



Available online at <http://scik.org>

Commun. Math. Biol. Neurosci. 2026, 2026:105

<https://doi.org/10.28919/cmbn/10130>

ISSN: 2052-2541

MATHEMATICAL MODEL ANALYSIS AND CONTROL OF CASSAVA MOSAIC AND CASSAVA BROWN STREAK CO-INFECTION

CHINENYE OMEH¹, OBIORA C. COLLINS^{2,*}, GODWIN C.E. MBAH¹

¹Department of Mathematics, University of Nigeria, Nsukka, Nigeria

²Institute of Systems Science, Durban University of Technology, Durban, 4000, South Africa

Copyright © 2026 the author(s). This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Abstract. Cassava is a major staple crop that contributes significantly to food security and economic development in many countries especially in Nigeria. However, its production is severely threatened by Cassava Mosaic Disease (CMD) and Cassava Brown Streak Disease (CBSD), which can result in significant yield losses. Cassava Mosaic disease (CMD) and Cassava Brown Streak disease (CBSD) co-infections is one of the most destructive infections of cassava that can lead to 90% yield loss if not controlled. In this study, a mathematical model that considered the co-infection of CMD and CBSD is use to analyse the dynamics and control of the co-infections. The model is formulated using a system of non-linear differential equations and then transformed into a non-dimensional form to facilitate mathematical analysis and numerical computation. The mathematical analysis of the model which is crucial for understanding the dynamics of the model are carried out accordingly. To enhance the biological relevance of the model, real-world data obtained from World Bank dataset and related agricultural datasets were utilized for model calibration. Unknown model parameters were estimated using the least-squares fitting technique, enabling the model to reproduce observed disease trends. The estimated parameter values were then employed to investigate the future dynamics of CMD-CBSD co-infection through predictive simulations. In addition, numerical experiments were conducted to evaluate the effectiveness of various disease control strategies, including interventions targeting infected plants and vector populations.

*Corresponding author

E-mail address: obiora.c.collins@gmail.com

Received July 14, 2026

Keywords: cassava mosaic disease; cassava brown streak disease; co-infection; host–vector; basic reproduction number; stability analysis; disease control.

2020 AMS Subject Classification: 92B05, 00A69.

1. INTRODUCTION

Millions of people worldwide rely heavily on cassava, one of the most significant staple food crops in the tropics, for their energy needs [1]. Because of its ability to adapt to a variety of agro-ecological circumstances, including drought and poor soils, cassava plays a vital role in food security, household income, and industrial growth in sub-Saharan Africa [2]. With a significant share of the world’s production, Nigeria is the world’s top producer of cassava. The crop is essential to rural farming’s economic viability, poverty alleviation, and food availability. Abiotic factors including rainfall, temperature, and soil type, as well as biotic factors like viral diseases, bacteria, fungi, white flies, and weeds, continue to limit cassava productivity despite its significance to humans [3, 4, 5]. Among these limitations, one of the biggest risks to cassava production is viral infections. In several cassava-growing locations, in particular, Cassava Mosaic Disease (CMD) and Cassava Brown Streak Disease (CBSD) have resulted in notable yield reductions [6, 7, 8]. Farmers suffer significant financial losses as a result of these illnesses, which are mostly spread by diseased stem cuttings and the whitefly vector (*Bemisia tabaci*). Co-infection, or the simultaneous presence of CMD and CBSD in the same cassava field, presents an even bigger problem for cassava production. Compared to plants infected by either disease alone, co-infected plants frequently exhibit more severe symptoms and larger output losses. The transmission of cassava mosaic disease, vector-host interactions, disease persistence, and the impact of management strategies have all been studied quantitatively and theoretically by existing mathematical models in the literature [9, 10, 11, 12, 13, 14, 15].

To the best of our knowledge, no mathematical model on the co-infection of CMD-CBSD using actual data validation has been done with Nigeria as a case study. In order to fill in these gaps, this study develop a mathematical model for the dynamics of CMD and CBSD co-infection in the form of non-linear ordinary differential equation that was later transformed to non dimensionalized. The paper builds a data-driven modelling framework using accessible agricultural and epidemiological data. The least-squares fitting method is used to estimate

model parameters, which improves the model's ability to properly represent observed illness trends. Using predictive simulations based on estimated parameter values, the fitted model is then used to examine the dynamics of cassava viral infections in the future. Numerical simulations are also used to analyze the course of the disease and the possible effects of control strategies.

2. MODEL FORMULATION

Mathematical model formulation for the co-infection of Cassava Mosaic Disease (CMD) and Cassava Brown Streak Disease (CBSD), is presented in this paper. The model formulation is based on the following assumptions. At time t , the model takes into account two populations that interact: the population of cassava plants, represented by $N_c(t)$, and the population of white-fly vectors, represented by $N_v(t)$. The population of cassava plants is partitioned into four compartments: susceptible/healthy cassava plants, $S(t)$; cassava plants infected with CMD, $I_m(t)$; cassava plants infected with CBSD, $I_b(t)$; and cassava plants co-infected with both CMD and CBSD, $I_{mb}(t)$. Similarly, the vector population is partitioned into four compartments namely: susceptible vectors, $X(t)$, vectors infected with CMD, $Y_m(t)$, vectors infected with CBSD, $Y_b(t)$, and vectors co-infected with both CMD and CBSD, $Y_{mb}(t)$. Note that the diseases are not vertically transmitted and that transmission does not occur directly between cassava plants or directly among vectors. Consequently, disease transmission occurs exclusively through interactions between infected vectors and susceptible cassava plants, and vice versa. The population of cassava plants is considered to increase logistically with intrinsic growth rate r to a carrying capacity K in the absence of disease. Susceptible cassava plants S get infected when it comes in contact with CMD-infected vectors Y_m or CBSD-infected vectors, Y_b at a rate β_m or β_b respectively. Additionally, plants that are already infected with one disease (I_m, I_b) may contract the second and become co-infected (I_{mb}) at a rate α_m and α_b respectively. Disease-induced mortality occurs at rates μ_m , μ_b , and μ_{mb} for I_m, I_b, I_{mb} , respectively. The vector population is recruited through migration at a constant rate Λ . Susceptible vectors acquire CMD and CBSD after feeding on infected cassava plants at rates σ_m and σ_b , respectively. Similarly, vectors infected with one disease may acquire the second disease after feeding on cassava plants infected with the alternate disease, thereby becoming co-infected at rates ρ_m and ρ_b . The virus is assumed to have

no adverse effect on the vector survival; however, vectors experience natural mortality at rate δ . The model incorporates four control measures to lower the spread of infections. The first is preventive control, denoted by c , which lowers the rate of disease transmission by the use of resistant cassava cultivars. The second is mechanical control, denoted as ζ , which reduces the spread of infection by removing infected cassava plants (rouged). Chemical control, η_1 , and biological control, η_2 , are the third and fourth controls, respectively, that lower the vector population by employing pesticides and biological trapping techniques respectively. As part of an integrated disease management approach, these control measures can be applied concurrently and independently.

A schematic representation of the CMD-CBSD co-infection model is given as a flow diagram below.

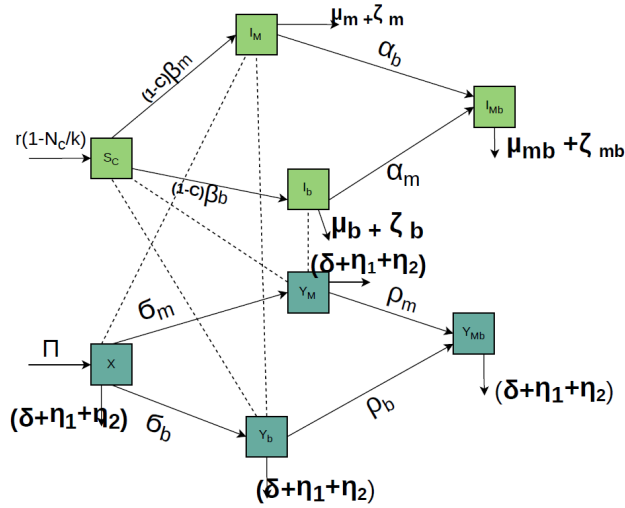


FIGURE 1. CMD-CBSD Co-infection of Cassava flow Digram

Based on the above assumptions and formulations, the CMD-CBSD co-infection model are described by the following system of ordinary differential equations.

$$\begin{aligned} \frac{dS}{dt} &= rS \left(1 - \frac{S}{K} \right) - (1-c)\tilde{\beta}_m Y_m S - (1-c)\tilde{\beta}_b Y_b S - (1-c)\tilde{\beta}_{mb} Y_{mb} S, \\ \frac{dI_m}{dt} &= (1-c)\tilde{\beta}_m Y_m S - (1-c)\tilde{\alpha}_b Y_b I_m - \tilde{\mu}_m I_m - \tilde{\zeta} I_m, \\ \frac{dI_b}{dt} &= (1-c)\tilde{\beta}_b Y_b S - (1-c)\tilde{\alpha}_m Y_m I_b - \tilde{\mu}_b I_b - \tilde{\zeta} I_b, \end{aligned}$$

$$\begin{aligned}
 (1) \quad \frac{dI_{mb}}{dt} &= (1-c)\tilde{\beta}_{mb}Y_{mb}S + (1-c)\tilde{\alpha}_bY_bI_m + (1-c)\tilde{\alpha}_mY_mI_b - \tilde{\mu}_{mb}I_{mb} - \tilde{\zeta}I_{mb} \\
 \frac{dX}{dt} &= \Lambda - \tilde{\sigma}_mXI_m - \tilde{\sigma}_bXI_b - \tilde{\sigma}_{mb}XI_{mb} - \tilde{\delta}X - \tilde{\eta}_1X - \tilde{\eta}_2X, \\
 \frac{dY_m}{dt} &= \tilde{\sigma}_mXI_m - \tilde{\rho}_mY_mI_b - \tilde{\delta}Y_m - \tilde{\eta}_1Y_m - \tilde{\eta}_2Y_m, \\
 \frac{dY_b}{dt} &= \tilde{\sigma}_bXI_b - \tilde{\rho}_bY_bI_m - \tilde{\delta}Y_b - \tilde{\eta}_1Y_b - \tilde{\eta}_2Y_b, \\
 \frac{dY_{mb}}{dt} &= \tilde{\sigma}_{mb}XI_{mb} + \tilde{\rho}_mY_mI_b + \tilde{\rho}_bY_bI_m - \tilde{\delta}Y_{mb} - \tilde{\eta}_1Y_{mb} - \tilde{\eta}_2Y_{mb}.
 \end{aligned}$$

The variables and parameters description are presented in Tables 1 and 2 respectively. The total number of cassava and vector are given by $N_c = S + I_m + I_b + I_{mb}$ and $N_v = X + Y_m + Y_b + Y_{mb}$.

