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CO-DYNAMICS OF CASSAVA VIRUS DISEASE WITH FARMING AWARENESS AND CONTROL MEASURES: A CASE OF LAKE ZONE IN TANZANIA

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Abstract: The cassava virus diseases co-dynamics model is formulated and analysed using ordinary differential equations theories. The modelling involves cassava plant and whitefly vector population, with aspects of Farmer's awareness level. The evaluation of farmers' awareness inverted numerically and found that increasing awareness levels among farmers decrease disease transmission and spread. The data from ten district councils in the Tanzania lake zone fit and estimate the model parameters. In addition, time-dependent controls were incorporated to reduce the burden on cassava farmers. The findings revealed that with limited resources, uprooting and burning the infected cassava plants and awareness campaign programs significantly reduce the transmission. Therefore, to overcome the burden caused by cassava virus diseases, the farmers are recommended to uproot and burn the infected cassava plants from the farm. Also, it should be updated on controlling disease through educational campaign programs to enhance farmers' awareness.

Keywords: cassava virus diseases; cassava mosaic disease; cassava brown streak disease; Farmer's awareness; optimal control; cost-effectiveness.

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1. INTRODUCTION

Cassava (*Manihot esculenta* Crantz) is a staple food crop in several developing countries and the fourth most important source of calories after sugar cane, maize, and rice [1]. Globally over 800 million people depend on cassava tubers as their foremost staple food [2]. Cassava is the primary security food in Africa, and it plays a more significant role in minimising or alleviating the food crisis [3]. Furthermore, it is the most important source of carbohydrates in some of Sub-Saharan Africa's poorest countries, such as Nigeria, Kenya, Senegal, Cameroon, Benin and Tanzania [4]. Sub-Saharan Africa is the world's biggest cassava producer, accounting for more than 51% of global production [5].

Tanzania has been the 11th cassava producer globally and Africa's sixth largest, with an estimated annual output of 5.4 million tonnes per year [5]. Humans consume over 84% of the cassava produced in Tanzania, the remainder going to animal feed, alcohol manufacture, and starch production [6]. In Tanzania, the average cassava output is 2.0 tons per hectare, lower than the potential yield of 20 tons per hectare [7]. Even though cassava is cultivated throughout the country, the lake zone in Tanzania is the leading area for cassava production [8]. Cassava is one of the country's most essential security food crops and the most important staple food in the Tanzanian lake zone (Kapinga et al., 1997). However, cassava yields in Tanzania's Lake Zone are still low due to various factors, including pests and diseases, according to NBS. In addition, cassava yields have been severely affected due to several diseases. However, cassava Virus Diseases (CVD), particularly Cassava Mosaic Disease (CMD) and Cassava Brown Streak Disease (CBSD), are the most devastating and pose the most significant danger to cassava production [10], [11].

This paper focuses on CVD co-infection, specifically CMD and CBSD, which are biotic threats to cassava productivity [11]. The number of CMD and CBSD cases reported in several districts threatens cassava production [4], [5]. The Cassava Mosaic Virus (CMV), which causes CMD, is spread by whitefly vectors (*Bemisia tabaci*) and through vegetative propagation [12]. Leaf yellowing, narrowing, deformation, stunted growth, and narrowing of root tubers indicate CMD dictated by the host plant's sensitivity, virus strain, and climatic conditions [13].

Cassava is also affected by severe CBSD, which degrades the quality of the roots in several Lake Zone districts (IITA, 2010). CBSD is caused by the Cassava Brown Streak Virus (CBSV), which is spread by planting infected cuttings and via the whitefly vector (*Bemisia tabaci*) [6]. Foliar chlorosis and a few stem lesions are among the CBSD symptoms [10], [15]. The CBSD also affects tuberous roots, which develop a yellow/brown, dry, corky necrosis within the starch-bearing tissues for a while, along with pitting and distortion visible from the outside [6]. According to disease research, the crop can lose up to 74% of its production, while in severely impacted areas, yield losses can reach 100% [6].

According to John (2011), CMD and CBSD co-infection in cassava cultivars lead to more severe symptoms, and such a condition could have devastating effects on cassava output. Therefore, management of CVD control techniques is recommended to prioritise both the CMV and CBSV viruses. CMD and CBSD are currently managed by using disease-free planting material, uprooting and burning diseased cassava plants, planting symptomless cassava, quarantining and scanning the fields for diseased plants, and planting resistant varieties [6], [15]–[21]. However, more study is still underway on creating tolerant/resistant cassava planting material variants. Although some farmers employ pesticides, pesticides are ineffective and inefficient.

Kinene et al. (2015) proposed a mathematical model for transmitting CBSD in Uganda and the most cost-effective control techniques. Researchers introduced two controls one involves spraying pesticides, and the second requires removal of the infected plants by uprooting and burning. Findings show that the most cost-effective strategy combines uprooting and burning infected cassava plants.

Awareness campaigns, in particular through radio, Television, social media and Seminars among cassava farmers, are required so that farmers will gain knowledge on a CVDs control approach. In their awareness, farmers can keep the crop under observation. Therefore, if correctly instructed, they will implement the control to reduce the spread of CVD co-infection. People working in agriculture need accurate and up-to-date information about crops, diseases and pests. Hence, the importance of electronic media in keeping farmers informed and providing them with crucial

agricultural details [22]–[24]. Accessible pesticide information campaigns help farmers know the severe risks pesticides have on human health and the environment and minimise adverse effects [25]. Adopting awareness programs to educate farmers results in better comprehensive development for the cultivars and farmers. Farmers primarily receive information on the use and risks of pesticides orally. Farmers who are self-aware use vastly enhanced agronomic techniques, protecting public health and lowering environmental hazards [26]. Therefore, awareness is essential in crop pest management. Television, radio, and mobile telephone are beneficial media providing agricultural practices and crop protection information. This article presents a mathematical model for CMD and CBSD (CVD co-infection), incorporating farming awareness levels. The main focus is to compare a fundamental advantage favouring the combined strategy by involving the following CVD co-infection control management. This is the immediate removal of the infected plants by uprooting and burning, killing the whitefly vectors through spraying pesticides and other means, and increasing farming awareness through the education campaign among cassava farmers through media to minimise the spread of CVD. The three-dependent optimal control problem is formulated and solved using the Pontryagin Minimum Principle to determine the optimal level of control strategies for cost-effectiveness.

2. MODEL DESCRIPTION AND FORMULATION

The deterministic mathematical model of Codynamics of Cassava mosaic disease (CMD) and Cassava brown streak disease (CBSD) was formulated and analysed following the theory of ordinary differential equations. The model includes the Cassava plant and whitefly vectors population. The awareness level among farmers $A(t)$ is incorporated to minimise disease transmission and spread. The Cassava population $N(t)$ is divided into five classes, namely: Susceptible (P), exposed (E), infected with CMD (M), infected with CBSD (S) and finally, CVD co-infection (B) class of plants infected with both CMD and CBSD. Thus

$$N(t) = P(t) + E(t) + M(t) + S(t) + B(t)$$

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The total whitefly vector population $N_v(t)$ has two classes: susceptible (V) and Infected (I).

Thus $N_v(t) = V(t) + I(t)$.

The compartmental diagram in Figure 1 summarises the transmission dynamics of CVD co-infection.

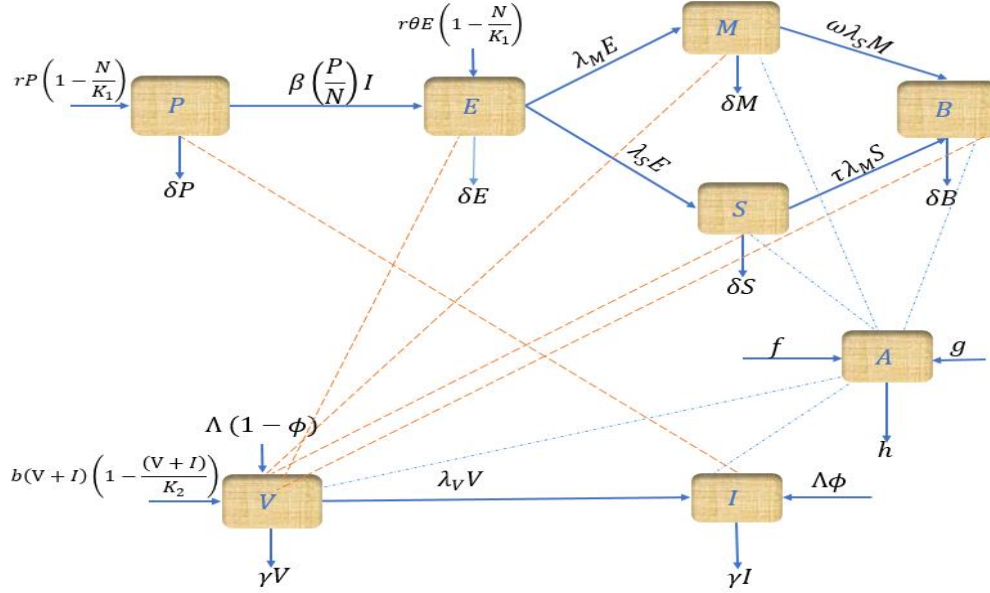


Figure 1: *Compartment model diagram model for the transmission dynamics of CVD co-infection with awareness. The solid lines symbolise the constant transmission from one compartment to another. The red dotted lines depict the regular interactions between different compartments, while the blue dashed dot lines indicate the activities of farmers due to disease awareness.*

The susceptible cassava plants contacted an infected whitefly vector and moved to the exposed class before becoming infected. Cassava plants can be infected with CMV, CBSV or both; the susceptible whitefly vector acquires viruses by inoculating the infected cassava plants and becomes infected. Due to the finite size of the cassava field, we assume logistic growth for the cassava population, with a net growth rate r and carrying capacity K_1 . The Farmer can replant both susceptible and exposed cassava cuttings stem without knowing. Therefore, θ specifies the

frequency to which exposed cassava is selected relative to susceptible cassava replanting material. Hence susceptible cassava plant population can be described by the following logistic population growth term $rp\left(1 - \frac{N}{K_1}\right)$ and by replanting exposed cuttings materials is $r\theta E\left(1 - \frac{N}{K_1}\right)$ where θE is the number of cassava material replanted with the viruses. The cassava plant moves to the exposed class after acquiring viruses through contact with the infective whitefly vector at a rate β . After developing the symptoms of one disease, the cassava plant infected with the single disease will become dually infected with the other Cassava Virus disease. The circumstance of a cassava plant being infected by both viruses simultaneously will be referred to as CVD co-infection. The CMV infectious class can move to the CBSV infectious class to form the CVD class at a rate ω , and the CBSV infectious class can move to the CMV infectious class to create the CVD class at the rate τ since each disease is joining the CVD class in a different time. All cassava classes are harvested at a rate δ during the harvesting cassava tubers period.

