cassava Mosaic Free Equilibrium

Due to non-linear nature of the model, it is not possible to obtain cassava mosaic equilibrium explicitly. To prove existence of cassava mosaic equilibrium, we state and prove the following theorem:

Theorem: Cassava mosaic equilibrium exists if $S_r^* > 0, S_C^* > 0, I_C^* > 0, S_V^* > 0, I_V^* > 0$.

Proof: Approach in Tumwine et al. [23] and Massawe et al: [16] is adopted in proving existence of cassava mosaic equilibrium. We use the sum of cassava plants and whitefly vectors when their rate of change is zero. When we consider total cassava plants at cassava mosaic equilibrium, we have:

$$r_1 S_r^* \left(1 - \frac{S_r^*}{k_1}\right) + r_2 S_C^* \left(1 - \frac{S_C^*}{k_2}\right) - \rho_1 S_r^* - \rho_2 S_C^* - (\rho_3 + a) I_C^*. \tag{34}$$

This lead to :

$$\rho_1 S_r^* + \rho_2 S_C^* + (\rho_3 + a) I_C^* = r_1 S_r^* \left(1 - \frac{S_r^*}{k_1}\right) + r_2 S_C^* \left(1 - \frac{S_C^*}{k_2}\right). \tag{35}$$

Since $S_r^* < k_1, S_C^* < k_2$ and all the parameters are positive.

Then:

$$r_1 S_r^* \left(1 - \frac{S_r^*}{k_1}\right) + r_2 S_C^* \left(1 - \frac{S_C^*}{k_2}\right) > 0, \tag{36}$$

showing that: $S_r^* > 0, S_C^* > 0$ and $I_C^* > 0$. Using the same approach for whitefly vector we have $S_V^* > 0$ and $I_V^* > 0$. This shows that cassava mosaic equilibrium exists.

5.1. Global Stability of Cassava Mosaic Equilibrium

The global stability of cassava mosaic equilibrium is investigated by logarithmic Lyapunov function which is given by:

$$L = \sum G_i \left(P_i - P_i^* ln P_i\right), \tag{37}$$

where G_1, is a positive constant which is to be chosen carefully, P_i is a variable in a compartment i and P^* present a compartment variable at equilibrium point. Using system (37) the Lyapunov function is defined by;

$$\begin{aligned} L(S_rS_C,I_C,S_V,I_V) = G_1\left(S_r - {S_r}^* lnS_r\right) \\ +G_2\left(S_C - {S_C}^* lnS_C\right) \\ +G_3\left(I_C - {I_C}^* lnI_C\right) \\ +G_4\left(S_V - {S_V}^* lnS_V\right) \\ +G_5\left(I_V - {I_V}^* lnI_V\right). \end{aligned} \tag{38}$$

Differentiate the Lyapunov function (38) above with respect to time, we get

$$\begin{aligned} \frac{dL}{dt} =& G_1\left(1-\frac{S_r^*}{S_r}\right)\frac{dS_r}{dt} + G_2\left(1-\frac{S_C^*}{S_C}\right)\frac{dS_C}{dt} + G_3\left(1-\frac{I_C^*}{I_C}\right)\frac{dI_C}{dt} \\ &+ G_4\left(1-\frac{S_V^*}{S_V}\right)\frac{dS_V}{dt} + G_5\left(1-\frac{I_V^*}{I_V}\right)\frac{dI_V}{dt} \end{aligned} \tag{39}$$

From equations (39), we have:

$$\begin{aligned} \frac{dL}{dt} =& G_1\left(1-\frac{S_r^*}{S_r}\right)\left(r_1S_r\left(1-\frac{S_r}{k_1}\right) - \beta_1S_rI_V - \rho_1S_r\right) \\ &+ G_2\left(1-\frac{S_C^*}{S_C}\right)\left(r_2S_C\left(1-\frac{S_C}{k_2}\right) - \beta_2S_CI_V - \rho_2S_C\right) \\ &+ G_3\left(1-\frac{I_C^*}{I_C}\right)\left(\beta_2S_CI_V + \beta_1S_rI_V - \rho_3I_C - aI_C\right) \\ &+ G_4\left(1-\frac{S_V^*}{S_V}\right)\left(b\left(S_V+I_V\right)\left(1-\frac{S_V+I_V}{k_3}\right) - \beta_3S_VI_C - \gamma S_V\right) \\ &+ G_5\left(1-\frac{I_V^*}{I_V}\right)\left(\beta_3\ S_VI_C - \gamma I_V\right). \end{aligned} \tag{40}$$

