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A MATHEMATICAL MODEL FOR THE DYNAMICS AND COST EFFECTIVENESS OF THE CURRENT CONTROLS OF CASSAVA BROWN STREAK DISEASE IN UGANDA

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Abstract. In this paper, Cassava brown streak disease (CBSD), transmitted from white fly vector to the host plant and vice versa, is a major threat to cassava production in Uganda and other cassava growing countries in Africa, e.g. Kenya, Tanzania, Malawi, Mozambique, e.t.c. The seriousness of the situation is that almost all varieties of cassava resistant to cassava mosaic disease (CMD) are susceptible to the new strain of CBSD. Numerous control measures are practiced by farmers, however, the cost effectiveness of these control measures have not been quantified. Therefore it is imperative that we formulate a mathematical model to investigate the transmission dynamics of CBSD and the cost-effectiveness of the control measures. In the analysis of the model we derived the basic reproduction number which helps us in establishing the stability of disease free and endemic equilibrium points. The model is then modified as an optimal control problem with an aim of minimizing the number of infected plants while keeping the cost low. Two time dependent controls are used in the model and an objective function which is a combination of the actual and relative costs associated with the controls is designed. Pontryagins Maximum Principle (PMP) is used to establish the necessary conditions for optimal control of the disease. The incremental cost-effectiveness ratio (ICER) is also computed and used to analyse the cost-effectiveness of the control strategies. Numerical results show that strategy B (uprooting and burning of infected plants) is cost effective, however if the government intervenes with massive spraying, strategy C (spraying with chemicals and uprooting and burning of infected plants) gives the farmer more yield.

Keywords: CBSD; Stability analysis; Optimal control; Cost-effectiveness.

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1. Introduction

Cassava is a woody shrub plant of the *Euphorbiaceae* (spurge) family originating from South America and is known under various names: *Manihot esculenta, monica, yucca* and *tapioca*. It is the third-largest source of carbohydrates in the tropics after rice and maize [5] and was introduced in East Africa via west Africa [8]. It is mainly grown in 26 districts in Uganda for food and it also serves as a cash crop to some Ugandan farmers. Nigeria is the best producer of cassava in Africa and Uganda is the sixth with current annual production of about 5.5 million tonnes [15]. Cassava production is constrained by many biotic factors of which cassava mosaic disease (CMD) and cassava brown streak disease (CBSD) are the major threats in Uganda.

Cassava brown streak disease (CBSD) is a viral disease currently caused by two virus species: The coastal endemic virus called cassava brown streak virus (CBSV) and the high land endemic virus known as Uganda cassava brown streak virus (UCBSV) [17]. It was named UCBSV by the international committee for taxonomy of viruses in June 2010 to avoid confusion with other gemini-viruses. Both viruses have (+)ssRNA genomes and belong to genus *Ipomovirus* in the family of *potyviridae* [26]. The disease causes rotting of the root which renders cassava tuber inedible for both animals and humans resulting in severe economic loss to farmers in Uganda.

The symptoms of CBSD are elusive, they may not appear until the cassava plant has stayed longer than 9 months. Farmers usually confuse them with those of CMD. According to Dr. Chris Omongo the coordinator of cassava project at National Crop Resources Research Institute (NaCRRI) at Namulonge, the symptoms of CBSD can be observed on leaves, stems and roots and some cassava plant varieties may not show symptoms on the leaves nor tubers, other varieties may only show symptoms on the leaves and not roots while others may not show symptoms on the leaves but on roots only [1]. Leaf symptoms vary greatly depending on altitude, rainfall quality, plant age and the virus species [18] and these include chlorosis and necrosis on the infected leaves which is associated with veins spanning from the mid veins, secondary and tertiary veins. According to Dr. Alicai Titus an expert in molecular virology and tissue culture at NaCRRI, brown streaks and spots appear as scratch like wounds and are prominent on the upper green portion of the stem [1]. A dry corky brown- necrotic rot of the cassava tuber may progress from a small lesion to the whole root and finally the tuber may become constricted

and malformed due to rotting of the root . UCBSD has mild symptoms than CBSD and lower pathogenicity [25]. The infected plant may not show all these symptoms because some of the symptoms may appear and disappear in young plants but appear again in cassava plants at a later stage.

The vector for CBSD is the silverleaf whitefly (*Bemisia tabaci* biotype B) [20]. This whitefly species is also considered to be the vector of CMD. The adult *B. tabaci* lives an average of 16 days and the maturation process from egg to adult is 30 days [9]. The eggs are deposited haphazardly or in spiral fashion on the leaf underside. Both juvenile and adult whiteflies feed on the phloem of the leaves by inserting a sucker mouth part into the leaf thus transmitting the virus to the plant. Saliva containing toxins is also injected into the plant in the process of feeding thus affecting plant growth.

Previously the disease was restricted to low altitude areas less than 1000m above sea level along coastal Kenya to the Mozambique, however CBSD is now accommodated in Mid-altitude levels (1200m-1500m) above sea level as it has been reported in Uganda [18]. In 2009 CBSD outbreaks were most prevalent in south-central Uganda. This was attributed to planting of infected stem cuttings. Currently the incidence is greatest in Uganda where there is CMD resistant cassava varieties [21]. Recent surveys demonstrated that of the 23 districts surveyed in Uganda, 70% had CMD resistant cassava varieties and all are vulnerable to CBSD causing viruses. These varieties hosted as many as 200 adult whiteflies on the top five cassava leaves [18] and this poses a serious threat to food security and income of farmers in Uganda.

According to Katama Doreen [1], CBSD is currently managed by: uprooting and burning infected cassava plants, planting of symptomless cassava, quarantining and scouting the gardens for diseases, planting tolerant varieties. However more research is still ongoing on breeding tolerant/resistant varieties. Some farmers spray with pesticides however, pesticides are not efficient or effective although farmers use them.

2. Formulation of the CBSD model

We considered an SEIR model for the dynamics of the disease in the cassava plants and SI for the dynamics in whitefly vectors. The total cassava population $N(t)$ is subdivided into

the following sub-populations; cassava plants that are susceptible to infection with CBSD $S(t)$, those exposed to CBSV $E(t)$, cassava plants with CBSD symptoms $I(t)$, removed cassava plants due to infection $R(t)$. The total whitefly vector population $N_V(t)$ is sub-divided into susceptible whitefly vector population $S_V(t)$ and infectious whitefly vector population $I_V(t)$. That is $N_V = S_V + I_V$, the transmission dynamics of CBSD is summarised in the compartmental diagram in Figure 1.

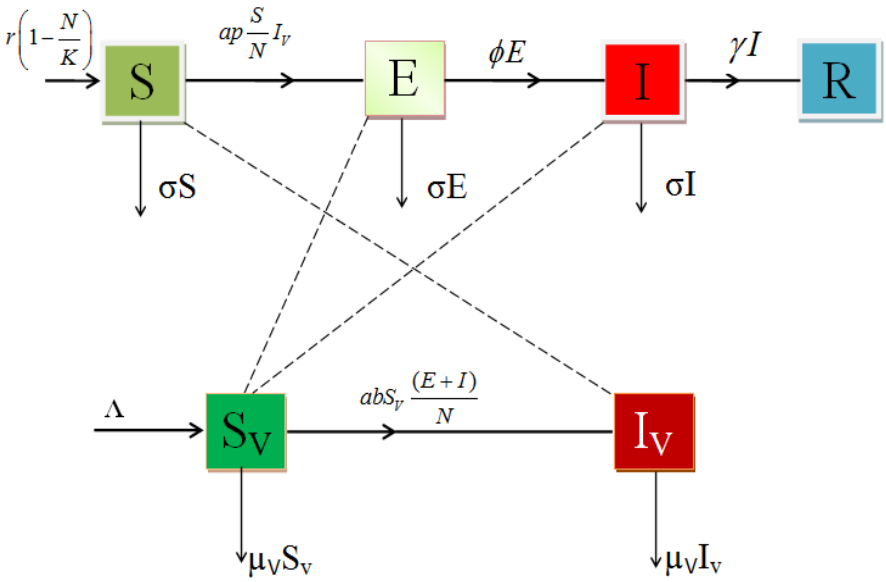

FIGURE 1. Compartmental diagram for the transmission dynamics of CBSD

It is assumed that healthy cassava plants are planted or replanted at a rate r. They are either harvested at a rate σ or move to the exposed class after acquiring CBSV through contact with the infectious whitefly vector at a rate ap, where p is the probability that a healthy plant will be inoculated by the virus during a single visit by an infected whitefly vector, a is the number of plants visited by either an infected whitefly or non infected whitefly per day. The exposed cassava plants are either harvested at a rate σ or move to the infectious class at a rate ϕ. Infectious cassava plants are assumed to be harvested or removed from the garden and burnt at a rate γ.

Susceptible whitefly vectors are recruited at a rate Λ. They either die naturally at a rate μ_V or move to the infectious class after acquiring CBSV from the infected cassava plants at a rate ab, where b is the probability that a non infectious vector will acquire the virus from an infected

cassava plant during a single visit. The infected whitefly vectors also die naturally at a rate μ_V. We assumed that farmers plant only healthy varieties of cassava in a garden of carrying capacity K, no death of cassava plants before harvesting and the vectors are assumed to remain infectious once they acquire the virus.