The exposed cassava plants can move to the CMD infectious class by acquiring CMV with λ_M transmission rate or CBSV infectious by acquiring CBSV with λ_S transmission rate. The transmission rates are defined as follows

$$\lambda_M = \eta \left(\frac{M + \rho B}{N} \right) \quad \text{and} \quad \lambda_S = \mu \left(\frac{S + \psi B}{N} \right)$$

where η and μ are the infectious rate of CMD and CBSV, respectively, while ρ and ψ are the transmission coefficient for the dually infected concerning CMD and CBSV, respectively [27]. The whitefly vector population increases due to birth and immigration/movement from one field to another. The whitefly vectors (*Bemisia tabaci*) population dynamics that transmit the viruses from infected to the susceptible cassava plants grow logistically with the vector birth rate b with carrying capacity K_2 . Both classes of Whitefly vectors will leave the population due to the natural death rate γ because the virus does not affect the whitefly. We consider the new whitefly vectors that flow into the population through immigration or movement among nearby cassava fields that

are either healthy or infective. The flow-through immigration/movement is assumed to occur constantly at the rate Λ with the infected whitefly vector fraction ϕ and the remaining fraction $(1-\phi)$. The healthy whitefly vector became an infected whitefly vector by acquiring viruses with λ_v the force of infection. That depends on the availability of healthy whitefly vectors and the abundance of the infected cassava plants. The force of infection described as

$$\lambda_v = \alpha \left(\frac{E+M+S+B}{N} \right) \text{ where } \alpha \text{ is the whitefly virus acquisition rate.}$$

The awareness level among farmers about the CMD, CBSD and CVD co-infection increases with the rate f through global sources. The local awareness rate about diseases g is proportional to the infected cassava plants. However, due to the fall in importance, the level of understanding of the CVD co-infection may decrease at the rate h [28], [29].

Aware farmers keep the cassava plant under observation. Thus, removing infected cassava plants from the field can be modelled via the following nonlinear term $\frac{aAM}{1+A}$ with the maximum activity rate a in diseased plants [30], [31]. Aware farmers' activities to eliminate infected and susceptible whitefly vectors can be implemented at a rate c with the nonlinear term $\frac{cAM}{1+A}$ [30], [31].

2.1 Equations of the model

Now using the model assumptions given above, the co-dynamics of CVD are described by the following systems of nonlinear differential equations:

$$\frac{dP}{dt} = rP \left(1 - \frac{N}{K_1} \right) - \beta \left(\frac{P}{N} \right) I - \delta P$$

$$\frac{dE}{dt} = r\theta E \left(1 - \frac{N}{K_1} \right) + \beta \left(\frac{P}{N} \right) I - \lambda_M E - \lambda_S E - \delta E$$

$$\frac{dM}{dt} = \lambda_M E - \omega \lambda_S M - \delta M - \frac{aAM}{1+A}$$

$$\begin{aligned}
\frac{dS}{dt} &= \lambda_S E - \tau \lambda_M S - \delta S - \frac{aAS}{1+A} \\
\frac{dB}{dt} &= \omega \lambda_S M + \tau \lambda_M S - \delta B - \frac{aAB}{1+A} \\
\frac{dV}{dt} &= b(V+I) \left(1 - \frac{(V+I)}{K_2} \right) + \Lambda(1-\phi) - \lambda_V V - \gamma V - \frac{cAV}{1+A} \\
\frac{dI}{dt} &= \lambda_V V + \Lambda \phi - \gamma I - \frac{cAI}{1+A} \\
\frac{dA}{dt} &= f + g(M+S+B+I) - hA
\end{aligned} \tag{1}$$

2.2 Invariant Region of the Solution

The CVD dynamic model system (1) has three subpopulations where all parameters and variables are nonnegative $\forall t \geq 0$.

Lemma 1:

Given the model of the system (1) \mathbb{R}_+^8 with initial conditions $P(t) \geq 0, E(t) \geq 0, M(t) \geq 0, S(t) \geq 0, B(t) \geq 0, V(t) \geq 0, I(t) \geq 0, A(t) \geq 0$, its solution enters the invariant region

$$H = H_C \times H_V \times H_A = \mathbb{R}_+^5 \times \mathbb{R}_+^2 \times \mathbb{R}_+^1 \quad \text{where;}$$

$$H_C = \{(P, E, M, S, B) \in \mathbb{R}_+^5 : P(t) + E(t) + M(t) + S(t) + B(t) = N\} \tag{2}$$

$$H_V = \{(V, I) \in \mathbb{R}_+^2 : V(t) + I(t) = N_V\}$$

$$H_A = \{A \in \mathbb{R}_+^1\}$$

Proof

We use the box invariant method to establish the feasible region of the CVD co-infection model solution [32]–[35]. In the co-infection dynamical system (1) $\dot{X} = Q(X, t)$, $X \in \mathbb{R}^n$, we assume its solution's continuity and Lipschitz properties. The model system (1) is reduced to the form

$$\frac{dX}{dt} = L(x)X + Q \tag{3}$$

where $X = (P, E, M, S, B, V, I, A)^T$ and a column vector $Q = (N, 0, 0, 0, 0, N_V, 0, A)^T$ thus

$$L(x) = \begin{bmatrix} -a_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ \frac{\beta I}{N} & -a_2 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \lambda_M & -a_3 & 0 & 0 & 0 & 0 & 0 \\ 0 & \lambda_S & 0 & -a_4 & 0 & 0 & 0 & 0 \\ 0 & 0 & \omega\lambda_S & \tau\lambda_M & -a_5 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -b_1 & b_2 & 0 \\ 0 & 0 & 0 & 0 & 0 & b_3 & -b_4 & 0 \\ 0 & 0 & g & g & g & 0 & g & -h \end{bmatrix} \quad (4)$$

$$\text{where: } a_1 = \frac{\beta I}{N} + \delta + r \left(\frac{N}{K_1} - 1 \right), a_2 = \delta + r\theta \left(\frac{N}{K_1} - 1 \right) + \lambda_M + \lambda_S, a_3 = \omega\lambda_S + \delta + \frac{aA}{1+A}$$

$$a_4 = \tau\lambda_M + \delta + \frac{aA}{1+A}, a_5 = \delta + \frac{aA}{1+A}, b_1 = \gamma + \lambda_V + b \left(\frac{V+I}{K_2} - 1 \right) + \frac{b(V+I)}{K_2} + \frac{cA}{1+A}$$

$$b_2 = b - \frac{2b(V+I)}{K_2} - \frac{cA}{1+A}, b_3 = \lambda_V, b_4 = \gamma + \frac{cA}{1+A}$$

In a reduced Metzler matrix $L(x)$, the principal diagonal has only negative values, and the remaining off-diagonal values are all positive. Thus, all variables enter and remain feasible in the invariant region H . This demonstrates the epidemiological significance and well-posedness of the CVD co-infection model system (1) in the invariant region H .

2.3 Positivity of the Solutions

Theorem 1

Let the initial values of the state variables of the model system be

$$\{(P(0), E(0), M(0), S(0), B(0), V(0), I(0), A(0)) \geq 0\} \in \Omega.$$

Then, the solution set $\{P(t), E(t), M(t), S(t), B(t), V(t), I(t), A(t)\}$ of the model system (1)

is nonnegative $\forall t > 0$.

Proof

We consider the first equation of model (1), representing our susceptible cassava plants. We develop the inequality as

$$\frac{dP}{dt} \geq \left(r \left(1 - \frac{N}{K_1} \right) - \left(\frac{\beta I}{N} + \delta \right) \right) P \quad (5)$$

By solving differential inequality, we get

$$P(t) \geq P(0) e^{-\left(\frac{\beta I}{N} + \delta\right)t}$$

Thus as $t \rightarrow \infty$ then, it follows that $P(t) \geq P(0) e^{-\left(\frac{\beta I}{N} + \delta\right)t} \geq 0$. Hence $P(t) \geq 0$

Applying the same procedures for the remaining state variables $\{E(t), M(t), S(t), B(t), V(t), I(t), A(t)\}$ are positive for all time $t > 0$. As a result, the CVD co-infection transmission model stated in the system (1) is epidemiologically significant and mathematically well-posed.

3. THEORETICAL ANALYSIS OF THE MODEL

3.1 CVD co-infection free equilibrium point C^0 (CVDCFEP)

The state in which there are no diseased plants in the cassava population and no infected whitefly in the whitefly vector population is known as CVD co-infection-free equilibrium. We get the CVDCFEP $C^0 = (P^0, E^0, M^0, S^0, B^0, V^0, I^0, A^0)$. Thus

$$C^0 = (P^0, E^0, M^0, S^0, B^0, V^0, I^0, A^0) = \left(\frac{K_1(r-\delta)}{r}, 0, 0, 0, 0, \frac{K_2(b-\gamma)}{b}, 0, \frac{f}{h} \right) \quad (6)$$

The basic reproduction number \mathfrak{R}_0 of the model (1), obtained using the next-generation method as

$$\mathfrak{R}_0 = \sqrt{\frac{r\alpha\beta K_2(b-\gamma)(f+h)}{b\delta K_1(r-\delta)(cf+(f+h)\gamma)}}$$

3.2 Local stability of CVD co-infection free equilibrium point (CVDCFEP)

Theorem 2

The CVD co-infection free equilibrium point is locally asymptotically stable if $\mathfrak{R}_0 < 1$ and unstable if $\mathfrak{R}_0 > 1$.