At cassava mosaic equilibrium, equation (40) becomes:

$$\begin{aligned}\frac{dL}{dt} =& -G_1\rho_1\frac{(S_r-S_r^*)^2}{S_r} - G_2\rho_2\frac{\left(S_C-S_C^*\right)^2}{S_C} - G_3(\rho_3+a)\frac{\left(I_C-I_C^*\right)^2}{I_C} - G_4\gamma\frac{(S_V-S_V^*)^2}{S_V}\\ &- G_5\gamma\frac{(I_V-I_V^*)^2}{I_V} - G_1\beta_1\frac{(S_r-S_r^*)\left(S_rI_V-S_r^*I_V^*\right)}{S_r} - G_2\beta_2\frac{(S_C-S_{C^*})\left(S_CI_V-S_C^*I_V^*\right)}{S_r}\\ &- G_4\beta_3\frac{(S_V-S_V^*)\left(S_VI_C-S_V^*I_V^*\right)}{S_V},\end{aligned} \tag{41}$$

this simplifies to:

$$\begin{aligned}\frac{dL}{dt} =& -G_1\rho_1\frac{(S_r-S_r^*)^2}{S_r} - G_2\rho_2\frac{\left(S_C-S_C^*\right)^2}{S_C} - G_3(\rho_3+a)\frac{\left(I_C-I_C^*\right)^2}{I_C} - G_4\gamma\frac{(S_V-S_V^*)^2}{S_V}\\ &- G_5\gamma\frac{(I_V-I_V^*)^2}{I_V} + F(\Omega),\end{aligned} \tag{42}$$

where:

$$\begin{aligned}F(\Omega) =& -G_1\beta_1\frac{(S_r-S_r^*)\left(S_rI_V-S_r^*I_V^*\right)}{S_r} - G_2\beta_2\frac{\left(S_C-S_C^*\right)\left(S_CI_V-S_C^*I_V^*\right)}{S_r}\\ &- G_4\beta_3\frac{(S_V-S_V^*)\left(S_VI_C-S_V^*I_C^*\right)}{S_V}.\end{aligned} \tag{43}$$

The function $F(\Omega)$ is negative or zero in Ω, therefore $\frac{dL}{dt} \leq 0$ in Ω and it is zero for $\Omega = \Omega^*$. Since $\frac{dL}{dt} = 0$ when $\Omega = \Omega^*$ and $\frac{dL}{dt} \leq 0$ in Ω then the largest invariant set in Ω when $\frac{dL}{dt} = 0$ is a singleton Ω^* which is cassava mosaic equilibrium point. By LaSalles invariant principle, the casssava mosaic equilibrium Ω^* is globally asymptotically stable when $R_0 > 1$.

6. Numerical Simulation of the Basic Model

In this section, we simulate model system (1) to determine the long term impact of cassava mosaic disease. We simulate the dynamics of cassava mosaic disease by considering sensitive parameters.

The dynamics of cassava mosaic disease is demonstrated in Figure 2. All susceptible vectors contract the disease before five months, this is reflected by susceptible cassava which also decrease due to the disease. Cassava resistant breed takes longer to get cassava mosaic disease as demonstrated in Figure 2. Figure 3 illustrates cassava and vector populations.