TABLE 1. Variable description

Variable	Description	Unit
N_c	Total population of cassava plants	Plants Ha^{-1}
S_c	Susceptible population of cassava plants	Plants Ha^{-1}
I_m	Population of cassava plants infected by CMD	Plants Ha^{-1}
I_b	Population of cassava plants infected by CBSD	Plants Ha^{-1}
I_{mb}	Population of cassava plants co-infected by CMD and CBSD	Plants Ha^{-1}
N_v	Total population of vectors	Vectors Ha^{-1}
X	Population of susceptible vectors	Vectors Ha^{-1}
Y_b	Population of vectors infected with CBSD	Vectors Ha^{-1}
Y_m	Population of vectors infected with CMD	Vectors Ha^{-1}
Y_{mb}	Population of vectors co-infected with CMD and CBSD	Vectors Ha^{-1}

TABLE 2. Parameter description

Parameter	Description	Unit
r	Growth rate of cassava plants	Day^{-1}
β_m	Contact rate of S_c with Y_m	$\text{Ha Vector}^{-1} \text{Day}^{-1}$
β_b	Contact rate of S_c with Y_b	$\text{Ha Vector}^{-1} \text{Day}^{-1}$
β_{mb}	Contact rate of susceptible with co-infection, Y_m and Y_b	$\text{Ha Vector}^{-1} \text{Day}^{-1}$
α_m	Transmission rate from Y_m to I_b	$\text{Ha Vector}^{-1} \text{Day}^{-1}$
α_b	Transmission rate from Y_b to I_m	$\text{Ha Vector}^{-1} \text{Day}^{-1}$
μ_m	Disease induced death rate of I_m	Day^{-1}
μ_b	Disease induced death rate of I_b	Day^{-1}
μ_{mb}	Disease induced death rate of I_{mb}	Day^{-1}
Λ	Recruitment rate of vectors	$\text{Vector Ha}^{-1} \text{Day}^{-1}$
ρ_m	transmission rate from Y_m to I_b	$\text{Ha Plant}^{-1} \text{Day}^{-1}$
ρ_b	transmission rate from Y_b to I_m	$\text{Ha Plant}^{-1} \text{Day}^{-1}$
σ_m	transmission rate from I_m to X	$\text{Ha Plant}^{-1} \text{Day}^{-1}$
σ_b	transmission rate from I_b to X	$\text{Ha Plant}^{-1} \text{Day}^{-1}$
δ	natural death rate	Day^{-1}
η_1	chemical control (insecticides)	Dimensionless
η_2	Biological control	Dimensionless
K	carrying capacity of cassava plant	Plant Ha^{-1}
c	use of resistant varieties	Dimensionless
ζ	Mechanical control (rogue of plant)	Dimensionless

3. MODEL ANALYSIS

In this section, the model analysis is carried out to improve our understanding of the dynamics of the model.

3.1. Dimensionless Model. Since model (1) comprises of two distinct populations (plants and vectors) with different measuring units, it is advisable to non-dimensionalize the model. By

making the following transformations, $\tau = rt$ $s = \frac{S}{K}$, $i_m = \frac{I_m}{K}$, $i_b = \frac{I_b}{K}$, $i_{mb} = \frac{I_{mb}}{K}$, $x = \frac{X}{N_v}$, $\Lambda = \frac{\tilde{\Lambda}}{rN_v}$, $y_m = \frac{Y_m}{N_v}$, $y_b = \frac{Y_b}{N_v}$, $y_{mb} = \frac{Y_{mb}}{N_v}$, $\beta_m = \frac{\tilde{\beta}_m N_v}{r}$, $\beta_b = \frac{\tilde{\beta}_b N_v}{r}$, $\beta_{mb} = \frac{\tilde{\beta}_{mb} N_v}{r}$, $\alpha_m = \frac{\tilde{\alpha}_m N_v}{r}$, $\alpha_b = \frac{\tilde{\alpha}_b N_v}{r}$, $\mu_m = \frac{\tilde{\mu}_m}{r}$, $\zeta = \frac{\tilde{\zeta}}{r}$, $\mu_b = \frac{\tilde{\mu}_b}{r}$, $\zeta = \frac{\tilde{\zeta}}{r}$, $\mu_{mb} = \frac{\tilde{\mu}_{mb}}{r}$, $\zeta = \frac{\tilde{\zeta}}{r}$, $\sigma_m = \frac{\tilde{\sigma}_m K}{r}$, $\sigma_b = \frac{\tilde{\sigma}_b K}{r}$, $\sigma_{mb} = \frac{\tilde{\sigma}_{mb} K}{r}$, $\rho_m = \frac{\tilde{\rho}_m K}{r}$, $\rho_b = \frac{\tilde{\rho}_b K}{r}$, $\delta = \frac{\tilde{\delta}}{r}$, $\eta_1 = \frac{\tilde{\eta}_1}{r}$, $\eta_2 = \frac{\tilde{\eta}_2}{r}$ a dimensionless version of model (2) is obtained and is given by

$$\begin{aligned}
 \frac{ds}{d\tau} &= s(1-s) - (1-c)\beta_m y_m s - (1-c)\beta_b y_b s - (1-c)\beta_{mb} y_{mb} s, \\
 \frac{di_m}{d\tau} &= (1-c)\beta_m y_m s - (1-c)\alpha_b y_b i_m - \mu_m i_m - \zeta i_m, \\
 \frac{di_b}{d\tau} &= (1-c)\beta_b y_b s - (1-c)\alpha_m y_m i_b - \mu_b i_b - \zeta i_b, \\
 \frac{di_{mb}}{d\tau} &= (1-c)\beta_{mb} y_{mb} s + (1-c)\alpha_b y_b i_m + (1-c)\alpha_m y_m i_b - \mu_{mb} i_{mb} - \zeta i_{mb}, \\
 \frac{dx}{d\tau} &= \Lambda - \sigma_m x i_m - \sigma_b x i_b - \sigma_{mb} x i_{mb} - \delta x - \eta_1 x - \eta_2 x, \\
 \frac{dy_m}{d\tau} &= \sigma_m x i_m - \rho_m y_m i_b - \delta y_m - \eta_1 y_m - \eta_2 y_m, \\
 \frac{dy_b}{d\tau} &= \sigma_b x i_b - \rho_b y_b i_m - \delta y_b - \eta_1 y_b - \eta_2 y_b, \\
 \frac{dy_{mb}}{d\tau} &= \sigma_{mb} x i_{mb} + \rho_m y_m i_b + \rho_b y_b i_m - \delta y_{mb} - \eta_1 y_{mb} - \eta_2 y_{mb}.
 \end{aligned}
 \tag{2}$$

3.2. Positivity and Boundedness of Solution. To determine the feasible region of model (2) where the model makes sense biologically, we determine the positivity and boundedness of solution. The positivity and boundedness of solution are summarize in Theorems 1 and 2.

Theorem 1. *The solution $\Omega = \{(s, i_m, i_b, i_{mb}, x, y_m, y_b, y_{mb}) \in \mathfrak{R}_+^8 : n_c \leq 1, n_v \leq 1 : s(0) \geq 0, i_m(0) \geq 0, i_b(0) \geq 0, x(0) \geq 0, y_m(0) \geq 0, y_b(0) \geq 0, y_{mb}(0) \geq 0\}$ of model (2) will remain positive for all $t \geq 0$.*

Proof. Here we will discuss positivity of the solution with initial conditions: $s(0) \geq 0, i_m(0) \geq 0, i_b(0) \geq 0, x(0) \geq 0, y_m(0) \geq 0, y_b(0) \geq 0, y_{mb}(0) \geq 0$.

From the first equation of the model (2)

$$\begin{aligned}
 \frac{ds}{d\tau} &= s[(1-s) - (1-c)(\beta_m y_m + \beta_b + \beta_{mb} y_{mb})] \\
 &\leq s(1-s).
 \end{aligned}$$

So, solving the logistic inequality gives

$$s(\tau) \leq \frac{De^\tau}{1 + De^\tau},$$

where $D = \frac{s(0)}{1-s(0)} > 0$. Since $D > 0$, then as $\tau \rightarrow \infty$, $\frac{De^\tau}{1+De^\tau} \rightarrow 1$. This shows that $s(\tau) \geq 0$. By a similar reasoning, we can show that $i_m \geq 0$, $i_b \geq 0$, $x \geq 0$, $y_m \geq 0$, $y_b \geq 0$, $y_{mb} \geq 0$. This complete the proof. $\square$

Theorem 2. *All solutions of the model (2) are bounded in a proper subset*

$$\Omega = \{(s, i_m, i_b, i_{mb}, x, y_m, y_b, y_{mb}) \in \mathfrak{R}_+^8 : n_c \leq 1, n_v \leq 1 : s(0) \geq 0, i_m(0) \geq 0, i_b(0) \geq 0, x(0) \geq 0, y_m(0) \geq 0, y_b(0) \geq 0, y_{mb}(0) \geq 0\}.$$

Proof. To show the boundedness of the solution, we have to show the existence of a lower bound and upper bound for the solution. At initial condition, we obtained $s(0) \geq 0$, $i_m(0) \geq 0$, $i_b(0) \geq 0$, $x(0) \geq 0$, $y_m(0) \geq 0$, $y_b(0) \geq 0$, $y_{mb}(0) \geq 0$, establishing the existence of a lower bound.

For the upper bound of model (2), we consider the total population: $n = n_c + n_v = s + i_m + i_b + i_{mb} + x + y_m + y_b + y_{mb}$ and we have: $\frac{dn}{d\tau} = \frac{ds}{d\tau} + \frac{di_m}{d\tau} + \frac{di_b}{d\tau} + \frac{di_{mb}}{d\tau} + \frac{dx}{d\tau} + \frac{dy_m}{d\tau} + \frac{dy_b}{d\tau} + \frac{dy_{mb}}{d\tau}$. By adding this system of differential equations and simplification yields

$$\begin{aligned} \frac{dn_c}{d\tau} &= s(1 - n_c) - \mu_i s, \\ \frac{dn_c}{d\tau} &\leq (1 - n_c)s, \\ \frac{dn_c}{1 - n_c} &\leq sd\tau. \end{aligned}$$

Integrating both sides and applying the initial condition $n_c(0) = n_{co}$ gives

$$(3) \quad n_c \leq 1.$$

This shows that the cassava population has an upper bound.

Using a similar argument for the vector population gives

$$\begin{aligned} \frac{dn_v}{d\tau} &= \Lambda - (\delta + \eta_1 + \eta_2)n_v, \\ \frac{dn_v}{1 - n_c} &= \delta d\tau. \end{aligned}$$

Integrating both sides and applying the initial condition $n_v(0) = n_{vo}$, gives

$$(4) \quad n_v \leq \frac{\Lambda}{\delta + \eta + \eta}.$$

This shows that the vector population also has an upper bound. Hence, all solutions of the model (2) are bounded completeing the proof $\square$

From Theorems 1 and 2, it implies that the feasible solution set of the system equation of the model enters and remains in the region $\Omega = \{(s, i_m, i_b, i_{mb}, y_m, y_b, y_{mb}) \in \mathfrak{R}_+^8 : n_c \leq 1, n_v \leq \frac{\Lambda}{\delta + \eta + \eta}\}$.

3.3. Model with cassava mosaics disease only. Here, we consider a special case where only Cassava Mosaic Disease (CMD) is present in the system and is obtained by setting $i_b = i_{bm} = y_b = 0$ in model (2) to get

$$(5) \quad \begin{aligned} \frac{ds}{d\tau} &= s(1-s) - (1-c)\beta_m y_m s, \\ \frac{di_m}{d\tau} &= (1-c)\beta_m y_m s - \mu_m i_m - \zeta I_m, \\ \frac{dx}{d\tau} &= \Lambda - \sigma_m x i_m - \delta x - \eta_1 x - \eta_2 x, \\ \frac{dy_m}{d\tau} &= \sigma_m x i_m - \delta y_m - \eta_1 y_m - \eta_2 y_m. \end{aligned}$$

The dynamical system analysis of model (5) is presented in this section.

