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Research article

Sensitivity analysis of cassava mosaic disease with saturation incidence rate model

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Abstract: Cassava mosaic disease (CMD) is caused by a virus transmitted by the whitefly. This disease can destroy cassava at any stage of its growth and it resulted in lower cassava yields. In this paper, we developed a mathematical model for the epidemic of cassava mosaic disease with a deterministic model which has saturation incidence rates. This model aims to explain the effect of vectors on cassava disease outbreaks. First, this model was analyzed using standard dynamic methods to determine the behavior of the solution. We found the existence and condition of disease-free and endemic steady state. The basic reproductive number (R_0) is obtained by using the next-generation method which R_0 helps assess the ability to spread infectious diseases. Second, the stability of the steady state was analyzed, then we obtain the condition of existence of local stability and global stability at each steady state of this model. Third, analysis of the sensitivity indices in the threshold number to determine the effect of the various parameters. Finally, the results of the theoretical model were validated by numerical simulations. It is represented by various graphs converging at a steady state and stable.

Keywords: basic reproductive number; cassava mosaic disease; geometric approach; stability analysis; sensitivity analysis
Mathematics Subject Classification: 34D20, 34D23

1. Introduction

Cassava is the fifth most important food crop after wheat, corn, rice and potatoes. It is an important food crop of tropical countries, especially African countries and South America [1]. In Asia, cassava is popularly grown in Indonesia and Thailand because it is easy to grow in low-nutrient soil and is drought tolerant, able to adapt to different climates [2]. Since 2000, the world production of cassava has increased by approximately 100 million tons due to the demand for cassava products in various regions increasing. Especially in Asia, cassava is used in animal feed production and industrial use. Among the cassava-growing regions of the world, Africa accounts for more than 50 percent [3]. Cassava has been attacked by complex arthropod pests in the tropical regions of the crop-growing world which found to have pest infestation problems causing severe damage to farmers. In addition to moina and mealybug, there is also another insect, the whitefly. The survey found that the number of whiteflies is increasing and this is likely to be a major pest that damages cassava farmers [4]. The main disease in cassava is cassava mosaic disease (CMD), which is caused by insects and results in lower yields. Cassava mosaic virus (CMV) contaminates the cassava leaves and is transmitted by the whitefly vector called Bemisia tabaci and through the movement of infected planting materials [5]. The first outbreak of this disease was seen in East Africa in the 1890s [6]. In Asia, the cassava mosaic disease outbreak in Cambodia in 2015 and in Thailand in 2016, respectively [7]. This disease causes stunted cassava tubers and small heads. If a pandemic of disease would result in reduced yields of 80–100 percent (National Agricultural Big Data Center, 2020). The severity of cassava mosaic disease is affected by environmental factors such as light intensity, wind, rainfall, density and plant temperature. Since the virus is transmitted by whitefly, its transmission depends largely on vector. In general, high temperatures are associated with a dramatic increase in the number of insects and the longer the whitefly lives [4, 5]. To solve the problem of cassava mosaic disease, farmers eliminate cassava plants and whitefly which are disease carriers to cut the cycle of disease transmission by burning the diseased cassava [8]. In this regard, mathematical processes can be used as a tool for determining factors contributing to outbreaks and as a guide to reducing disease outbreaks.

Mathematical models provide a tool used to understand the dynamics of disease spread through a population and in decision-making in regard to disease prediction and disease control [9–12]. The researchers studied the dynamics of disease plants, including disease patterns, disease transmission and disease control strategies [13]. In the past, many researchers have been interested in plant disease which it is transmitted by a vector. They have developed the model of plant disease using mathematical models to analyze and explain plant disease epidemic dynamics. For instance, Jeger et al. analyzed the model of the propagation characteristics of the virus in plants through vector mating and to examine the transmission effect on plant disease control [14]. Shi et al. offered model of a vector-borne plant disease model by considering co-infection between infected plants and infected insects [15]. Kinene et al. developed a model of cassava brown streak disease with vectors in Uganda and applied the optimal control theory to reduce the number of cassava infections. They created a control strategy, which was to increase vector mortality by spraying pesticides, uprooting and burning infected cassava [16]. In 2019, Florence et al. developed a cassava mosaic virus model by considering resistant and susceptible breed [17]. Basir et al. presented a model of cassava mosaic and the role of vector growth in which he was interested in how delayed vector growth affects vector population in 2021 [18]. In 2022, Erickab et al. have studied the transmission of cassava mosaic disease in cassava and non-cassava

plants. They found the mortality rate of whiteflies and the carrying capacity of whiteflies affect disease transmission [19].

In the study of the behavior of infectious disease, the incidence function plays a significant role in describing epidemics' rise and fall. It indicates the number of new infections per unit of time. The general epidemic model frequently uses the bilinear incidence rate and the standard incidence rate. The bilinear incidence rate βSI where β is a parameter used to measure the rate of infection of the disease [20]. It describes an epidemic in which the number of host contacts increases linearly with population density. The bilinear incidence rate is not suitable for large populations because it cannot clearly explain the phenomenon of the disease. For standard incidence rate $\frac{\beta SI}{N}$ describes an epidemic where the population is constant and regardless of population density, but infection depends only on the frequency of exposure to the infected population [16, 18, 21]. This incidence is appropriate for a population that is large enough. In 1978, Capasso and Serio [22] found that the number of infections between infected and healthy populations did not always increase linearly. They proposed saturation incidence rate $\frac{\beta SI}{1+\alpha I}$ which tends to reach saturation levels when I reach the maximum number of effective infections between the infected and susceptible populations, where βI measures the force of infection when the disease enters a fully susceptible population, α is the inhibition constant, and $\frac{1}{1+\alpha I}$ measures the inhibitory effect from changes in the behavior of susceptible populations as their numbers increase or from the overcrowding of infected individuals [23–26]. From considering the above incidence rates, we can be seen that the saturation incidence rate is appropriate for the actual situation because behavioral changes and the impact of the infected population are taken into account. In addition, there is a scope for infection rates as the uninfected or infected population increases. Therefore, it is this incidence rate that interests us and is used in the development of this model.

In this paper, we consider a model with a nonlinear saturation incidence rate in analyzing the dynamics of cassava mosaic disease. The research aims to measure the spreading of the disease and to study factor that the effect of whitefly on the spread of cassava mosaic disease. We believe that the results of this research will be useful in controlling the spread of this disease. This paper is organized as follows: In Section 2, analyze the mathematical model and find the basic reproductive number which is the threshold value by using the next-generation method. Considering local stability and global stability of each steady state are derived from the Jacobian matrix. The Castillo-Chovez method is used to consider the global stability at a disease-free steady state when $R_0 < 1$. For the global stability at an endemic steady state when $R_0 > 1$, we use the geometric approach method to help created global stability. This method is a generalization of the Lyapunov theory. In Section 3, we show the results of numerical simulations to prove the theoretical results. The sensitivity analysis was used to study the effects of the parameters in our model. Section 4 presents a discussion. The last section contains the summary of this paper.

2. Formulation and analysis of the model

In this section, we defined the cassava mosaic model by dividing the population into two groups such as cassava population density and whitefly vector population density. The total cassava population density (K) and the total whitefly population density (P) are positive constants. The assumption is that the plants in the area are fixed since we must replace the dead cassava with new cassava. In addition, we assume that emigrates the number of vectors per time unit is a constant. The cassava population density

is divided into two subclasses: healthy and infectious cassava at time t which is H and I, respectively. The whitefly vector population density is divided into two subclasses: healthy and infectious vector which is X and Z, respectively. The healthy vector can be infected by infected cassava only and that it will be infected for the rest of your life. Our parameters used in the model can be described in Table 1.

