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Research article

Mathematical modelling and analysis of coffee berry disease dynamics on a coffee farm

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Abstract: This paper focuses on a mathematical model for coffee berry disease infestation dynamics. This model considers coffee berry and vector populations with the interaction of fungal pathogens. In order to gain an insight into the global dynamics of coffee berry disease transmission and eradication on any given coffee farm, the assumption of logistic growth with a carrying capacity reflects the fact that the amount of coffee plants depends on the limited size of the coffee farm. First, we show that all solutions of the chosen model are bounded and non-negative with positive initial data in a feasible region. Subsequently, endemic and disease-free equilibrium points are calculated. The basic reproduction number with respect to the coffee berry disease-free equilibrium point is derived using a next generation matrix approach. Furthermore, the local stability of the equilibria is established based on the Jacobian matrix and Routh Hurwitz criteria. The global stability of the equilibria is also proved by using the Lyapunov function. Moreover, bifurcation analysis is proved by the center manifold theory. The sensitivity indices for the basic reproduction number with respect to the main parameters are determined. Finally, the numerical simulations show the agreement with the analytical results of the model analysis.

Keywords: mathematical model; coffee berry; colletotrichum kahawae; bifurcation analysis; numerical simulations

1. Introduction

Coffee is a plantation crop that is well adapted to different eco-physiological conditions of tropical and subtropical highlands. It is the favourite beverage and second most traded commodity next to crude oil in the world [1]. The coffee industry is estimated to be over $100 billion worldwide with an average consumption of 500 billion cups per year. It is mostly consumed in the developed nations and produced by tropical countries, often less developed, being a big source of their economy [1, 2]. However,

coffee production has been challenged by diseases, weeds and pests. For instance, coffee berry disease (CBD) caused by *Colletotrichum kahawae* is a major challenge to Arabica coffee production in African countries [3]. It is a fungal plant pathogen [4, 5]. The symptoms of CBD include black depressed wounds on coffee berries (see Figure 1) [6].

Figure 1. Coffee berries infected with coffee berry disease (CBD).

The first report of CBD (*Colletotrichum kahawae*) dates back to 1922 in western Kenya [7]. Soon after, the fungus quickly spread throughout most of the African countries (see Figure 2) [8]. The disease attacks flowers and fruits at all stages of growth, but it is more destructive to young berries especially during the expanding period 4–l6 weeks after flowering [9]. This disease decreases both the quality and yield of coffee berries. For instance, Ethiopia, Kenya and Cameroon annually lose up to 29.9, 75 and 60%, respectively. Losses may reach up to 100% [2, 10] in high rainfall, humidity and altitude areas.

Figure 2. Spread of CBD across Africa.

Because CBD can become very severe and there is a lack of effective control measures, there is much concern that the fungus could spread to coffee-growing areas on other continents. Currently, however, the disease is only prevalent in Africa at high altitudes with relatively high humidity [11]. There are various applications for CBD management such as chemical treatments, cultural practices, the use of resistant varieties and biological control [12–18]. Several chemical products (fungicides) have been evaluated and recommended for CBD control measures such as different copper fungicides, organic fungicides and mixtures of the two. For instance, fungicide spray against CBD starting six weeks after the main flowering for six rounds at a four-week interval during a crop season was recommended in Ethiopia. Cultural practices include a variety of management tactics, such as mixed cropping with shade plants, pruning infected branches, the destruction of infected material and the removal of mummified berries to create environmental conditions that limit CBD development [14]. Chemical control seems the most effective method but if incorrectly used, it causes ecological risks. On the contrary, the use of resistant varieties and cultural practices is cost-effective, biologically safe and environmentally friendly.