3.3.1. Disease-Free Equilibrium of Model (5). To determine the disease-free equilibrium (DFE) of model (5), we assume the absence of infection in both the cassava plant and vector populations. Thus, the infected compartments satisfy $i_m = y_m = 0$. Substituting these conditions into model (5) and simplifying the resulting system yields

$$(6) \quad E^0 = (s^0, i^0, x^0, y^0) = \left(1, 0, \frac{\Lambda}{\delta + \eta_1 + \eta_2}, 0\right).$$

3.3.2. Basic Reproduction Number of Model (5). The basic reproduction number of model (5) is the average number secondary infection generated by an infection in a population where everyone is susceptible. To calculate basic reproduction of CMD model 5, we used the next

generation matrix method to obtain [16, 17]

$$F = \begin{pmatrix} 0 & (1-c)\beta_m \\ \frac{\sigma_m \Lambda}{\delta + \eta_1 + \eta_2} & 0 \end{pmatrix}, \quad V^{-1} = \begin{pmatrix} \frac{1}{\mu_m + \zeta} & 0 \\ 0 & \frac{1}{\delta + \eta_1 + \eta_2} \end{pmatrix}.$$

The dominant eigenvalue of the next generation matrix (FV^{-1}) becomes the basic reproduction number and is given by

$$(7) \quad R_{0m} = \sqrt{\frac{(1-c)\beta_m \sigma_m \Lambda}{(\delta + \eta_1 + \eta_2)^2 (\mu_m + \zeta)}}.$$

3.3.3. Local stability of model 5 about DFE. In this section, we carried out the stability of the subsystem of the model 6 about DFE. The Jacobian of the sub-system at (E^0) is

$$(8) \quad J(E^0) = \begin{pmatrix} -1 & 0 & 0 & -(1-c)\beta_m \\ 0 & -(\mu_m + \zeta) & 0 & (1-c)\beta_m \\ 0 & -\frac{\sigma_m \Lambda}{\delta + \eta_1 + \eta_2} & -(\delta + \eta_1 + \eta_2) & 0 \\ 0 & \frac{\sigma_m \Lambda}{\delta + \eta_1 + \eta_2} & 0 & -(\delta + \eta_1 + \eta_2) \end{pmatrix}.$$

From the Jacobian matrix, two eigenvalues are: $\lambda_1 = -1 < 0$, $\lambda_2 = -(\delta + \eta_1 + \eta_2) < 0$.

The remaining eigenvalues are obtained from the sub matrix as follows:

$$(9) \quad \begin{vmatrix} -(\mu_m + \zeta) - \lambda & (1-c)\beta_m \\ \frac{\sigma_m \Lambda}{\delta + \eta_1 + \eta_2} & -(\delta + \eta_1 + \eta_2) - \lambda \end{vmatrix} = 0.$$

The characteristic equation of the sub matrix is:

$$(10) \quad \lambda^2 + ((\mu_m + \zeta)(\delta + \eta_1 + \eta_2))\lambda + (\mu_m + \zeta)(\delta + \eta_1 + \eta_2) - \frac{(1-c)\beta_m \sigma_m \Lambda}{\delta + \eta_1 + \eta_2} = 0.$$

For local asymptotic stability, all eigenvalues must have negative real parts. This holds if $(\mu_m + \zeta)(\delta + \eta_1 + \eta_2) > 0$ and this implies $\Rightarrow R_{0m} < 1$. Thus, the subsystem model 5 is locally asymptotically stable about the DFE provided $R_{0m} < 1$. The epidemiological implication of this results is that Cassava Mosaic Disease (CMD) will be eradicated if the initial infected populations are within the neighbourhood of the DFE and $R_{0m} < 1$. To show that disease eradication is independent of the initial infected population, it is crucial to establish the global stability of the model about the DFE.

3.3.4. Global stability of model 5 about DFE. The global stability of model 6 about DFE will be established using a global stability results which is stated in Lemma 1.

Lemma 1. *Following [16], the system can be written as:*

$$(11) \quad \begin{aligned} \frac{dZ_1}{d\tau} &= F(Z_1, Z_2) \\ \frac{dZ_2}{d\tau} &= G(Z_1, Z_2), \quad G(Z_1, 0) = 0 \end{aligned}$$

where $Z_1 \in \mathbb{R}^m$ and $Z_2 \in \mathbb{R}^n$, $Z_0 = (Z_1^*, 0)$ denotes the disease-free equilibrium of the system. Assume that

(H1) For $\frac{dZ_1}{d\tau} = F(Z_1, 0)$, Z_1^* is globally asymptotically stable;

(H2) $G(Z_1, Z_2) = AZ_2 - \hat{G}(Z_1, Z_2) \geq 0$ for $(Z_1, Z_2) \in \Omega$, where the Jacobian $A = \frac{\partial G}{\partial Z_2}(Z_1, 0)$ is an M-matrix (the off diagonal elements of A are non negative) and Ω is the region where the model makes biological sense. Then the disease-free equilibrium Z_0 is globally asymptotically stable provided that $R_0 < 1$ (Castillo Chavez et al, 2002).

Theorem 3. *The subsystem model 6 is globally asymptotically stable about the DFE provided $R_{0m} < 1$.*

Proof. [16] Using Lemma 1, we only need to show that conditions (H1) and (H2) hold when $R_{0m} < 1$. From model 5 $Z_1 = (s(\tau), x(\tau))$, $Z_2 = (i(\tau), y_m(\tau))$. The disease free model is given by

$$(12) \quad \frac{dZ_1}{d\tau} = F(Z_1, 0) = \begin{pmatrix} s(1-s) \\ \Lambda - (\delta + \eta_1 + \eta_2)x \end{pmatrix}.$$

Equation (12) is a logistic equation whose solution converges to $s = 1$. Similarly, second equation (12) is linear and has the solution $x(\tau) = x(0)e^{-(\delta + \eta_1 + \eta_2)\tau} + \frac{\Lambda}{\delta + \eta_1 + \eta_2} \left(1 - e^{-(\delta + \eta_1 + \eta_2)\tau}\right)$. Hence, $\lim_{\tau \rightarrow \infty} x(\tau) = \frac{\Lambda}{\delta + \eta_1 + \eta_2}$. Therefore, the equilibrium $(s^0, x^0) = \left(1, \frac{\Lambda}{\delta + \eta_1 + \eta_2}\right)$ is globally asymptotically stable. Thus, condition (H1) is satisfied. To show that condition (H2) holds, we consider

$$(13) \quad \frac{dZ_2}{d\tau} = G(Z_1, Z_2) = \begin{pmatrix} (1-c)\beta_m y_m s - (\mu_m + \zeta)i_m \\ \sigma_m x i_m - (\delta + \eta_1 + \eta_2)y_m \end{pmatrix}.$$

By computing the Jacobian of 14 about the DFE, we get

$$(14) \quad A = \begin{pmatrix} -(\mu_m + \zeta) & (1-c)\beta_m \\ \frac{\sigma_m \Lambda}{\delta + \eta_1 + \eta_2} & -(\delta + \eta_1 + \eta_2) \end{pmatrix}.$$

Obviously, A is an M -matrix with all its off diagonal elements non-negatives. From equation 13 and 14, we got this

$$(15) \quad \hat{G}(Z_1, Z_2) = \begin{pmatrix} (1-c)\beta_m y_m (1-s) \\ \sigma_m i_m \left(\frac{\Lambda}{\delta + \eta_1 + \eta_2} - x \right) \end{pmatrix}.$$

Clearly, $\hat{G}(Z_1, Z_2) \geq 0$, since $1 \geq s$ and $\frac{\Lambda}{\delta + \eta_1 + \eta_2} \geq x$. Hence (H2) holds completing the proof. $\square$

Epidemiologically, this result indicate that cassava mosaic disease could possibly be eradicated irrespective of the size of population of the infected cassava plants and white-flies at the beginning of the outbreak, provided the control measures are effective to keep R_{0m} below unity.

3.3.5. Endemic equilibrium (E^*). The endemic equilibrium (E^*) of CMD model is obtained by equating the system of equation to zero. The existence of (E^*) is given by $E^* = (s^*, i_m^*, x^*, y_m^*)$ where

$$(16) \quad s^* = 1 - (1-c)\beta_m y_m^*,$$

$$(17) \quad i_m^* = \frac{(1-c)\beta_m y_m^* s^*}{\mu_m + \zeta},$$

$$(18) \quad x^* = \frac{\Lambda}{\sigma_m i_m^* + (\delta + \eta_1 + \eta_2)},$$

$$(19) \quad y_m^* = \frac{\sigma_m x^* i_m^*}{\delta + \eta_1 + \eta_2}$$

3.3.6. Local stability of endemic equilibrium of CMD model. In this section, the local asymptotic stability of the endemic equilibrium point of the CMD model is investigated using the Jacobian matrix approach together with the Routh–Hurwitz stability criterion. Let the endemic equilibrium point of the system be denoted by $E^* = (s^*, i_m^*, x^*, y_m^*)$, where $s^* > 0$, $i_m^* > 0$, $x^* > 0$, $y_m^* > 0$.

The Jacobian matrix of the CMD model evaluated at the E^* is given by

$$J(E^*) = \begin{pmatrix} 1 - 2s^* - (1 - c)\beta_m y_m^* & 0 & 0 & -(1 - c)\beta_m s^* \\ (1 - c)\beta_m y_m^* & -(\mu_m + \zeta) & 0 & (1 - c)\beta_m s^* \\ 0 & -\sigma_m x^* & -\sigma_m i_m^* - (\delta + \eta_1 + \eta_2) & 0 \\ 0 & \sigma_m x^* & \sigma_m i_m^* & -(\delta + \eta_1 + \eta_2) \end{pmatrix}.$$

The characteristic equation is obtained from $|J(E^*) - \lambda I| = 0$.

Thus,

$$\begin{vmatrix} 1 - 2s^* - (1 - c)\beta_m y_m^* - \lambda & 0 & 0 & -(1 - c)\beta_m s^* \\ (1 - c)\beta_m y_m^* & -(\mu_m + \zeta) - \lambda & 0 & (1 - c)\beta_m s^* \\ 0 & -\sigma_m x^* & -\sigma_m i_m^* - (\delta + \eta_1 + \eta_2) - \lambda & 0 \\ 0 & \sigma_m x^* & \sigma_m i_m^* & -(\delta + \eta_1 + \eta_2) - \lambda \end{vmatrix} = 0.$$

After simplification, the characteristic polynomial becomes

$$\lambda^4 + a_1 \lambda^3 + a_2 \lambda^2 + a_3 \lambda + a_4 = 0,$$

where a_1, a_2, a_3 , and a_4 are functions of the model parameters and endemic equilibrium components. Applying the Routh–Hurwitz stability criterion, the endemic equilibrium E^* is locally asymptotically stable provided that

$$a_1 > 0, \quad a_2 > 0, \quad a_3 > 0, \quad a_4 > 0,$$

and

$$a_1 a_2 a_3 > a_3^2 + a_1^2 a_4.$$

Hence, all eigenvalues of the Jacobian matrix have negative real parts provided $R_{0m} > 1$. The local asymptotic stability of the endemic equilibrium implies that cassava mosaic disease persists within the cassava and vector populations whenever the system is slightly perturbed around the endemic state. Consequently, the disease remains endemic in the population and returns to the equilibrium level after small disturbances, indicating continuous transmission dynamics of CMD within the host–vector system.

3.4. Cassava Brown Streak Disease (CBSD) Sub-model. Here, we consider a special case where only CBSD is present in the system and obtained by $i_m = i_{mb} = y_m = 0$.

$$\begin{aligned}
(20) \quad \frac{ds}{d\tau} &= s(1-s) - (1-c)\beta_b y_b s \\
\frac{di_b}{d\tau} &= (1-c)\beta_b y_b s - \mu_b i_b - \zeta i_b, \\
\frac{dx}{d\tau} &= \Lambda - \sigma_b x i_b - \delta x - \eta_1 x - \eta_2 x \\
\frac{dy}{d\tau} &= \sigma_b x i_b - \delta y_b - \eta_1 y_b - \eta_2 y_b
\end{aligned}$$

The dynamics system analysis of the model of equation 20 is presented in this section.