Table 1. Description of parameters of the cassava mosaic model.

parameter symbol	Description	Unit
b	replanting rate of cassava	day^{-1}
σ	removal rate of infected cassava	day^{-1}
μ	mortality rate of cassava or harvest rate of cassava	day^{-1}
β_p	acquisition rate of the infected vectors due to healthy cassava	$vector^{-1}day^{-1}$
β_v	inoculation rate of the infected cassava due to healthy vectors	$m^{-2}day^{-1}$
α	the birth rate of vector	day^{-1}
m	the death rate of vector	day^{-1}
γ_p	saturation constant of cassava due to vectors	$vector^{-1}$
γ_v	saturation constant of vectors due to cassava	m^{-2}
V	emigrates number of vectors	$vectorday^{-1}$

Each variable describes the population at time t. The model is formulated as follow:

$$\frac{dH}{dt} = bK - \frac{\beta_p HZ}{1+\gamma_p Z} - \mu H \tag{2.1}$$

$$\frac{dI}{dt} = \frac{\beta_p HZ}{1+\gamma_p Z} - \sigma I - \mu I \tag{2.2}$$

$$\frac{dX}{dt} = \alpha P + V - \frac{\beta_v IX}{1+\gamma_v I} - \frac{V}{P}X - mX \tag{2.3}$$

$$\frac{dZ}{dt} = \frac{\beta_v IX}{1+\gamma_v I} - \frac{V}{P}Z - mZ \tag{2.4}$$

where $H + I = K$ and $X + Z = P$

From the vector population density is constant, then we have $\frac{dP}{dt} = 0$ that is $\alpha = m$ (birth rate of vector equal to death rate of vector).

We get host-vector model which is dynamic Eqs (2.1)–(2.4) as follows:

$$\frac{dH}{dt} = \mu K - \frac{\beta_p HZ}{1+\gamma_p Z} - \mu H \tag{2.5}$$

$$\frac{dI}{dt} = \frac{\beta_p HZ}{1+\gamma_p Z} - \sigma I - \mu I \tag{2.6}$$

$$\frac{dX}{dt} = \alpha P + V - \frac{\beta_v IX}{1+\gamma_v I} - \frac{V}{P}X - \alpha X \tag{2.7}$$

$$\frac{dZ}{dt} = \frac{\beta_v IX}{1+\gamma_v I} - \frac{V}{P}Z - \alpha Z. \tag{2.8}$$

The feasible region is

$$\Omega = \Big\{(H, I, X, Z) \in \mathbb{R}^4 : H + I \leq K, X + Z \leq P\Big\}. \tag{2.9}$$

2.1. Positivity and boundedness of the solution

To confirm that the model has epidemiological meaning, it must necessary to prove that the system of equations ODEs is non-negative and has a boundary for all time $t \geq 0$.

Theorem 1. *Let $t \geq 0$. In the model, $H(t), I(t), X(t), Z(t)$ be the solution of Eqs (2.5)–(2.8) with positive initial conditions in $H(0), I(0), X(0), Z(0)$. Then, the bounded solution which is positively invariant set of the model given by*

$$\Omega = \{(H, I, X, Z) \in \mathbb{R}^4_+ : N_1(t) \leq K, N_2(t) \leq P\}.$$

Proof. For this model, we set Eqs (2.5)–(2.8) by

$$\begin{aligned} N(t) &= (N_1(t), N_2(t)) \\ &= (H + I, X + Z). \end{aligned}$$

Then, differentiating the function $N(t)$ with respect to t

$$\begin{aligned} \frac{dN(t)}{dt} &= \left(\frac{dN_1(t)}{dt}, \frac{dN_2(t)}{dt}\right) \\ &= \left(\mu K - \frac{\beta_p HZ}{1 + \gamma_p Z} - \mu H + \frac{\beta_p HZ}{1 + \gamma_p Z} - \sigma I - \mu I, \alpha P + V - \frac{\beta_v IX}{1 + \gamma_v I} - \frac{V}{P}X - \alpha X + \frac{\beta_v IX}{1 + \gamma_v I} - \frac{V}{P}Z - \alpha Z\right) \\ &= \Big(\mu K - \sigma I - \mu(H + I), \alpha P - \alpha(X + Z)\Big) \\ &\leq \Big(\mu K - \mu N_1, \alpha P - \alpha N_2\Big). \end{aligned}$$

Therefore, we obtained $\frac{dN_1(t)}{dt} = \mu K - \mu N_1 \leq 0$ for $N_1(t) \geq K$ and $\frac{dN_2(t)}{dt} = \alpha P - \alpha N_2 \leq 0$ for $N_2(t) \geq P$ Using the integration of the above equation is

$$0 \leq (N_1(t), N_2(t)) \leq \Big(K + (N_1(0) - K)e^{-\mu t}, P + (N_2(0) - P)e^{-\alpha t}\Big).$$

As $t \to \infty$, then $0 \leq (N_1(t), N_2(t)) \leq (K, P)$. Hence, The feasible solution set of all solutions of Ω in $\mathbb{R}^4_+$ and given by

$$\Omega = \{(H, I, X, Z) \in \mathbb{R}^4_+ : N_1(t) \leq K, N_2(t) \leq P\}.$$

Thus, Ω is positively invariant set for all $t \geq 0$. In other words, Eqs (2.5)–(2.8) are described as non-negative in the $\mathbb{R}^4_+$. □

2.2. The existence of steady state

The steady state (H^*, I^*, X^*, Z^*) can be obtained from setting the right hand side of the Eqs (2.5)–(2.8) equal to zero. We get 2 steady state, namely
i) disease-free steady state: $E_0(H^*, I^*, X^*, Z^*) = E_0(K, 0, P, 0)$
ii) endemic steady state: $E_1(H^*, I^*, X^*, Z^*)$
where

$$H^* = \frac{\mu K(1+\gamma_p Z^*)}{\beta_p Z^* + \mu(1+\gamma_p Z^*)}$$
$$I^* = \frac{\mu\beta_p K Z^*}{(\sigma+\mu)(\beta_p Z^* + \mu(1+\gamma_p Z^*))}$$
$$X^* = \frac{(V+\alpha P)P((\sigma+\mu)(\beta_p Z^* + \mu(1+\gamma_p Z^*)) + \mu\gamma_v\beta_p K Z^*)}{\mu\beta_v\beta_p P K Z^* + (V+\alpha P)((\sigma+\mu)(\beta_p Z^* + \mu(1+\gamma_p Z^*)) + \mu\gamma_v\beta_p K Z^*)}$$
$$Z^* = \frac{\mu\beta_v\beta_p K P^2 - \mu(\sigma+\mu)(V+\alpha P)}{\mu\beta_v\beta_p P K + (V+\alpha P)((\sigma+\mu)(\beta_p+\mu\gamma_p) + \mu\gamma_v\beta_p K)}.$$

2.3. The basic reproductive number

The basic reproductive number or R_0 is the threshold parameter that measures the expected number of secondary infections caused by one new infected individual introduced into susceptible population group. It represents the average number of infected cassava and infected vector that will cross-infection between one infected cassava plant or one infected vector in group of only susceptible populations. We calculate the basic reproductive number using the next-generation method [27, 28]. The population is divided into m compartments and there are n infected population compartment where $m < n$. We consider only the infected compartments in the form:

$$\frac{dx_i}{dt} = F_i(x) - V_i(x). \tag{2.10}$$

F_i is the rate at which new infections are created in compartment i where $i = 1, 2, 3, ..., m$,
V_i is the rate of transfer into and out of the class of infected state in compartment i where $i = 1, 2, 3, ..., m$.

In this case $x = \begin{bmatrix} I & Z \end{bmatrix}^t$ and we obtained

$$\mathbb{F} = \frac{\partial F_i(E_0)}{\partial x_j} = \begin{bmatrix} 0 & \beta_p K \\ \beta_v P & 0 \end{bmatrix} \tag{2.11}$$

$$\mathbb{V} = \frac{\partial V_i(E_0)}{\partial x_j} = \begin{bmatrix} \sigma+\mu & 0 \\ 0 & \frac{V+\alpha P}{P} \end{bmatrix} \tag{2.12}$$

where $i = 1, 2$ and $j = 1, 2$.
The inverse of $\mathbb{V}$ is

$$\mathbb{V}^{-1} = \begin{bmatrix} \frac{1}{\sigma+\mu} & 0 \\ 0 & \frac{P}{V+\alpha P} \end{bmatrix}. \tag{2.13}$$

Hence, The next generation matrix is

$$\mathbb{F}\mathbb{V}^{-1} = \begin{bmatrix} 0 & \frac{\beta_p K P}{V+\alpha P} \\ \frac{\beta_v P}{\sigma+\mu} & 0 \end{bmatrix}. \tag{2.14}$$

Therefore, the basic reproductive number is given by the spectral radius of matrix $\mathbb{F}\mathbb{V}^{-1}$ is

$$R_0 = \rho(\mathbb{F}\mathbb{V}^{-1}) = P\sqrt{\frac{\beta_v\beta_p K}{(\sigma+\mu)(V+\alpha P)}}. \tag{2.15}$$

2.4. Stability analysis

2.4.1. Local stability of the steady state

The local stability of each steady state, we can analyze the system by linearization from considering Jacobian matrix.