Putting the above description and assumptions of the dynamics of CBSD together, gives the following host-vector model:

The host equations

$$\begin{aligned}
\frac{dS}{dt} &= r(1-\frac{N}{K}) - ap\frac{S}{N}I_V - \sigma S,\\
\frac{dE}{dt} &= ap\frac{S}{N}I_V - \phi E - \sigma E,\\
\frac{dI}{dt} &= \phi E - \gamma I - \sigma I,\\
\frac{dR}{dt} &= \gamma I. \qquad (1)
\end{aligned}$$

Vector equations

$$\begin{aligned}
\frac{dS_V}{dt} &= \Lambda - abS_V\frac{(E+I)}{N} - \mu_V S_V,\\
\frac{dI_V}{dt} &= abS_V\frac{(E+I)}{N} - \mu_V I_V. \qquad (2)
\end{aligned}$$

For the purpose of this paper, we refer to system of equations (1) and (2) as our model. Since R does not play any role in the dynamics of the disease, we shall ignore it in the analysis of the model, thus we have $N = S+E+I$. The equation of the total population of cassava plants and the total population of the whitefly vectors are:

$$\frac{dN}{dt} = r(1-\frac{N}{K}) - \sigma N - \gamma I, \qquad (3)$$

$$\frac{dN_V}{dt} = \Lambda - \mu_V N_V. \qquad (4)$$

3. Analysis of the CBSD model

3.1. Positivity and boundedness of the solution

For the CBSD model to be epidemiogically meaningful, it is important to show that the state variables are non-negative for all times $t \geq 0$. i.e. the solutions of the model system (1) and (2) with non negative initial data in Ω will remain non-negative for all times.

Theorem 3.1. *The solution sets of the CBSD model with nonnegative initial data in* $\mathbb{R}^5$ *are feasible for all* $t > 0$ *if they enter the invariant region* $\Omega = \Omega_h \times \Omega_V \subset \mathbb{R}^3_+ \times \mathbb{R}^2_+$ *where* $\Omega_h = \{(S(t), E(t), I(t)) \in \mathbb{R}^3_+ : N(t) \leq \frac{rK}{r+\sigma K}\}$ *and* $\Omega_V = \{(S_v(t), I_v(t)) \in \mathbb{R}^2_+ : N_V(t) \leq \frac{\Lambda}{\mu_V}\}$.

Proof. Let $\Omega = (S, E, I, S_V, I_V) \in \mathbb{R}^5_+$ be any solution of the CBSD model system (1) and (2) with non-negative initial conditions. From equation (3) we have

$$\frac{dN}{dt} \leq r(1 - \frac{N}{K}) - \sigma N.$$

Thus

$$N(t) \leq \frac{r}{\theta} + (N_0 - \frac{r}{\theta})e^{-\theta t},$$

where $\theta = \frac{r}{K} + \sigma$, $\frac{r}{\theta} < K$ and $\frac{r}{\theta} = \frac{rK}{r+\sigma K}$ is the equilibrium population of cassava plants. For $N_0 \leq \frac{r}{\theta}$ we have the population of the cassava plants increasing to $\frac{r}{\theta}$ as $t \to \infty$. Then $N_0 \leq N(t) \leq \frac{r}{\theta}$ for all t, Therefore $\Omega_h = \{(S(t), E(t), I(t)) \in \mathbb{R}^3_+ : N_0 \leq N(t) \leq \frac{r}{\theta}\}$. Similarly from equation eqn (4) we have

$$N_V(t) = \frac{\Lambda}{\mu_V} + (N_V(0) - \frac{\Lambda}{\mu_V})e^{-\mu_V t}.$$

When $N_V(0) \geq \frac{\Lambda}{\mu_V}$, the population of the whitefly vector $N_V(t)$ reduces to the equilibrium $\frac{\Lambda}{\mu_V}$ as $t \to \infty$. Then $\frac{\Lambda}{\mu_V} \leq N_V(t) \leq N_V(0)$ for all t. For $N_V(0) \leq \frac{\Lambda}{\mu_V}$, the whitefly vector population $N_V(t)$ increases to $\frac{\Lambda}{\mu_V}$ as $t \to \infty$. Then $N_V(0) \leq N_V(t) \leq \frac{\Lambda}{\mu_V}$ for all t. Generally the feasible solution set of the whitefly vector population enters the region $\Omega_V = \{(S_v(t), I_v(t)) \in \mathbb{R}^2_+ : N_V(0) \leq N_V(t) \leq \frac{\Lambda}{\mu_V}\}$. Therefore the feasible solutions set for the CBSD model given by $\Omega = \Omega_h \times \Omega_V$ is positively invariant, epidemiologically meaningful and mathematically well-posed in the domain Ω according to Hethcote (2000) [7]. Therefore it is sufficient to consider the dynamics of the flow generated by the model system (1) and (2) in Ω, thus every solution of the model system (1) and (2) with initial conditions in Ω remain in Ω for all $t > 0$.

3.2. Existence and stability of equilibrium points

Since the whitefly vector recruitment term Λ and the cassava planting or replanting rate r are not zero, the population will not be extinct. This implies that there is no trivial equilibrium point, thus $E_0(S^*,E^*,I^*,S_V^*,I_V^*) \neq (0,0,0,0,0)$. We find equilibrium points by setting the right-hand sides of system model equations (1) and (2) equal to zero.

3.2.1. Disease free equilibrium point (E_0)

This is a steady state solution where we assume that there is no CBSD in plant population and no CBSV in the whitefly vector population. In absence of the disease we assume that $(E = I = I_V = 0)$ and $N = S$. Therefore the disease free equilibrium point is given by:

$$E_0(S^*,E^*,I^*,S_V^*,I_V^*) = (\frac{rK}{r+\sigma K},0,0,\frac{\Lambda}{\mu_V},0). \tag{5}$$

3.2.2. The basic reproduction number R_0

To analyze the stability of the disease free equilibrium point, we compute the basic reproduction number R_0 for the model. The basic reproduction number R_0, is defined as the total number of infections arising from one newly infected individual introduced into a health population. We calculate the basic reproduction number R_0 of the system by applying the next generation operator approach as laid out by Driessche & Watmough (2002) [4]. We obtain.

$$R_0 = \sqrt{ap(\frac{1}{K}+\frac{\sigma}{r})\frac{(\gamma+\sigma+\phi)}{(\phi+\sigma)(\gamma+\sigma)}(\frac{ab\Lambda}{\mu_V^2})}. \tag{6}$$

R_0 is a threshold parameter that represents the average number of infected vectors and infected hosts caused by a cross-infection of one cassava plant host or one whitefly vector when the other population consist of only susceptibles [4]. The square root arises from the fact that two generations are required for transmission of CBSD to take place, i.e. From an infectious cassava plant to a susceptible whitefly vector and then from an infectious whitefly vector to susceptible cassava plant(host). The square on a results from the two cycles of feeding/bitting necessary for an infection to occur. We interpret the terms in the basic reproduction number epidemiologically as follow;

- The term $\frac{1}{\phi+\sigma}$ is the average length of time a cassava plant spends while exposed to the infection during its lifetime.

- The term $\frac{1}{\gamma+\sigma}$ is the average length of time the cassava plant spends while infected during its life time.
- The term $\frac{1}{\mu_V}$ is the average length of time a vector spends while infected during its life time.
- $\frac{ap}{\mu_v}$ is expected number of new infections in the exposed cassava class produced by the infected vector originally introduced into compartment I_V.
- The term $\frac{ab\Lambda(r+\sigma K(\gamma+\sigma+\phi))}{\mu_V Kr(\phi+\sigma)(\gamma+\sigma)}$ is the expected number of new infections in infected vector population (compartment I_V) produced by the exposed cassava originally introduced in the exposed class (compartment E).

3.3. Local stability of the disease free equilibrium point

The local stability of the disease free equilibrium can be analyzed by linearizing the system using the Jacobian matrix of the model at the disease free equilibrium point. The local stability is then determined basing on the signs of the eigenvalues of the Jacobian. The equilibrium E_0 is locally asymptotically stable if the real part of the eigenvalues are all negative.

Theorem 3.2. *The disease free equilibrium E_0 is locally asymptotically stable in Ω if $R_0 < 1$ but unstable if $R_0 > 1$, where R_0 is the basic reproduction number.*

Proof. We linearize the system (1)-(2) using the Jacobian technique. At the disease free equilibrium point E_0, the jacobian matrix is

$$J(E_0) = \begin{pmatrix} \frac{r}{K}-\sigma & -\frac{r}{k} & -\frac{r}{k} & 0 & -ap \\ 0 & -(\phi+\sigma) & 0 & 0 & ap \\ 0 & \phi & -(\gamma+\sigma) & 0 & 0 \\ 0 & -\frac{ab\Lambda}{\mu_V N^*} & -\frac{ab\Lambda}{\mu_V N^*} & -\mu_V & 0 \\ 0 & \frac{ab\Lambda}{\mu_V N^*} & \frac{ab\Lambda}{\mu_V N^*} & 0 & -\mu_V \end{pmatrix}. \tag{7}$$

It is clear from equation (7) that the first and second eigenvalues are $\lambda_1 = \frac{r}{K} - \sigma$ and $\lambda_2 = -\mu_V$. Therefore the matrix (7) reduces to a 3×3 matrix shown in matrix (8).

$$J(E_0) = \begin{pmatrix} -(\phi+\sigma) & 0 & ap) \\ \phi & -(\gamma+\sigma) & 0 \\ \frac{ab\Lambda}{\mu_V N^*} & \frac{ab\Lambda}{\mu_V N^*} & -\mu_V \end{pmatrix}. \tag{8}$$