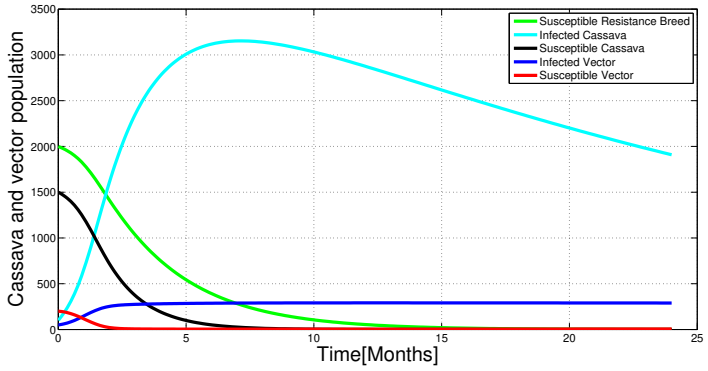
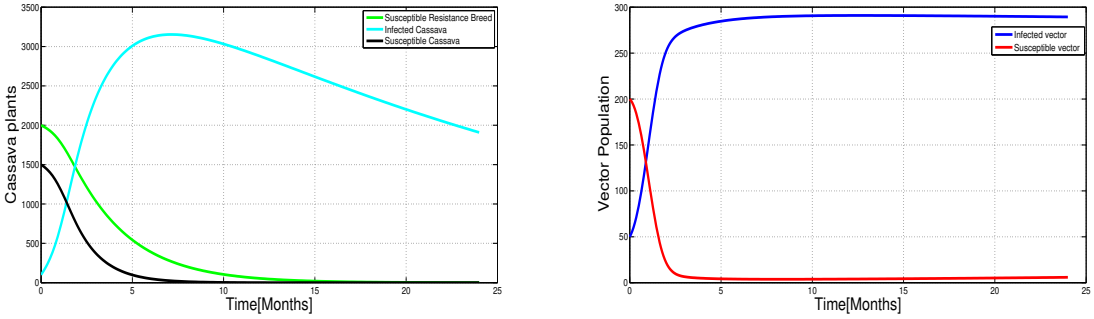


FIGURE 2. Total Population



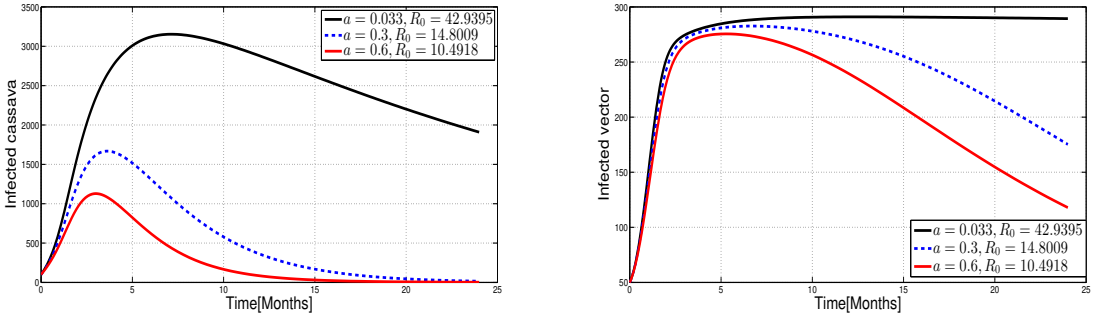
(A) (B)

FIGURE 3. Vector and Cassava Population

The variation of sensitive parameters shows that cassava mosaic disease increase proportionally to recruitment rate of whitefly vectors and decreases as the rate of loosing infected cassava increases. All classes are demonstrated in Figures below as follows.

Figure 4 demonstrates the variation of the rate of loss of infected cassava to the infected classes. It shows the behavior of infected cassava and infected vectors when the parameter a vary, the increase of a lead to the decrease of infected cassava and the decrease of infected

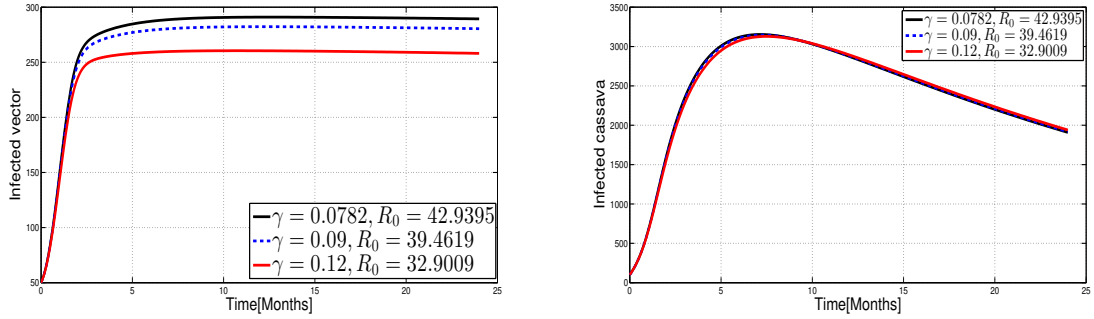
vector.



(A) Infected cassava. (B) Infected vector.

FIGURE 4. Variation of loss of infected cassava rate in infected class.