Mathematics is becoming an important tool for studying the evolution of plant pests and diseases. Some mathematical models have been formulated and analyzed to explain the dynamics of plant disease transmission and assess their controls. For example, Fotsa et al. [19] introduced the mathematical modelling and optimal control of Anthracnose disease. Anthracnose attacks a wide range of commercial crops, including coffee, mango, banana, blueberry, cherry, and strawberry. Cunniffe and Gilligan [20] investigated epidemiological models for plant pathogens. These models ensured maximum transferability across the widest range of host-pathogen systems by using the common currency of the field; they illustrated the results in a class of model that has been experimentally tested for plant disease. Fotso et al. [21] introduced and analyzed a mathematical model of coffee berry borer with optimal control strategies. Among coffee diseases, CBD, coffee wilt disease, and coffee leaf rust are the most destructive diseases threatening coffee production in developing countries like Ethiopia. However, according to the survey results, mostly CBD was more widespread than the other coffee diseases [22]. In all the previous studies, the mathematical model for CBD epidemics concerning fungal pathogen and vector population was not considered. In the present work, we developed a nonlinear deterministic mathematical model for the dynamics of CBD infestation on a coffee farm; and also we also applied their qualitative analysis.

2. Materials and methods

2.1. Biological background and pathosystem

The reproduction of the coffee tree involves flowers containing both females and males and their pollination occurs mainly by wind and to a lesser extent by insects [23]. CBD is locally spread between coffee trees and branches by wind and rain [11]. But, the common vectors of long- and medium-distance CBD dispersal are insects, birds, and coffee harvesters [6]. Hence, we assumed those common vectors that, after contacting a fungal pathogen from the environment or already CBD infected coffee berries, then contact healthy coffee berries as responsible vectors for the spread of CBD. The fungal pathogen in the environment may be transported to the coffee plant via infected vectors; once the coffee berry becomes infected, the fungus increases within the infected coffee and destroys it. Twig bark, flower cushions, and mummified berries are considered to be sources of primary inoculum [24].

Most diseased berries drop prematurely, but those that stick to the branches are the main sources of the secondary inoculum (new *Colletotrichum kahawae*) conidia that are dispersed to contaminate other healthy berries.

The CBD is highly dependent upon climatic factors: humidity, rainfall, and temperature [25–27]. These climatic conditions conducive to *Colletotrichum kahawae* typically occur at high elevations greater than 1600 m (i.e., the altitude at which coffee Arabica is grown); disease incidence is minimal below 1000 m [24]. The optimal temperatures for conidium germination and mycelium growth are in the range of 20 to 22°C for CBD [28]. An increase of 0.61°C in the global mean temperature has been recorded since the beginning of the twentieth century and the predicted warming of 2–6°C by 2100 have direly increased the need to understand the impacts of climate change [29]. Most insects in temperate climates have optimum development at temperatures between 20 and 35°C. The total development time from egg to adult was 89.6 days at 20°C, 63.10 days at 25°C and 55.81 days at 30°C, and no development at 35°C [30]. Whereas at temperatures below 15°C mating is limited, and movement such as flying becomes stalled [31]. Most conidia of *Colletotrichum kahawae* can be effectively dispersed by an optimum rainfall of 10 mm but heavy rainfall leads more to their leaching from the coffee tree canopy to the soil [32, 33].