Theorem 2. *The disease-free equilibrium point $E_0(K, 0, P, 0)$ is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$*

Proof. We found the Jacobian matrix of Eqs (2.5)–(2.8) is

$$J = \begin{pmatrix} -\frac{\beta_p Z^*}{1+\gamma_p Z^*} - \mu & 0 & 0 & -\frac{\beta_p H^*}{(1+\gamma_p Z^*)^2} \\ \frac{\beta_p Z^*}{1+\gamma_p Z^*} & -(\sigma+\mu) & 0 & \frac{\beta_p H^*}{(1+\gamma_p Z^*)^2} \\ 0 & -\frac{\beta_v X^*}{(1+\gamma_v I^*)^2} & -\frac{V+\alpha P}{P} - \frac{\beta_v I^*}{(1+\gamma_v I^*)} & 0 \\ 0 & \frac{\beta_v X^*}{(1+\gamma_v I^*)^2} & \frac{\beta_v I^*}{1+\gamma_v I^*} & -\frac{V+\alpha P}{P} \end{pmatrix}. \tag{2.16}$$

Disease-free equilibrium point $E_0(K, 0, P, 0)$ is

$$J = \begin{pmatrix} -\mu & 0 & 0 & -\beta_p K \\ 0 & -(\sigma+\mu) & 0 & \beta_p K \\ 0 & -\beta_v P & -\frac{V+\alpha P}{P} & 0 \\ 0 & \beta_v P & 0 & -\frac{V+\alpha P}{P} \end{pmatrix}. \tag{2.17}$$

Therefore, we obtained the characteristic equation is given by

$$(\mu+\lambda)(V+\alpha P+\lambda)(\lambda^2 + a_1\lambda + a_0) = 0. \tag{2.18}$$

Where $a_1 = \frac{V+\alpha P}{P} + \sigma + \mu$
$a_2 = \frac{(V+\alpha P)(\sigma+\mu)}{P}(1 - R_0^2)$.
From the characteristic Eq (2.18), two eigenvalues are $\lambda = -\mu$ and $\lambda = -\frac{V+\alpha P}{P}$ which they are always negative real parts. So, other eigenvalues are quadratic equation that can be obtained solving the equations as follows

$$\lambda^2 + a_1\lambda + a_2 = 0. \tag{2.19}$$

We only need consider the roots of quadratic equation is negative real parts when they satisfy the Routh-Hurwitz criterion which are:

$$i) \quad a_1 > 0 \tag{2.20}$$

and

$$ii) \quad a_2 > 0. \tag{2.21}$$

Clearly, a_1 and a_2 are always positive that satisfies with condition (2.20) to (2.21) of Routh-Hurwitz criterion. Hence, disease-free equilibrium point is locally asymptotically stable when $R_0 < 1$. □

Theorem 3. *If $R_0 > 1$, The endemic equilibrium point $E_1(H^*, I^*, X^*, Z^*)$ of Eqs (2.5)–(2.8) is locally asymptotically stable.*

Proof. endemic equilibrium point $E_1(H^*, I^*, X^*, Z^*)$ where

$Z^* = \frac{\mu\beta_v\beta_p KP^2 - \mu(\sigma+\mu)(V+\alpha P)}{\mu\beta_v\beta_p KP + (V+\alpha P)((\sigma+\mu)(\beta_p+\mu\gamma_p)+\mu\gamma_v\beta_p K)}$

$H^* = \frac{\mu K(1+\gamma_p Z^*)}{\beta_p Z^* + \mu(1+\gamma_p Z^*)}$

$I^* = \frac{\mu\beta_p KZ^*}{(\sigma+\mu)(\beta_p Z^* + \mu(1+\gamma_p Z^*))}$

$X^* = \frac{(V+\alpha P)P((\sigma+\mu)(\beta_p Z^* + \mu(1+\gamma_p Z^*))+\mu\gamma_v\beta_p KZ^*)}{\mu\beta_v\beta_p PKZ^* + (V+\alpha P)((\sigma+\mu)(\beta_p Z^* + \mu(1+\gamma_p Z^*))+\mu\gamma_v\beta_p KZ^*)}$

we get

$$J = \begin{pmatrix} -\frac{\beta_p Z^*}{1+\gamma_p Z^*} - \mu & 0 & 0 & -\frac{\beta_p H^*}{(1+\gamma_p Z^*)^2} \\ \frac{\beta_p Z^*}{1+\gamma_p Z^*} & -(\sigma+\mu) & 0 & \frac{\beta_p H^*}{(1+\gamma_p Z^*)^2} \\ 0 & -\frac{\beta_v X^*}{(1+\gamma_v I^*)^2} & -\frac{V+\alpha P}{P} - \frac{\beta_v I^*}{(1+\gamma_v I^*)} & 0 \\ 0 & \frac{\beta_v X^*}{(1+\gamma_v I^*)^2} & \frac{\beta_v I^*}{1+\gamma_v I^*} & -\frac{V+\alpha P}{P} \end{pmatrix}. \tag{2.22}$$

Therefore, the characteristic equation of J can be obtained from

$$\left(\frac{V+\alpha P}{P} + \lambda\right)(\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3) = 0. \tag{2.23}$$

Where

$$a_1 = \sigma + \mu + \frac{(V+\alpha P)\Big(g_3 + \beta_p P(\sigma+\mu)\Big)}{g_4 P} + \frac{\mu\Big(\mu R_0^2(\sigma+\mu)(1+\gamma_p P+\beta_p P) + \mu\gamma_v\beta_p KP\Big)}{g_2}$$

$$a_2 = \frac{(V+\alpha P)g_3\Big(\sigma(R_0^2-1)\Big(\mu R_0^2(\sigma+\mu)(1+\gamma_p P) + \mu\gamma_v\beta_p KP\Big) + \mu g_1(2R_0^2-1)\Big)}{g_2 g_4 R_0^2 P} + \frac{\mu g_1(\sigma+\mu)}{g_2}$$

$$a_3 = \frac{\mu g_1 g_3(\sigma+\mu)(V+\alpha P)(R_0^2-1)}{g_2 g_4 P R_0^2}$$

with coefficients

$$\begin{aligned} g_1 &= (\sigma+\mu)R_0^2(\beta_p P + \mu + \mu\gamma_p P) + \mu\gamma_v\beta_p KP \\ g_2 &= \mu R_0^2(\sigma+\mu)(1+\gamma_p P) + \beta_p P(\sigma+\mu+\mu\gamma_v K) \\ g_3 &= (\sigma+\mu)(\beta_p P + \mu\gamma_p P + \mu R_0^2) + \mu\gamma_v\beta_p KP \\ g_4 &= (\sigma+\mu)(\beta_p P + \mu + \mu\gamma_p P) + \mu\gamma_v\beta_p KP. \end{aligned}$$

From the characteristic Eq (2.23), we get 1 eigenvalue is $\lambda = -\frac{V+\alpha P}{P}$ which it always negative real part. The remaining eigenvalue is cubic equation which in this form.

$$\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0. \tag{2.24}$$

Considering the roots of the cubic equation have negative real parts when conditions satisfy the Routh-Hurwitz criterion which are:

$$i) \quad a_1 > 0 \tag{2.25}$$

$$ii) \quad a_3 > 0 \tag{2.26}$$

and

$$iii) \quad a_1a_2 - a_3 > 0. \tag{2.27}$$

For a_1 and a_3 is always positive, then we consider conditions (2.27).
Finally, we obtain

$$\begin{aligned} a_1a_2 - a_3 = &\left(\sigma + \mu + \frac{(V + \alpha P)\beta_p(\sigma + \mu)}{g_4} + \frac{\mu^2\Big(R_0^2(\sigma + \mu)(1 + \gamma_p P + \beta_p P) + \gamma_v\beta_p KP\Big)}{g_2}\right) \\ &\mu\Bigg(\frac{(V + \alpha P)g_3\Big(\sigma(R_0^2 - 1)\Big(R_0^2(\sigma + \mu)(1 + \gamma_p P) + \gamma_v\beta_p KP\Big) + g_1(2R_0^2 - 1)\Big)}{g_2g_4R_0^2P} \\ &+ \frac{g_1(\sigma + \mu)}{g_2}\Bigg) + \frac{(V + \alpha P)\mu g_3}{g_4P}\Bigg(\frac{(V + \alpha P)g_3\Big(R_0^2(\sigma + \mu)(1 + \gamma_p P + \beta_p P) + \gamma_v\beta_p KP\Big)}{g_2g_4R_0^2P} \\ &+ g_1(2R_0^2 - 1) + \frac{g_1(\mu + \sigma)}{g_2R_0^2}\Bigg) \\ > &\, 0. \end{aligned}$$

It can be seen that $a_1 > 0$, $a_3 > 0$, and $a_1a_2 - a_3 > 0$, which corresponds to condition of Routh Hurwitz criterion for the third order polynomial equation. Therefore, The endemic equilibrium point is locally asymptotically stable when $R_0 > 1$.