Proof. To prove this theorem, let us first find the Jacobian matrix of system (1) at the CVD co-infection free equilibrium point: we get

$$J(C^0) = \begin{bmatrix} -z_1 & 0 & 0 & 0 & 0 & 0 & \beta & 0 \\ 0 & -\delta & 0 & 0 & 0 & 0 & \beta & 0 \\ 0 & 0 & -z_2 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -z_3 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -z_4 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -z_5 & -(\gamma - b) & z_8 \\ 0 & z_7 & z_7 & z_7 & z_7 & 0 & -z_6 & 0 \\ 0 & 0 & g & g & g & 0 & g & -h \end{bmatrix} \quad (7)$$

Where: $z_1 = r - \delta$, $z_2 = \delta + \frac{af}{f+h}$, $z_3 = \delta + \frac{af}{f+h}$, $z_4 = \delta + \frac{af}{f+h}$, $z_5 = b - \gamma + \frac{cf}{f+h}$,
 $z_6 = \gamma + \frac{cf}{f+h}$, $z_7 = \frac{\alpha r K_2 (b - \gamma)}{b K_1 (r - \delta)}$, $z_8 = \frac{K_2 (f + h)(b - \gamma)}{bh}$

By using the basic properties of matrix algebra and from the characteristic polynomial of $J(C^0)$ the following are the eigenvalues $\lambda_1 = -z_1$, $\lambda_2 = -z_2$, $\lambda_3 = -z_3$, $\lambda_4 = -z_4$, $\lambda_5 = -z_5$ and $\lambda_6 = -h$ have a negative real part, and the remaining reduced matrix is

$$J(C_1^0) = \begin{pmatrix} -\delta & \beta \\ \frac{\alpha r K_2 (b - \gamma)}{b K_1 (r - \delta)} & -\left(\gamma + \frac{cf}{f+h}\right) \end{pmatrix} \quad (8)$$

Here, we employ the determinant and trace method to examine the local stability of the CVD co-infection-free equilibrium point. However, first, we must prove that the determinant and trace of the matrix (8) are positive and negative, respectively.

The determinant of the matrix (8) is then calculated using the software Mathematica; we get

$$\text{Det}(J(C_1^0)) = \left(\frac{cf + (f+h)\gamma}{f+h}\right) \delta (1 - \mathfrak{R}_0^2) \quad (9)$$

As we can see $\left(\frac{cf + (f+h)\gamma}{f+h}\right) \delta > 0$, it is the product of positive parameters and the

$\text{Det}\left(J\left(C_1^0\right)\right)$ to-be >0 we should have $\mathfrak{R}_0 < 1$.

Also, it is clear that the trace of the matrix (20) is negative, which is

$$\text{Trace}\left(J\left(C_1^0\right)\right) = -\left(\delta + \gamma + \frac{cf}{f+h}\right) < 0 \quad (10)$$

Hence, the CVD co-infection-free equilibrium point is locally asymptotically stable if $\mathfrak{R}_0 < 1$. \square

3.3 Global stability of CVD co-infection free equilibrium point (CVDCFEP).

To investigate the issue of the global stability of CVD co-infection free equilibrium point, we use the Metzler matrix method, as stated by [36]. So, we pose the following theorem:

Theorem 2

The CVDCFEP (C^0) of the model system (1) is globally asymptotically stable (GAS) if $\mathfrak{R}_0 \leq 1$ and unstable if $\mathfrak{R}_0 \geq 1$.

Proof:

We separate the model system (1) into compartments for transmitting (E, M, S, B, I) and non-transmitting (P, V, A) individuals. Therefore, we let X_n be the vector for non-transmitting compartments, X_i the vector for transmitting compartments, and $X_{C^0, n}$ the vector of diseases free equilibrium point. Then

$$\begin{cases} \frac{dX_n}{dt} = C_1(X_n - X_{C^0, n}) + C_2 X_i \\ \frac{dX_i}{dt} = C_3 X_i \end{cases} \quad (11)$$

To prove the global stability of the CVDCFEP, we are required to show that matrix C_1 has real negative eigenvalues and C_3 is a Metzler matrix containing all nonnegative off-diagonal entries.

Based on the model system (1), we will have $X_n = (P, V, A)^T$, $X_i = (E, M, S, B, I)$

$$X_{C^0, n} = \left(\frac{K_1(r-\delta)}{r}, \frac{K_2(b-\gamma)}{b}, \frac{f}{h} \right) \quad (12)$$

$$X_n - X_{c^0,n} = \begin{pmatrix} P - \frac{K_1(r-\delta)}{r} \\ V - \frac{K_2(b-\gamma)}{b} \\ A - \frac{f}{h} \end{pmatrix} \quad (13)$$

We will now have the matrices below employing the general system non-transmitting and transmitting compartments.

$$C_1 = \begin{pmatrix} r\left(1 - \frac{N}{K_1}\right) - \frac{IB}{N} - \delta & 0 & 0 \\ 0 & b - \alpha - \gamma - \frac{2b(V+I)}{K_2} - \frac{cA}{1+A} & -\frac{cAV}{(1+A)^2} \\ 0 & 0 & -h \end{pmatrix} \quad (14)$$

Eigenvalues of C_1 are

$$\left(-\left(\frac{rN}{K_1} + \frac{\beta I}{N} + \delta - r\right), -\left(\frac{2b(V+I)}{K_2} + \lambda_v + \gamma - b\right), -h \right) \quad (15)$$

$$C_2 = \begin{pmatrix} 0 & 0 & 0 & 0 & \frac{\beta P}{N} \\ -\frac{\alpha V}{N} & -\frac{\alpha V}{N} & -\frac{\alpha V}{N} & -\frac{\alpha V}{N} & b - \frac{2b(V+I)}{K_2} \\ 0 & g & g & g & g \end{pmatrix} \quad (16)$$

$$C_3 = \begin{pmatrix} -\left(\delta + r\theta\left(\frac{N}{K_1} - 1\right) + \lambda_M + \lambda_S\right) & 0 & 0 & 0 & \frac{\beta P}{N} \\ \lambda_M & -\left(\omega\lambda_S + \delta + \frac{aA}{1+A}\right) & 0 & 0 & 0 \\ \lambda_S & 0 & -\left(\tau\lambda_M + \delta + \frac{aA}{1+A}\right) & 0 & 0 \\ 0 & \omega\lambda_S & \tau\lambda_M & -\left(\delta + \frac{aA}{1+A}\right) & 0 \\ \frac{\alpha V}{N} & \frac{\alpha V}{N} & \frac{\alpha V}{N} & \frac{\alpha V}{N} & -\left(\gamma + \frac{cA}{1+A}\right) \end{pmatrix} \quad (17)$$

Now when we calculate eigenvalues for the matrix C_1 The results indicate that the eigenvalues are negative and real, proving that the system $\frac{dX_n}{dt} = C_1(X_n - X_{c^0,n}) + C_2X_i$ is globally asymptotically stable at X_{c^0} . Furthermore, considering the matrix, C_3 we find it is a Metzler stable matrix since all of its off-diagonal elements are positive. Therefore, CVDCFEP for system (1) is globally asymptotically stable.

4. OPTIMAL CONTROL

This section aims to introduce the control mechanisms that possibly reduce the burden on farmers of cassava. Historically, most local farmers have insufficient knowledge about farming practices, especially the proper implication of the control strategies in cassava disease management. This study considers farmers' awareness levels intending to educate local farmers to gain in-depth knowledge and accurate information about a cassava disease to help set up correct control strategies during an epidemic eruption. The model system (1) is modified by introducing three time-dependent controls $u_1(t), u_2(t)$ and $u_3(t)$ reducing or eliminating the CVD co-infection transmission. The first control $u_1(t)$ aims to stop infection of the vector by the plant, which involves immediate removal of the infected plants by uprooting and burning. The second control $u_2(t)$ proposed to reduce the transmission of CVD co-infections from the infected whitefly vector to the cassava plants in the field. It involves killing the whitefly vectors through spraying pesticides and other means. The third control $u_3(t)$ aimed to increase farming awareness through the education campaign among cassava farmers through media. The goal is to reduce the number of cases of infected cassava plants at the minimum possible cost, compare the controls, and determine their cost-effectiveness. Therefore the following modifications were made to the system (1);

$$\frac{dP}{dt} = rP \left(1 - \frac{N}{K_1} \right) - \beta(1 - u_3) \left(\frac{P}{N} \right) I - \delta P$$

$$\begin{aligned}
\frac{dE}{dt} &= r\theta E \left(1 - \frac{N}{K_1}\right) + \beta(1-u_3) \left(\frac{P}{N}\right) I - \lambda_M(1-u_3)E - \lambda_S(1-u_3)E - \delta E \\
\frac{dM}{dt} &= \lambda_M(1-u_3)E - \omega\lambda_S(1-u_3)M - \delta M - (u_1+a) \frac{AM}{1+A} \\
\frac{dS}{dt} &= \lambda_S(1-u_3)E - \tau\lambda_M(1-u_3)S - \delta S - (u_1+a) \frac{AS}{1+A} \\
\frac{dB}{dt} &= \omega\lambda_S(1-u_3)M + \tau\lambda_M(1-u_3)S - \delta B - (u_1+a) \frac{AB}{1+A} \\
\frac{dV}{dt} &= b(V+I) \left(1 - \frac{(V+I)}{K_2}\right) + \Lambda(1-\phi) - \lambda_V V - \gamma V - (u_2+c) \frac{AV}{1+A} \\
\frac{dI}{dt} &= \lambda_V V + \Lambda\phi - \gamma I - (u_2+c) \frac{AI}{1+A} \\
\frac{dA}{dt} &= u_3 f + g(M+S+B) - hA
\end{aligned} \tag{18}$$

These control strategies need to be adjusted to minimise the number of infectious cassava plants, whitefly vectors and the cost of implementing the control strategies. It is ought to consider the optimal control problem with objective functional of the form

$$J = \underset{u}{Min} \int_0^{tf} \left(W_1 M + W_2 S + W_3 B + W_4 I + \sum_{i=1}^3 \frac{G_i}{2} u_i^2 \right) \tag{19}$$

where tf is the final time, and $W_j, j=1 \dots 4$ are the positive weight constants associated with the number of CMD, CBSD, CVD infectious cassava plants and infected whitefly vectors. In contrast, $G_i, i=1,2,3$ there are positive control weights relative to its cost implications. The quadratic expressions $\frac{G_1}{2} u_1^2, \frac{G_2}{2} u_2^2$, and $\frac{G_3}{2} u_3^2$ represent the costs of control efforts on removing the infected cassava plants by uprooting and burning, killing the whitefly vectors by spraying pesticides and farming awareness campaigns, respectively. In this article, the objective functional of controls $u_i; i=1,2,3$ are quadratic because these interventions' costs are nonlinear. This assumption follows studies that propose nonlinear relationships between the effects of interventions and the cost of interventions for infective populations. Moreover, several authors have often used such quadratic costs, e.g. Kinene et al. [11]. The purpose is to minimise the

objective function J ; consequently, it is required to find the optimal control such that

$$J(u^*) = \min J(u | u \in U) \quad (20)$$

where $U = \{(u_1, u_2, u_3) | u_i \text{ is Lebesgue measurable with } 0 \leq u_i \leq 1, \text{ for } t \in [0, t_f], i = 1, 2, 3\}$ is the set of admissible controls. Note that in all cases, setting the control to zero indicates that no effort is being made to control the disease, and setting it to one indicates that the most effort possible is being made. Now we apply the following theorem to show the existence of optimal control for systems (18) and (19).