2.2. *Mathematical model*

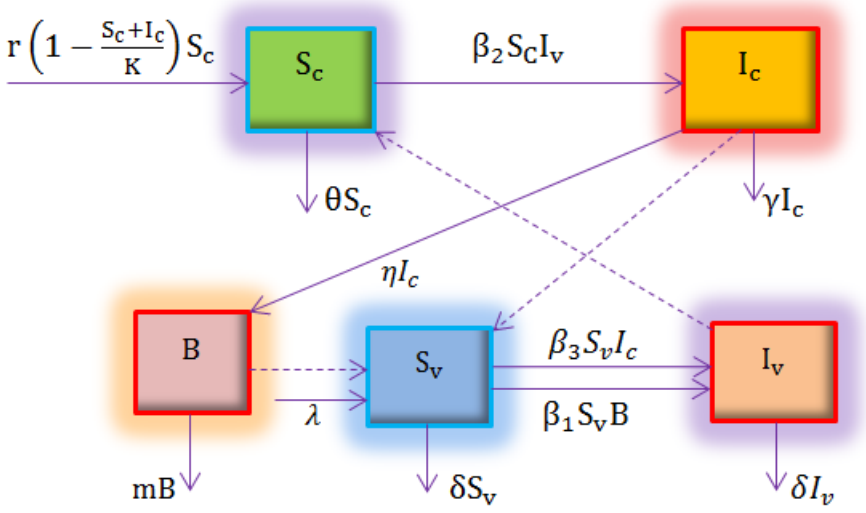


Figure 3. Flow diagram of CBD transmission.

This study considers coffee berry and vector populations with the interaction of a fungal pathogen (B). The coffee berry population is considered into two forms: the susceptible coffee berry (S_c) and infected coffee berry (I_c). Due to the limited size of a coffee plantation, we adopted a logistic growth function for the biomass density of coffee berries. The coffee berry population grows logistically [34] at a growth rate r and an environmental carrying capacity K. Susceptible coffee berries move to the infected class following contacts with an infected vector at a per capita rate β_2; its mortality rate is θ. The infected coffee berry's death rate is γ due to the disease. The coffee berry, once becoming

infected, never recovers, and gives no or very low yield of coffee. The vector population is divided into two form: the susceptible vector (S_v) and infected vector (I_v). The infected vectors (insect, birds) are assumed to transport the fungal pathogen to the coffee berry. The vectors can be infected from the fungal pathogen in the environment at rate β_1 or from the infected coffee berry at rate β_3. The susceptible vector is recruited at rate λ. The vectors' death rate is represented by δ. The fungal pathogens in the environment may be transported to the coffee plant via the infected vectors, once the coffee berry becomes infected, the fungal increases within the infected coffee and destroy it. This invariably adds to the fungal pathogens in the environment at rate η and the infected coffee berry fungal pathogen contribution to the environment is (ηI_c). The fungal pathogen's decay rate is m. The flow diagram of CBD transmission is illustrated in Figure 3. For further information, the parameters and their biological meanings are given in Table 1.

The governing mathematical model is given by

$$\begin{cases} \dfrac{dS_c}{dt} = rS_c\left(1 - \dfrac{S_c + I_c}{K}\right) - \beta_2 S_c I_v - \theta S_c, \\ \dfrac{dI_c}{dt} = \beta_2 S_c I_v - (\gamma + \eta) I_c, \\ \dfrac{dS_v}{dt} = \lambda - (\beta_1 B + \beta_3 I_c) S_v - \delta S_v, \\ \dfrac{dI_v}{dt} = (\beta_1 B + \beta_3 I_c) S_v - \delta I_v, \\ \dfrac{dB}{dt} = \eta I_c - mB. \end{cases} \tag{2.1}$$

Together with initial condition:

$$S_c(0) = S_{c^0} > 0,\ I_c(0) = I_{c^0} \geq 0,\ S_v(0) = S_{v^0} > 0,\ I_v(0) = I_{v^0} \geq 0,\ B(0) = B_0 \geq 0. \tag{2.2}$$

Table 1. Meaning of the parameters of model (2.1) with corresponding values.

Parameter	Description	Unit	Value	Source
β_1	Contact rate of vector with pathogen environment	day^{-1}	0.000209818	Estimated
β_2	Contact rate of coffee berries with infected vector	day^{-1}	0.000795455	Estimated
β_3	Contact rate of vector with infected coffee berries	day^{-1}	0.000149091	Estimated
θ	Mortality rate of healthy coffee berries	day^{-1}	0.01	[35]
γ	Removal rate of infected coffee berries	day^{-1}	0.005	Estimated
r	Growth rate of new coffee berries	day^{-1}	0.12	[36]
K	Carrying capacity	m^{-2}	150	Estimated
η	Induced rate of infected coffee berries	$\mho$	0.009	Estimated
λ	Recruitment rate of vector	day^{-1}	0.488364	Estimated
m	Decay rate of pathogen	day^{-1}	0.0900982	Estimated
δ	Death rate of vector	day^{-1}	0.009	Estimated

Note that in the Table 1, the unit of η is to be Cells(ml^{-1})(Individual^{-1})(day^{-1})=$\mho$.