From this model, we get 2 equilibrium point such as disease-free equilibrium point and endemic equilibrium point. The disease-free equilibrium point is stable when $R_0 < 1$ and the endemic equilibrium point is stable when $R_0 > 1$. This means that to reduce the spread of cassava mosaic disease that we must be set value of the parameter according conditions described above. □

2.4.2. Global stability of the steady state

2.4.2.1. disease-free steady state

For our model in Eqs (2.5)–(2.8), we will to investigate the global stability of disease-free equilibrium point by technique of Castillo-Chavez [29]. We rewrite our model into two subsystems which is in the following form:

$$\begin{aligned} \frac{dY_1}{dt} &= F(Y_1, Y_2) \\ \frac{dY_2}{dt} &= G(Y_1, Y_2). \end{aligned} \tag{2.28}$$

Where Y_1 represent uninfected populations and Y_2 represent infected populations, respectively, that is $Y_1 = (H, X) \in R^2$ and $Y_2 = (I, Y) \in R^2$. E^0 denotes the disease-free equilibrium point and set as $E^0 = (Y_1^0, 0)$. The existence of the globally stability at disease-free equilibrium point for our model must satisfies the following two conditions:
i) For $\frac{dY_1}{dt} = F(Y_1, 0)$, Y_1^0 is globally asymptotically stable.
ii) $G(Y_1, Y_2) = AY_2 - \widehat{G}(Y_1, Y_2)$ where $\widehat{G}(Y_1, Y_2) \leq 0$ for all $(Y_1, Y_2) \in \Omega$.

Where $A = D_{Y_2}G(Y_1^*, 0)$ is M-matrix that is the off diagonal elements are nonnegative and Ω is feasible region. If Eqs (2.5)–(2.8) satisfies with two conditions above then the following the statement holds.

Theorem 4. *If $R_0 < 1$, then the equilibrium point $E_0 = (Y_1^*, 0)$ of the Eqs (2.5)–(2.8) is globally asymptotically stable at the disease-free equilibrium point E_0 and unstable otherwise.*

Proof. Let $Y_1 = (H, X)$ and $Y_2 = (I, Z)$. From Eqs (2.5)–(2.8), we get $F(Y_1, Y_2)$ and $G(Y_1, Y_2)$ is

$$\begin{aligned} F(Y_1, Y_2) &= \begin{bmatrix} \mu K - \frac{\beta_P ZH}{1+\gamma_P Z} - \mu H \\ \alpha P + V - \frac{\beta_V IX}{1+\gamma_V I} - \frac{V}{P}X - \alpha X \end{bmatrix} \\ G(Y_1, Y_2) &= \begin{bmatrix} \frac{\beta_P ZH}{1+\gamma_P Z} - \sigma I - \mu I \\ \frac{\beta_V IX}{1+\gamma_V I} - \frac{V}{P}Z - \alpha Z \end{bmatrix}. \end{aligned} \tag{2.29}$$

Then, we defined $E_0 = (Y_1^0, 0)$ where $Y_1^0 = (K, P)$.
Consider $H = H^0, X = X^0$ and $F(Y_1, 0) = 0$. Therefore, we be obtained

$$F(Y_1, 0) = \begin{bmatrix} \mu K - \mu H \\ \alpha P + V - \frac{V}{P}X - \alpha X \end{bmatrix} = 0. \tag{2.30}$$

Then, we solve Eq (2.30) which we get $Y_1 \to Y_1^0$ as $t \to \infty$.
Hence, it mean that the convergence of Eqs (2.5)–(2.8) is globally asymptotically stable in Ω.
For $G(Y_1, Y_2) = AY_2 - \widehat{G}(Y_1, Y_2)$ and we show that $\widehat{G}(Y_1, Y_2) \geq 0$.
Now, we compute

$$A = \begin{bmatrix} -(\sigma + \mu) & \beta_p K \\ \beta_v P & -\left(\frac{V}{P} + \alpha\right) \end{bmatrix} \tag{2.31}$$

and

$$\widehat{G}(Y_1, Y_2) = \begin{bmatrix} \beta_p KZ - \frac{\beta_p ZH}{1+\gamma_P Z} \\ \beta_v PI - \frac{\beta_V IX}{1+\gamma_V I} \end{bmatrix}. \tag{2.32}$$

That is $K \geq \frac{H}{1+\gamma_P Z}$ and $P \geq \frac{X}{1+\gamma_V I}$. So, $\widehat{G}(Y_1, Y_2) \geq 0$ for all $(Y_1, Y_2) \in \Omega$. Obviously, A is M-matrix and that means both conditions are proved. Therefore, the disease-free equilibrium point E_1 is globally asymptotically stable when $R_0 < 1$. □

2.4.2.2. Endemic steady state

In this section, we consider global stability at endemic equilibrium point E_1^* using geometrical approach [30]. This method is used to investigate and find sufficient conditions for global stability at endemic equilibrium point E_1^*. We describe this method as follows. We consider differential equation

$$\dot{x} = f(x) \tag{2.33}$$

where $x \in \Omega \subset R^n$ is an open set and is simply connected and $f(x) : \Omega \to R^n$ is a continuous and differentiable function in Ω. Let $x(t, x_0)$ is a solution of Eq (2.33) which determined initial value $x(0, x_0) = x_0$. We will hypothesis as follows:

(a) There exists a compact absorbing set $K \subset \Omega$.

(b) System (2.33) has a unique equilibrium $x^* \in \Omega$.

The basic concept of this method is that if x^* is locally stable, then stability must be consistent with condition (a) and (b) and the nonexistence of non-constant periodic solutions of system (2.33). Therefore, the sufficient condition of $f(x)$ can preclusion the existence of such solutions and we found the solution.

Suppose that the condition (a) and (b) are satisfied and if Bendixson criterion which is robust under C^1 local perturbation of f at all non-equilibrium, non-wandering is also satisfied with Eq (2.33), then x^* is globally asymptotically stable in Ω provides it is stable. Let $x \mapsto P(x)$ is a nonsingular $\binom{n}{1} \times \binom{n}{1}$ matrix valued function which is C^1 for $x \in \Omega$.

Assume that P^{-1} exist and continuous for $x \in K$. We give

$$B = P_f P^{-1} + P J^{[3]} P^{-1} \tag{2.34}$$

where P_f is the matrix obtained by substituting in each element p_{ij} in P by the direction derivative in the direction of f and the matrix $J^{[3]}$ is the third additive compound matrix of the Jacobian matrix J that is $J(x) = Df(x)$.

Let $\ell(B)$ is the Lozinskii measure ℓ of B with respect to the vector norm $\|\cdot\|$ in R^n defined by

$$\ell(B) = \lim_{x \to 0^+} \frac{|I + Bx| - 1}{x}. \tag{2.35}$$

Define a quantity $\bar{q}$ is

$$\bar{q} = \lim_{t \to \infty} \sup \sup_{x_0 \in K} \frac{1}{t} \int_0^t \ell(B) dt. \tag{2.36}$$

It is proven in [30] which if Ω is simply connected then $\bar{q} < 0$ eliminate the existence of any orbit that produces a simple closed rectifiable curve that is periodic orbits, homoclinic orbits, and heteroclinic cycles.