3.4.1. Disease-Free Equilibrium (DFE) of CBSD Sub-model. The DFE of CBSD sub-model (20) corresponds to the state where no CBSD exists in the system. Thus, we set the infected compartments to zero that is $i = y = 0$ and substituting into the system (20) and simplifying gives

$$(21) \quad E_0 = (s^*, i^*, x^*, y^*) = \left(1, 0, \frac{\Lambda}{\delta + \eta_1 + \eta_2}, 0\right).$$

3.4.2. Basic Reproduction Number of (CBSD) Sub-model. The Basic Reproduction Number of CBSD sub-model 20 using the next generation matrix gives

$$F = \begin{pmatrix} 0 & (1-c)\beta_b \\ \frac{\sigma_b \Lambda}{\delta + \eta_1 + \eta_2} & 0 \end{pmatrix} \quad V^{-1} = \begin{pmatrix} \frac{1}{\mu_b + \zeta} & 0 \\ 0 & \frac{1}{\delta + \eta_1 + \eta_2} \end{pmatrix}.$$

The spectral radius of the next generation matrix FV^{-1} which is the basic reproduction number is

$$(22) \quad R_{0b} = \sqrt{\frac{(1-c)\beta_b \sigma_b \Lambda}{(\delta + \eta_1 + \eta_2)^2 (\mu_b + \zeta)}}.$$

3.4.3. Local Stability about DFE of CBSD Sub-model. To analyse the local stability of CBSD Sub-model about the DFE, we compute the Jacobian matrix of system 20 and Substituting $(E_0 = (1, 0, \frac{\Lambda}{\delta + \eta_1 + \eta_2}, 0))$ gives

$$J(E_0) = \begin{pmatrix} -1 & 0 & 0 & -(1-c)\beta_b \\ 0 & -(\mu_b + \zeta) & 0 & (1-c)\beta_b \\ 0 & -\frac{\sigma_b \Lambda}{\delta + \eta_1 + \eta_2} & -(\delta + \eta_1 + \eta_2) & 0 \\ 0 & \frac{\sigma_b \Lambda}{\delta + \eta_1 + \eta_2} & 0 & -(\delta + \eta_1 + \eta_2) \end{pmatrix}.$$

Two of the four eigenvalues are immediately obtained as

$$\lambda_1 = -1, \quad \lambda_2 = -(\delta + \eta_1 + \eta_2).$$

The remaining two eigenvalues are obtained from the infected subsystem

$$(23) \quad \begin{pmatrix} -(\mu_b + \zeta) & (1-c)\beta_b \\ \frac{\sigma_b \Lambda}{\delta + \eta_1 + \eta_2} & -(\delta + \eta_1 + \eta_2) \end{pmatrix}.$$

The characteristic equation is

$$\lambda^2 + [(\mu_b + \zeta)(\delta + \eta_1 + \eta_2)]\lambda + (\mu_b + \zeta)(\delta + \eta + \eta) - \frac{(1-c)\beta_b \sigma_b \Lambda}{\delta + \eta_1 + \eta_2} = 0.$$

$$\implies \lambda^2 + a_1 \lambda + a_2$$

For local asymptotic stability, the Routh–Hurwitz criteria require $a_1 > 0$ and $a_2 > 0$.

$$(24) \quad (\mu_b + \zeta)(\delta + \eta + \eta) - \frac{(1-c)\beta_b \sigma_b \Lambda}{\delta + \eta_1 + \eta_2} > 0.$$

Using the definition of the basic reproduction number

$$R_{0b}^2 = \frac{(1-c)\beta_b \sigma_b \Lambda}{(\delta + \eta_1 + \eta_2)^2(\mu_m + \zeta)},$$

we obtain $a_2 = (\mu_b + \zeta)(\delta + \eta_1 + \eta_2)(1 - R_0^2)$. The above condition becomes $R_0 < 1$. Thus, the CBSD Sub-model is locally asymptotically stable about the DFE if $R_{0b} < 1$. The epidemiological implication of this results is that CBSD will be eradicated if the initial infected populations are within the neighbourhood of the DFE and $R_{0b} < 1$. To show that disease eradication is independent of the initial infected population, it is crucial the establish the global stability of the model about the DFE.

3.4.4. Global Stability of CBSD Sub-model about DFE.

Theorem 4. *The CBSD Sub-model (20) is globally stable about DFE if $R_{0b} < 1$.*

Proof. [16] Using Lemma 1, we only need to show that conditions (H_1) and (H_2) hold when $R_0 < 1$. From model Equation 6 $Z_1 = (s(\tau), x(\tau))$, $Z_2 = (i(\tau), t(\tau))$. The disease free model

$$(25) \quad \frac{dZ_1}{d\tau} = F(Z_1, 0) = \begin{pmatrix} s(1-s) \\ \Lambda - (\delta + \eta + \eta)x \end{pmatrix}$$

is a set of linear ordinary differential equations and its exact solution can be calculated as $s(\tau) = \frac{Pe^\tau}{1+Pe^\tau}$, $x(\tau) = 1 - a_5 e^{-\delta\tau}$, where $P = \frac{s(0)}{1-s(0)}$, $a_5 = 1 - x_0$. Clearly, $s(\tau) \rightarrow s_0 = 1$, $x(\tau) \rightarrow x_0 = 1$ as $\tau \rightarrow \infty$. Thus, Z_1^* is globally asymptotically stable, confirming that condition (H1) holds. To show that condition (H2) holds, we consider

$$(26) \quad \frac{dZ_2}{d\tau} = G(Z_1, Z_2) = \begin{pmatrix} (1-c)\beta_b s y_b - (\mu_b + \zeta)i_m \\ \sigma_b x i - (\delta + \eta_1 + \eta_2)y_b \end{pmatrix}$$

By computing the Jacobian of 20 about the DFE, we got

$$(27) \quad A = \begin{pmatrix} -(\mu_b + \zeta) & (1-c)\beta_b \\ \frac{\sigma_b \Lambda}{\delta + \eta_1 + \eta_2} & -(\delta + \eta_1 + \eta_2) \end{pmatrix}.$$

Obviously, A is an M-matrix with all its off diagonal elements non-negatives. From equation 27 and 28, we got this

$$(28) \quad \hat{G}(Z_1, Z_2) = \begin{pmatrix} (1-c)\beta_b y_b (1-s) \\ \sigma_b i_b \left(\frac{\Lambda}{\delta + \eta_1 + \eta_2} - x \right) \end{pmatrix}.$$

Obviously, $\hat{G}(Z_1, Z_2) \geq 0$, since $1 \geq s$ and $\frac{\Lambda}{\delta + \eta_1 + \eta_2} \geq x$. Hence (H2) holds completing the proof $\square$

Epidemiologically, this result indicate that cassava Brown Streak disease(CMBD) could possibly be eradicated irrespective of the size of population of the infected cassava plants and white flies at the beginning of the outbreak, provided the control measures are effective to keep $R_{0b} < 1$.

3.4.5. Endemic equilibrium (E^*). The endemic equilibrium (E^*) of CBSD model by equating the system of equation to zero. The existence of the endemic equilibrium is given by $E^* = (s^*, i_b^*, x^*, y_b^*)$ where

$$(29) \quad s^* = 1 - (1-c)\beta_b y_b^*,$$

$$i_b^* = \frac{(1-c)\beta_b y_b^* s^*}{\mu_b + \zeta},$$

$$(30) \quad x^* = \frac{\Lambda}{\sigma_b i_b^* + (\delta + \eta_1 + \eta_2)}$$

$$(30) \quad y_b^* = \frac{\sigma_b x^* i_b^*}{\delta + \eta_1 + \eta_2}.$$

3.4.6. Local stability of CBSD Sub-model about E^* . In this section, the local asymptotic stability of the endemic equilibrium point of the CBSD model is investigated using the Jacobian matrix approach together with the Routh–Hurwitz stability criterion. Let the endemic equilibrium point of the system be denoted by $E^* = (s^*, i_b^*, x^*, y_b^*)$, where $s^* > 0$, $i_b^* > 0$, $x^* > 0$, $y_b^* > 0$. The Jacobian matrix of the CBSD model evaluated at the endemic equilibrium E^* is given by

$$J(E^*) = \begin{pmatrix} 1 - 2s^* - (1 - c)\beta_b y_b^* & 0 & 0 & -(1 - c)\beta_b s^* \\ (1 - c)\beta_b y_b^* & -(\mu_b + \zeta) & 0 & (1 - c)\beta_b s^* \\ 0 & -\sigma_b x^* & -\sigma_b i_b^* - (\delta + \eta_1 + \eta_2) & 0 \\ 0 & \sigma_b x^* & \sigma_b i_b^* & -(\delta + \eta_1 + \eta_2) \end{pmatrix}.$$

The characteristic equation is obtained from $|J(E^*) - \lambda I| = 0$.

Thus,

$$\begin{vmatrix} 1 - 2s^* - (1 - c)\beta_b y_b^* - \lambda & 0 & 0 & -(1 - c)\beta_b s^* \\ (1 - c)\beta_b y_b^* & -(\mu_b + \zeta) - \lambda & 0 & (1 - c)\beta_b s^* \\ 0 & -\sigma_b x^* & -\sigma_b i_b^* - (\delta + \eta_1 + \eta_2) - \lambda & 0 \\ 0 & \sigma_b x^* & \sigma_b i_b^* & -(\delta + \eta_1 + \eta_2) - \lambda \end{vmatrix} = 0.$$

After simplification, the characteristic polynomial becomes

$$\lambda^4 + a_1 \lambda^3 + a_2 \lambda^2 + a_3 \lambda + a_4 = 0,$$

where a_1, a_2, a_3 , and a_4 are functions of the model parameters and endemic equilibrium components.

Applying the Routh–Hurwitz stability criterion, the endemic equilibrium E^* is locally asymptotically stable provided that

$$a_1 > 0, \quad a_2 > 0, \quad a_3 > 0, \quad a_4 > 0,$$

and

$$a_1 a_2 a_3 > a_3^2 + a_1^2 a_4.$$

Hence, all eigenvalues of the Jacobian matrix have negative real parts which implies that the model is stable about E^* if $R_{0b} > 1$. The local asymptotic stability of the endemic equilibrium implies that cassava brown streak disease persists within the cassava and vector populations whenever the system is slightly perturbed around the endemic state. Consequently, the disease remains endemic in the population and returns to the equilibrium level after small disturbances, indicating continuous transmission dynamics of CBSD within the host–vector system when $R_{0b} > 1$.

3.5. Analysis of CMD-CBSD co-infection model. Here we present the dynamical system analysis of the CMD-CBSD co-infection model (2).