3. Results

3.1. Model analysis

3.1.1. Positivity and boundedness of solutions

In this section, we need to prove that the solutions of system (2.1) are nonnegative for all $t \geq 0$. This will be stated as follows.

Theorem 1. *Every solution of system* (2.1) *with initial condition* (2.2) *will remain positive in* $\mathbb{R}^5_+$ *for all* $t > 0$.

Proof. From the system (2.1), we obtain

$$\left.\frac{dS_c}{dt}\right|_{[S_c=0]} \geq 0, \quad \left.\frac{dI_c}{dt}\right|_{[I_c=0]} = \beta_2 S_c I_v \geq 0, \quad \left.\frac{dS_v}{dt}\right|_{[S_v=0]} = \lambda > 0,$$
$$\left.\frac{dI_v}{dt}\right|_{[I_v=0]} = (\beta_1 B + \beta_3 I_c) S_v \geq 0, \qquad \left.\frac{dB}{dt}\right|_{[B=0]} = \eta I_c \geq 0.$$

This implies that all the solutions with positive initial data remains nonnegative in $\mathbb{R}^5_+$ for all $t \geq 0$. This means, the region attracts all solutions of the governing system (2.1). □

Moreover, it is easy to verify that there exist an invariant region Ω where a solution set for the system (2.1) is bounded.

Theorem 2. *Every solution of system* (2.1) *initiating in* $\mathbb{R}^5_+$ *is uniformly bounded in the region* $\Omega = \Omega_c \times \Omega_v \times \Omega_p$, *where*

$$\Omega_c = \left\{(S_c,\ I_c) \in \mathbb{R}^2_+ : N_c \leq \frac{M(1+r)}{h}\right\}, \Omega_v = \left\{(S_v,\ I_v) \in \mathbb{R}^2_+ : N_v \leq \frac{\lambda}{\delta}\right\},$$
$$\Omega_p = \left\{B \in \mathbb{R}_+ : 0 \leq B \leq \frac{M(1+r)\eta}{mh}\right\},$$

with $M = \max\{S_c(0),\ K\}$, $h = \min\{1, \gamma + \eta\}$.

Proof. Let $N_c(t) = S_c(t) + I_c(t)$ be the total population of coffee berry. Then differentiating $N_c(t)$ with respect to time t and adding the first two equations of system (2.1), we get

$$\frac{dN_c}{dt} \leq rS_c - (\gamma + \eta)I_c = (1 + r)S_c - S_c - (\gamma + \eta)I_c. \tag{3.1}$$

The last inequality in Eq (3.1) can be rewritten as

$$\frac{dN_c}{dt} + hN_c \leq M(1 + r). \tag{3.2}$$

Integrating Eq (3.2) by separation of variables and then as $t \to \infty$, the feasible region of the system (2.1) for coffee population is given by

$$\Omega_c = \left\{(S_c,\ I_c) \in \mathbb{R}^2_+ : N_c \leq \frac{M(1+r)}{h}\right\}.$$

We also consider the total population of vector as $N_v(t) = S_v(t) + I_v(t)$. Then the derivative of $N_v(t)$ with respect to t is given by

$$\frac{dN_v}{dt} = \lambda - \delta N_v. \tag{3.3}$$

By solving Eq (3.3) and then as $t \to \infty$, we get the feasible region of the system (2.1) for vector population:

$$\Omega_v = \left\{(S_v,\ I_v) \in \mathbb{R}^2_+ : N_v \leq \frac{\lambda}{\delta}\right\}. \tag{3.4}$$

From the fifth equation of system (2.1) and by the comparison, we have

$$\frac{dB}{dt} = \eta I_c - mB \leq \eta(S_c + I_c) - mB \leq \frac{M(1+r)\eta}{h} - mB. \tag{3.5}$$

Solving the last inequality of Eq (3.5) and $t \to \infty$ results $0 \leq B(t) \leq M(1+r)\eta/mh$. Consequently, the feasible region of the system (2.1) is given by $\Omega = \Omega_c \times \Omega_v \times \Omega_p$ is positively invariant. □

Hence, the solutions of the model are bounded. Therefore, the model is suitable to conduct the study, and the analysis of the system dynamics can be considered in the region Ω.