Lemma 1. *Suppose that Ω is simply connected and condition (a) and (b) are satisfied then the unique equilibrium x^* of Eq (2.33) is globally asymptotically stable in Ω if $\bar{q} < 0$.*

Theorem 5. *If $\frac{(H^*)^2}{1+\gamma_p Z^*}(\frac{1}{X^*} + \frac{1}{Z^*}) < Z^*$, $\frac{\beta_v (I^*)^2}{H^*(1+\gamma_v I^*)} < \frac{\beta_p Z^*}{1+\gamma_p Z^*}$, $\frac{(X^*)^2}{(1+\gamma_v I^*)}(\frac{1}{H^*} + \frac{1}{I^*}) < I^*$, and $\frac{\beta_p (Z^*)^2}{X^*(1+\gamma_p Z^*)} < \frac{\beta_v I^*}{1+\gamma_v I^*}$ and if $R_0 > 1$, then the Eqs (2.5)–(2.8) is globally asymptotically stable at the endemic equilibrium E^* and unstable otherwise.*

Proof. We prove the globally asymptotically stability of this model which is Eqs (2.5)–(2.8) with endemic equilibrium point, we consider the nonlinear equations in Eqs (2.5)–(2.8).

$$\begin{aligned}
\frac{dH}{dt} &= \mu K - \frac{\beta_P Z H}{1 + \gamma_p Z} - \mu H, \\
\frac{dI}{dt} &= \frac{\beta_p Z H}{1 + \gamma_p Z} - \sigma I - \mu I, \\
\frac{dX}{dt} &= \alpha P + V - \frac{\beta_v I X}{1 + \gamma_v I} - \frac{V}{P} X - \alpha X, \\
\frac{dZ}{dt} &= \frac{\beta_v I X}{1 + \gamma_v I} - \frac{V}{P} Z - \alpha Z
\end{aligned} \tag{2.37}$$

for which the Jacobian matrix at disease-endemic equilibrium points is:

$$J(E^*) = \begin{bmatrix} -\mu - \frac{Z^*\beta_p}{1+\gamma_p Z^*} & 0 & 0 & -\frac{\beta_p H^*}{(1+\gamma_p Z^*)^2} \\ \frac{\beta_p Z^*}{1+\gamma_p Z^*} & -\mu-\sigma & 0 & \frac{\beta_p H^*}{(1+\gamma_p Z^*)^2} \\ 0 & -\frac{\beta_v X^*}{(1+\gamma_v I^*)^2} & -\alpha - \frac{V}{P} - \frac{\beta_v I^*}{1+\gamma_v I^*} & 0 \\ 0 & \frac{\beta_v X^*}{(1+\gamma_v I^*)^2} & \frac{\beta_v I^*}{1+\gamma_v I^*} & -\alpha - \frac{V}{P} \end{bmatrix}. \tag{2.38}$$

Furthermore, the general form of third additive compound matrix $J^{|3|}$, is given by

$$J^{|3|} = \begin{bmatrix} j_{11} + j_{22} + j_{33} & j_{34} & -j_{24} & j_{14} \\ j_{43} & j_{11} + j_{22} + j_{44} & j_{23} & -j_{13} \\ -j_{42} & j_{32} & j_{11} + j_{33} + j_{44} & j_{12} \\ j_{41} & -j_{31} & j_{21} & j_{22} + j_{33} + j_{44} \end{bmatrix} \tag{2.39}$$

from matrix Eqs (2.38) and (2.39) implies that

$$J^{|3|} = \begin{bmatrix} -j_{11} & 0 & -\frac{\beta_p H^*}{(1+\gamma_p Z^*)^2} & -\frac{\beta_p H^*}{(1+\gamma_p Z^*)^2} \\ \frac{\beta_v I^*}{1+\gamma_v I^*} & -j_{22} & 0 & 0 \\ -\frac{\beta_v X^*}{(1+\gamma_v I^*)^2} & -\frac{\beta_v X^*}{(1+\gamma_v I^*)^2} & -j_{33} & 0 \\ 0 & 0 & \frac{\beta_p Z^*}{1+\gamma_p Z^*} & -j_{44} \end{bmatrix}. \tag{2.40}$$

Where $j_{11} = \alpha+2\mu+\sigma+\frac{V}{P}+\frac{\beta_v I^*}{1+\gamma_v I^*}+\frac{\beta_p Z^*}{1+\gamma_p Z^*}$, $j_{22} = \alpha+2\mu+\sigma+\frac{V}{P}+\frac{\beta_p Z^*}{1+\gamma_p Z^*}$, $j_{33} = 2\alpha+\mu+\frac{2V}{P}+\frac{\beta_v I^*}{1+\gamma_v I^*}+\frac{\beta_p Z^*}{1+\gamma_p Z^*}$, and $j_{44} = 2\alpha + \mu + \sigma + \frac{2V}{P} + \frac{\beta_v I^*}{1+\gamma_v I^*}$.

Consider $P(X) = diag\{H(t), I(t), X(t), Z(t)\}$, the inverse of $P(X)$ is given as $P^{-1}(X) = diag\{1/H(t), 1/I(t), 1/X(t), 1/Z(t)\}$, the derivative with respect to time is $P_f(X) = diag\{\dot{H}(t), \dot{I}(t), \dot{X}(t), \dot{Z}(t)\}$, while $P_f P^{-1} = diag\{\dot{H}(t)/H(t), \dot{I}(t)/I(t), \dot{X}(t)/X(t), \dot{Z}(t)/Z(t)\}$, and

$$PJ^{|3|}P^{-1} = \begin{bmatrix} -j_{11} & 0 & a_{13} & a_{14} \\ \frac{\beta_v (I^*)^2}{H^*(1+\gamma_v I^*)} & -j_{22} & 0 & 0 \\ a_{31} & a_{32} & -j_{33} & 0 \\ 0 & 0 & \frac{\beta_p (Z^*)^2}{X^*\left(1+\gamma_p Z^*\right)} & -j_{44} \end{bmatrix}. \tag{2.41}$$

Where $a_{13} = -\frac{H^*}{X^*}\left(\frac{\beta_p H^*}{(1+\gamma_p Z^*)^2}\right)$, $a_{14} = -\frac{H^*}{Z^*}\left(\frac{\beta_p H^*}{(1+\gamma_p Z^*)^2}\right)$, $a_{31} = -\frac{X^*}{H^*}\left(\frac{\beta_v X^*}{(1+\gamma_v I^*)^2}\right)$, and $a_{32} = -\frac{X^*}{I^*}\left(\frac{\beta_v X^*}{(1+\gamma_v I^*)^2}\right)$.

Furthermore, $B = P_f P^{-1} + PJ^{|3|}P^{-1}$ that is

$$B = \begin{bmatrix} \frac{\dot{H}(t)}{H(t)} - j_{11} & 0 & a_{13} & a_{14} \\ \frac{\beta_v (I^*)^2}{H^*(1+\gamma_v I^*)} & \frac{\dot{I}(t)}{I(t)} - j_{22} & 0 & 0 \\ a_{31} & a_{32} & \frac{\dot{X}(t)}{X(t)} - j_{33} & 0 \\ 0 & 0 & \frac{\beta_p (Z^*)^2}{X^*\left(1+\gamma_p Z^*\right)} & \frac{\dot{Z}(t)}{Z(t)} - j_{44} \end{bmatrix}. \tag{2.42}$$

Now, consequently we are to find $\hbar_i(t)$, $i = 1, 2, 3, 4$, by assuming that B_{ij} are the entries of matrix B,

such that

$$\begin{aligned}
\hbar_1(t) &= B_{11} + \sum_{j\neq 1\wedge j=2}^{4} |B_{1j}| \\
\hbar_2(t) &= B_{22} + \sum_{j=1\wedge j\neq 2}^{4} |B_{2j}| \\
\hbar_3(t) &= B_{33} + \sum_{j=1\wedge j\neq 3}^{4} |B_{3j}| \\
\hbar_4(t) &= B_{44} + \sum_{j=1\wedge j\neq 4}^{4} |B_{4j}|.
\end{aligned} \tag{2.43}$$