The characteristic equation of (8) is found from $det(J(E_0) - \lambda I) = 0$, that is

$$\lambda^3 + \lambda^2(\gamma+2\sigma+\phi+\mu_V) + \lambda\left((\gamma+2\sigma+\phi)\mu_V + (\gamma+\sigma)(\phi+\sigma) - \frac{a^2bp\Lambda(r+\sigma K)}{\mu_V rK}\right)$$
$$+(\gamma+\sigma)(\phi+\sigma)\mu_V - \left(\frac{a^2bp\Lambda(r+\sigma K)}{\mu_V rK}\right)(\phi+\gamma+\sigma) = 0. \tag{9}$$

From Routh-Hurwitz criteria, with a polynomial of degree three i.e $\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0$, the necessary and sufficient condition for local stability of the system is, that all eigenvalues must have negative real part. Therefore the following conditions must hold; $a_1 > 0$, $a_2 > 0$, $a_3 > 0$ and $a_1a_2 > a_3$. From the polynomial in equation (9) we have:

$$\begin{aligned} a_1 &= (\gamma+2\sigma+\phi+\mu_V). \\ a_2 &= (\gamma+2\sigma+\phi)\mu_V + (\gamma+\sigma)(\phi+\sigma) - (\frac{a^2bp\Lambda(r+\sigma K)}{\mu_V rK}). \\ a_3 &= (\gamma+\sigma)(\phi+\sigma)\mu_V - (\frac{a^2bp\Lambda(r+\sigma K)}{\mu_V rK})(\gamma+\sigma+\phi). \end{aligned}$$

From the fact that all model parameters are considered positive, its is clear that a_1 is positive. For a_2 to be positive, set;

$$(\gamma+2\sigma+\phi)\mu_V + (\gamma+\sigma)(\phi+\sigma) - (\frac{a^2bp\Lambda(r+\sigma K)}{\mu_V rK}) > 0,$$

which simplifies to

$$\frac{(\gamma+2\sigma+\phi)}{(\gamma+\sigma)(\phi+\sigma)} + \frac{1}{\mu_V} > \frac{a^2bp\Lambda(r+\sigma k)}{(\mu_V^2 rk)}.$$

Now comparing the right hand side of the above inequality with the expression for R_0 in (6) we deduce

$$\frac{(\gamma+2\sigma+\phi)}{(\gamma+\sigma)(\phi+\sigma)} + \frac{1}{\mu_V} > \frac{R_0^2}{(\gamma+\sigma+\phi)}.$$

For a_3 to be positive, set

$$(\gamma+\sigma)(\phi+\sigma)\mu_V - (\frac{a^2bp\Lambda(r+\sigma K)}{\mu_V rK})(\gamma+\sigma+\phi) \quad > \quad 0,$$

which simplifies to

$$1 > \frac{a^2bp\Lambda(r+\sigma K)(\gamma+\sigma+\phi)}{\mu_V^2 rK(\gamma+\sigma)(\phi+\sigma)}. \tag{10}$$

It can be clearly seen that the right side of (10) is R_0^2 so equation (10) reduces to $1 > R_0^2$. This can be written as $R_0 < 1$. Thus $a_3 > 0$ whenever $R_0 < 1$.

Next we consider $a_1a_2 - a_3 = (\gamma+2\sigma+\phi+\mu_V)((\gamma+2\sigma+\phi)\mu_V + (\gamma+\sigma)(\phi+\sigma) - (\frac{a^2bp\Lambda(r+\sigma K)}{\mu_V rK}))$
$-(\gamma+\sigma)(\phi+\sigma)\mu_V + (\frac{a^2bp\Lambda(r+\sigma K)}{\mu_V rK})(\gamma+\sigma+\phi)$.

On simplification we get $a_1a_2 - a_3 > 0$. Hence by Routh-Hurwitz criteria all eigenvalues have negative real parts if $R_0 < 1$ thus making the disease free equilibrium locally asymptotically stable.

3.4. Global stability of the disease free equilibrium point

Definition: If all solutions of system that start out near an equilibrium point stay near the equilibrium point over indefinite time, then the point is considered to be globally asymptotically stable. Previous scholars have used Lyapunov functions to prove global stability. In this paper we investigate global asymptotic stability of the disease free equilibrium using the theorem by Castillo-Chavez, Blower, Driessche, Kirschner & Yakubu (2002) [2]. Thus we rewrite our model and list two conditions, if met will guarantee global asymptotic stability of the disease free equilibrium.

$$\begin{cases} \frac{dX}{dt} = F(X,Z) \\ \frac{dZ}{dt} = G(X,Z), \qquad G(X,0) = 0, \end{cases} \tag{11}$$

where $X = (S, S_V) \in \mathbb{R}^2$ denotes uninfected populations and $Z = (E, I, I_V) \in \mathbb{R}^3$ denotes the infected population. $E_0 = (X^*, 0)$ represents the disease free equilibrium of this system. The conditions (i) and (ii) below guarantee Global asymptotic stability:

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

(ii): $G(X,Z) = D_z G(X^*,0)Z - \widehat{G}(X,Z), \qquad \widehat{G}(X,Z) \geq 0$, for $(X,Z) \in \Omega$,

where $D_z G(X^*,0)$ is an M-matrix (the off diagonal elements are nonnegative) and is also the Jacobian of $G(X,Z)$ taken in (E,I,I_v) and evaluated at $(X^*,0) = (\frac{rK}{r+\sigma K}, \frac{\Lambda}{\mu_V}, 0, 0, 0)$. If the system (11) satisfies the above conditions, then according to Castillo-Chavez *et al.* (2002) [2] the following theorem holds.

Theorem 3.3. *The equilibrium point $E_0 = (X^*,0)$ of the system (11) is globally asymptotically stable if $R_0 \leq 1$ and the conditions (i) and (ii) are satisfied.*

Proof. We begin our proof by defining new variables and breaking the system into subsystems. $X = (S, S_V)$ and $Z = (E,I,I_V)$. From equation (11) we have two vector valued functions $G(X,Z)$ and $F(X,Z)$ given by:

$$F(X,Z) = \begin{pmatrix} r(1-\frac{N}{K}) - ap\frac{S}{N}I_V - \sigma S \\ \Lambda - abS_V\frac{(E+I)}{N} - \mu_V S_V \end{pmatrix},$$

$$G(X,Z) = \begin{pmatrix} ap\frac{S}{N}I_V - (\phi+\sigma)E \\ \phi E - (\gamma+\sigma)I \\ abS_V\frac{(E+I)}{N} - \mu_V I_V \end{pmatrix}.$$

Now we consider the reduced system $\frac{dX}{dt} = F(X,0)$ from condition (i).

$$\begin{cases} \frac{dS}{dt} = r(1-\frac{N}{K}) - \sigma S, \\ \frac{dS_V}{dt} = \Lambda - \mu_V S_V. \end{cases} \tag{12}$$

$X^* = (\frac{rK}{r+\sigma K}, \frac{\Lambda}{\mu_V})$ is globally asymptotically stable equilibrium point for the reduced system $\frac{dX}{dt} = F(X,0)$. Clearly when we solve the second equation from (12) we obtain $S_V(t) = \frac{\Lambda}{\mu_V} + (S_V(0) - \frac{\Lambda}{\mu_V})e^{-\mu_V t}$ which approaches $\frac{\Lambda}{\mu_V}$ as $t \to \infty$. Similarly from the first equation of (12) we get $S(t) = \frac{rk}{r+\sigma K} + (S(0) - \frac{rK}{r+\sigma K})e^{-(\frac{r+\sigma K}{K})t}$ which approaches $\frac{rK}{r+\sigma K}$ as $t \to \infty$. We note that this asymptomatic dynamics is independent of the initial conditions in Ω , therefore the convergence of the solutions of the reduced system (12) is global in Ω. We compute $G(X,Z) =$

$D_zG(X^*,0)Z-\widehat{G}(X,Z)$ and show that $\widehat{G}(X,Z)\geq 0$.

$$D_zG(X^*,0) = \begin{pmatrix} -(\phi+\sigma) & 0 & ap \\ \phi & -(\gamma+\sigma) & 0 \\ \frac{ab\Lambda(r+\sigma K)}{rK\mu_V} & \frac{ab\Lambda(r+\sigma K)}{rK\mu_V} & -\mu_V \end{pmatrix},$$

$$\widehat{G}(X,Z) = \begin{pmatrix} apI_V(1-\frac{S}{N}) \\ 0 \\ ab(E+I)(\frac{\Lambda(r+\sigma K)}{\mu_V rK}-\frac{S_V}{N}) \end{pmatrix}.$$

Using Theorem 3.1, $\frac{\Lambda(r+\sigma K)}{\mu_V rK} > \frac{S_V}{N}$ thus the term $ab(E+I)(\frac{\Lambda(r+\sigma K)}{\mu_V rK}-\frac{S_V}{N})$ is non-negative. Therefore $\widehat{G}(X,Z)\geq 0$ and hence Theorem 3.3 holds.