3.1.2. Existence and uniqueness of solutions

In this section, we provide the following results which guarantee that the CBD model governed by system (2.1) is epidemiologically and mathematically well posed.

Theorem 3. *(Existence-uniqueness of solution). Let Θ be the domain:*

$$|t - t_0| \leq a, \|x - x_0\| \leq b, x = (x_1, x_2, ..., x_n), x_0 = (x_{10}, x_{20}, ..., x_{n0}) \tag{3.6}$$

and suppose that $f(t, x)$ satisfies the Lipschitz condition:

$$\|f(t, x_1) - f(t, x_2)\| \leq \kappa \|x_1 - x_2\|, \tag{3.7}$$

where (t, x_1), $(t, x_2) \in \Theta$, and $\kappa > 0$. Then, there exist a constant $\nu > 0$ such that there exists a unique continuous vector solution $x(t)$ of the system (2.1) in the interval $|t - t_0| \leq \nu$. From Eq (3.7), f is satisfied by requirement that $\partial f_i/\partial x_j$, $i, j = 1, 2, ..., 5$ be continuous and bounded in Θ.

If $f(t, x)$ has continuous partial derivative $\partial f_i/\partial x_j$ on a bounded closed convex domain $\mathbb{R}$, then it satisfies a Lipschitz condition in $\mathbb{R} \in (0, \infty)$. Our concern is in the domain:

$$1 \leq \epsilon \leq \mathbb{R}. \tag{3.8}$$

Let Θ denote the region defined in Eq (3.6) such that Eqs (3.7) and (3.8) hold. Then, there exist a solution of system (2.1) which is bounded in Θ.

Proof. Suppose that the right side of system (2.1) is re-written as:

$$f_1 = rS_c\left(1 - \frac{S_c + I_c}{K}\right) - \beta_2 S_c I_v - \theta S_c, \tag{3.9}$$

$$f_2 = \beta_2 S_c I_v - (\gamma + \eta) I_c, \tag{3.10}$$

$$f_3 = \lambda - (\beta_1 B + \beta_3 I_c) S_v - \delta S_v, \tag{3.11}$$

$$f_4 = (\beta_1 B + \beta_3 I_c) S_v - \delta I_v, \tag{3.12}$$
$$f_5 = \eta I_c - mB. \tag{3.13}$$

Since the functions in Eqs (3.9)–(3.13) are polynomial, then they are infinitely differentiable. Thus, for the detailed proof, we can follow the proof of classical Cauchy-Lypschitz theorem [43]. Therefore, all the partial derivatives exist and are finite, then there exists a solution for the model and hence, we can say that there exist a unique solution of system (2.1) in the domain Θ. □

3.1.3. Disease-free equilibrium point (DFE)

At DFE, there is no infections (i.e., $I_c = I_v = B = 0$) in the populations. To find DFE, we equate the right hand side of system (2.1) to zero and solving for the noninfected state variables we get $S_c = 0$ or $S_c = K - K\theta/r,\ S_v = \lambda/\delta$. Thus, the equilibrium points of the system (2.1) are $(0,\ 0,\ \lambda/\delta,\ 0,\ 0)$ and $(K(r-\theta)/r,\ 0,\ \lambda/\delta,\ 0,\ 0)$. Hence, the DFE is given by

$$E_0 = \left(\frac{K(r-\theta)}{r},\ 0,\ \frac{\lambda}{\delta},\ 0,\ 0\right). \tag{3.14}$$