Now, for $\hbar_1(t)$ if $\frac{(H^*)^2}{1+\gamma_p Z^*}(\frac{1}{X^*}+\frac{1}{Z^*}) < Z^*$, then

$$\begin{aligned}
\hbar_1(t) &= \frac{\dot{H}(t)}{H(t)} - (\alpha+2\mu+\sigma+\frac{V}{P}) - \left(\frac{\beta_v I^*}{1+\gamma_v I^*} + \frac{\beta_p Z^*}{1+\gamma_p Z^*}\right) + \left|-\frac{H^*}{X^*}\left(\frac{\beta_p H^*}{(1+\gamma_p Z^*)^2}\right)\right| \\
&\quad + \left|-\frac{H^*}{Z^*}\left(\frac{\beta_p H^*}{(1+\gamma_p Z^*)^2}\right)\right| \\
\hbar_1(t) &= \frac{\dot{H}(t)}{H(t)} - (\alpha+2\mu+\sigma+\frac{V}{P}) - \left(\frac{\beta_v I^*}{1+\gamma_v I^*} + \frac{\beta_p Z^*}{1+\gamma_p Z^*}\right) + H^*\left(\frac{\beta_p H^*}{\left(1+\gamma_p Z^*\right)^2}\right) \\
&\quad \times\left(\frac{1}{X^*}+\frac{1}{Z^*}\right) \\
\hbar_1(t) &\leq \frac{\dot{H}(t)}{H(t)} - (\alpha+2\mu+\sigma+\frac{V}{P}).
\end{aligned} \tag{2.44}$$

For $\hbar_2(t)$ if $\frac{\beta_v (I^*)^2}{H^*(1+\gamma_v I^*)} < \frac{\beta_p Z^*}{1+\gamma_p Z^*}$, then

$$\begin{aligned}
\hbar_2(t) &= \frac{\dot{I}(t)}{I(t)} - (\alpha+2\mu+\sigma+\frac{V}{P}) - \frac{\beta_p Z^*}{1+\gamma_p Z^*} + \left|\frac{\beta_v (I^*)^2}{H^*(1+\gamma_v I^*)}\right| \\
\hbar_2(t) &= \frac{\dot{I}(t)}{I(t)} - (\alpha+2\mu+\sigma+\frac{V}{P}) - \frac{\beta_p Z^*}{1+\gamma_p Z^*} + \frac{\beta_v (I^*)^2}{H^*(1+\gamma_v I^*)} \\
\hbar_2(t) &\leq \frac{\dot{I}(t)}{I(t)} - (\alpha+2\mu+\sigma+\frac{V}{P}).
\end{aligned} \tag{2.45}$$

If, $\frac{(X^*)^2}{(1+\gamma_v I^*)}(\frac{1}{H^*}+\frac{1}{I^*}) < I^*$, then

$$\begin{aligned}
\hbar_3(t) &= \frac{\dot{X}(t)}{X(t)} - (2\alpha+\mu+\frac{2V}{P}) - \left(\frac{\beta_v I^*}{1+\gamma_v I^*} + \frac{\beta_p Z^*}{1+\gamma_p Z^*}\right) + \left|-\frac{X^*}{H^*}\left(\frac{\beta_v X^*}{(1+\gamma_v I^*)^2}\right)\right| \\
&\quad + \left|-\frac{X^*}{I^*}\left(\frac{\beta_v X^*}{(1+\gamma_v I^*)^2}\right)\right|
\end{aligned}$$

$$\hbar_3(t) = \frac{\dot{X}(t)}{X(t)} - (2\alpha + \mu + \frac{2V}{P}) - \left(\frac{\beta_v I^*}{1+\gamma_v I^*} + \frac{\beta_p Z^*}{1+\gamma_p Z^*}\right) + \left(\frac{\beta_v (X^*)^2}{(1+\gamma_v I^*)^2}\right) \times \left(\frac{1}{H^*} + \frac{1}{I^*}\right)$$

$$\hbar_3(t) \leq \frac{\dot{X}(t)}{X(t)} - (2\alpha + \mu + \frac{2V}{P}) \tag{2.46}$$

and, similarly for $\frac{\beta_p (Z^*)^2}{X^*\left(1+\gamma_p Z^*\right)} < \frac{\beta_v I^*}{1+\gamma_v I^*}$, then

$$\hbar_4(t) = \frac{\dot{Z}(t)}{Z(t)} - (2\alpha + \mu + \sigma + \frac{2V}{P}) - \frac{\beta_v I^*}{1+\gamma_v I^*} + \left|\frac{\beta_p (Z^*)^2}{X^*\left(1+\gamma_p Z^*\right)}\right|$$

$$\hbar_4(t) = \frac{\dot{Z}(t)}{Z(t)} - (2\alpha + \mu + \sigma + \frac{2V}{P}) - \frac{\beta_v I^*}{1+\gamma_v I^*} + \frac{\beta_p (Z^*)^2}{X^*\left(1+\gamma_p Z^*\right)}$$

$$\hbar_4(t) \leq \frac{\dot{Z}(t)}{Z(t)} - (2\alpha + \mu + \sigma + \frac{2V}{P}). \tag{2.47}$$

Now, in R^4 we assume a vector (b_1, b_2, b_3, b_4). The Lozinski measure $\ell(B)$ is defined as $\ell(B) = \hbar_i$, $i = 1, 2, 3, 4$. The integration of the Lozinski measure $\ell(B)$ and taking the limits as $t \to \infty$ lead to the following equations:

$$\bar{q_1} = \lim_{t\to\infty} \sup \sup \frac{1}{t} \int_0^t \hbar_1(t)dt,$$

$$\leq \lim_{t\to\infty} \sup \sup \frac{1}{t} \int_0^t \frac{\dot{H}(t)}{H(t)} - \left(\alpha + 2\mu + \sigma + \frac{V}{P}\right)dt,$$

$$\bar{q_1} < -\left(\alpha + 2\mu + \sigma + \frac{V}{P}\right). \tag{2.48}$$

$$\bar{q_2} = \lim_{t\to\infty} \sup \sup \frac{1}{t} \int_0^t \hbar_2(t)dt,$$

$$\leq \lim_{t\to\infty} \sup \sup \frac{1}{t} \int_0^t \frac{\dot{I}(t)}{I(t)} - \left(\alpha + 2\mu + \sigma + \frac{V}{P}\right)dt,$$

$$\bar{q_2} < -\left(\alpha + 2\mu + \sigma + \frac{V}{P}\right). \tag{2.49}$$

$$\bar{q_3} = \lim_{t\to\infty} \sup \sup \frac{1}{t} \int_0^t \hbar_3(t)dt,$$

$$\leq \lim_{t\to\infty} \sup \sup \frac{1}{t} \int_0^t \frac{\dot{X(t)}}{X(t)} - \left(2\alpha + \mu + \frac{2V}{P}\right)dt,$$

$$\bar{q_3} < -\left(2\alpha + \mu + \frac{2V}{P}\right). \tag{2.50}$$

$$\bar{q_4} = \lim_{t\to\infty} \sup \sup \frac{1}{t} \int_0^t \hbar_4(t)dt$$
$$\leq \lim_{t\to\infty} \sup \sup \frac{1}{t} \int_0^t \frac{\dot{Z(t)}}{Z(t)} - \left(2\alpha + \mu + \sigma + \frac{2V}{P}\right)dt,$$
$$\bar{q_4} < -\left(2\alpha + \mu + \sigma + \frac{2V}{P}\right). \tag{2.51}$$

Now, the combination of the last four inequalities from Eqs (2.48)–(2.51)

$$\bar{q} = \lim_{t\to\infty} \sup \sup_{E_1\in K} \frac{1}{t} \int_0^t \ell(B)dt < 0. \tag{2.52}$$

The system containing only four non-linear equations of model Eqs (2.5)–(2.8) is globally asymptotically stable around its interior equilibrium (H^*, I^*, X^*, Z^*) when $R_0 > 1$. □

3. Numerical results

In this section, we consider numerical solutions of transmission of the cassava mosaic disease by Matlab which shown disease-free and endemic regions. The parameter values in Table 2 are derived from literature and some values have been assumed. Normally, cassava takes about 375 days to harvest. Therefore, we set $\mu = 1/375$ per day. The whitefly lives for about 37.5 days, so $\alpha = 1/37.5$ per day. The initial value of our model is $H(0) = 800, I(0) = 200, X(0) = 150$ and $Z(0) = 50$.

Table 2. Show the parameter values for model of cassava mosaic disease.