3.5.1. Disease -Free Equilibrium (DFE) for Co-infection. (E^0) In order to calculate disease free equilibrium point, at which the epidemic is eradicated from the population, we set $i_m = i_b = i_{mb} = x = y_m = y_b = y_{mb} = 0$, in the system of differential equation 2. Simplifying the model at the disease free equilibrium, gives

$$(31) \quad E^0 = (s^0, i_m^0, i_b^0, x^0, y_m^0, y_b^0, y_{mb}^0) = \left(1, 0, 0, 0, \frac{\Lambda}{\delta + \eta_1 + \eta_2}, 0, 0, 0\right).$$

3.5.2. Basic Reproduction Number, R_0 . The Basic Reproduction number R_0 can be understand as a measure of the potential spread of the disease. It is the average number secondary infection generated or produced by a typical case of an infection in a population where everyone is susceptible . To calculate basic reproduction of our CMD-CBSD Co-infection model, we used the next generation matrix method [17]to obtain

$$(32) \quad FV^{-1} = \begin{pmatrix} 0 & 0 & 0 & \frac{(1-c)\beta_m}{\delta + \eta_1 + \eta_2} & 0 & 0 \\ 0 & 0 & 0 & 0 & \frac{(1-c)\beta_b}{\delta + \eta_1 + \eta_2} & 0 \\ 0 & 0 & 0 & 0 & 0 & \frac{(1-c)\beta_{mb}}{\delta + \eta_1 + \eta_2} \\ \frac{\sigma_m x^*}{\mu_m + \zeta} & 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{\sigma_b x^*}{\mu_b + \zeta} & 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{\sigma_{mb} x^*}{\mu_{mb} + \zeta} & 0 & 0 & 0 \end{pmatrix},$$

where, $x^* = \frac{\Lambda}{\delta + \eta_1 + \eta_2}$,

$$F = \begin{pmatrix} 0 & 0 & 0 & (1-c)\beta_m & 0 & 0 \\ 0 & 0 & 0 & 0 & (1-c)\beta_b & 0 \\ 0 & 0 & 0 & 0 & 0 & (1-c)\beta_{mb} \\ \sigma_m x^* & 0 & 0 & 0 & 0 & 0 \\ 0 & \sigma_b x^* & 0 & 0 & 0 & 0 \\ 0 & 0 & \sigma_{mb} x^* & 0 & 0 & 0 \end{pmatrix},$$

$$V^{-1} = \begin{pmatrix} \frac{1}{\mu_m + \zeta} & 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{1}{\mu_b + \zeta} & 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{1}{\mu_{mb} + \zeta} & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{1}{\delta + \eta_1 + \eta_2} & 0 & 0 \\ 0 & 0 & 0 & 0 & \frac{1}{\delta + \eta_1 + \eta_2} & 0 \\ 0 & 0 & 0 & 0 & 0 & \frac{1}{\delta + \eta_1 + \eta_2} \end{pmatrix}.$$

The overall basic reproduction number is the dominant positive eigenvalue of the next generation matrix (FV^{-1}) , and is calculated as

$$(33) \quad R_0 = \max \{R_{0m}, R_{0b}, R_{0mb}\},$$

where $R_{0m} = \sqrt{\frac{(1-c)\beta_m \sigma_m x^*}{(\mu_m + \zeta)(\delta + \eta_1 + \eta_2)}}$, $R_{0b} = \sqrt{\frac{(1-c)\beta_b \sigma_b x^*}{(\mu_b + \zeta)(\delta + \eta_1 + \eta_2)}}$, $R_{0mb} = \sqrt{\frac{(1-c)\beta_{mb} \sigma_{mb} x^*}{(\mu_{mb} + \zeta)(\delta + \eta_1 + \eta_2)}}$. Note R_{0m} , R_{0b} and R_{0mb} are basic reproduction associated with CMD, CBS, and CMD-CBS co-infection respectively.

3.5.3. Stability analysis of CMD-CBS co-infection model about DFE.

Theorem 5. *The CMD-CBS co-infection model is not globally stable about the DFE if $R_0 < 1$.*

Proof. [16] Using Lemma 1, we only need to show that at least one of the conditions (H_1) or (H_2) fails to hold when $R_0 < 1$. From model Equation 2 $Z_1 = (s(\tau), x(\tau))$, $Z_2 = (i(\tau), t(\tau))$. The disease free model

$$(34) \quad \frac{dZ_1}{d\tau} = F(Z_1, 0) = \begin{pmatrix} s(1-s) \\ \Lambda - (\delta + \eta + \eta)x \end{pmatrix}$$

is a set of linear ordinary differential equations and its exact solution can be calculated as $s(\tau) = \frac{Pe^\tau}{1+Pe^\tau}$ $x(\tau) = 1 - a_5 e^{-\delta\tau}$, where $P = \frac{s(0)}{1-s(0)} a_5 = 1 - x_0$. Clearly, $s(\tau) \rightarrow s_0 = 1$, $x(\tau) \rightarrow x_0 = 1$ as $\tau \rightarrow \infty$. Thus, Z_1^* is globally asymptotically stable, confirming that condition (H1) holds. To show that condition (H2) fails to hold, we consider

$$\frac{dZ_2}{d\tau} = G(Z_1, Z_2) = \begin{pmatrix} (1-c)\beta_m y_m s - (1-c)\alpha_b y_b i_m - \mu_m i_m - \zeta i_m \\ (1-c)\beta_b y_b s - (1-c)\alpha_m y_m i_b - \mu_b i_b - \zeta i_b \\ (1-c)\beta_{mb} y_{mb} s + (1-c)\alpha_b y_b i_m + (1-c)\alpha_m y_m i_b - \mu_{mb} i_{mb} - \zeta i_{mb} \\ \sigma_m x i_m - \rho_m y_m i_b - \delta y_m - \eta_1 y_m - \eta_2 y_m \\ \sigma_b x i_b - \rho_b y_b i_m - \delta y_b - \eta_1 y_b - \eta_2 y_b \\ \sigma_{mb} x i_{mb} + \rho_m y_m i_b + \rho_b y_b i_m - \delta y_{mb} - \eta_1 y_{mb} - \eta_2 y_{mb} \end{pmatrix}$$

By computing the Jacobian of 2 about the DFE, we got

$$A(35) = \begin{pmatrix} -(\mu_m + \zeta) & 0 & 0 & (1-c)\beta_m & 0 & 0 \\ 0 & -(\mu_b + \zeta) & 0 & 0 & (1-c)\beta_b & 0 \\ 0 & 0 & -(\mu_{mb} + \zeta) & 0 & 0 & (1-c)\beta_{mb} \\ \sigma_m x^* & 0 & 0 & -(\delta + \eta_1 + \eta_2) & 0 & 0 \\ 0 & \sigma_b x^* & 0 & 0 & -(\delta + \eta_1 + \eta_2) & 0 \\ 0 & 0 & \sigma_{mb} x^* & 0 & 0 & -(\delta + \eta_1 + \eta_2) \end{pmatrix}$$

Obviously, A is an M-matrix with all its off diagonal elements non-negatives. From equation 35 and 35, we got this

$$(36) \quad \hat{G}(Z_1, Z_2) = \begin{pmatrix} (1-c)\alpha_b y_b i_m \\ (1-c)\alpha_m y_m i_b \\ -(1-c)\alpha_b y_b i_m - (1-c)\alpha_m y_m i_b \\ \rho_m y_m i_b \\ \rho_b y_b i_m \\ -\rho_m y_m i_b - \rho_b y_b i_m \end{pmatrix}.$$

Obviously, $\hat{G}(Z_1, Z_2) \geq 0$ fail to hold since $\rho_m y_m i_b - \rho_b y_b i_m < 0$ and

$-(1-c)\alpha_b y_b i_m - (1-c)\alpha_m y_m i_b < 0$. Hence (H2) fail to hold, completing the proof. $\square$

Epidemiologically, this result indicate that CMD-CMBD co-infection could be difficult to be eradicated irrespective of the size of population of the infected cassava plants and white flies at

the beginning of the outbreak, even when the control measures are effective to keep R_0 below unity. This illustrates the severity of co-infection, thus more effective intervention is required to eradicate the diseases in a co-infected scenario.

4. NUMERICAL SIMULATION

In this section, numerical simulations of the proposed CMD–CBSD co-infection model are performed to complement and validate the analytical results obtained in the preceding sections. The simulations are used to illustrate the dynamics of disease transmission, examine the stability behaviour of the model about the various equilibria, and assess the influence of key epidemiological parameters on the spread of infection. Specifically, the effectiveness of various intervention strategies, including the use of resistant cassava varieties, rouging of infected plants, and vector control measures are investigated numerically. The simulations provide valuable insights into the long-term behaviour of the system and the potential impact of integrated disease management strategies on reducing the burden of CMD–CBSD co-infection in cassava production systems. The parameter values used for the model simulations are given in Tables 3.

Table 3: Parameters values used in the simulation

Parameters	Value	References
K	$[0.1 * 1,000, 0.1 * 20,000]$	[14, 9, 15]
r	$[0.025, 0.1]$	[18]
β_m	$0.002 - 0.032$	[14, 9, 15]
β_b	$0.002 - 0.032$	[14, 9, 15]
β_{mb}	$0.2 - 0.8]$	Assumed
μ_m	$0.002 - 0.004$	[14, 9]
μ_b	$0.002 - 0.004$	[14, 9]
μ_{mb}	$0.01 - 0.06$	Estimated
ϕ	0.50	Estimated
Λ	$0.1 - 0.5$	[11]
α_m	$0.002 - 0.032$	[14, 9]
α_b	$0.002 - 0.032$	[14, 9]

Parameters	Value	References
σ_m	0.002 - 0.032	[14, 9, 15]
σ_b	0.002 - 0.032	[14, 9, 15]
δ	0.06 - 0.18	[14, 9, 15]
ρ_b	0.002 - 0.004	Estimated
μ_{mb}	0.01 - 0.06	Estimated
ζ	0.01 - 1.06	Estimated
η_1	0 - 0.3	Estimated
η_2	0 - 0.3	Estimated
c	0 - 1	Estimated

4.1. Model fitting and parameter estimation. For the numerical simulation, we consider real data on cassava yield in Nigeria from 2000 to 2018 extracted from World bank data base and is presented in Figure 2. By fitting model (2) to these data, the important parameters such as the control parameters are estimated using the least square method while the remaining parameters are taken from the literature as presented in Table 3.

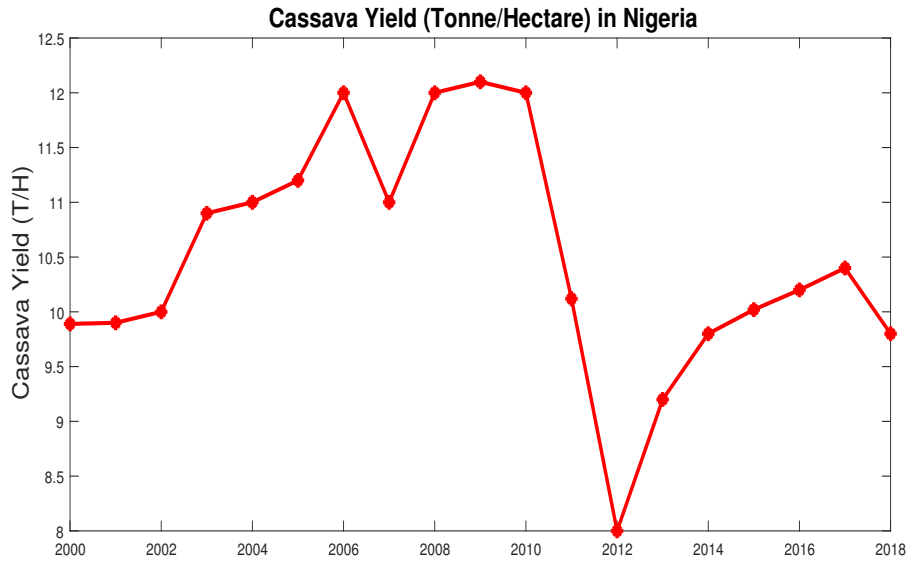


FIGURE 2. Plot illustrating cassava yield in Nigeria (Tonne/Ha) from 2000 to 2018 [19, 20].

Figure 2 presents the temporal trend of cassava yield in Nigeria from 2000 to 2018, expressed in tonnes per hectare (T/Ha)[20, 21]. This reveals that cassava yield experienced moderate fluctuations throughout the study period, with production levels ranging approximately between 8 and 12.5 T/Ha. The figure further demonstrates a gradual improvement in cassava yield over time, despite the occurrence of periodic fluctuations. In the early years of the study period (2000–2003), cassava yield remained relatively low, with values below 11.0 T/Ha. However, from 2004 onwards, a progressive increase in yield was observed, with production exceeding 11.0 T/Ha in the later years. This upward trend may be attributed to the adoption of improved cassava varieties, enhanced agronomic practices, increased awareness of disease management strategies, and government-supported agricultural interventions aimed at improving cassava productivity in Nigeria.