3.5. Existence of the endemic equilibrium point

In the presence of CBSD, $E(t)\neq 0, I(t)\neq 0, I_v\neq 0$ our model has an equilibrium point called endemic equilibrium point denoted by $E_1=(S^*,E^*,I^*,S_V^*,I_V^*)\neq 0$. E_1 is the steady state solution where CBSD persist in the population of cassava plants. For the existence of E_1 , the elements must satisfy; $0<S^*,0<E^*,0<I^*,0<S_V^*,0<I_V^*$. We find the endemic equilibrium point by setting the right side of the the model system equations (1) and (2) equal to zero. Thus;

$$\begin{aligned}
S^* &= \frac{r}{\sigma}(1-\frac{N^*}{K})-\frac{(\gamma+\sigma)(\phi+\sigma)}{\phi\sigma}I^*, \\
E^* &= \frac{\gamma+\sigma}{\phi}I^*, \\
I^* &= \phi[\frac{\Lambda pa^2rb(\gamma+\sigma+\phi)(1-\frac{N^*}{K})-\mu_V^2N^{*2}\sigma(\gamma+\sigma)(\phi+\sigma)}{ab(\gamma+\sigma+\phi)(\Lambda pa+\sigma N^*\mu_V)(\phi+\sigma)(\gamma+\sigma)}], \\
S_V^* &= \frac{\Lambda\phi N^*}{abI^*(\gamma+\sigma+\phi)+\mu_V\phi N^*}, \\
I_V^* &= \frac{abI^*\Lambda(\gamma+\sigma+\phi)}{ab\mu_V I^*(\gamma+\sigma+\phi)+\mu_V^2\phi N^*}.
\end{aligned}$$

For a positive endemic equilibrium point, the conditions $\Lambda pa^2rb(\gamma+\sigma+\phi)(1-\frac{N^*}{K})>\mu_V^2N^{*2}\sigma(\gamma+\sigma)(\phi+\sigma)$ and $\frac{r}{\sigma}(1-\frac{N^*}{K})>\frac{(\gamma+\sigma)(\phi+\sigma)}{\phi\sigma}I^*$ must hold.

The local stability of the endemic equilibrium point was not included because farmers are only interested in quantifying the cost-effectiveness of the current control measures that minimize the disease. Therefore we describe optimal control analysis in the next section.

4. Analysis of optimal control

In this section we modify the model system (1) and (2) by introducing two time dependent controls $u_1(t)$ and $u_2(t)$. With the first control $u_1(t)$ aimed at reducing the transmission from the vector to the cassava plant. This involves killing the vector through spraying pesticides and other means. The second control $u_2(t)$ is aimed at stopping infection to the vector by the plant. This involves immediate removal of the infected plants by uprooting and burning. $\alpha \in [0,1]$ is the efficacy of the pesticide spray and αu_1 is the rate at which vectors are controlled through spraying. The the objective is to minimize the number of the infected plants at the minimum cost possible and then compare the controls and determine their cost effectiveness. Therefore system (1) and (2) are modified as follow;

$$
\begin{cases}
\frac{dS}{dt} = r(1-\frac{N}{K}) - \frac{apSI_V}{N} - \sigma S, \\
\frac{dE}{dt} = \frac{apSI_V}{N} - (\phi+\sigma)E, \\
\frac{dI}{dt} = \phi E - (u_2\gamma+\sigma)I, \\
\frac{dR}{dt} = u_2\gamma I, \\
\frac{dS_V}{dt} = \Lambda - abS_V\frac{(E+I)}{N} - (\mu_V+\alpha u_1)S_V, \\
\frac{dI_V}{dt} = abS_V\frac{(E+I)}{N} - (\mu_V+\alpha u_1)I_V.
\end{cases} \tag{13}
$$

The objective functional subject to the state system (13) is given by:

$$
J(u_1,u_2) = \int_0^{t_f} (AI(t) + \frac{A_1u_1^2(t)}{2} + \frac{A_2u_2^2(t)}{2})dt, \tag{14}
$$

where the quantity A represents the weight constant on the infected cassava plant $I(t)$ and A_1, A_2 represent the related weights of enforcing the interventions. The term $A_1\frac{u_1^2(t)}{2}$ is the cost of control efforts on reducing transmission from the whitefly vector to the cassava plant and the term $A_2\frac{u_2^2(t)}{2}$ is the cost of the control effort of stopping infection of the vector by the plant.

We choose a quadratic cost on the controls as an estimate for nonlinear function based on the assumption that the cost take nonlinear form and also to avoid the bang bang or singular optimal control cases [12]. t_f is the total time of prevention. Optimal control function (u_1^*, u_2^*) need to be found such that.

$$J(u_1^*, u_2^*) = min\{J(u_1, u_2)|(u_1, u_2) \in U\}, \tag{15}$$

where $U = \{J(u_1, u_2)|(u_i(t)\ is\ lebesgue\ measurable\ on\ [0, t_f],\ 0 \leq u_i(t) \leq 1, i = 1, 2\}$ is a control set. Note that in all cases when the control is set to zero, it means that there is no effort invested in controlling the disease and when it is set to one, it means that there is maximum effort invested in controlling the disease. To show the existence of an optimal control for system (13) and (15) we use the following theorem [16].

Theorem 4.1. *There exist an optimal control pair* (u_1^*, u_2^*) *such that* $J(u_1^*, u_2^*) = min\{J(u_1, u_2)|(u_1, u_2) \in U\}$ *subject to system (13)*

Proof. Since the control and state values are nonnegative, the space U is closed and convex by definition. Therefore the integrand $L(I, u_1, u_2)$ of the objective functional is convex on the control pair (u_1, u_2) if there exist a constant $P > 1$, numbers $\omega_1 \geq 0$ and $\omega_2 > 0$ such that $L(I, u_1, u_2) \geq \omega_1 + \omega_2(|u_1|^2 + |u_2|^2)^{\frac{P}{2}}$ then this completes the proof for the existence of an optimal control pair.

To find the optimal solution, we apply Pontryagin's Maximum Principle [24] to the constrained problem (13) and (14). Then the principle converts the system into a problem of minimizing the Hamiltonian H point-wise with respect to the controls u_1 and u_2.

Theorem 4.2. *Let* $u_i^* \in U$ *be an optimal control. Then there exists an adjoint function* $\lambda : \mathbb{R} \to \mathbb{R}^n$ *such that* $x(t, u_i^*), u_i^*, \lambda$ *satisfy the state system (13) with intimal conditions and the adjoint system. For* $i = 1, 2$*:*

$$\begin{cases} \lambda'(t) = -\frac{\partial H}{\partial x}, \\ \lambda(t_f) = 0, \end{cases} \tag{16}$$

where the Hamiltonian H is given by $H(t, x, u_i) = f(t, x, u_i) + \lambda g(t, x, u_i)$*, f is the integrand of the objective function (equation 14), g is the state system in equation (13) and* $x = \{S, E, I, R, S_V, I_V\}$

Proof. Assume (u_1^*, u_2^*) is an optimal control and S, E, I, R, S_V and I_V are the state responses x to the optimal control. The Hamiltonian is given by:

$H = AI + A_1\frac{u_1^2}{2} + A_2\frac{u_2^2}{2} + \lambda_1(r(1-\frac{N}{k}) - \frac{apSI_V}{N} - \sigma S) + \lambda_2(\frac{apSI_V}{N} - (\phi+\sigma)E) + \lambda_3(\phi E - (u_2\gamma + \sigma)I) + \lambda_4(u_2\gamma I) + \lambda_5(\Lambda - abS_V\frac{(E+I)}{N} - (\mu_V + \alpha u_1)S_V) + \lambda_6(abS_V\frac{(E+I)}{N} - (\mu_V + \alpha u_1)I_V)$.

From $\lambda_1' = -\frac{\partial H}{\partial S}, \lambda_2' = -\frac{\partial H}{\partial E}, \lambda_3' = -\frac{\partial H}{\partial I}, \lambda_4' = -\frac{\partial H}{\partial R}, \lambda_5' = -\frac{\partial H}{\partial S_V}, \lambda_6' = -\frac{\partial H}{\partial I_V}$, there exist adjoint variables or shadow prices λ_i, $i = 1,2,3,4,5,6$. satisfying

$$
(17)\begin{cases}
\lambda_1' = \lambda_1(\frac{r}{K} + \frac{apI_V(N-S)}{N^2} + \sigma) - \frac{apI_V(N-S)}{N^2}\lambda_2 - \frac{abS_V(E+I)}{N^2}\lambda_5 + \frac{abS_V(E+I)}{N^2}\lambda_6, \\
\lambda_2' = \lambda_1(\frac{r}{K} - \frac{apSI_V}{N^2}) + (\frac{apSI_V}{N^2} + \phi + \sigma)\lambda_2 - \phi\lambda_3 + (\lambda_5 - \lambda_6)\frac{abS_VS}{N^2}, \\
\lambda_3' = -A + \lambda_1(\frac{r}{K} - \frac{apSI_V}{N^2}) + \frac{apSI_V}{N^2}\lambda_2 + (u_2\gamma + \sigma)\lambda_3 - u_2\gamma\lambda_4 + (\lambda_5 - \lambda_6)\frac{abS_VS}{N^2}, \\
\lambda_4' = 0, \\
\lambda_5' = ab\frac{(E+I)}{N}(\lambda_5 - \lambda_6) + (\mu_V + \alpha u_1)\lambda_5, \\
\lambda_6' = ap\frac{S}{N}(\lambda_1 - \lambda_2) + (\mu_V + \alpha u_1)\lambda_6.
\end{cases}
$$

with boundary conditions $\lambda_1(t_f) = \lambda_2(t_f) = \lambda_3(t_f) = ... = \lambda_6(t_f) = 0$.