Theorem 3.

Give $J(u)$ subject to the system (18) with $(P^0, E^0, M^0, S^0, B^0, V^0, I^0, A^0) \geq (0, 0, 0, 0, 0, 0, 0, 0)$ then there exists an optimal control u^* and corresponding $(P^*, E^*, M^*, S^*, B^*, V^*, I^*, A^*)$ that minimises $J(u)$ over U .

Proof

Since the set of controls and state variables are nonnegative and the measurable control set is convex and closed. Each right-hand side of the state system is continuous, bounded above by a sum of the bounded control and the state, and can be written as a linear function of u with coefficients depending on time and the state. The integrand $z(q, u)$ of the objective functional is convex. There exist constants $C_1, C_2 > 0$, and $\beta^* \geq 1$ such that the integrand of the objective

functional satisfies $z \geq C_1 \left(|u_1|^2 + |u_2|^2 + |u_3|^2 \right)^{\frac{\beta^*}{2}} - C_2$

The existence results in Lukes (1982) [Theorem 9.2.1 page 182] for the state system to verify that the first property is satisfied. The control set is convex and closed from the definition of a convex set U ; thus, the second property also holds. Given that the state solutions of a linear state system in u_i are bounded, then the right-hand side is bounded by a linear function. Finally, there are $C_1, C_2 \geq 0$ and $\beta \geq 1$ satisfying

$$W_1 M + W_2 S + W_3 B + W_4 I + G_1 u_1^2(t) + G_2 u_2^2(t) + G_3 u_3^2(t) \geq C_1 \left(|u_1|^2 + |u_2|^2 + |u_3|^2 \right)^{\frac{\beta^*}{2}} - C_2 \quad (21)$$

since the state variables are bounded. Hence, the existence of optimal control follows from the existing results [37].

4.1 CHARACTERISATION OF THE OPTIMAL CONTROLS

The representation of the optimal controls depends on Pontryagin's Maximum Principle [38]. To achieve this, one needs to transform the optimal control problem into the problem of minimising point-wise a Hamiltonian H with respect to u_i . Let x be the set of state variables, U be the set of controls, L be the set of adjoint variables, and z be the right-hand side of the differential of the i^{th} state variable. Then Our problem's Lagrangian function is the integrand of the objective functional, the inner product of the right-hand side of the state equations, and the adjoint variables $(L_1, L_2, L_3, L_4, L_5, L_6, L_7, L_8)$.

In more compact, the Lagrangian is defined by

$$H = W_1M + W_2S + W_3B + W_4I + \sum_{i=1}^3 \frac{G_i}{2} u_i^2 + Lz(t, x(t), u_i(t)) \quad (22)$$

Then, the expanded form of the Lagrangian is given by

$$\begin{aligned} H = & W_1M + W_2S + W_3B + W_4I + G_1u_1^2(t) + G_2u_2^2(t) + G_3u_3^2(t) \geq C_1 \left(|u_1|^2 + |u_2|^2 + |u_3|^2 \right)^{\frac{\beta^*}{2}} - C_2 \\ & + L_1 \left(rP \left(1 - \frac{N}{K_1} \right) - \beta(1-u_3) \left(\frac{P}{N} \right) I - \delta P \right) \\ & + L_2 \left(r\theta E \left(1 - \frac{N}{K_1} \right) + \beta(1-u_3) \left(\frac{P}{N} \right) I - \lambda_M(1-u_3)E - \lambda_S(1-u_3)E - \delta E \right) \\ & + L_3 \left(\lambda_M(1-u_3)E - \omega\lambda_S(1-u_3)M - \delta M - (u_1+a) \frac{AM}{1+A} \right) \\ & + L_4 \left(\lambda_S(1-u_3)E - \tau\lambda_M(1-u_3)S - \delta S - (u_1+a) \frac{AS}{1+A} \right) \\ & + L_5 \left(\omega\lambda_S(1-u_3)M + \tau\lambda_M(1-u_3)S - \delta B - (u_1+a) \frac{AB}{1+A} \right) \\ & + L_6 \left(b(V+I) \left(1 - \frac{(V+I)}{K_2} \right) + \Lambda(1-\phi) - \lambda_VV - \gamma V - (u_2+c) \frac{AV}{1+A} \right) \\ & + L_7 \left(\lambda_VV + \Lambda\phi - \gamma I - (u_2+c) \frac{AI}{1+A} \right) \\ & + L_8 (u_3f + g(M+S+B) - hA) \end{aligned} \quad (23)$$

where $L_i, i=1,2,3,4,5,6,7,8$ are the co-state variables associated by P, E, M, S, B, V, I, A .

Theorem 4.

Since u_i^* is the set of optimal control and x^* the corresponding set of solutions for the state system (18) that minimises J over Ω ; then there exist adjoint variables L such that

$$\frac{dL}{dt} = -\frac{dH}{dx}, \text{adjoint conditions and} \quad (24)$$

$$L(t_f) = 0, \text{transversality conditions. Furthermore;} \quad (25)$$

$$\frac{dH}{dt} = 0, \text{at } u^* \text{ optimality conditions:} \quad (26)$$

Proof.

We are taking the partial derivative of the Lagrangian H with respect to state variables. That is,

$$\begin{aligned} \frac{dL_1}{dt} &= L_1 \left(\delta + r \left(\frac{N}{K_1} - 1 \right) - \left(\frac{\beta(u_3 - 1)I^*}{N} \right) \right) + \left(\frac{\beta(u_3 - 1)I^*L_2}{N} \right) \\ \frac{dL_2}{dt} &= L_2 \left(\delta + r\theta \left(\frac{N}{K_1} \right) \right) - \left(\frac{\mu(S^* + B^*\psi)(u_3 - 1)}{N} \right) - \left(\frac{\eta(M^* + B^*\rho)(u_3 - 1)}{N} \right) + \frac{\alpha V^* L_6}{N} - \left(\frac{\alpha V^* L_7}{N} \right) \\ &+ \left(\frac{\eta L_3 (M^* + B^*\rho)(u_3 - 1)}{N} \right) + \left(\frac{\mu L_4 (S^* + B^*\psi)(u_3 - 1)}{N} \right) \\ \frac{dL_3}{dt} &= L_3 \left(\delta + \left(\frac{(u_1 + a)A^*}{A^* + 1} \right) \right) + \left(\frac{\eta(u_3 - 1)E^*}{N} \right) + \left(\frac{\mu\omega(S^* + B^*\psi)}{N} \right) - gL_8 - W_1 + L_5 \left(\frac{\eta\tau(u_3 - 1)S^*}{N} \right) \\ &+ \left(\frac{\mu\omega(S^* + B^*\psi)(u_3 - 1)}{N} \right) + \frac{\alpha L_6 V^*}{N} - \left(\frac{\alpha L_7 V^*}{N} \right) - \left(\frac{\eta(u_3 - 1)L_2 E^*}{N} \right) - \left(\frac{\eta(u_3 - 1)\tau L_4 S^*}{N} \right) \\ \frac{dL_4}{dt} &= L_4 \left(\delta + \left(\frac{(u_1 + a)A^*}{A^* + 1} \right) \right) + \frac{\mu(u_3 - 1)E^*}{N} - \left(\frac{\eta\tau(M^* + B^*\rho)(u_3 - 1)}{N} \right) - gL_8 - W_2 + L_5 \left(\frac{\mu\omega(u_3 - 1)M^*}{N} \right) \\ &+ \left(\frac{\eta\tau(M^* + B^*\rho)(u_3 - 1)}{N} \right) + \frac{\alpha V^* L_6}{N} - \frac{\alpha V^* L_7}{N} + \frac{\mu\omega M^* L_3}{N} - \frac{\mu(u_3 - 1)E^* L_2}{N} \end{aligned}$$

$$\begin{aligned}
\frac{dL_5}{dt} &= L_5 \left(\delta + \left(\frac{(u_1 + a)A^*}{A^* + 1} \right) \right) + \left(\frac{\mu(u_3 - 1)\omega\psi M^*}{N} \right) + \frac{\eta(u_3 - 1)\tau\rho S^*}{N} - gL_8 - W_3 + L_4 \left(\frac{\mu(u_3 - 1)\psi E^*}{N} \right) \\
&\quad - \frac{\eta(u_3 - 1)\rho\tau S^*}{N} + L_3 \left(\frac{\mu(u_3 - 1)\omega\psi M^*}{N} \right) + \frac{\eta(u_3 - 1)\rho E^*}{N} - L_2 \left(\frac{\eta(u_3 - 1)\rho E^*}{N} \right) + \frac{\mu(u_3 - 1)\psi E^*}{N} + \frac{L_6\alpha V^*}{N} - \frac{L_7\alpha V^*}{N} \\
\frac{dL_6}{dt} &= L_6 \left(\gamma + b \left(\frac{(V^* + I^*)}{K_2} - 1 \right) + b \left(\frac{(V^* + I^*)}{K_2} \right) + \frac{(u_2 + c)A^*}{A^* + 1} + \left(\frac{\alpha(E^* + M^* + S^* + B^*)}{N} \right) \right) - hL_8 \\
&\quad - \left(\frac{\alpha L_7(E^* + M^* + S^* + B^*)}{N} \right) \\
\frac{dL_7}{dt} &= L_7 \left(\gamma + \frac{(u_2 + c)A^*}{A^* + 1} \right) - gL_8 - W_4 + L_6 \left(b \left(\frac{V^* + I^*}{K_2} - 1 \right) \right) + b \left(\frac{V^* + I^*}{K_2} \right) - \frac{\beta(u_3 - 1)P^* L_1}{N} + \frac{\beta(u_3 - 1)P^* L_2}{N} \\
\frac{dL_8}{dt} &= hL_8 + B^* L_5 \left(\frac{(u_1 + a)}{A^* + 1} - \frac{(u_1 + a)A^*}{(A^* + 1)^2} \right) + M^* L_3 \left(\frac{(u_1 + a)}{A^* + 1} - \frac{(u_1 + a)A^*}{(A^* + 1)^2} \right) + I^* L_7 \left(\frac{(u_2 + c)}{A^* + 1} - \frac{(u_2 + c)A^*}{(A^* + 1)^2} \right) \\
&\quad + S^* L_4 \left(\frac{(u_1 + a)}{A^* + 1} - \frac{(u_1 + a)A^*}{(A^* + 1)^2} \right) + V^* L_6 \left(\frac{(u_2 + c)}{A^* + 1} - \frac{(u_2 + c)A^*}{(A^* + 1)^2} \right)
\end{aligned} \tag{27}$$

with transversality conditions (or final time conditions)

$$L_1(T) = 0, L_2(T) = 0, L_3(T) = 0, L_4(T) = 0, L_5(T) = 0, L_6(T) = 0, L_7(T) = 0 \text{ and } L_8(T) = 0$$