Remark 1. *The disease-free equilibrium poin E_0 in Eq* (3.14) *is biologically feasible when $r > \theta$.*

3.1.4. Basic reproduction number (R_0)

The basic reproduction number is the number of secondary infections that one infectious individual would create over the duration of the infectious period [37]. The expression of R_0 for the system (2.1) can be derived using the next generation matrix method [38]. The first step to find R_0 is rewriting the model equations starting with newly infective classes:

$$\begin{cases} \dfrac{dI_c}{dt} = \beta_2 S_c I_v - (\gamma + \eta) I_c, \\ \dfrac{dI_v}{dt} = (\beta_1 B + \beta_3 I_c) S_v - \delta I_v, \\ \dfrac{dB}{dt} = \eta I_c - mB. \end{cases} \tag{3.15}$$

The right hand side of system (3.15) is written as $f - g$, where

$$f = \begin{bmatrix} \beta_2 S\, cIv \\ (\beta_1 B + \beta_3 Ic) S\, v \\ 0 \end{bmatrix},\quad g = \begin{bmatrix} (\gamma + \eta) Ic \\ \delta Iv \\ -\eta Ic + mB \end{bmatrix}. \tag{3.16}$$

Then by linearization approach, the associated matrices of f and g at E_0 are given by

$$\mathbb{F} = \begin{bmatrix} 0 & \dfrac{K(r-\theta)\beta_2}{r} & 0 \\ \dfrac{\beta_3 \lambda}{\delta} & 0 & \dfrac{\beta_1 \lambda}{\delta} \\ 0 & 0 & 0 \end{bmatrix},\quad \mathbb{G} = \begin{bmatrix} \gamma + \eta & 0 & 0 \\ 0 & \delta & 0 \\ -\eta & 0 & m \end{bmatrix}.$$

The basic reproduction number, $R_0 = \rho(\mathbb{F}\mathbb{G}^{-1})$ is the spectral radius of the product $\mathbb{F}\mathbb{G}^{-1}$. Thus, it is given by

$$R_0 = \sqrt{\frac{K\beta_2\lambda\left[m\beta_3 + \beta_1\eta\right](r-\theta)}{rm\delta^2(\gamma+\eta)}}. \tag{3.17}$$

3.1.5. Local stability of disease-free equilibrium

In this section, we shall investigate the local stability of disease-free equilibrium point based on the basic reproduction number, R_0.

Theorem 4. *If $R_0 < 1$, then E_0 of the system* (2.1) *is locally asymptotically stable in* Ω.

Proof. The Jacobian matrix of the system (2.1) at E_0 is given by

$$J(E_0) = \begin{bmatrix} \theta - r & -(r-\theta) & 0 & -\frac{\beta_2 K(r-\theta)}{r} & 0 \\ 0 & -\gamma-\eta & 0 & \frac{\beta_2 K(r-\theta)}{r} & 0 \\ 0 & -\frac{\beta_3\lambda}{\delta} & -\delta & 0 & -\frac{\beta_1\lambda}{\delta} \\ 0 & \frac{\beta_3\lambda}{\delta} & 0 & -\delta & \frac{\beta_1\lambda}{\delta} \\ 0 & \eta & 0 & 0 & -m \end{bmatrix}. \tag{3.18}$$

From the Jacobian matrix (3.18), we obtain characteristic polynomial:

$$(\delta+\Lambda)(r-\theta+\Lambda)(\Lambda^3 + A_2\Lambda^2 + A_1\Lambda + A_0) = 0. \tag{3.19}$$

From Eq (3.19), we obtain that $\Lambda_1 = -\delta < 0$, $\Lambda_2 = -(r-\theta) < 0$ (see Remark 1, $r > \theta$) and

$$\Lambda^3 + A_2\Lambda^2 + A_1\Lambda + A_0 = 0, \tag{3.20}$$

where

$$A_2 = \gamma + \eta + m + \delta, \;\; A_1 = m(\delta+\gamma+\eta) + \frac{\delta(\gamma+\eta)\eta\beta_1}{m\beta_3+\eta\beta_1} + \frac{\delta(\gamma+\eta)m\beta_3}{m\beta_3+\eta\beta_1}(1-\mathcal{R}_0^2),$$
$$A_0 = \delta(\gamma+\eta)m(1-R_0^2).$$