The back and forth movement observed in the yield trend may be linked to several biotic and abiotic factors affecting cassava production. These include the prevalence of Cassava Mosaic Disease (CMD), Cassava Brown Streak Disease (CBSD), whitefly vector infestation, climatic variability, soil fertility constraints, and inconsistencies in farm management practices. Such factors play significant roles in reducing cassava productivity and contributing to variations in annual yield performance.

Although the results indicate a noticeable improvement in cassava production during the study period, the yield values remained below the optimum attainable yield of 20 T/Ha. This suggests that cassava diseases and other environmental limitations continue to pose major challenges to cassava production in Nigeria.

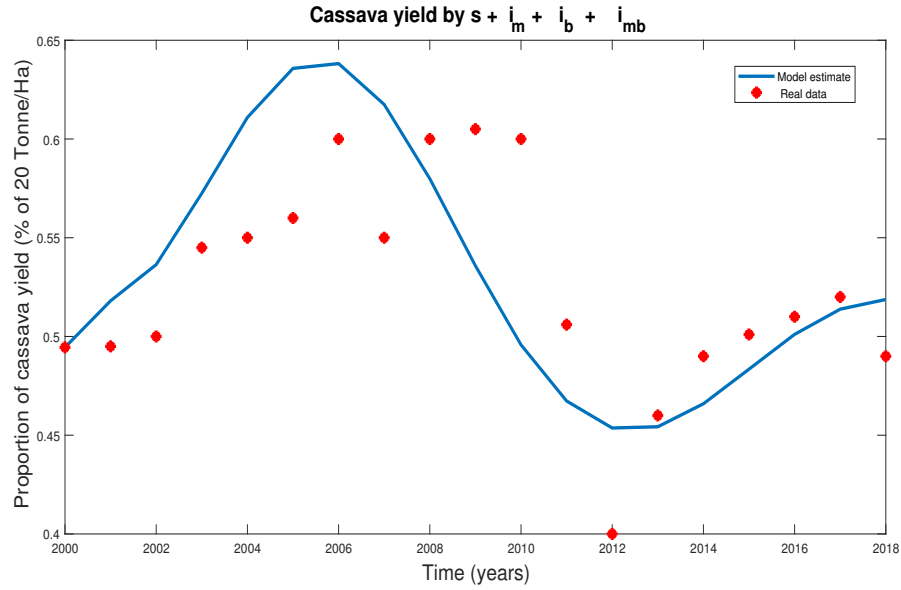


FIGURE 3. Model fit of the proportion of cassava yield in Nigeria (% of 20 Tonne/Ha) from 2000 to 2018, where bold lines represent the model fit and stars mark the actual data.

Figure 3 presents the model fitting of cassava yield in Nigeria from 2000 to 2018, where the cassava yield is expressed as a proportion of the standard yield of 20 Tonne/Ha. The figure compares the simulated model output with the observed real data obtained for cassava production in Nigeria [World Bank, 2024][20]. The result shows that the model estimates closely agree with the observed data throughout the study period, indicating that the developed model adequately captures the dynamics of cassava yield under the influence of cassava diseases and vector transmission. Although slight fluctuations are observed in some years, the overall trend of the simulated data follows the same pattern as the real data, demonstrating the reliability and predictive capability of the model. Furthermore, the cassava yield remained below the maximum attainable yield of 20 Tonne/Ha during the study period, suggesting that cassava diseases and other environmental factors significantly affected cassava productivity in Nigeria. The good agreement between the model predictions and the observed data validates the applicability of the model for numerical simulations, forecasting, and evaluation of disease control strategies aimed at improving cassava production.

4.2. Model prediction. The model prediction of cassava yield in Nigeria (% of 20 Tonne/Ha) over a long period of time is presented using the estimated parameter values. This model prediction reveals the future dynamics of the disease which is crucial for proper management and disease control. The outcome of the model prediction is presented in Figures 4 and 5.

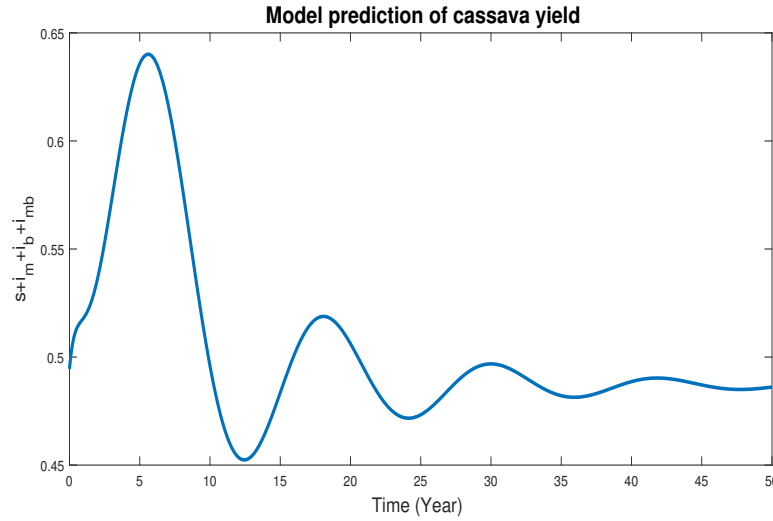


FIGURE 4. Plot showing a possible long-term dynamics of the proportion of cassava yield in Nigeria (% of 20 Tonne/Ha).

Figure 4 presents the model prediction of cassava yield over time under the combined effects of susceptible cassava plants, plants infected with Cassava Mosaic Disease(CMD), plants infected with Cassava Brown Streak Disease(CBSD), and co-infected cassava plants. The graph illustrates the temporal variation in cassava yield within the period considered in the simulation study. The result indicates that cassava yield initially increases gradually with time before exhibiting moderate fluctuations as the disease dynamics evolve. The yield values range approximately between 0.45 and 0.65, representing the proportion of attainable cassava yield under disease pressure. The observed fluctuations suggest that the interaction between healthy cassava plants, infected plants, and vector transmission significantly influences cassava productivity. Furthermore, the decline observed in some portions of the graph may be attributed to increased prevalence of CMD, CBSD, and co-infection effects, which reduce the productivity of cassava plants and consequently lower the overall yield. The presence of infected and co-infected cassava compartments contributes to disease persistence within the cassava population, thereby affecting the long-term stability of cassava production.

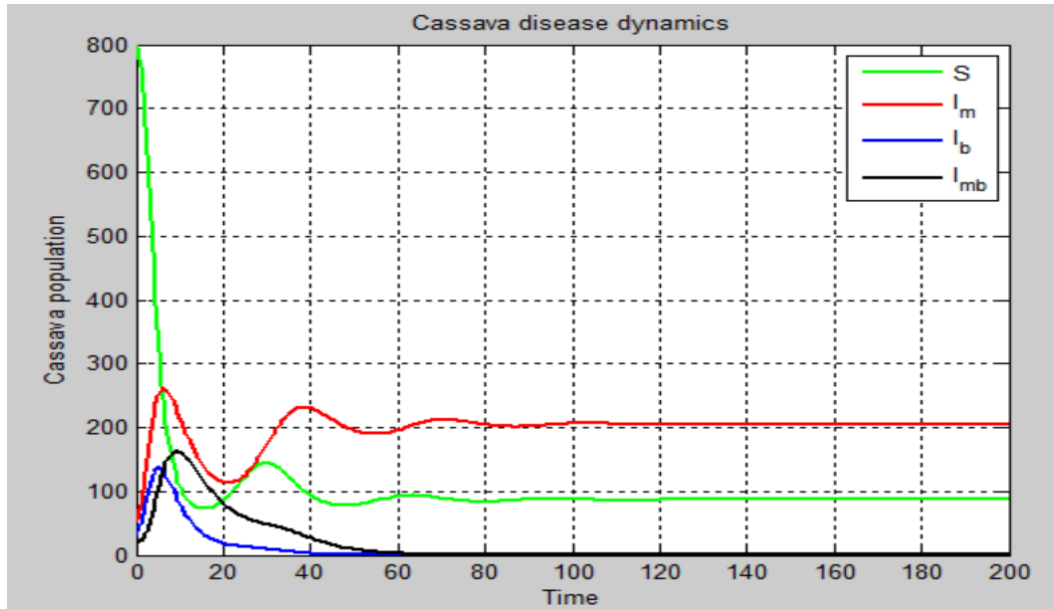


FIGURE 5. Plot showing a possible long-term dynamics of the proportion of cassava yield in Nigeria (% of 20 Tonne/Ha) for various variables.

Figure 5 presents the numerical simulation results of the cassava disease model, showing the temporal dynamics of healthy cassava plants, cassava plants infected with Cassava Mosaic Disease (CMD), cassava plants infected with Cassava Brown Streak Disease (CBSD), and cassava plants co-infected with both CMD and CBSD over time (0-50 years). The first graph illustrates the dynamics of healthy cassava plants. It is observed that the proportion of healthy cassava plants initially increases gradually before approaching a relatively stable state over time. This behaviour suggests that despite the presence of disease transmission within the cassava population, a considerable proportion of cassava plants remain susceptible and productive throughout the simulation period.

The second graph represents cassava plants infected with CMD. The result shows that the proportion of CMD-infected cassava plants remains relatively low throughout the simulation period, although slight fluctuations are observed. This indicates that CMD persists within the cassava population but does not completely dominate the system. The relatively controlled level of infection may be attributed to the effects of control measures, resistant cassava varieties, or reduced transmission efficiency.

Similarly, the third graph describes the dynamics of cassava plants infected with CBSD. The proportion of CBSD-infected plants also remains low during the simulation period, indicating that the disease persists at an endemic level within the cassava population. The behaviour of the CBSD compartment suggests that although the disease continues to spread through vector transmission, its prevalence remains limited under the assumed model conditions.

The fourth graph presents the dynamics of cassava plants co-infected with both CMD and CBSD. The result reveals that the proportion of co-infected cassava plants gradually increases with time before approaching a steady state. Compared to the single-infection compartments, the co-infected class exhibits relatively higher values, indicating that simultaneous infection by CMD and CBSD may contribute significantly to cassava yield reduction and disease burden within the cassava population. This behaviour highlights the epidemiological importance of co-infection dynamics in cassava disease transmission. Overall, the numerical simulation results demonstrate that CMD, CBSD, and co-infection can coexist persistently within the cassava population over time.

4.3. Impact of control measures. In this section, the impact of various control measures on the dynamics of model (2) is explored using the estimated parameter values.