The characterisations of the optimal controls $u^*(t)$ and corresponding $u_1^*(t), u_2^*(t), u_3^*(t)$ that is, the optimality equations, are based on the conditions:

$$\frac{\partial H}{\partial u_1} = \frac{\partial H}{\partial u_2} = \frac{\partial H}{\partial u_3} = 0$$

Thus, using the bounds of the control $u_i(t); i=1,2,3$, its optimal control is given by

$$u_1^* = \max \left\{ 1, \min \left\{ 0, \frac{\left(\frac{L_5 A^* B^*}{A^* + 1} + \frac{L_3 A^* M^*}{A^* + 1} + \frac{L_4 A^* S^*}{A^* + 1} \right)}{G_1} \right\} \right\} \tag{28}$$

$$u_2^* = \max \left\{ 1, \min \left\{ 0, \frac{\left(\frac{L_7 A^* I^*}{A^* + 1} + \frac{L_6 A^* V^*}{A^* + 1} \right)}{G_2} \right\} \right\} \quad (29)$$

$$u_3^* = \max \left\{ 1, \min \left\{ 0, \frac{\left(\begin{aligned} & -L_8 f + L_4 \left(\frac{\mu(S^* + B^* \psi) E^*}{N} \right) + \left(\frac{\eta \tau (M^* + B^* \rho) S^*}{N} \right) - L_2 \left(\frac{\eta (M^* + B^* \rho) E^*}{N} \right) - \left(\frac{\mu (S^* + B^* \psi) E^*}{N} \right) \\ & + \frac{\beta P^* I^*}{N} + L_5 \left(\frac{\eta \tau (M^* + B^* \rho) S^*}{N} \right) - \left(\frac{\mu \omega (S^* + B^* \psi) M^*}{N} \right) - \frac{\beta L_1 P^* I^*}{N} + \left(\frac{\eta L_3 (M^* + B^* \rho) E^*}{N} \right) \end{aligned} \right)}{G_3} \right\} \right\} \quad (30)$$

The optimality system consists of the state system (18) coupled with the adjoint system with initial and transversal conditions and the characterisation of optimal control. It can be noticed that

$$\frac{\partial^2 H}{\partial u_1^2} = G_1 > 0, \quad \frac{\partial^2 H}{\partial u_2^2} = G_2 > 0, \quad \frac{\partial^2 H}{\partial u_3^2} = G_3 > 0 \quad (31)$$

which implies that the second partial derivative of the Hamiltonian H with respect to controls u_1, u_2 and u_3 are positive. Therefore, the optimal problem is minimum at controls u_1, u_2 , and u_3 .

5. NUMERICAL SIMULATIONS AND DISCUSSION

5.1 Parameter estimation of CVD co-infection model

This section presents the numerical analysis using the data obtained from ten district councils in two regions (Mwanza and Geita) in Tanzania for 2014-2018, as summarised in Tables 1 and 2. The maximum likelihood estimation (MLE) approach was used to estimate parameters and actual data of infected CMD and CBSD plant cases from Magu, Kwimba, Misungwi, Sengerema, Ukerewe, Chato, Geita rural, Geita Town, Mbogwe and Nyang'hwale districts councils were used.

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Table 1: Average CMD infected plants cases per hectare per district council for 2014-2018

District Council	Year				
	2014	2015	2016	2017	2018
Magu	3100	3238	3100	3322	3319
Kwimba	3238	3315	3097	3168	3017
Misungwi	3295	3018	2937	3044	3105
Sengerema	3274	3323	2840	3014	3182
Ukerewe	3183	3127	3287	3306	3418
Geita Town	3134	3283	2907	2851	3129
Geita	3319	3144	3242	3363	2837
Chato	3218	2828	3428	3043	3071
Nyang'hwale	3167	3039	3115	3228	3055
Bukombe	3152	2962	3148	3313	3273

Table 2: Average CBSD infected plants cases per hectare per district council for 2014-2018

District Councils	Year				
	2014	2015	2016	2017	2018
Magu	2190	2215	2199	2314	2288
Kwimba	2094	2292	2073	2241	2416
Misungwi	2149	2082	2132	2245	2342
Sengerema	2227	2223	2028	2317	2131
Ukerewe	2331	2443	2451	2263	2319
Geita Town	2035	2124	2088	2259	2252
Geita	2227	2123	2328	2262	2128
Chato	2313	2195	2071	2228	2326
Nyang'hwale	2196	2139	2045	2157	2291
Bukombe	2315	2221	2239	2418	2491

The maximum-likelihood estimator (MLE)

The maximum likelihood estimation (MLE) was used to estimate the model's parameters. The goal of the maximum likelihood technique is to maximise the likelihood function, and we do so in this paper by minimising the sum of squares of residuals (SSR), which defined as

$$L(\theta) = \sum_{i=1}^N (y_i - y_i^{est})^2$$

where $\{y_i\}_{i=1}^N$ is the actual data, and $\{y_i^{est}\}_{i=1}^N$ is the solution of model equations (1) at a given parameter value. Some of the initial parameter values were derived from a variety of sources in the literature, such as Holt et al. [39], Kinene et al. [11], Magoyo et al. [40], Chapwanya et al. [41] and Jittama et al., [42], while others were assumed based on the environmental implications. The numerical results for MLE for the CVD co-infection model parameters are summarised in Table 3.

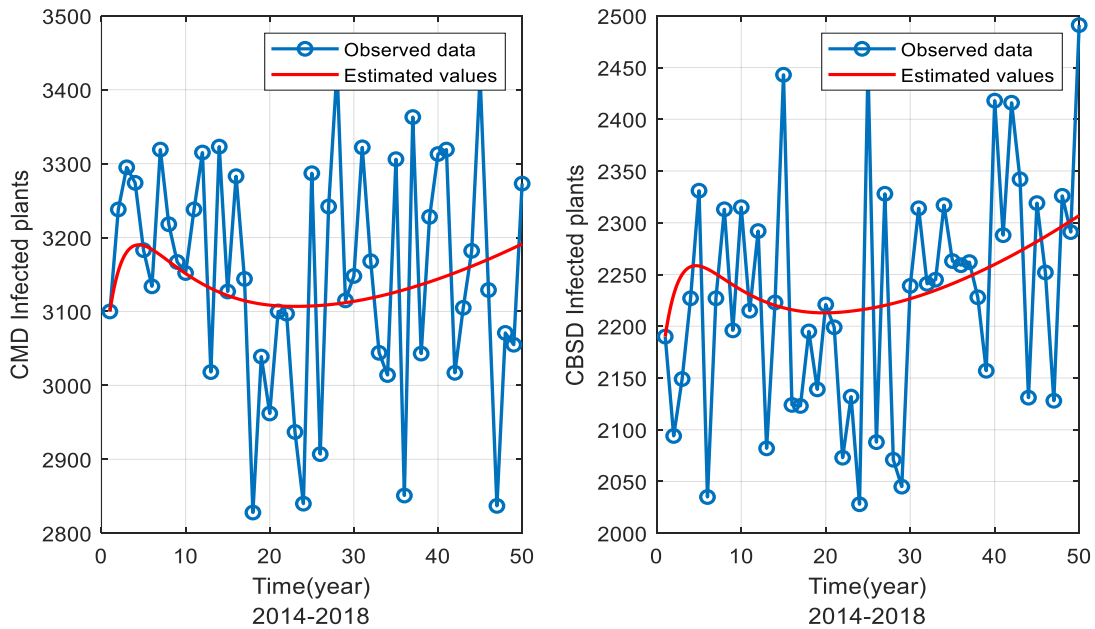


Figure2. *The Average CMD and CBSD infected plant cases per hectare per district council for 2014-2018 and the best-fitted solution of the model (1).*

Table 3: Parameter values that give the best fit to the data in the model

Parameter	Literature value	Estimated Parameter value	Parameter	Literature value	Estimated Parameter value
K_1	10000	10000	a	0.022	0.00899444
K_2	2510	3600	c	0.025	0.024362
r	0.07	0.0768663	f	0.001	0.00128288
δ	0.0024	0.0026751	h	0.024	0.0223494
β	0.024	0.0216701	g	0.05	0.0418727
α	0.042	0.051725	ψ	0.0029	0.00735984
θ	0.032	0.0312163	b	0.192	0.229256
Λ	0.62	0.607043	ϕ	0.82	0.279803
γ	0.04	0.0337999	η	0.081	0.0559998
ω	0.06	0.0430805	μ	0.06	0.0575963
τ	0.035	0.0321168	ρ	0.0062	0.00809025

5.2 Sensitivity Analysis of Model Parameters

Sensitivity analysis was obtained on the fundamental parameters to check and identify parameters influencing the basic reproductive number. We used the approach specified by Blower, S. M. and Dowlatabadi, H. [43] to conduct sensitivity analysis, as described in [44]. The sensitivity indices are given as

$$\Psi_p^{\mathfrak{R}_0} = \frac{\partial \mathfrak{R}_0}{\partial p} \times \frac{p}{\mathfrak{R}_0} \quad \text{where } p \text{ is the model parameter.} \quad (29)$$

The sensitivity indices of the model are presented in figure 3 below

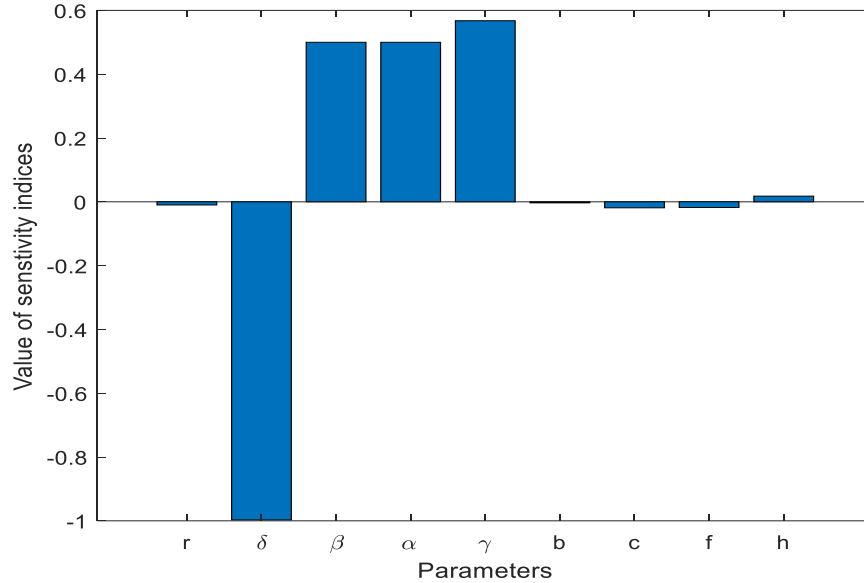


Figure 3: Sensitivity analysis of \mathcal{R}_0 with respect to each model parameter

5.3 Impact of Farmer's Awareness toward cassava virus disease

The impact of the farmers' awareness is evaluated through model (1) with the variables a : a maximum activity rate in diseased plants and c a maximum activity rate in infected whitefly vectors. Whereby the graphs of the variables against time with different values of farmers' activity rate due to the farmers' awareness of infected cassava plants and infected whitefly vector populations. For example, Figure 4(a)- (c) shows that CMD, CBSD and CVD co-infection incidence in the cassava plants population decrease with an increase in farmers' awareness activities on the infected plants due to the farmers' awareness. On the other hand, the number of infective whitefly vectors initially decreases with time due to the small number of infected plants that reduce is associated with the increase of the farmers' awareness activities in infected plants, as displayed in figure 4(d).