Parameter symbol	Value	Reference
μ	$\frac{1}{375}$	[31]
σ	0.1	[16]
β_v	0.0002	Assumption
α	$\frac{1}{37.5}$	[18]
γ_p	0.01	[32]
γ_v	0.01	[32]
V	10	Assumption
β_p	0-1	Assumption

The equations of the model defined (2.5) to (2.8) were analyzed using the parameter values in Table 2. We examine the behavior of system and show the globally stability of the disease-free and endemic state. From Figure 1, we calculate and get $R_0 = 0.71283 < 1$. We can conclude that the model exists a globally asymptotically stable at the disease free equilibrium point consistent with Theorem 4. Similarly, we obtained $R_0 = 5.96432 > 1$ which is shown in Figure 2. It shows the numerical trajectory of the existence of the globally asymptotically stable at the endemic equilibrium point consistent with Theorem 5.

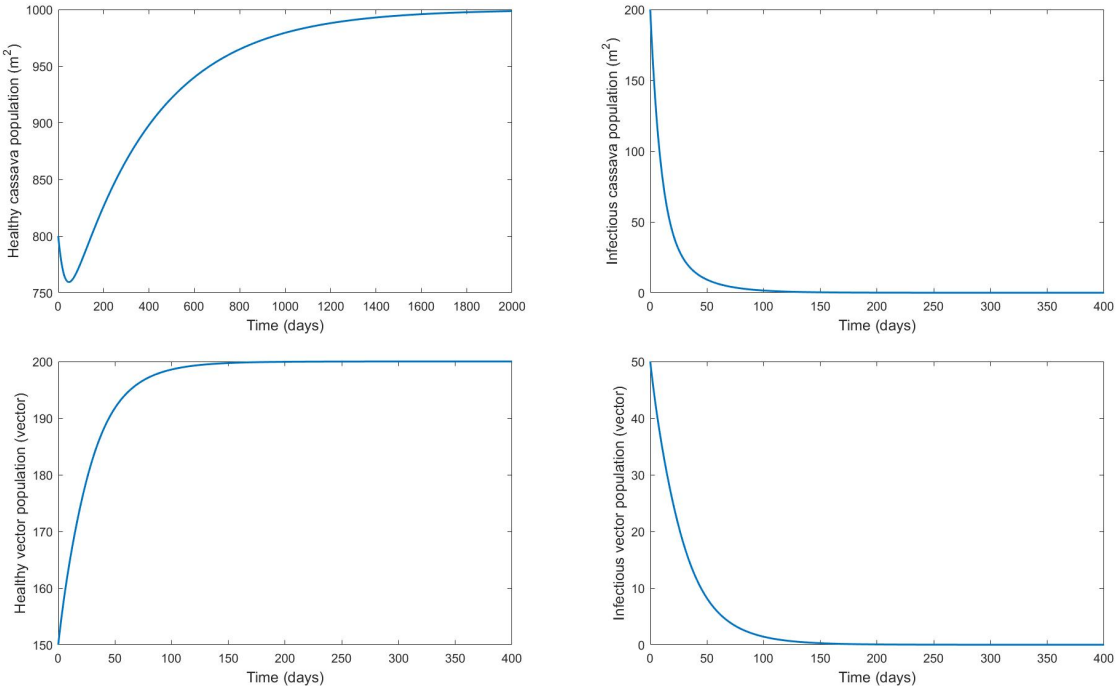


Figure 1. Time series of healthy cassava, infectious cassava, healthy vector and infectious vector, for $R_0 < 1$. The value of $\beta_p = 0.0001$ and $R_0 = 0.71287$. The fractions of populations approach to the disease-free equilibrium point $(1000, 0, 200, 0)$.

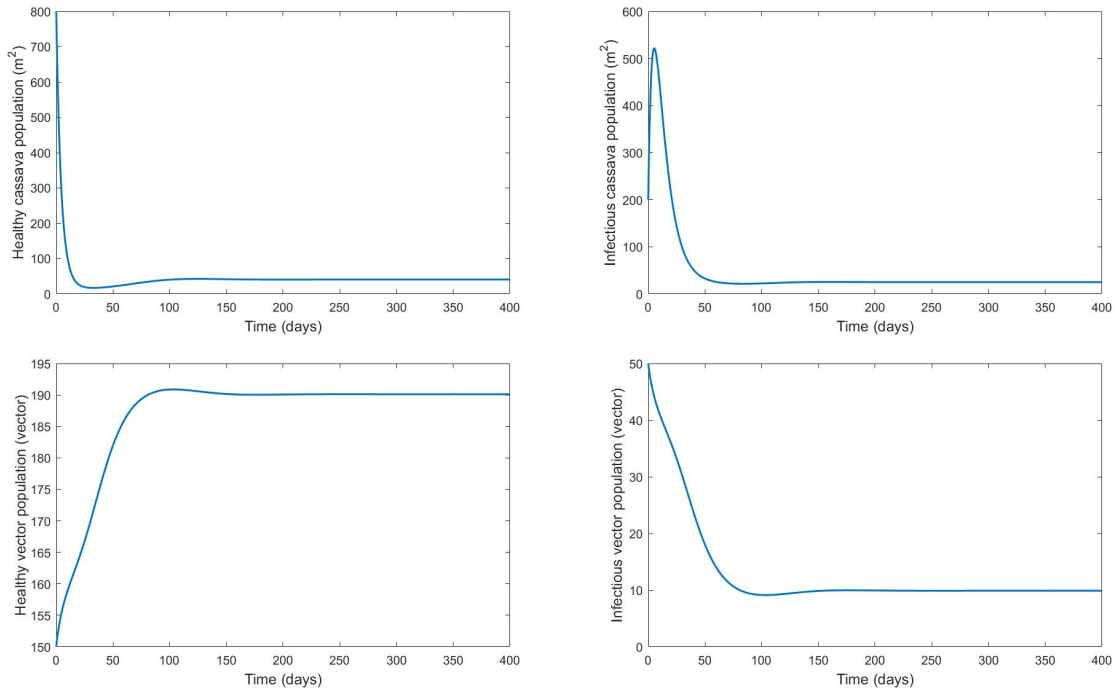


Figure 2. Time series of healthy cassava, infectious cassava, healthy vector and infectious vector, for $R_0 > 1$. The value of $\beta_p = 0.007$ and $R_0 = 5.96432$. The fractions of populations approach to the endemic equilibrium point $(40.5986, 24.9195, 190.1069, 9.8931)$.

3.1. Sensitivity analysis of the model parameter

Sensitivity analysis is also considered the best method to reduce mortality and the incidence of cassava mosaic disease in cassava. We performed a sensitivity analysis to find the correlation between the model parameters on the spread of disease. The analysis demonstrated the effect of parameters on the basic reproduction number. The explicit expression of R_0, is given by

$$R_0 = P\sqrt{\frac{\beta_v\beta_p K}{(\sigma+\mu)(V+\alpha P)}}. \tag{3.1}$$

We analyzed it following Rodrigues et al. [33]. This technique was developed and obtained a formula for obtaining the parameter sensitivity index.

Definition 1. The normalized forward sensitivity index of R_0, which depends differentiable on a parameter, Λ is defined by

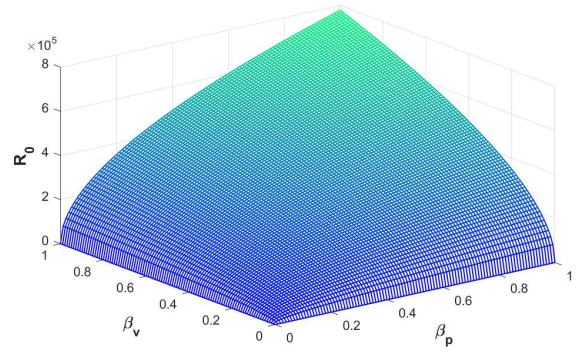
$$\Upsilon_{\Lambda}^{R_0} = \frac{\partial R_0}{\partial \Lambda} \times \frac{\Lambda}{R_0}. \tag{3.2}$$

All sensitivity indices have been operated and are shown in Table 2.