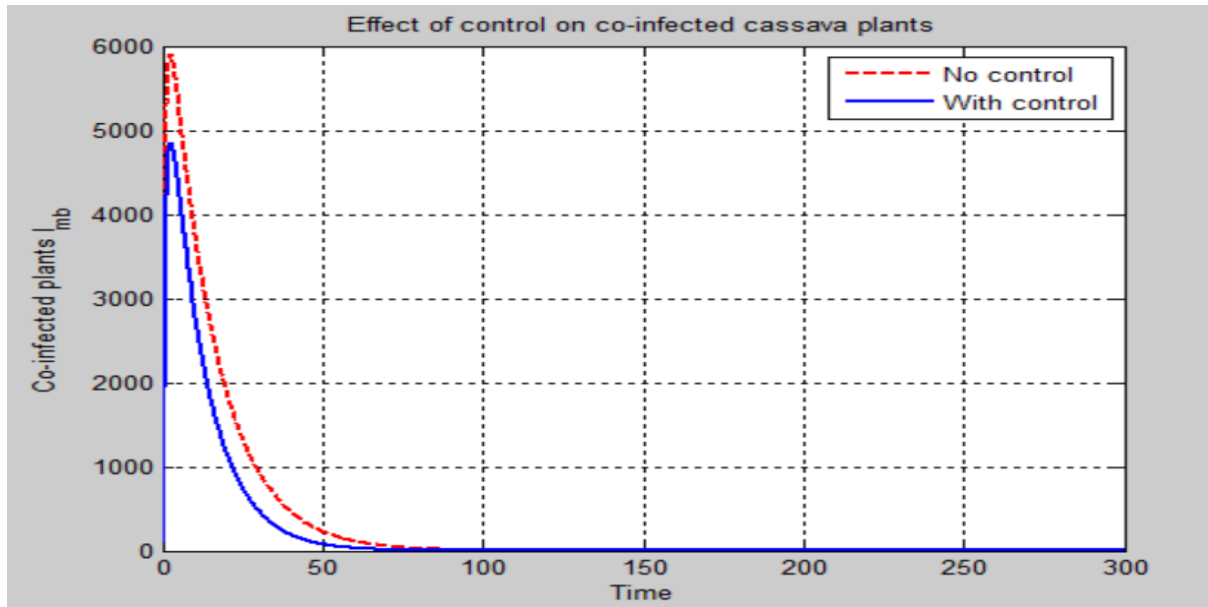


FIGURE 6. Plot showing the impact of use of resistant varieties (c) on cassava model dynamics.

Figure 6 presents the simulation results showing the influence of different levels of control effort c on the transmission dynamics of model (2). Specifically, the first sub-plot illustrates the behaviour of the healthy cassava population (s). It is observed that the proportion of healthy cassava plants increases as the control parameter increases. When no control strategy is applied ($c = 0$), the healthy cassava population remains comparatively low due to persistent disease spread. However, as the level of control increases from $c = 0.25$ to $c = 1.0$, the number of healthy cassava plants rises steadily and approaches its maximum level. This indicates that effective disease intervention strategies enhance the survival and sustainability of healthy cassava plants.

The second sub-plot describes the dynamics of cassava plants infected with CMD (i_m). The results show that the CMD-infected population is highest in the absence of control measures. As the control effort increases, the infected population decreases progressively. At higher control levels, especially when $c = 1.0$, the CMD infection is drastically reduced, tending toward eradication. This suggests that the implemented control strategy is highly effective in suppressing the transmission of CMD.

The third sub-plot represents cassava plants infected with CBSD (i_b). A similar trend is observed, where increasing the control effort significantly reduces the prevalence of CBSD infection. Without control, the infected population persists at a relatively high level, whereas stronger intervention measures lead to a continuous decline in the number of infected plants. Under full control implementation, the CBSD-infected population becomes minimal.

The fourth sub-plot illustrates the dynamics of cassava plants co-infected with both CMD and CBSD (i_{mb}). The figure reveals that co-infection is most severe when no control is applied. However, increasing the control parameter substantially reduces the co-infected population over time. At maximum control effort, the co-infected class approaches zero, demonstrating the strong effectiveness of the intervention measures against simultaneous infections.

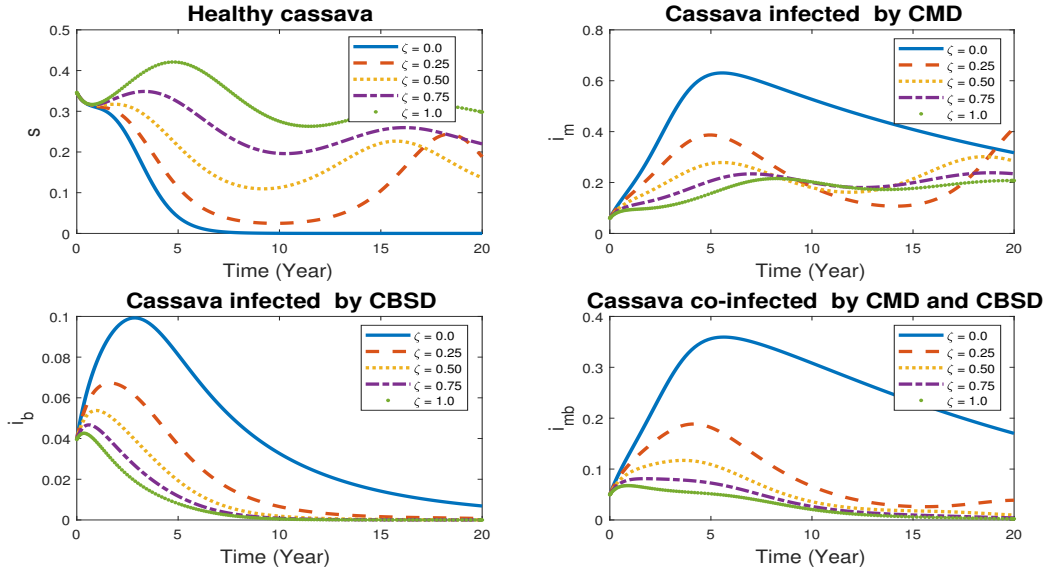


FIGURE 7. Plot showing the impact of mechanical control (ζ) on cassava model dynamics.

Figure 7 illustrates the impact of mechanical control (ζ) on the dynamics of model (2) over a period of time. The first sub-plot represents the healthy cassava population (s). The simulation results show that the number of healthy cassava plants decreases when the control effort is low. In the absence of effective control, disease transmission remains active, leading to a reduction in healthy cassava plants. However, as the control parameter (ζ) increases, the healthy cassava population improves gradually, indicating that stronger intervention measures help maintain and sustain healthy cassava production.

From the second sub-plot, it is observed that the CMD-infected population increases rapidly when there is little or no control. As the control effort increases, the number of infected plants decreases significantly. At higher levels of control, the infection burden becomes much lower, demonstrating that the implemented intervention strategy is effective in reducing the transmission and prevalence of CMD.

The third sub-plot reveal that the CBSD-infected population follows a similar pattern to that of CMD infection. Without adequate control measures, the infection persists within the cassava population. However, increasing the control parameter leads to a steady reduction in CBSD infection levels, showing that stronger control strategies contribute greatly to disease suppression.

The fourth sub-plot indicate that co-infection levels are highest when no mechanical control strategy is applied. As the control effort increases, the co-infected population declines progressively over time. Under maximum control implementation, the co-infected class is reduced substantially, suggesting that the intervention measures are highly effective in minimizing multiple infections within the cassava population.

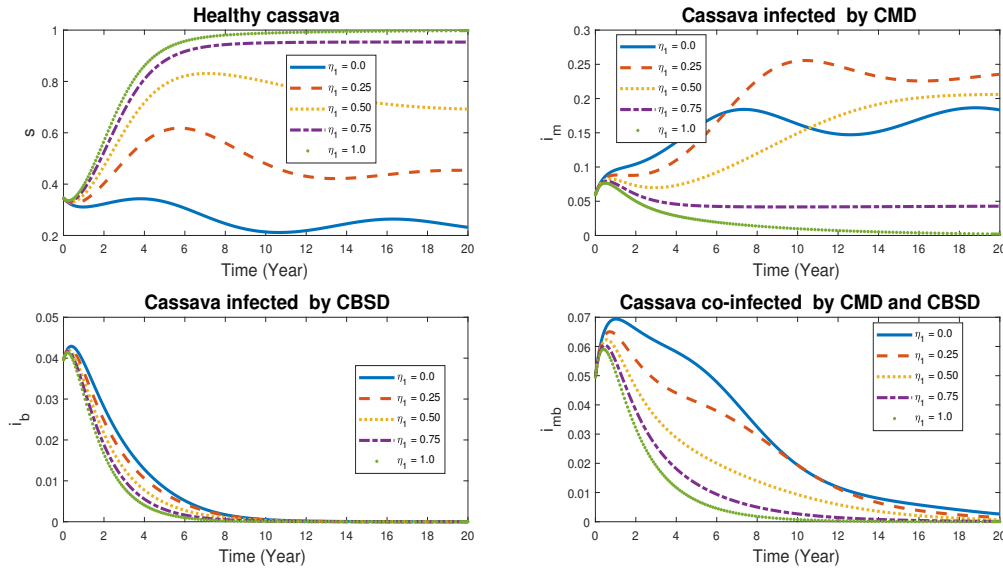


FIGURE 8. Plot showing the impact of chemical control (η_1) on cassava model dynamics.

Figure 8 presents the numerical simulation results showing the effect of chemical control on the dynamics of model (2). The simulations were carried out for different values of the control parameter, namely ($\tau_1 = 0.0, 0.25, 0.50, 0.75,$) and (1.0).

The first sub-plot indicates that the proportion of healthy cassava plants increases significantly as the level of control increases. When no control is applied ($\tau_1 = 0$), the healthy cassava population remains relatively low. However, as the control intensity increases, the number of healthy cassava plants rises steadily and approaches its maximum level under full implementation of the control strategy ($\tau_1 = 1.0$). This suggests that effective control measures play a crucial role in preserving healthy cassava plants and enhancing cassava productivity.

The second sub-plot show that CMD prevalence decreases considerably with increasing control effort. In the absence of control, the CMD-infected population remains high throughout

the simulation period. Conversely, higher control levels lead to a substantial reduction in the number of infected cassava plants, demonstrating the effectiveness of the intervention strategy in suppressing CMD transmission within the cassava population.

Similarly, the third sub-plot reveals that increasing the control parameter significantly reduces the prevalence of CBSD over time. The reduction in the CBSD-infected compartment indicates that the implemented control strategy effectively limits disease transmission and reduces the burden of infection within the cassava population.

The fourth sub-plot shows that the proportion of co-infected cassava plants is highest when no control is implemented and decreases progressively as the control level increases. At higher levels of control, the co-infected population approaches near elimination. This behaviour highlights the impact of chemical control in disease management strategies in reducing the combined effects of CMD and CBSD on cassava production.

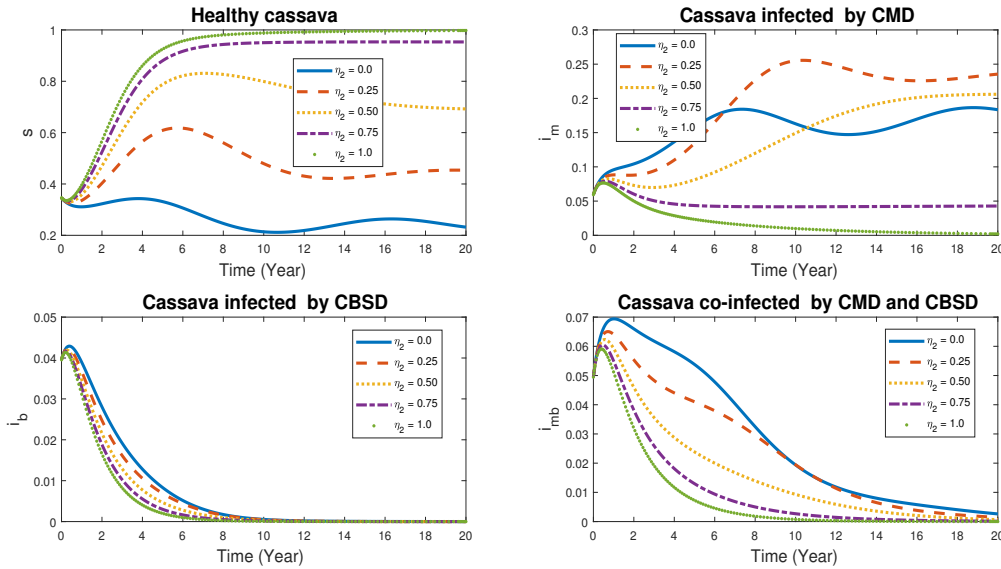


FIGURE 9. Plot showing the impact of biological (η_2) on cassava model dynamics.