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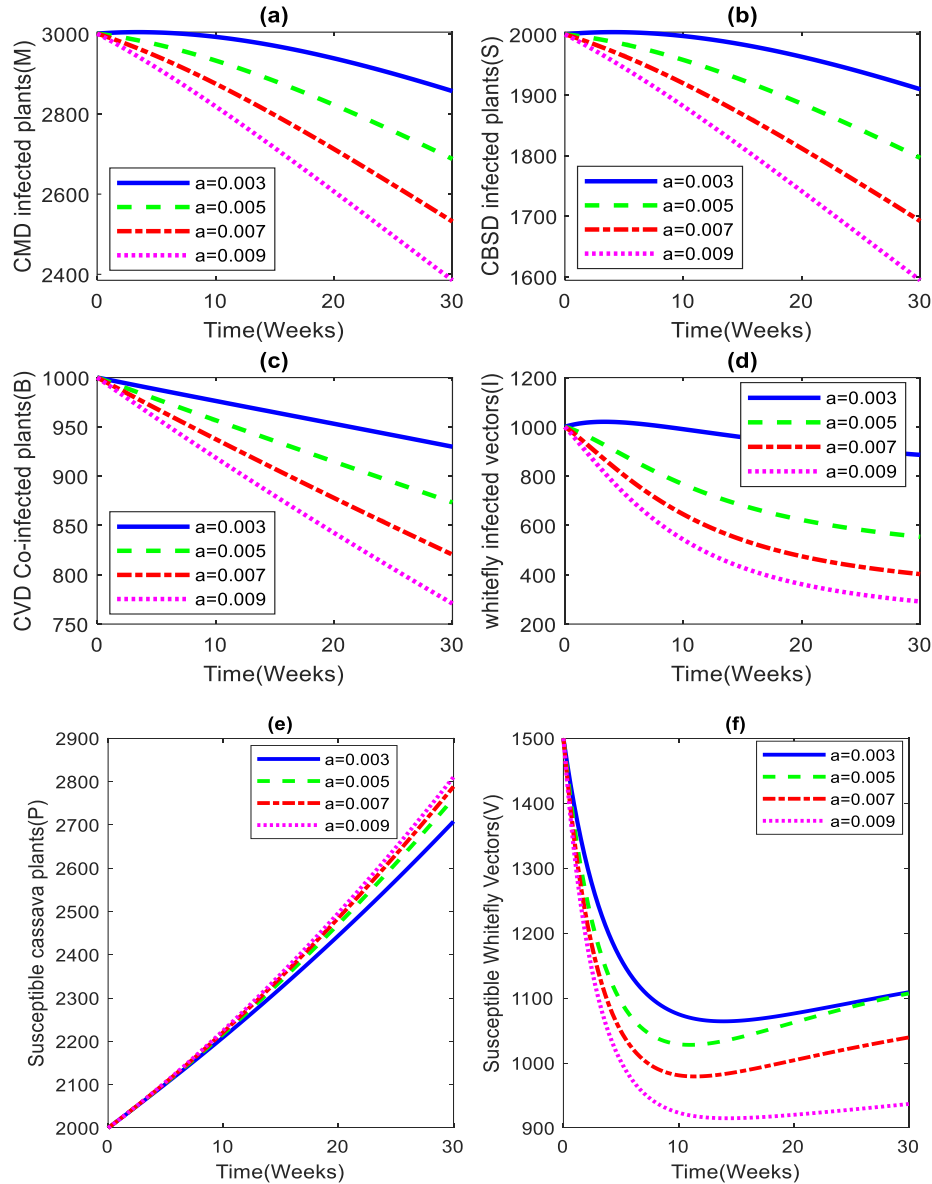


Figure 4. The impact of different values of the farmers' activity rate a in infected cassava plants due to Farmer's awareness of (a)CMD, (b) CBSD, (c)CVD co-infection, (d) infected whitefly vectors, (e)susceptible plants and (f) susceptible whitefly vectors populations with respect to time. Furthermore, figure 4 (e) shows that the susceptible cassava plants subpopulation increases slowly as the Farmer's activities increases due to the Farmer's awareness level. On the other hand, figure 4(f) shows that the infective whitefly population decreases with time due to the Farmer's awareness activities associated with the Farmer's awareness level. Similarly, from figure 5(a)- (c), we see that CMD, CBSD CVD co-infection in the cassava population decreases as the Farmer's activity on

infected whitefly vectors increases due to Farmer's awareness. The same applied to the infected whitefly vectors is reduced as the Farmer's activities increase due to the Farmer's awareness level, as depicted in figure 5(d).

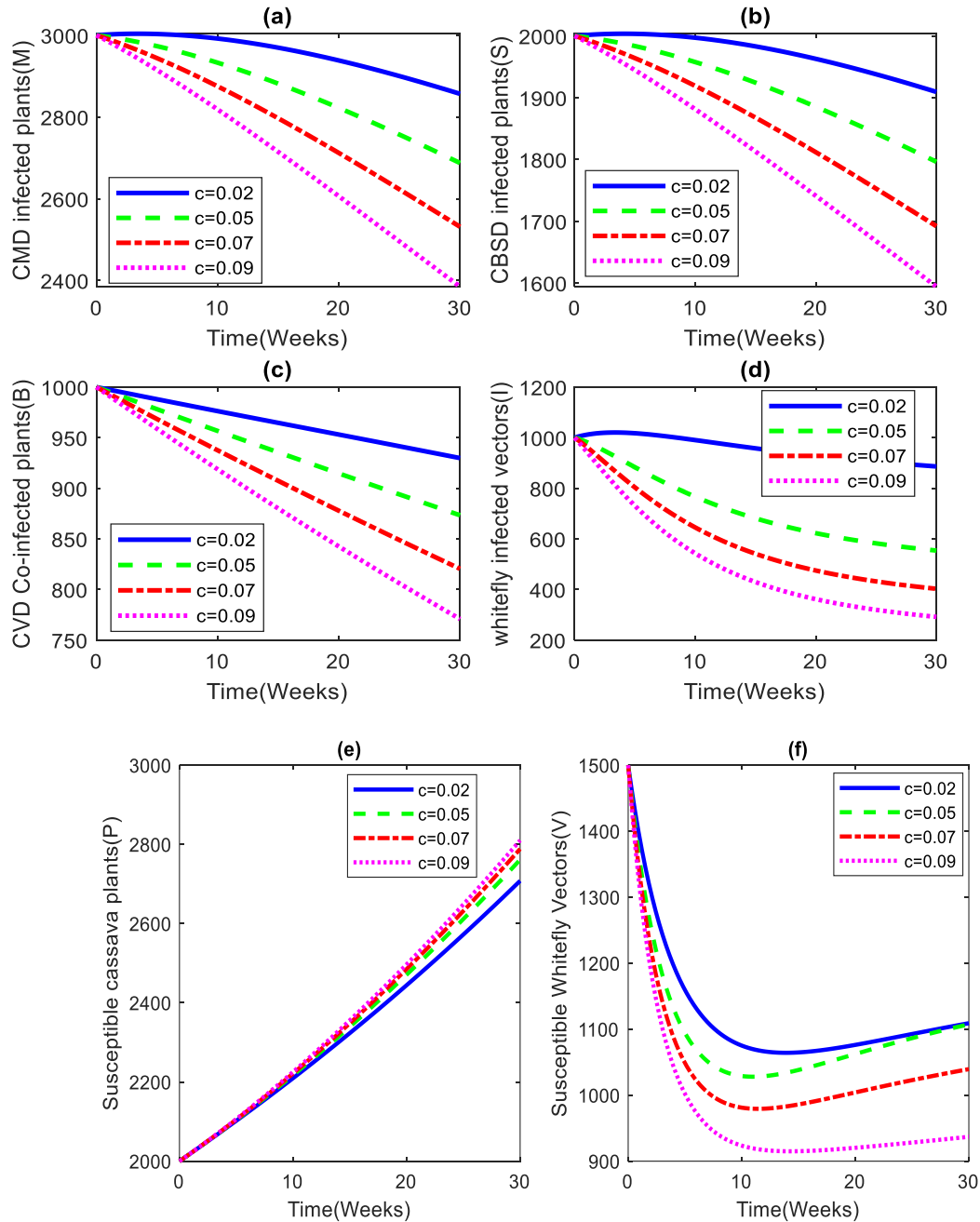


Figure 5. The impact of different values of the farmers' activity rate c in infected whitefly vectors due to Farmer's awareness of (a)CMD, (b) CBSD, (c)CVD co-infection, (d) infected whitefly vectors, (e)susceptible plants and (f) susceptible whitefly vectors populations with respect to time.

Moreover, figure 5(e) shows that farmers' activities on infected whitefly vectors increase the number of susceptible cassava plants due to farmers' awareness. In addition, figure 5(f) illustrates that farmers' activities due to awareness of infected whitefly vectors significantly contribute to reducing or eliminating susceptible whitefly vectors.

5.2 Control Scenarios

The following four combination optimal control strategies were considered and evaluated for their impact on the eradication of CMD, CBSD and CVD-coinfection:

Strategy A: Control by uprooting and burning, and spraying pesticides ($u_1 \neq 0, u_2 \neq 0, u_3 = 0$).

Strategy B: Control with uprooting and burning and farming awareness ($u_1 \neq 0, u_2 = 0, u_3 \neq 0$).

Strategy C: Control by spraying pesticides and farming awareness ($u_1 = 0, u_2 \neq 0, u_3 \neq 0$).