Using Routh-Hurwitz criteria [38], Eq (3.20) has eigenvalues which are negative real part whenever $A_0 > 0, A_1 > 0, A_2 > 0$, $A_1A_2 - A_0 > 0$ and $A_0(A_1A_2 - A_0) > 0$. Therefore, E_0 is locally asymptotically stable for $R_0 < 1$. □

3.1.6. Global stability of disease-free equilibrium

Theorem 5. *If $R_0 < 1$, then E_0 of the system* (2.1) *is globally asymptotically stable in* Ω.

Proof. To proof this theorem, we first define the linear Lyapunov function:

$$U = \epsilon_1 I_c + \epsilon_2 I_v + \epsilon_3 B,$$

where ϵ_1, ϵ_2 and ϵ_3 are positive constants to be computed.

Then differentiating U with respect to t, we obtain

$$\frac{dU}{dt} = (-\epsilon_1(\gamma+\eta) + \epsilon_2\beta_3 S_v^0 + \epsilon_3\eta)I_c + (\epsilon_1\beta_2 S_c^0 - \epsilon_3\delta)I_v + (\epsilon_2\beta_1 S_v^0 - \epsilon_3 m)B, \tag{3.21}$$

with $S_c^0 = K(r-\theta)/r$ and $S_v^0 = \lambda/\delta$. The $\epsilon_1, \epsilon_2, \epsilon_3$ can be chosen such that

$$\begin{aligned} -\epsilon_1(\gamma+\eta) + \epsilon_2\beta_3 S_v^0 + \epsilon_3\eta = 0,\\ \epsilon_1\beta_2 S_c^0 - \epsilon_3\delta = 0,\\ \epsilon_2\beta_1 S_v^0 - \epsilon_3 m = 0. \end{aligned} \tag{3.22}$$

If we choose $\epsilon_1 = 1$, then $\epsilon_2 = \beta_2 K(r-\theta)/r\delta$ and $\epsilon_3 = \beta_1\beta_2 K(r-\theta)\lambda/r\delta^2 m$. Substituting ϵ_1, ϵ_2 and ϵ_3 into Eq (3.21) leads to

$$\begin{aligned} \frac{dU}{dt} &= (-\epsilon_1(\gamma+\eta) + \epsilon_2\frac{\beta_3\lambda}{\delta} + \epsilon_3\eta)I_c + \left(\frac{\beta_2 K(r-\theta)}{r} - \frac{\beta_2 K(r-\theta)}{r}\right)I_v \\ &\quad + \left(\frac{\beta_1\beta_2 K(r-\theta)\lambda}{r\delta^2} - \frac{m\beta_1\beta_2 K(r-\theta)\lambda}{r\delta^2 m}\right)B, \\ &= (\gamma+\eta)\left(R_0^2 - 1\right)I_c. \end{aligned} \tag{3.23}$$

Since $R_0 < 1$ and $R_0 \geq 0$ imply that $R_0^2 < 1$, we have $dU/dt \leq 0$.

Moreover, the largest compact invariant set in $\{(S_c,\ I_c,\ S_v,\ I_v,\ B) \in \Omega : dU/dt = 0\}$ is the singleton $\{E_0\}$. By LaSalle [39], it then implies that E_0 is globally asymptotically stable in Ω. □