Figure 9 presents the biological control on the dynamics of model (2). The simulations were performed for different values of the control parameter ($\tau_2 = 0.0, 0.25, 0.50, 0.75,$) and (1.0).

The first sub-plot indicates that the proportion of healthy cassava plants increases progressively as the control parameter (τ_2) increases. In the absence of control ($\tau_2 = 0$), the healthy

cassava population remains relatively low due to continuous disease transmission within the cassava population. However, as the level of control increases, the healthy cassava compartment rises steadily and approaches its maximum level when the control effort reaches ($\tau_2 = 1.0$). This suggests that increasing the intensity of the control strategy contributes significantly to the protection of cassava plants from infection and promotes higher cassava productivity.

The second sub-plot illustrates the prevalence of CMD decreases substantially with increasing values of (τ_2). When no control measure is implemented, CMD infection remains comparatively high throughout the simulation period. However, stronger control interventions result in a rapid decline in the CMD-infected population, indicating the effectiveness of the control strategy in suppressing CMD transmission within the cassava population.

Similarly, the third sub-plot reveal that increasing the control parameter (τ_2) leads to a significant reduction in the prevalence of CBSD over time. This decline demonstrates the ability of the implemented control measures to reduce disease transmission and limit the persistence of CBSD within the cassava population.

The fourth sub-plot shows that co-infection is highest when no control is applied and decreases steadily as the control parameter increases. At higher control levels, the co-infected population approaches near elimination, highlighting the importance of effective control strategies in reducing the combined burden of CMD and CBSD infections.

5. DISCUSSION

The co-infection dynamics of Cassava Mosaic Disease (CMD) and Cassava Brown Streak Disease (CBSD) in cassava plants and whitefly vectors were studied using a deterministic mathematical model. In order to ensure that the model is both mathematically well-posed and biologically relevant, fundamental features such as positivity and boundedness of solutions were defined and established. The sub-models for CMD and CBSD were examined in order to provide additional epidemiological understanding for a situation where either of the infection is invading the system. The epidemiological features of the model such as the disease-free equilibrium and basic reproduction numbers were determined. The method in [17] was used to establish the global stability of the model about the disease-free equilibrium, whereas the Jacobian matrix approach was used for local stability. These showed that it is possible to eradicate

Cassava Mosaic Disease (CMD) or Cassava brown streak disease irrespective of the initial infected populations are within the neighbourhood of the DFE provided that the control measures are effective enough such that $R_{0m} < 1$ or $R_{0b} < 1$. To show that disease eradication is independent of the initial infected population for the sub-system. On the contrary, it was established that the CMD-CMBD co-infection model is not globally asymptotically stable. This indicates that CMD-CMBD co-infection could be difficult to be eradicated irrespective of the size of population of the infected cassava plants and white flies at the beginning of the outbreak, even when the control measures are effective to keep R_0 below unity which illustrates the severity of co-infection. Thus, more effective intervention is required to eradicate the diseases in a co-infected scenario.

Numerical simulations were used to supplement the theoretical findings. Using data on cassava production from the World Bank database, model parameters were estimated using a least-squares fitting technique. The fitted model's capacity to represent the underlying dynamics of the disease was demonstrated by its strong agreement with the observed data. To investigate the future development of CMD-CBSD co-infection under various epidemiological scenarios, long-term model predictions were also carried out. The efficacy of various control strategies, such as roguing sick plants, chemical control, biological control of vectors, and resistant cassava varieties, was also assessed. According to the simulation results, applying control measures greatly lowers the prevalence of illness and increases cassava productivity, with integrated intervention approaches offering the biggest advantages. The results indicate that the fitted model provides a satisfactory representation of observed disease dynamics and can be used as a reliable tool for disease forecasting. Furthermore, simulation outcomes suggest that combined control strategies are more effective in reducing disease prevalence and improving cassava productivity than single control measures implemented independently. The study demonstrates the importance of integrating mathematical modelling, parameter estimation, data fitting, and forecasting in understanding and managing cassava co-infections. The proposed framework provides useful insights for policy makers, researchers, and agricultural stakeholders seeking sustainable strategies for enhancing cassava production and food security in Nigeria.

In summary, this study advances our knowledge of the dynamics of CMD–CBSD co-infection transmission in cassava. The model can be used to determine efficient methods for lowering infection and increasing cassava output since it illustrates how various control techniques impact the spread of disease. The results could aid in the development of sustainable strategies for controlling cassava viral infections and enhancing food security by farmers, agricultural researchers, and legislators.

CONFLICT OF INTERESTS

The author(s) declare that there is no conflict of interests.

REFERENCES

- [1] A.W. Borku, T.T. Tora, M. Masha, Cassava in Focus: A Comprehensive Literature Review, Its Production, Processing Landscape, and Multi-Dimensional Benefits to Society, *Food Chem. Adv.* 7 (2025), 100945. <https://doi.org/10.1016/j.focha.2025.100945>.
- [2] H.K. Gibbs, A.S. Ruesch, F. Achard, M.K. Clayton, P. Holmgren, et al., Tropical Forests Were the Primary Sources of New Agricultural Land in the 1980s and 1990s, *Proc. Natl. Acad. Sci. U.S.A.* 107 (2010), 16732–16737. <https://doi.org/10.1073/pnas.0910275107>.
- [3] B. Devi, M.N. Kumar, M. Chutia, N. Bhattacharyya, Abiotic and Biotic Stress Challenges of Cassava in Changing Climate and Strategies to Overcome: A Review, *Sci. Hortic.* 305 (2022), 111432. <https://doi.org/10.1016/j.scienta.2022.111432>.
- [4] O.D. Omodara, O.A. Ige, O. Oluwasola, A.T. Oyebanji, O.O. Afape, Factors Influencing Cassava Farmers' Choice of Climate Change Adaption Practices and Its Effect on Cassava Productivity in Nigeria, *Heliyon* 9 (2023), e14563. <https://doi.org/10.1016/j.heliyon.2023.e14563>.
- [5] V. Alonso Chavez, A.E. Milne, F. van den Bosch, J. Pita, C.F. McQuaid, Modelling Cassava Production and Pest Management under Biotic and Abiotic Constraints, *Plant Mol. Biol.* 109 (2022), 325–349. <https://doi.org/10.1007/s11103-021-01170-8>.
- [6] R.R. Shirima, E.N. Wosula, A.A. Hamza, N.A. Mohammed, H. Mouigni, et al., Epidemiological Analysis of Cassava Mosaic and Brown Streak Diseases, and Bemisia tabaci in the Comoros Islands, *Viruses* 14 (2022), 2165. <https://doi.org/10.3390/v14102165>.
- [7] C.M. Casinga, R.R. Shirima, A. Kangela, E.N. Wosula, B. Bashizi, et al., Effects of Cassava Brown Streak Disease and Harvest Time on Two Cassava Mosaic Disease-Resistant Varieties in Eastern Democratic Republic of the Congo, *Agronomy* 15 (2025), 2891. <https://doi.org/10.3390/agronomy15122891>.

- [8] P.C. Chikoti, M. Tembo, Expansion and Impact of Cassava Brown Streak and Cassava Mosaic Diseases in Africa: A Review, *Front. Sustain. Food Syst.* 6 (2022), 1076364. <https://doi.org/10.3389/fsufs.2022.1076364>.
- [9] B. Erick, M. Mayengo, Modelling the Dynamics of Cassava Mosaic Disease with Non-Cassava Host Plants, *Inform. Med. Unlocked* 33 (2022), 101086. <https://doi.org/10.1016/j.imu.2022.101086>.
- [10] S. Sangsawang, U.W. Humphries, A. Khan, P. Pongsumpun, Sensitivity Analysis of Cassava Mosaic Disease with Saturation Incidence Rate Model, *AIMS Math.* 8 (2023), 6233–6254. <https://doi.org/10.3934/math.2023315>.
- [11] P. Jittamai, N. Chanlawong, W. Atisattapong, W. Anlamlert, N. Buensanteai, Reproduction Number and Sensitivity Analysis of Cassava Mosaic Disease Spread for Policy Design, *Math. Biosci. Eng.* 18 (2021), 5069–5093. <https://doi.org/10.3934/mbe.2021258>.
- [12] F. Minhós, A. Raza, U. Shafique, M. Mohsin, Modeling, Analysis, and Transmission Dynamics of Cassava Mosaic Disease Through Stochastic Fractional Delay Differential Equations, *Mathematics* 13 (2025), 383. <https://doi.org/10.3390/math13030383>.
- [13] T.Q.S. Abdullah, G. Huang, W. Al-Sadi, Y. Aboelmagd, W. Mobarak, Fractional Dynamics of Cassava Mosaic Disease Model with Recovery Rate Using New Proposed Numerical Scheme, *Mathematics* 12 (2024), 2386. <https://doi.org/10.3390/math12152386>.
- [14] J. Holt, M.J. Jeger, J.M. Thresh, G.W. Otim-Nape, An Epidemiological Model Incorporating Vector Population Dynamics Applied to African Cassava Mosaic Virus Disease, *J. Appl. Ecol.* 34 (1997), 793–806. <https://doi.org/10.2307/2404924>.
- [15] O.C. Collins, K.J. Duffy, O.A. Olanrewaju, Mathematical Model Analysis for Dynamics and Control of African Cassava Mosaic Virus and East African Cassava Mosaic Cameroon Virus Co-infections, *Front. Appl. Math. Stat.* 12 (2026), 1770887. <https://doi.org/10.3389/fams.2026.1770887>.
- [16] C. Castillo-Chavez, Z. Feng, W. Huang, On the Computation of R_0 and its Role on Global Stability, in: C. Castillo-Chavez, S. Blower, P. van den Driessche, D. Kirschner, A.A. Yakubu, (eds) *Mathematical Approaches for Emerging and Reemerging Infectious Diseases: An Introduction*, The IMA Volumes in Mathematics and its Applications 125, Springer, New York, pp. 229–250, (2002). https://doi.org/10.1007/978-1-4757-3667-0_13.
- [17] P. van den Driessche, J. Watmough, Reproduction Numbers and Sub-Threshold Endemic Equilibria for Compartmental Models of Disease Transmission, *Math. Biosci.* 180 (2002), 29–48. [https://doi.org/10.1016/S0025-5564\(02\)00108-6](https://doi.org/10.1016/S0025-5564(02)00108-6).
- [18] M.J. Jeger, J. Holt, F. van den Bosch, L.V. Madden, Epidemiology of Insect-Transmitted Plant Viruses: Modelling Disease Dynamics and Control Interventions, *Physiol. Entomol.* 29 (2004), 291–304. <https://doi.org/10.1111/j.0307-6962.2004.00394.x>.

- [19] I. Adjei, O.A. Oyebamiji, Climate Change Impact on Migration and Food Security: An Existential Threat in Africa, *Int. J. Earth Sci. Knowl. Appl.* 6 (2024), 90–106.
- [20] E.W. Mghenyi, A.R. Sebastian, Nigeria - Policy Reforms and Investment Priorities in the Cassava Value Chain, Policy Note 185966, World Bank, Washington, D.C., 2021.
- [21] W.G. Adebayo, Cassava Production in Africa: A Panel Analysis of the Drivers and Trends, *Heliyon* 9 (2023), e19939. <https://doi.org/10.1016/j.heliyon.2023.e19939>.