The optimality equations are obtained by finding the partial derivatives of the Hamiltonian equation with respect to each control variable.

$$\frac{\partial H}{\partial u_1} = A_1u_1 - \alpha S_V\lambda_5 - \alpha I_V\lambda_6, \tag{18}$$

$$\frac{\partial H}{\partial u_2} = A_2u_2 - \gamma I\lambda_3 + \gamma I\lambda_4. \tag{19}$$

Solving for u_i^* in (18) and (19) where the derivative vanishes i.e $\frac{\partial H}{\partial u_i}|_{u_i^*} = 0$ for $i = 1,2$ we get

$$u_1^* = \frac{\alpha}{A_1}(S_V\lambda_5 + I_V\lambda_6), \qquad u_2^* = \frac{\gamma I}{A_2}(\lambda_3 - \lambda_4). \tag{20}$$

It is clear that the optimal control (u_1^*, u_2^*) can be characterized as:

$$u_1^* = \max\{0, \min\{1, \frac{\alpha}{A_1}(S_V\lambda_5 + I_V\lambda_6)\}\}, \tag{21}$$

$$u_2^* = \max\{0, \min\{1, \frac{\gamma I}{A_2}(\lambda_3 - \lambda_4)\}\}. \tag{22}$$

Therefore the optimal control (u_1^*, u_2^*) that minimizes the objective function over a control set U is given by equation (21) and (22). We note that since the solutions of the state system (13)

and adjoint system (17) are bounded and satisfy *Lipschitz* conditions, the optimality system is unique for some small t_f. Thus the restriction on the length of time interval $[0,t_f]$ in the control problem is to guarantee uniqueness of the optimality system [6].

5. Numerical simulations

In this section we aim at verifying some of the analytical results on the model system (1)-(2) and (13)-(14). This is done by using a set of parameter values derived from literature, Uganda Ministry of Agriculture units and realistic assumptions. We investigate numerically the effect of applying various optimal control strategies in controlling the spread of CBSD. The following strategies are considered.

Strategy A: Spraying the whitefly vector with pesticides $(u_1 \neq 0, u_2 = 0)$.

Strategy B: Uprooting and burning infected cassava, this involves scouting the garden for infected plants. $(u_1 = 0, u_2 \neq 0)$.

Strategy C: Combination of both strategies A and B. $(u_1 \neq 0, u_2 \neq 0)$.

The optimal control is obtained by solving the optimality system consisting of the state system and the adjoint system (17), (21)-(22). We use the following algorithm for solving the optimality system.

Algorithm 1 Optimal Control

[19]

(1). Subdivide the time interval $[t_0, t_f]$ into N equal sub intervals. Set the state variable at different time as $\overrightarrow{x} = x(t)$. Assume a piecewise constant control $u_j^{(0)}(t)$, $t \in [t_k, t_{k+1}]$ where $k = 0, 1..., N-1$ and $j = 1, 2$.

(2). Apply the assumed control $u_j^{(0)}(t)$ to integrate the state system with an initial condition $\overrightarrow{x}(t_0) = \{S(0), E(0), I(0), S_V(0), I_V(0)\}$ forward in time $[0, t_f]$ using the fourth-order Runge-Kutta scheme.

(3). Apply the assumed control $u_j^{(0)}(t)$ to integrate the adjoint system with transversality condition $\overrightarrow{\lambda}(t_f) = \{\lambda_1(t_f), \lambda_2(t_f), \lambda_3(t_f), \lambda_4(t_f), \lambda_5(t_f), \lambda_6(t_f)\}$ backward in time $[t_f, 0]$ using the fourth-order Runge-Kutta scheme.

(4). Update the control by entering the new state and adjust solutions $\overrightarrow{x}$ and $\overrightarrow{\lambda}$ respectively through the characterization equation (21) and (22).

(5). Stop the algorithm if $\frac{\|\overrightarrow{x}^{i+1} - \overrightarrow{x}^{i}\|}{\|\overrightarrow{x}^{i+1}\|} < \varepsilon$ [13], otherwise update the control using a convex combination of the current and previous control and Go to Step (2). Here $\overrightarrow{x}^{i}$ is the i^{th} iterative solution of the state variable and ε is the arbitrarily small positive number.

The model systems are simulated using the fourth order Runge Kutta scheme and we consider the following initial conditions $S(0) = 2000$, $E(0) = 0$, $I(0) = 0$, $S_V(0) = 800$, $I_V(0) = 50$. The rest of the parameter values are shown in the Table 1 below.

TABLE 1. Parameter values for the model

Parameter	Description	Value day^{-1}	Reference
K	Carrying capacity	10000	Estimated
r	Planting/replanting rate	0.05	Holt *et al.* (1997)
a	Number of plants visited by vectors per day	100	Legg (1995)
p	Probability of acquisition of virus by plant	0.0033	Estimated
b	Probability of acquisition of virus by vector	0.0033	Estimated
ϕ	Rate of development to infection	0.05-0.008	Wagaba *et al.* (2013)
σ	Harvest rate of cassava	0.003	Holt *et al.* (1997)
γ	Removal rate of infected cassava	0.03-0.1	Estimated
Λ	Constant vector recruitment rate	0.2	Jeger *et al.* (2004)
μ_V	Vector death rate	0.0166-0.0142	CUES (2013)
A_1	Per area unit cost of pesticide	35000/-	Estimated
A_2	per area unit cost of uprooting and burning	15000/-	Estimated

STRATEGY A

Under this strategy, we use the control on the mortality rate of the whitefly vector population u_1 to optimize the objective function J. This is done by killing the whitefly vector population through spraying pesticides and others means, while the second control on uprooting and burning of infected cassava plants u_2 is set at Zero. The numerical results in Figure 2 (a) show that with the case when the control u_1 is active, by the time of harvest at 240 days we have about 148 plants out of 2000 that are still healthy when the efficacy level of the pesticide spray is 3%, 316 plants when the level of efficacy is set to 5% and 464 plants when the level of efficacy of the pesticide spray is set to 8%. We note that number of healthy plants increases with increase in the level of efficacy of the pesticide. For the case when the control u_1 is not active we have only 11 healthy cassava plants by 90 days.

For the case when the control is active Figure 2 (b) shows a significant decrease in the number of exposed cassava plants from 799 plants to 566 plants in 30 days when the level of efficacy of the pesticide spray is set to 3%, 458 plants in 30 days when the level of efficacy of the pesticide spray is set to 5% and 386 exposed cassava plants with the level of efficacy of the pesticide spray is set to 8%.

Figure 2 (c) we observe that when the control is active, we have a decrease in the infected cassava plants from 1480 plants in 90 days to 1192 plants with the efficacy level (α) set to 3%; 957 infected plants with the efficacy of 5% and 766 infected plants with an efficacy level of 8%. At the harvest time we still note that we have high infection despite the fact that our control is active. That is to say, 890 infected plants at the time of harvest with the efficacy level set to 3%, 717 infected plants at the time of harvest with efficacy level set to 5% and 568 infected plants with an efficacy level of 8%. For the case without control we have 1040 infected cassava plants at the time of harvest. This is a very big loss for the farmer. Figure 2 (d) shows zero removed plants, this is simply because with this strategy control u_2 is set to zero (not active) since we are only investigating the effect of increasing the mortality rate of the whitefly vectors in this strategy.

Figure 2 (e) we observe that under this strategy control u_1 increases the mortality rate of the vectors thus the slight drop of the vector population compared to the case when the control u_1 is not active. The loss of susceptible vectors in 43 days in Figure 2 (e) is also due to the fact that the infection in whitefly population is too high implying that by 43 days all vectors are infected regardless of the pesticide spray and the level of efficacy of the spray. So we have a shift to the infectious class of vectors.

Figure 2 (f) shows significant decrease in the infected vector population from 477 infected vectors in 30 days to; 223 infected vectors in 25 days when the level of efficacy is set to 3%, 158 infected vectors in 22 days with the level of efficacy of 5% and 131 infected vectors in 19 days with the level of efficacy of 8%. We can observe in Figure 2 (f) when the control is active with the efficacy level of the pesticide spray set to 8% that the infection in the whitefly vector is brought down faster. However we are interested in bringing down the infection of cassava plants in the shortest time possible and with this strategy we observe from numerical simulation

results that there is still a big challenge in bringing down infection in cassava plants. So the strategy is not effective.