Strategy D: Control with all three controls: uprooting and burning, spraying pesticides and farming awareness ($u_1 \neq 0, u_2 \neq 0, u_3 \neq 0$).

The parameter used in the simulation is shown in Table 3. However, the following initial values were employed to model optimal control: $P(0) = 2000$, $E(0) = 1000$, $M(0) = 3000$, $S(0) = 2000$, $B(0) = 1000$, $V(0) = 1500$ and $I(0) = 1000$. Furthermore, the coefficients of the state variables and controls are $W_1 = 0.2$, $W_2 = 0.4$, $W_3 = 0.7$, $W_4 = 0.3$, $G_1 = 10000$, $G_2 = 3000$ and $G_3 = 5000$. The weight values used in the simulations are entirely theoretical, chosen randomly to illustrate the control mechanisms suggested in this paper.

Strategy A: Control by uprooting and burning, and spraying pesticides ($u_1 \neq 0, u_2 \neq 0, u_3 = 0$)

Figures 6(a) and 6(b) show that the number of CMD and CBSD infectious plants decreases when applying strategy D. Furthermore, Figure 6(c) indicates that the CVD co-infectious plants reduced suggestively with the implementation of the same technique. Figure 6(d) shows that implementing this strategy affects infected whitefly vectors. This is because the number of infected whitefly vectors tends to decrease with this strategy. This approach significantly reduces the number of

CMD, CBSD, and CVD co-infectious populations and infected whitefly vectors. However, additional control measures are required to limit the disease.

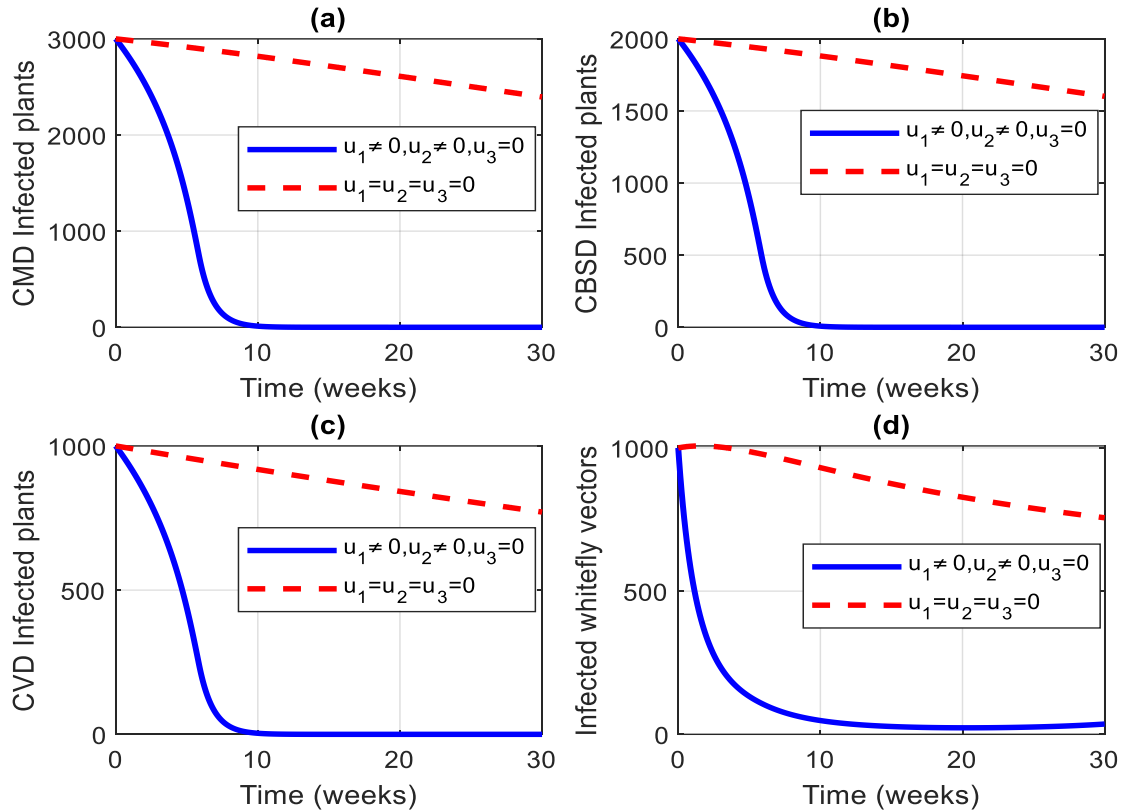


Figure 6: *Impacts of uprooting and burning, and spraying pesticides controls on CVD co-infection transmission dynamics.*

Strategy B: Control by uprooting and burning and farming awareness ($u_1 \neq 0, u_2 = 0, u_3 \neq 0$)

Figures 7(a)-(c) show a marked decrease in the number of CMD, CBSD and CVD co-infectious cassava plant populations at any given moment. Furthermore, Figure 7(d) indicates that applying the strategy is also more helpful in reducing the population of infected whitefly vectors from the field. Finally, it should be noted that uprooting and burning diseased cassava plants along with farmer awareness campaigns greatly reduce infections by minimising the transmission of the virus to another plant through whitefly vectors.

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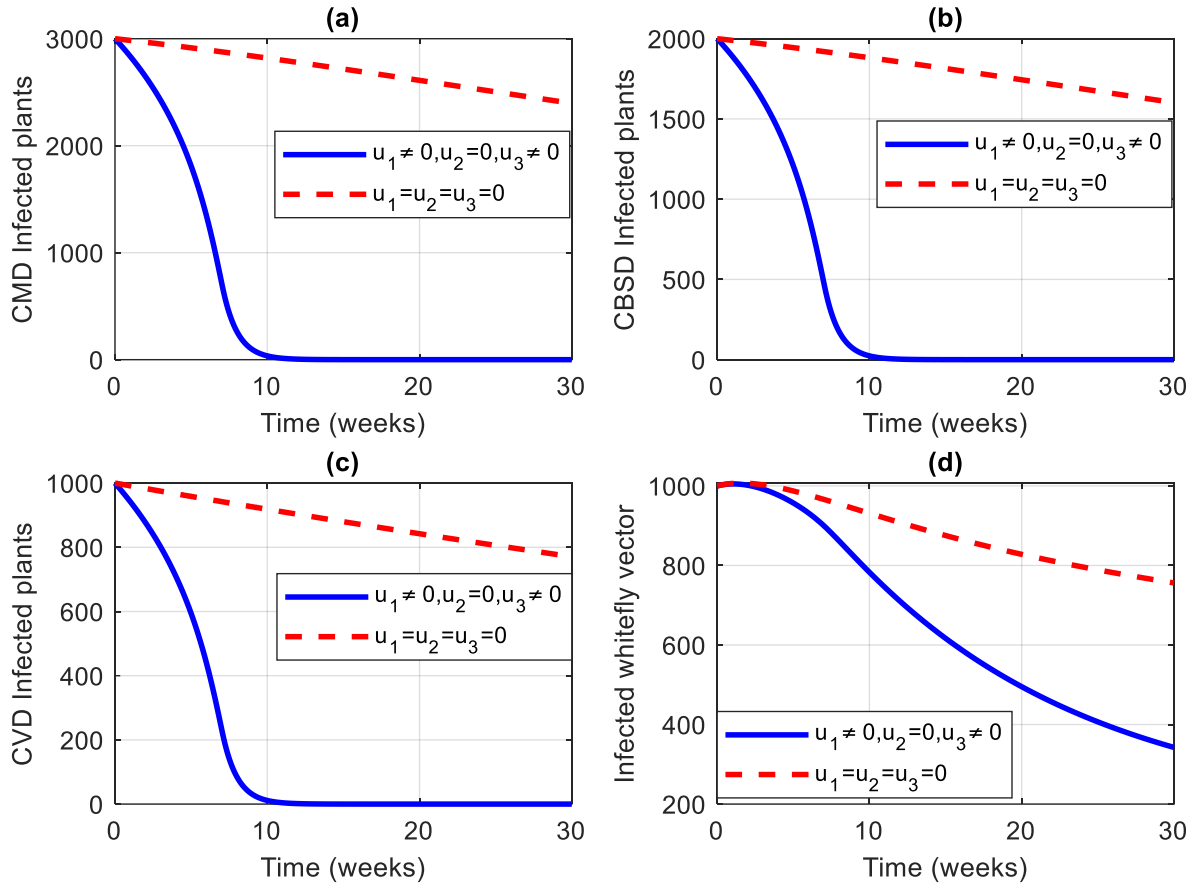


Figure 7: *Impacts of uprooting and burning and farming awareness controls on CVD co-infection transmission dynamics.*

Strategy C: Control by spraying pesticides and farming awareness ($u_1 = 0, u_2 \neq 0, u_3 \neq 0$)

It can be noted from Figure 8(a)-(c) that there is no decrease in the number of CMD, CBSD and CVD co-infectious, respectively, as a result of the spraying pesticides and farming awareness application. It means that spraying pesticides and farming awareness measures are not intended to remove infected cassava plants within the field. However, from Figure 8(d), applying such efforts may reduce the infected whitefly vectors from the environment. Therefore, other strategies targeting CMD, CBSD, and CVD co-infectious populations should be attempted to eradicate these diseases.