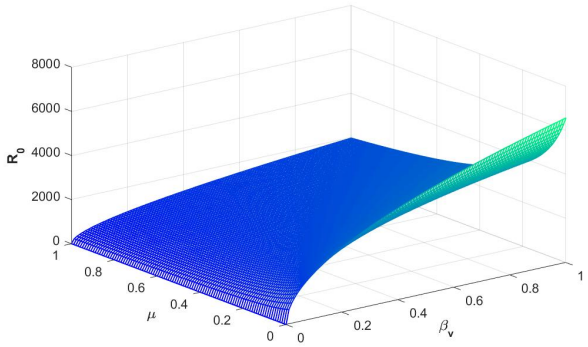
From the sensitivity indices of the basic reproductive number with respect to the parameter in Table 2 then we obtained the sensitivity index for parameter values shown in Table 3. The parameters that have positive sensitivity indices, i.e., β_v and β_p have a positive effect on the basic reproductive number. As these parameters increase, the basic reproductive number also increases, so the average number of secondary infections increases. It means that β_v and β_p increase make the number of the infectious cassava population and the infectious vector population may lead to an outbreak. Furthermore, the parameters that have negative sensitivity indices, i.e., μ, V, σ and α have a negative effect on the basic reproductive number. It means that μ, V, σ and α is increase the basic reproductive number is decrease. Therefore, if μ, V, σ and α increases while the others parameter are constant then it results in a reduction in the spread of this disease. We want to show how each parameter affects R_0 which each sensitivity parameter is compared with R_0, which is shown in Figure 3.

Table 3. Sensitivity indices (3.2) of the basic reproductive number by the parameters used in the calculation of endemic equilibrium point.

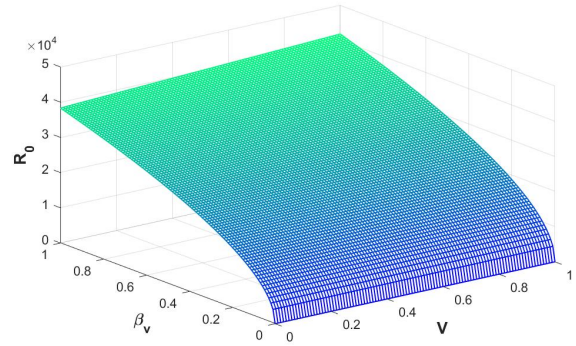
Parameter symbol	Sensitivity index	Sensitivity index for parameter values
β_v	0.5	0.5
β_p	0.5	0.5
μ	$-(2\mu+\sigma)\sqrt{\frac{\mu+\sigma}{\mu}}$	-0.653576
V	$-\frac{V}{2(V+\alpha P)}$	-0.326087
α	$-\frac{\alpha P}{2(V+\alpha P)}$	-0.173913
σ	$-\frac{\sigma}{\sigma+\mu}$	-0.974026



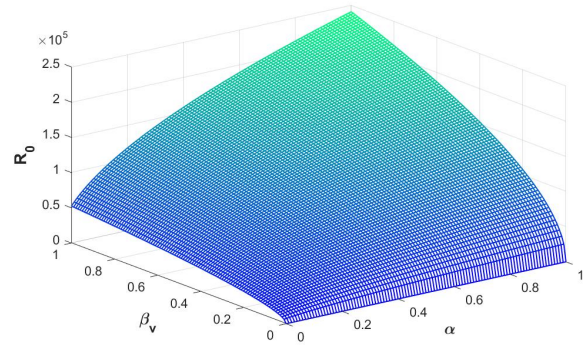
(a) parameter β_v and β_p are compared with R_0



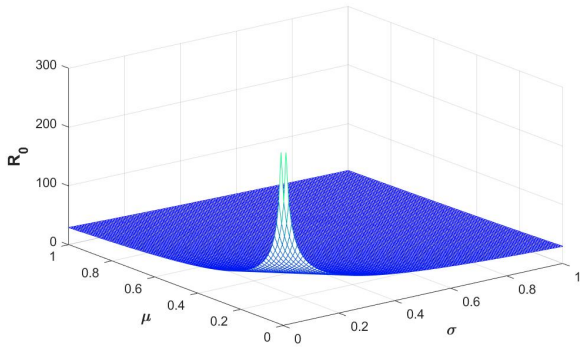
(b) parameter μ and β_v are compared with R_0.



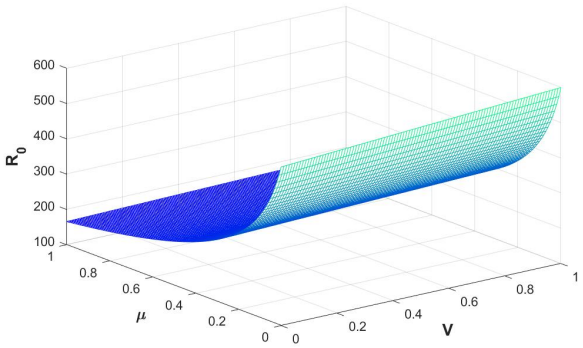
(c) parameter β_v and V are compared with R_0.



(d) parameter β_v and α are compared with R_0.



(e) parameter μ and σ are compared with R_0.



(f) parameter μ and V are compared with R_0.

Figure 3. The simulation results of the sensitivity analysis were compared with different parameters.

4. Discussion

In this paper, we have presented and analyzed a differential system of the model of cassava mosaic disease. We considered the transmission response of the disease with a saturation incidence rate, which is biological truth. For this model, our primary objective is to study the effect of whitefly on

the spread of cassava mosaic disease. From the epidemiological model, We found 2 steady state: disease-free steady state and endemic steady state. We obtain the basic reproduction number or R_0 which is a method used to measure the spread of disease by using the next-generation method. The basic reproduction number for this model is $P\sqrt{\frac{\beta_v\beta_p K}{(\sigma+\mu)(V+\alpha P)}}$. Our results show that R_0 is less than 1 in which the infected cassava population decreases and the disease are eventually eliminated. For R_0 is greater than 1, the infected cassava population will increase sequentially (multiple) and this will cause an epidemic. We have obtained the necessary conditions for locally asymptotically stability when $R_0 < 1$ in Theorem 2 which is the disease-free steady state. For $R_0 > 1$, we have shown in Theorem 3 which is the endemic steady state. Furthermore, Theorems 4 and 5 confirmed the existence of globally asymptotically stability by application of Castillo-Chavez's method for disease-free steady state and geometric approach method for endemic steady state. Finally, numerical simulations with true parameters were used to confirm the theoretical analysis results. It shows that over time we can control and eliminate cassava mosaic disease.

Our goal is to describe cassava mosaic disease by determining the severity of the disease and identifying the parameters that most affect the stability of the model. Parameter sensitivity analyses revealed parameters that influence the spread of this disease. It makes known which parameters cause the disease to persist and disappear. We can see that $\beta_v = 0.5$ and $\beta_p = 0.5$ is positive, thus affecting the increase of basic reproductive number. An increase in the basic reproductive number has resulted in increased outbreaks and indicates that the transmission of the disease largely depends on the infection rate (acquisition rate and inoculation rate). Therefore, we should try to minimize contact with infected cassava and the susceptible whitefly, as well as healthy cassava and infected whitefly. Moreover, the increase of parameters σ and μ made the basic reproductive number lower. It means that if we increase the mortality rate of whitefly and the removal rate of infected cassava then fewer cassava mosaic disease outbreaks. This will be a guideline for increasing the policy to reduce the epidemic. Therefore, the whitefly should be eradicated with pesticides to reduce the spread of infected whitefly and get rid of the infected cassava by burning them in the field before the whitefly arrives. In addition to the analysis of factors affecting the outbreak of mosaic cassava disease and control strategies. We see that the best and most cost-effective disease control strategies, as well as optimal costs, are interesting. It is necessary for farmers and they must consider using strategies reasonably in choosing ways to reduce mosaic cassava disease outbreaks. Therefore, the optimal control method will be considered in the next paper.

5. Conclusions

We developed a mathematical model to describe the epidemic dynamics of cassava mosaic disease. The basic reproductive number is obtained which was established as criteria for the stability analysis of the disease and showed that the disease would be stable both locally and globally. The theoretical possibility of our model was confirmed by numerical simulations. Finally, the parameters affecting the persistence and extinction of this disease can be identified through sensitivity analysis and have been shown. We found that the acquisition rate and inoculation rate influenced the increase in disease outbreaks.

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Conflict of interest

The authors declare no conflicts of interest.

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