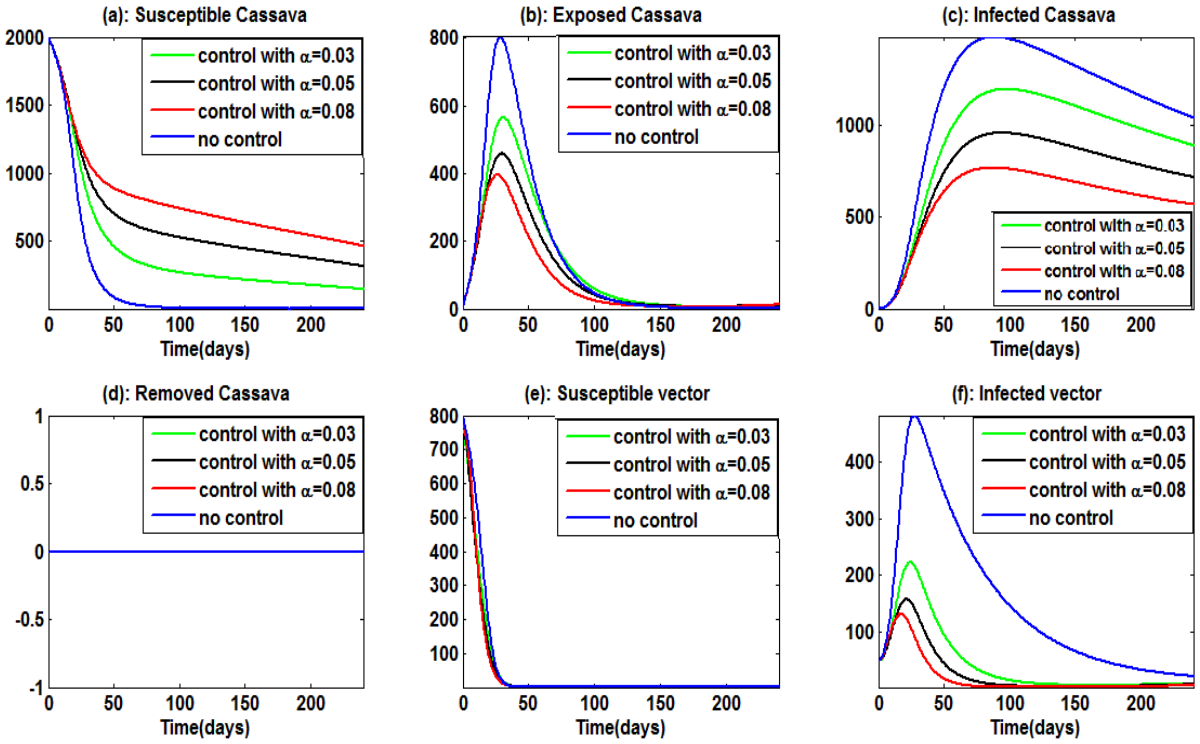

FIGURE 2. Strategy A $u_1 \neq 0$, $u_2 = 0$

The control profile Figure 3 (g) shows that the control u_1 on the mortality rate of vectors at the lower bound on the 1^{st} day of the intervention and then at the upper bound for 148 days of the intervention when the efficacy level is set to 3%, then reduces to zero by 230 days. With efficacy level set to 5% we have the control at the lower bound of 0% for 4 days , then raises to the upper bound of 100% and stays at the upper bound for 142 days, the control is then reduced gradually to 0% by 235 days. With the efficacy level set to 8% we have the control at the lower bound of 0% for 8 days, then raises to the upper bound and stays at the upper bound for 90 days and then slowly reduces to the lower bound of 0% by 235 days. Figure 3 (h) shows control u_2 at zero because in this strategy the control is inactive.

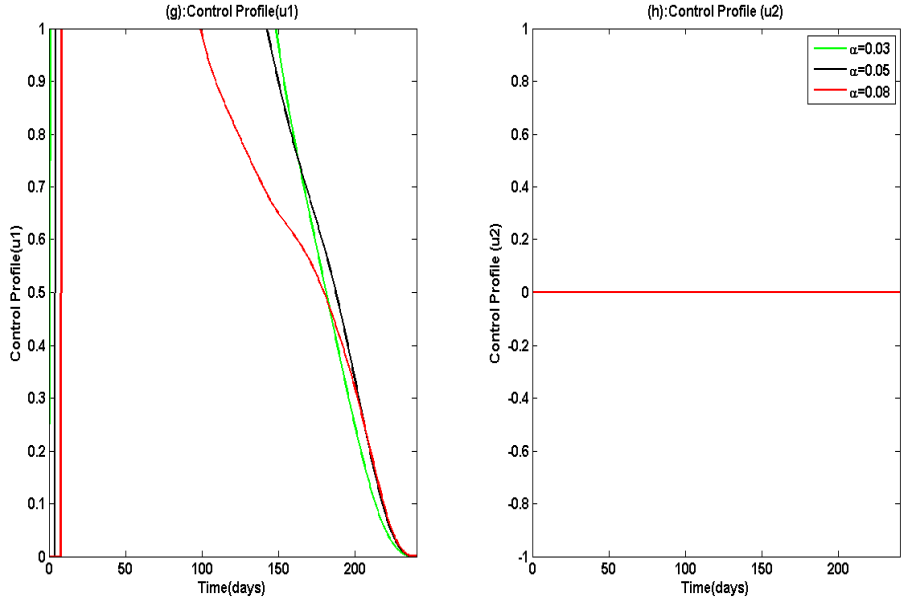

FIGURE 3. Control profile for strategy A $u_1 \neq 0$, $u_2 = 0$

STRATEGY B

Under this strategy, the second control u_2 which involves scouting gardens, uprooting and burning of infected cassava plants is used to optimize the objective function J while setting the control on the mortality rate of the vectors u_1 to zero. Figure 4 (a) shows that when the control u_2 is active, there is significant decrease in the number of susceptible cassava plants regardless of the different rates of removal of infected cassava plants. This is because removing infected plants from the garden does not immediately affect vector population, so more pressure is put on the susceptible plants that remain in the garden by the vectors in the process of feeding and this explains the drop in the number of susceptible plants when the control is active.

Figure 4 (b) show significant increase in the number of exposed cassava plants from 803 exposed cassava plants to 812 plants in 29 days with the removal rate set to 3%, 820 exposed cassava plants with the removal rate set to 6% and 825 exposed cassava plants with the removal rate set to 10% in 29 days. This is attributed to the competition for resources by the vectors when some of the infected cassava plants are removed from the garden, Thus vector feed on ones that remain in the garden and in the course of feeding more cassava plants are exposed to the infection. By 64 days the number of exposed cassava plants reduces compared to the case

when the control is not active. This is because all the plants that where exposed to the infection have developed symptoms and moved to the infectious class.

In Figure4 (c) we observe significant decrease in the number of infected cassava plants from 1480 plants in 90 days to 450 infected plants in 90 days with the removal rate(γ) set to 3%, 177 infected plants in 90 days with the removal rate set to 6% and 97 infected cassava plants in 90 days with the removal rate set to 10%. We also observe that in the long run the disease does not phase out completely even when every plant is harvested.

Figure 4 (d) shows that by about 140 days; 1713 infected cassava plants are removed from the garden with a removal rate of 10%, 1673 plants are removed from the garden with the removal rate set to 6% and 1493 infected cassava plants are removed from the garden with the removal rate of 3%. We note that although this strategy tries to control the disease, the farmer incurs a very big loss.

In Figure 4 (e) and Figure 4 (f) we observe that regardless of the removal rate of the infected cassava plants, we have superposition of the curves representing the susceptible vector population. This implies that with this strategy the control u_2 does not immediately affect the vector population.

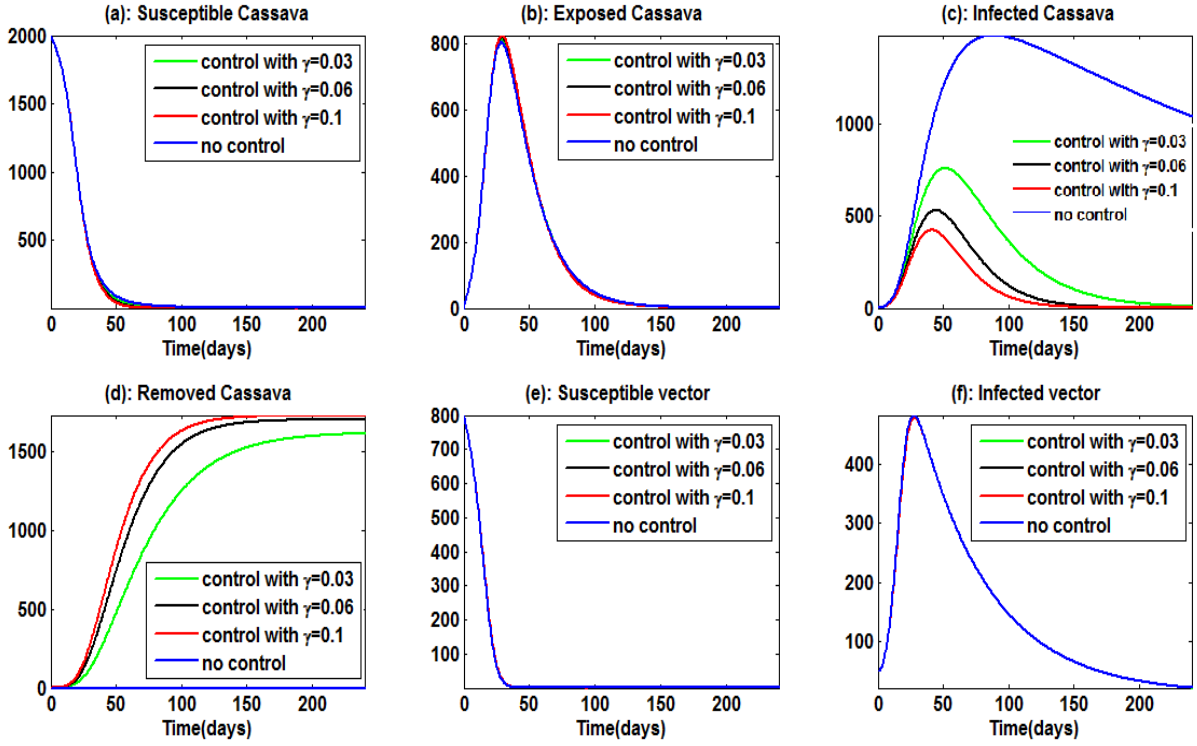

FIGURE 4. Strategy B $u_1 = 0, u_2 \neq 0$

The control profile Figure 5 (g) shows control u_1 at zero. i.e in this strategy the control is not active. The control profile Figure 4 (h) shows the second control u_2 with the removal rate set to 3% starts from 0%, then raises to its upper bound in 3 days and stays at its upper bound for 223 days then reduces slowly to the lower bound of 0% in 14 days. With the removal rate set to 6% the control u_2 starts at the lower bound and raises to its upper bound in 4 days and stays up for162 days then reduces to the lower bound of 0% in 74 days. With the removal rate set to 10% the control u_2 starts at the lower bound of 0%, then raises to its upper bound in 5 days and then is stays at its upper bound for 130 days, then reduces slowly to its lower bound in 105 days. The control profiles show us the extent to which the disease can be controlled using this particular strategy.