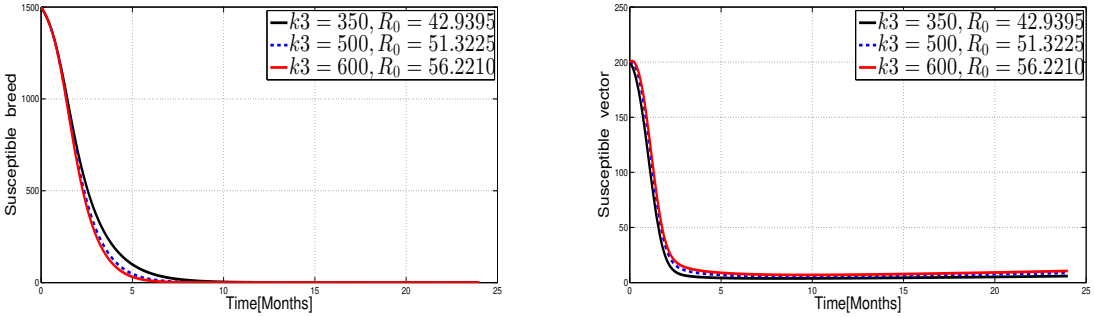
Figure 5, shows the variation of vector mortality rate γ to the infectious vector and infected cassava class, if the rate of vector mortality increase the number of infected vector and infected cassava decreases.



(A) Infected vector. (B) Infected cassava.

FIGURE 5. Variation of vector mortality rate in infectious class.

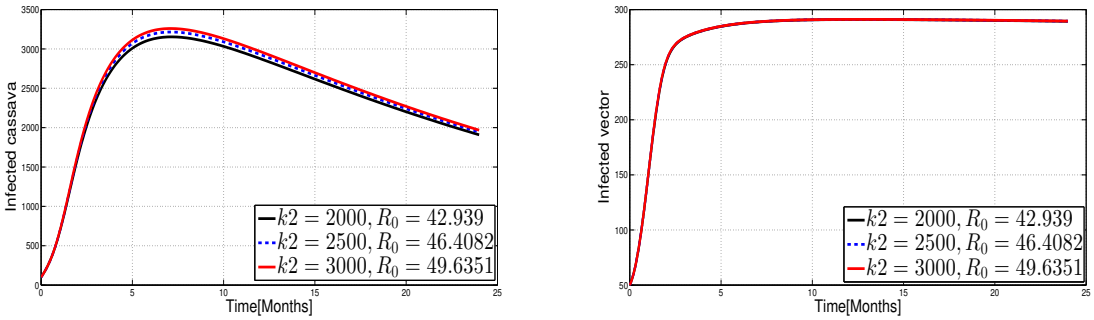
From Figure 6 the graphs demonstrate the variation of vector carrying capacity k_3 to the susceptible class of cassava and susceptible class of vector. The graphs show as the carrying capacity of whitefly vectors increase the number of susceptible cassava breed decrease, the number of susceptible vector increase.



(A) Susceptible breed. (B) Susceptible vector.

FIGURE 6. Variation of vector carrying capacity to the susceptible class.

Figure 7 shows the impact of varying the carrying capacity of susceptible breed of cassava to the infected cassava class and infected vector . It shows that as the carrying capacity of susceptible breed increases the number of infected cassava and infected vector increases.



(A) Infected cassava. (B) Infected vector.

FIGURE 7. Variation of cassava susceptible breed carrying capacity to the infected class.

7. Conclusion

In this paper, the deterministic model for transmission dynamics of CMD which includes population of cassava and whitefly vector is presented and analyzed. The sensitivity analysis was performed to identify sensitive parameters. Analysis shows that the number of vectors that can be supported, the rates at which vectors acquire disease and the carrying capacity of susceptible cassava breed, play important role in the transmission dynamics of cassava mosaic

disease. New infections will increase as the carrying capacity of susceptible cassava and the rate of infection of vectors increases. To improve cassava productivity, campaigns to eradicate cassava mosaic disease should focus on strategies which reduce vectors' population. These strategies include spraying insecticide, use of vector-resistant varieties, phytosanitation which involve the removal of infected cassava plants from the place that will be used for the new plantings, crop hygiene and the use of free stem cutting method.

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Conflict of Interests

The authors declare that there is no conflict of interests.

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