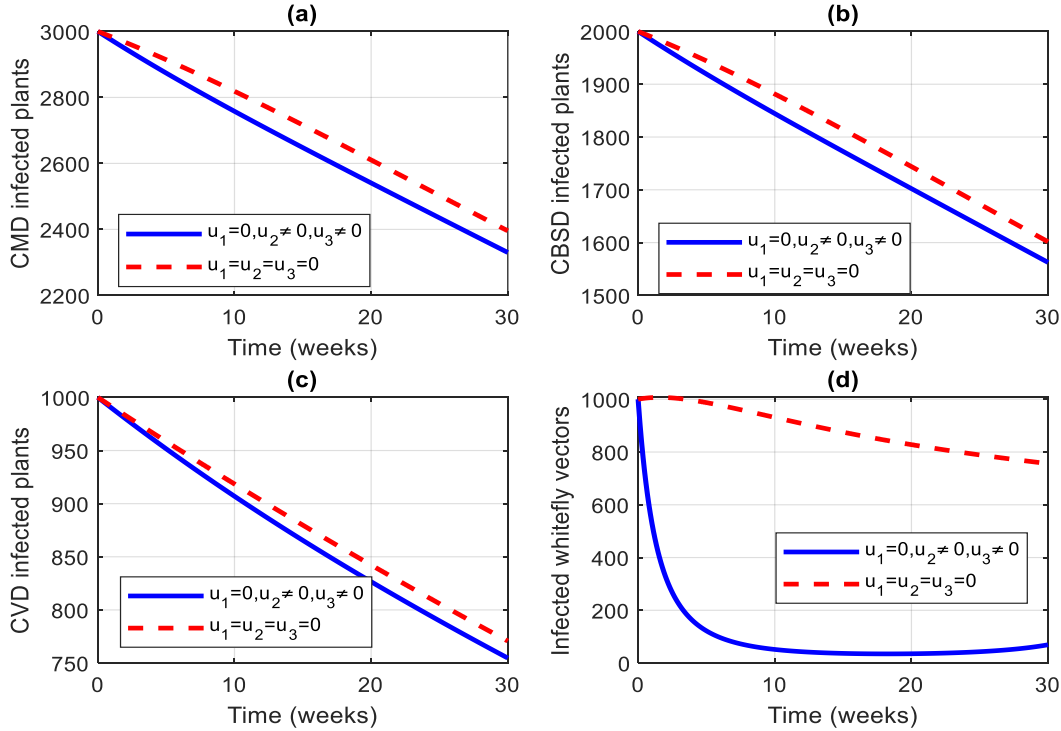


Figure 8: *Impacts of spraying pesticides and farming awareness controls on CVD-coinfection transmission dynamics.*

Strategy D: Control by uprooting and burning, spraying pesticides and farming awareness
 $(u_1 \neq 0, u_2 \neq 0, u_3 \neq 0)$

The results show that uprooting and burning infected cassava plants in the system, spraying pesticides and farming awareness will reduce the spread of the disease. We can observe this tendency from 9(a)-(c), which displays that the CMD, CBSD and CVD co-infected cassava decreases by intensifying the removal of the infected plants and the infected whitefly vector decreases by strengthening pesticides application due to the increase of farmers awareness. Moreover, Figure 9(d) shows that the infected whitefly vector decreases when applied control. The combination of strategies u_1, u_2 and u_3 gives the best results to optimise the objective function (J) .

CO-DYNAMICS OF CASSAVA VIRUS DISEASE

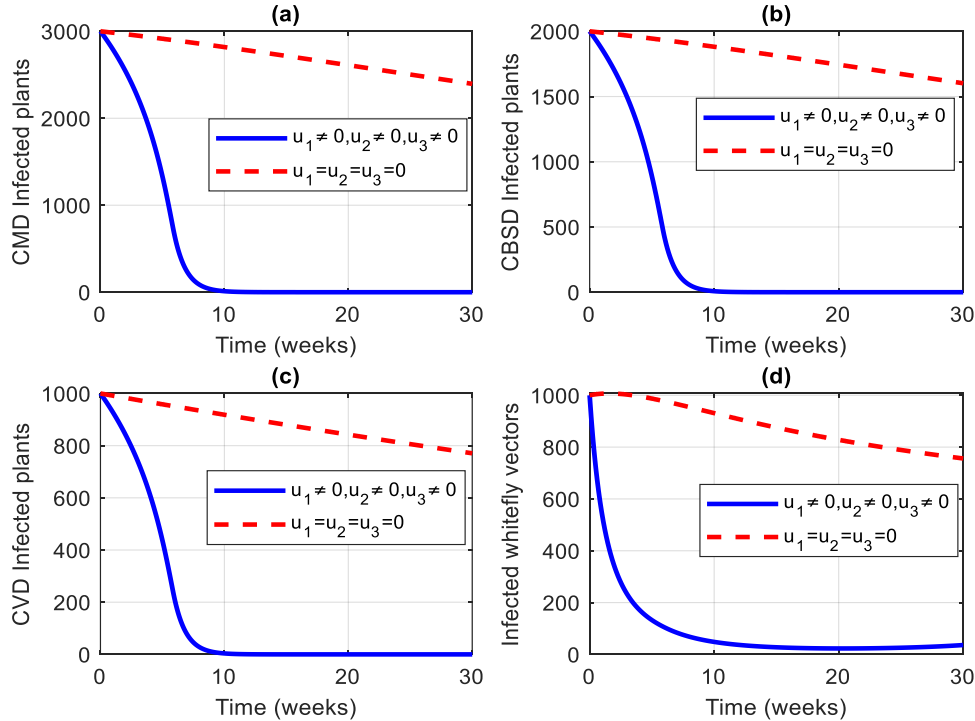


Figure 9: Impacts of uprooting and burning, spraying pesticides and farming awareness controls on CVD-coinfection transmission dynamics.

6. COST-EFFECTIVENESS ANALYSIS

The Incremental Cost-Effectiveness Ratio (ICER), as suggested by Kinene et al. [11], is a more formal approach to evaluating the cost-effectiveness of the strategies. The ICER compares two competing alternative strategies using limited resources and limited resources. When comparing two intervention strategies in ICER, one strategy should be compared incrementally with the next-less-effective alternative. It is termed additional costs per additional health outcome. In other words, ICER can be defined as the ratio of the cost difference between two strategies to the averted difference between the total number of infections. That is,

$$ICER(X) = \frac{\text{Cost of intervention } X - \text{Cost of Intervention } Y}{\text{Effect of intervention } X - \text{Effect of Intervention } Y} = \frac{\Delta C_T}{\Delta E} \quad (46)$$

where X and Y are the two intervention strategies compared. The incremental cost is denoted by ΔC_T while the incremental effect is represented by ΔE . Furthermore, C_T it indicates the total costs incurred by implementing a specific strategy, whereas E it means the effectiveness of a

particular strategy. Finally, the total number of infections averted (E) is estimated for each strategy by subtracting total infections with control from those without control.

An ICER is calculated based on the strategies: A, B, C and D. Parameter values from Table 3 were used to estimate the total cost and infections averted presented in Table 4. For example, consider strategy A: where the estimated total number of infections is 713610. Furthermore, the total number of infections was calculated as 3901500 when there was no control strategy. To attain the total number of averted infections for Strategy A, deduct the total number of infections when there was no control strategy (status quo) from the number of infections when strategy A was considered, i.e. $3901500 - 713610 = 3187890$. Therefore, for Strategy A, the number of averted infections is 3187890, and the total cost is \$17912, whereas the cost of a status quo strategy is \$0.

Table 4: Number of infections averted and total cost of each strategy.

Strategies	Infections	Infection averted (E)	Cost (\$) (C_T)
	3901500		0
A	713610	3187890	17912.00
B	1120400	2781100	15553.00
C	3602600	298900	2406.40
D	1105500	2796000	15913.00

First, reorder the control strategies in Table 4 in increasing order of effectiveness (E). Next, compute incremental effectiveness (ΔE) and incremental cost (ΔC_T). Finally, the ICER is calculated by dividing incremental costs (ΔC_T) by incremental effectiveness (ΔE).

$$\text{ICER}(C) = \frac{2406.4}{298900} = 0.008051$$

$$\text{ICER}(B) = \frac{13146.6}{2482200} = 0.005296$$

Table 5: Incremental cost-effectiveness ratios of different optimal control strategies.

Strategies	E	ΔE	C_T	ΔC_T	ICER($\Delta C_T / \Delta E$)
C	298900	298900	2406.4	2406.4	0.008051
B	2781100	2482200	15553	13146.6	0.005296
D	2796000	14900	15913	360	0.024161
A	3187890	391890	17912	1999	0.005101

Comparing strategies C and B, strategy B's ICER is less than strategy C's ICER. Therefore, strategy C is more costly and less effective than strategy B. Thus, strategy C is excluded from alternatives, and ICER is recalculated for the remaining strategies. With Strategy C dropped, Table 5 deduced the ICER, which was determined as

$$\text{ICER(B)} = \frac{15553}{2781100} = 0.005592$$

$$\text{ICER(D)} = \frac{360}{14900} = 0.024161$$

Table 6: Incremental cost-effectiveness ratios of different optimal control strategies excluding strategy C.

Strategies	E	ΔE	C_T	ΔC_T	ICER($\Delta C_T / \Delta E$)
B	2781100	2781100	15553	15553	0.005592
D	2796000	14900	15913	360	0.024161
A	3187890	391890	17912	1999	0.005101

Also, this comparison shows that Strategy B is cheaper than Strategy D. Strategy D is thus excluded, and proceeds to compare Strategy B to Strategy A.

From Table 7, one attains

$$\text{ICER(B)} = \frac{15553}{2781100} = 0.005592$$

$$\text{ICER(A)} = \frac{2359}{406790} = 0.005799$$

Table 7: Incremental cost-effectiveness ratios for optimal control strategies B and A.

Strategies	E	ΔE	C_T	ΔC_T	ICER ($\Delta C_T / \Delta E$)
B	2781100	2781100	15553	15553	0.005592
A	3187890	406790	17912	2359	0.005799

Finally, the findings show that strategy B is less expensive than strategy A. Strategy B (uprooting and burning infected plants and a farmer's awareness campaign) is the best of all possible strategies due to its cost-effectiveness and health benefits.

7. CONCLUSION

This work develops and discusses a mathematical model tracing the impact of farming awareness on CVD co-infection. In the first place, basic properties of the model, such as boundedness of the biological feasibility and positivity of the solution of the model, proved that all variables that enter and remain feasible in the invariant region were positive. Then, the basic reproduction number is computed with respect to the CVD co-infection-free equilibrium point using the next-generation matrix approach. The Jacobian and Metzler matrices were used to check CVD co-infection-free equilibrium local and global stability. The model's CVD co-infection-free equilibrium points are locally and globally asymptotically stable if the basic reproduction number is less than one. The sensitivity analysis of the basic reproduction numbers was obtained, and their biological interpretation was provided. The effect of the farming awareness level in the CVD co-infection model was performed numerically. Then the later mathematical model was modified to incorporate three time-dependent control to obtain an optimal control problem. The existence of optimal control is proved and later analysed. Finally, the optimality system is numerically solved, and its results are presented. The findings suggest that a strategy that includes a combination of two controls (uprooting and burning and a farmer's awareness campaign) is effective and plays a significant role in minimising the epidemic.

Similarly, it was found that any strategy under consideration, combined with awareness campaigns, appeared to be more effective than that ignored awareness. Finally, the cost-effectiveness of the

control strategies was developed and analysed using the ICER method. It was noted that the most cost-effective strategy was integrating the combination of two control efforts uprooting and burning, and an awareness campaign. Thus, uprooting and burning infected plants and Farmer's awareness campaigns about the CVD co-infection is the cost-effective optimal control strategy and is sufficient to combat the epidemic of Cassava virus diseases with limited resources.

CONFLICT OF INTERESTS

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

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