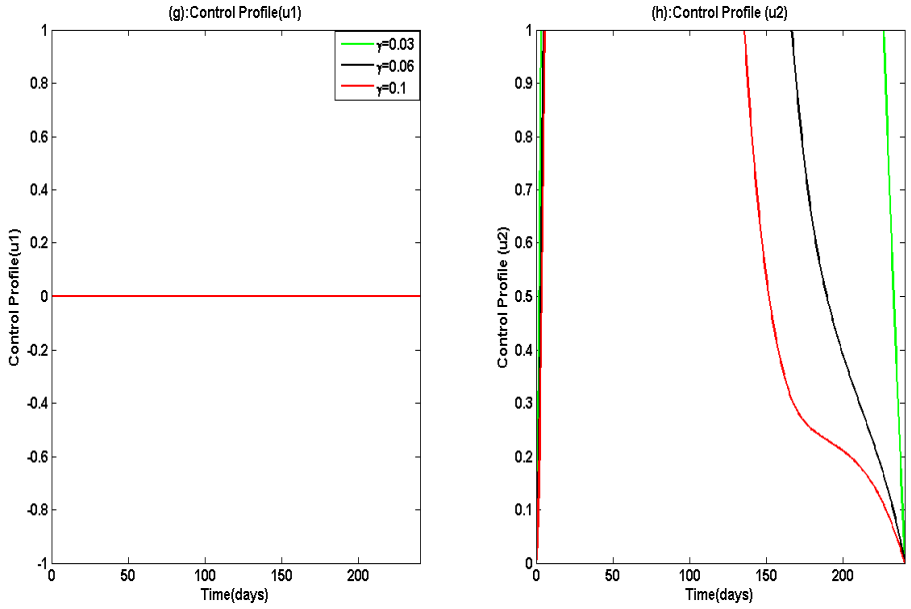

FIGURE 5. Control profile for strategy B $u_1 = 0, u_2 \neq 0$

STRATEGY C

Under this strategy; the control on the mortality rate of vectors u_1 through spray with pesticides and other means and the second control on uprooting and burning of infected plants u_2 are used to optimize the objective function J. From numerical simulation results Figure 6 (a) shows significant increase in the number of susceptible plants from 4 plants at the time of harvest (240 days) when the controls are not active to 451 plants at the harvest time when the controls are active.

Figure 6 (b) we observe a significant decrease in the number of exposed cassava plants from 803 plants in 29 days, the case when both controls are not active to 398 cassava plants for the case when both controls are active. This implies that applying both controls manages the spread of the disease to a certain level, but the disease is not phased out completely. By 130 days we have more exposed cassava plants when both controls are active compared to the case when both controls are inactive. This means that its better to let cassava stay longer in the exposed class upto the time of harvest than in the infected class. This is because cassava exposed to infection has no root symptoms and therefore edible.

Figure 6 (c) shows significant decrease in the number of infected cassava plants from 1480 infected plants in 90 days (the case when both controls are inactive) to 43 infected plants in 90 days when both controls are active. At the time of harvest (240 days) we notice 9 infected plants in the garden for the case when both controls are active compared to 1040 infected cassava plants for the case without the control. We also notice that with both controls active the disease can be managed however there is still evidence of a few infected plants in the long run.

Figure 6 (d) show an exponential increase in the number of removed plants due to the infection. At the time of harvest we observe 931 infected plants removed from the garden. This is a very big loss to the farmer and threatens cassava production in Uganda. For the case without the control we have no removed cassava plants from the garden.

Figure 6 (e) shows a slight drop in the number of susceptible vectors. This is because with both controls active, control u_1 increases the mortality rate of the vectors and control u_2 increases the removal rate of infected plants and this leads to competition for resources in the vector population and the out competed vectors also die, thus the drop in Figure 6 (e). We also observe that by 42 days we only have 1 susceptible vector implying that the infection is too high, all the other vectors have become infectious.

Figure 6 (f) shows significant decrease in the number of infected vectors from 483 infected vectors in 28 days for the case without the control to 70 infected vectors for the case with control. We notice that the vector population is reducing with time, this is as a result of harvesting cassava plants. At the time of harvest we have 21 infected vectors for the case without control and 2 infected vectors for the case with control and this remains constant in the long run, implying that the disease can not die out (remains endemic).

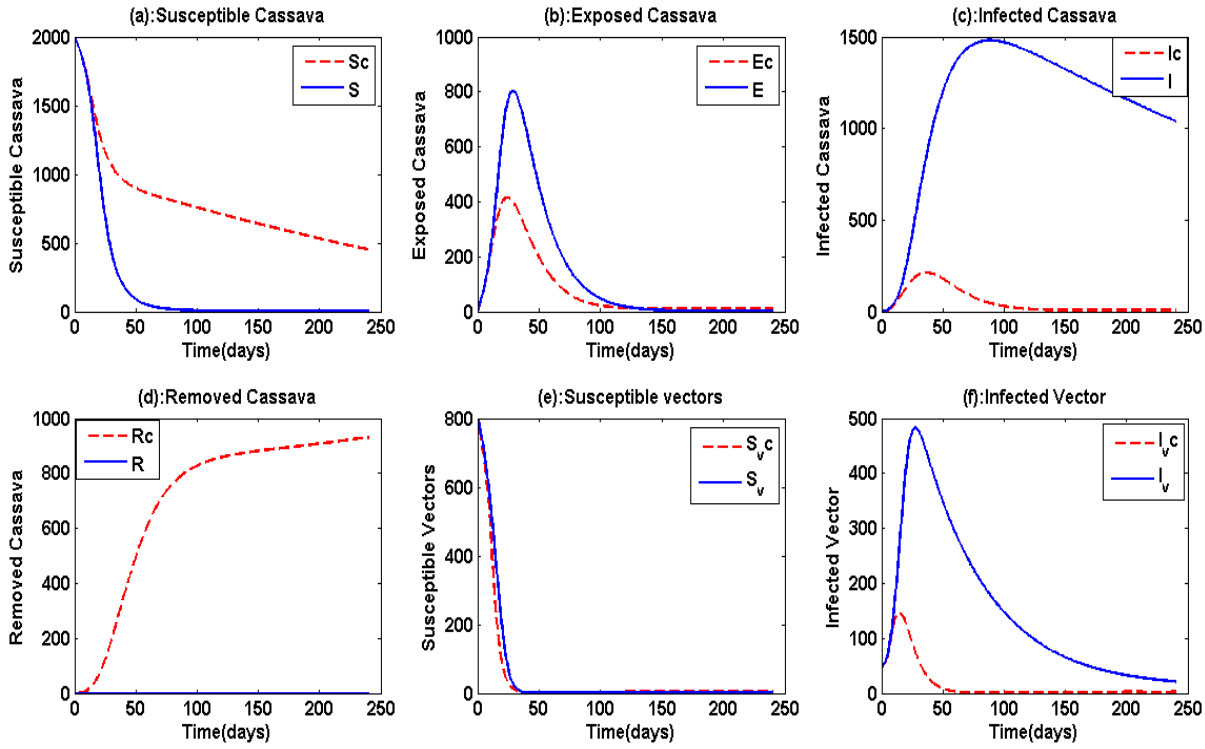

FIGURE 6. Strategy C $u_1 \neq 0$, $u_2 \neq 0$

The control profile Figure 6 (g) shows control u_1 at the lower bound for 10 days then increases to its upper bound in 1 day, stays at the upper bound for 73 days then reduces to lower bound of 0% in 3 days and then stays at the lower bound for 71 days then increases again to the upper bound of 100% in 7 days, stays up for 33 days and then reduces slowly to a lower bound in 38 days.

The control profile Figure 6 (h) the control u_2 is at the lower bound of 0% then raises to the upper bound of 100% and stays up for 237 days, reduces slowly to the lower bound in 3 days. This implies that the second control u_2 is applied up to almost the time of harvest. This can be a great loss to farmer since they usually don't want to invest for a long time.

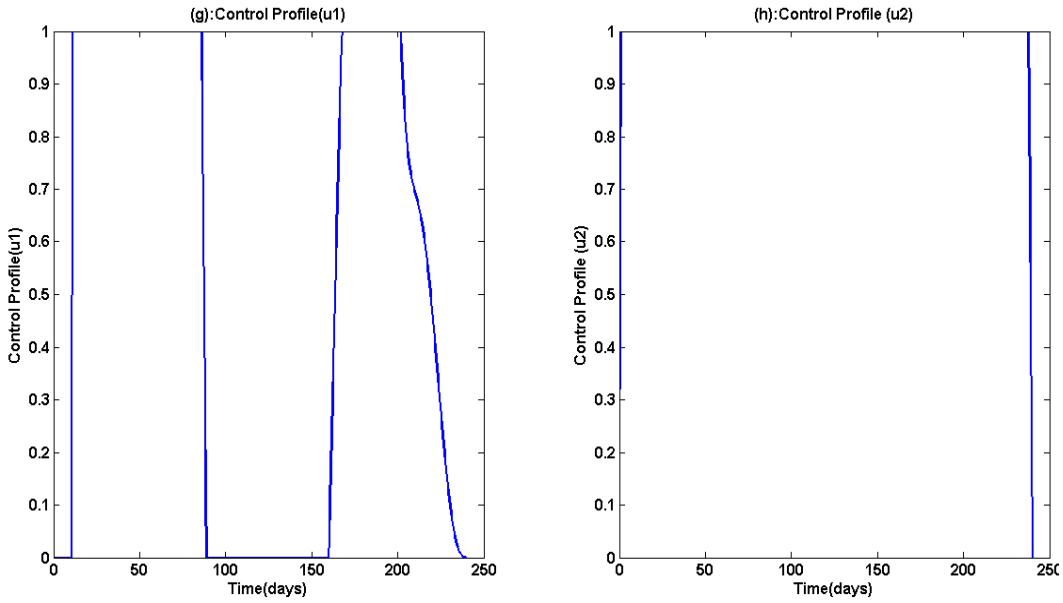

FIGURE 7. Control profile for strategy C $u_1 \neq 0$, $u_2 \neq 0$

5.1. Cost-effectiveness analysis

In order to quantify the cost-effectiveness of the control measures, we examine the cost effectiveness ratio of the strategies employed so that we can draw our conclusions. There are three types of cost-effectiveness ratios;

(1) The Average Cost-Effectiveness Ratio (ACER); This deals with a single intervention and evaluates the intervention against its baseline option. It is calculated by dividing the net cost of the intervention by the total number of health outcomes prevented by the intervention.

(2) The Marginal Cost-Effectiveness Ratio (MCER). This deals with assessment of the specific changes in cost and effect when a program is expanded or contracted.

(3) The Incremental Cost-Effectiveness Ratio (ICER). This provides a means of comparing the differences between the costs and health outcomes of two alternative intervention strategies that compete for the same resources and it is generally described as the additional cost per additional health outcome [23]. The ICER formula is given by

$$\text{ICER} = \frac{\text{difference in intervention costs}}{\text{difference in the total number of infection averted}} \tag{23}$$

The total number of infection averted is computed by estimating the difference between the total number of infection cases without control and the number of infection cases with control.

For purposes of this study, we consider the Incremental Cost-Effectiveness ratio (ICER) because it allows us to compare the cost-effectiveness of the combination of at least two of the control strategies. When comparing two competing intervention strategies using (ICER), one intervention is compared with the next less-effective alternative to determine one that provides the most cost-effective therapy. The numerator in the ICER formula includes differences in intervention costs, averted disease costs, costs of prevented cases and averted productivity loses if applicable. The denominator in the ICER formula includes the difference in health outcomes for example total number of infection averted, number of susceptibility cases prevented [22]. Conclusively (ICER) provides information to policy makers on where limited resources should be allocated in controlling the disease in question. Basing on our model simulation results, we rank the strategies in order of increasing effectiveness.

TABLE 2. Ranking control strategies in order of increasing effectiveness

Strategies	Total infection averted	Total cost (Ugx)	ICER
No strategy	0	0	-
Strategy A	266070	2263900	8.509
Strategy C	430170	3039900	4.729
Strategy B	517880	914160	-24.236

The ICER is calculated from equation (23) as follow:

$$\begin{aligned} ICER(A) &= \frac{2263900}{266070} = 8.509 \\ ICER(C) &= \frac{3039900 - 2263900}{430170 - 266070} = 4.729 \\ ICER(B) &= \frac{914160 - 3039900}{517880 - 430170} = -24.236 \end{aligned}$$

The comparison between strategy A and C shows a cost saving of UGx 4.729 for strategy C over strategy A. The high ICER for strategy A indicates that strategy A is strongly dominated. That is Strategy A is more costly and less effective than strategy C. Therefore strategy A is excluded

from the set of alternatives that it does not consume limited resources.

We recalculate ICER

TABLE 3. Computation of ICER after excluding Strategy A

Strategies	Total infection averted	Total cost	ICER
Strategy C	430170	3039900	7.067
Strategy B	517880	914160	-24.236

The comparison between strategy C and B shows a cost saving of UGx 24.236 for strategy B over strategy A. The negative ICER for strategy B indicates that Strategy C is strongly dominated, that is strategy C is more costly and less effective than strategy B. Therefore with this result we can conclude that strategy B (scouting gardens, Uprooting and burning of infected cassava plants) is more cost effective than all the other strategies.

6. Discussion, conclusion and recommendations

6.1. Discussion

In this study we derived and analyzed the deterministic model for the transmission dynamics of Cassava Brown Streak Disease (CBSD) in Uganda. We calculated the basic reproduction number, investigated the existence and stability of equilibria. The results show that for a positive level of infective vectors, exposed and infective plants the model has an endemic equilibrium point. When all the vectors and the plants are susceptible, the model predicts a disease free equilibrium point. We also reformulated our model to accommodate control measures that are being employed by farmers to manage the disease. That is, control u_1 (increasing mortality rate of vectors through spraying with pesticides and other means), the second control u_2 (uprooting and burning of infected cassava plants, this may also involve scouting the gardens for infected plants, educating farmers on symptoms and how to uproot the diseased plants). The study also analyzed the optimal control of the model using Pontryagins Maximum principle where we derived and analyzed the necessary conditions for optimal control of the disease. Three control strategies where numerically studied, that is strategy A, B, and C. Our numerical simulation

results show that strategy A (increasing the mortality rate of vector through spraying with pesticide) does not bring down the infection to a desired level in cassava plants. We observed that the efficacy level of the pesticide spray is also key in controlling the vector population i.e the higher the efficacy of the pesticide spray, the more death of vectors realized. Results show that with the level of efficacy set to 10%, the control effort of 100% is applied for 3 months, then the percentage of the effort applied reduces slowly up to the harvest time. This makes strategy A less effective and very expensive for the farmers since even pesticides, equipments for spraying and labour are very expensive.

Strategy B (uprooting of infected cassava plants), results show that the infection in cassava plants can be put down although the infection in vector remains high. This is a threat because we can not have disease free equilibrium when the infective vector population is high. It is evident that although the infection is down in cassava plants, the farmer loses a lot of plants in the process of removing infected cassava plants. With the removal rate set to 10% the farmer must apply 100% effort when using this control from the 5^{th} day for 130 days, then the effort is reduced slowly up to the time of harvest as shown in Figure 5.4. This strategy is more effective than strategy A.

Strategy C (Combination of pesticide spray and uprooting and burning of infected plants). Results show that this strategy is very effective in managing the disease in both vectors and cassava plants. However its is also very expensive to the farmer compared to other strategies. We observed 931 infected plants removed from the garden at the time of harvest and this was practically less than 1713 removed plants while using strategy B. It is evident in Figure 5.6 (g) that the farmer under this strategy must apply 100% effort when using control u_1 for 73 days, then rest for 71 days, apply maximum effort again of 100% for 33 days and then reduces the effort to zero at time of harvest. Figure 5.6 (h) shows that maximum effort of 100% is applied for 237 days while using control u_2 in this strategy. Therefore this strategy can manage the disease and give the farmer more yield of cassava but more expensive because of the additional cost of spraying involved.

We investigated the cost-effectiveness of the controls to determine the most cost effective strategy for minimizing cassava brown streak disease (CBSD) with a minimum cost. Using

the Incremental Cost-Effectiveness Ratio (ICER) we found that the total cost of the objective function for using control strategy A (increasing the mortality rate of vectors through spraying with pesticide) is Ugx 2263900. This is very costly compared to the total infection averted see Table (2). The total cost of the objective function for choosing strategy B (uprooting and burning of infected cassava plants) is Ugx 914160, this strategy is very cost effective in managing the disease and the total number of infection averted with this strategy is 517880 which is higher compared with the other strategies. However we note that although strategy B is cost effective, there is a reduction in the yield of cassava as 1713 infected plants are removed from the garden at the time of harvest see Figure 5.3 (d). The total cost of the objective function for choosing all the two intervention strategies (strategy C) is Ugx 3039900, this is very costly compared to all other strategies and the total number of infection averted (430170). Results showed that strategy C is very costly but it can manage the disease better than the other two strategies see Figure 5.5

The challenges of implementing the optimal control strategies analyzed in this study are not to be underestimated. These include Uganda's economic crisis which has led to the increase of taxes on agricultural inputs, the development of pesticide resistance and inefficiency, inadequate education given to farmers about symptoms and how they can manage the disease, hesitation of farmers to uproot infected plants because they fear losses and insufficient data from MUZARDI, NARO and NACCRI about CBSD which led to use of literature data to estimate some parameters.

6.2. Conclusion

We conclude that according to our model we have an endemic equilibrium point and the disease remains endemic as long as we have a positive population of vectors. We also note that with parameter values in Table 1, $R_0 = 10.59$ hence the disease is endemic. The most cost-effective strategy of all combinations is strategy B (uprooting and burning infected cassava plants). However the best way would be coming up with resistant varieties of cassava, other than that research should focus on finding the best pesticide that can kill the vector.

6.3. Recommendations

According to our model, it is very hard to achieve the disease free equilibrium when the vector population is positive, therefore we suggest that the Government of Uganda through Ministry of Agriculture units should help our farmers in controlling vectors which spread the disease by spraying massively. In this case strategy C can be adopted by policy makers over strategy B because with this strategy the farmers realize more yield of cassava but the problem is that it has an additional cost of spraying vectors. The best way to manage CBSD would be coming up with resistant varieties because its not easy to eliminate the whitefly vector completely.

Conflict of Interests

The authors declare that there is no conflict of interests.

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