3.1.7. Endemic equilibrium point (EEP)

Endemic equilibrium point (E^*) exists when CBD persist in the populations. To obtain EEP, we set each right hand equation in the system (2.1) equal to zero. That is,

$$\begin{aligned} rS_c^*\left(1 - \frac{S_c^* + I_c^*}{K}\right) - \beta_2 S_c^* I_v^* - \theta S_c^* &= 0,\\ \beta_2 S_c^* I_v^* - (\gamma+\eta)I_c^* &= 0,\\ \lambda - (\beta_1 B^* + \beta_3 I_c^*)S_v^* - \delta S_v^* &= 0,\\ (\beta_1 B^* + \beta_3 I_c^*)S_v^* - \delta I_v^* &= 0,\\ \eta I_c^* - mB^* &= 0. \end{aligned} \tag{3.24}$$

From the first equation of Eq (3.24) we find $S_c^* = 0$ or $r(1 - (S_c^* + I_c^*)/K) - \beta_2 I_v^* - \theta = 0$.
Case i: $S_c^* = 0$, we obtain $I_c^* = \delta m/(\beta_1\eta + m\beta_3)$, $S_v^* = \lambda/2\delta$, $I_v^* = \lambda/2\delta$, $B^* = \eta\delta/(\beta_1\eta + m\beta_3)$. This endemic point is a point where the disease kills all coffee berries.
Case ii: $r(1 - (S_c^* + I_c^*)/K) - \beta_2 I_v^* - \theta = 0$, then we get $S_c^*,\ S_v^*, I_v^*$ and B^*:

$$\begin{aligned} S_c^* &= \frac{\delta(\gamma+\eta)(m + (\beta_1\eta + m\beta_3)I_c^*)}{\lambda\beta_2(\beta_1\eta + m\beta_3)}, \qquad & S_v^* &= \frac{\lambda m}{\delta(m + (\beta_1\eta + \beta_3 m)I_c^*)},\\ I_v^* &= \frac{\lambda(\beta_1\eta + m\beta_3)I_c^*}{\delta(m + (\beta_1\eta + m\beta_3)I_c^*)}, & B^* &= \frac{\eta I_c^*}{m}, \end{aligned} \tag{3.25}$$

where I_c^* is the positive root of

$$f(I_c^*) = A(I_c^*)^2 + BI_c^* + C = 0, \tag{3.26}$$

with

$$A = \frac{r^3m^2\delta^5(\delta(\gamma+\eta)+\beta_2\lambda)(\gamma+\eta)^2R_0^4}{K^2\beta_2^2\lambda^2(r-\theta)^2} > 0,$$
$$B = \frac{r^2m^2\delta^3(\gamma+\eta)}{K\beta_2\lambda(r-\theta)}\left[\beta_2\lambda + 2\delta(\gamma+\eta) + \frac{\delta(\beta_2\lambda - r\delta)(\gamma+\eta)R_0^2}{r-\theta}\right]R_0^2,$$
$$C = r\delta^2m^2(\gamma+\eta)\left[\frac{r-\theta-r\delta R_0^2}{r-\theta}\right].$$

The feasibility of the endemic equilibrium is determined by the next proposition.

Proposition 1. *(Feasibility of the endemic equilibrium).*
Let $E^* = (S_c^*, I_c^*, S_v^*, I_v^*, B^*)$ *be as Eq* (3.25).
(i) If $B > 0$ *and* $C < 0$ *or* $B < 0$ *and* $C < 0$*, then there is a unique endemic equilibrium* E^*.
(ii) If $B < 0$ *or* $C > 0$*, then there exist two endemic equilibrium* E^* *of the system.*

Proof. The result follows Descartes' rule of signs to $f(I_c^*)$ given in Eq (3.26). □

3.1.8. Local stability of endemic equilibrium point

Theorem 6. *If* $R_0 > 1$, $B_i > 0, i = 1, 2, ..., 5$, $B_1B_2 - B_3 > 0, B_1(B_2B_3 + B_5) - B_3^2 - B_1^2B_4 > 0, (B_2B_3 - B_1(B_2^2 - 2B_4))B_5 - B_4^2(-B_1B_2B_3 + B_3^2 + B_1^2B_4) - B_5^2 > 0, B_5(-B_4(-B_1B_2B_3 + B_3^2 + B_1^2B_4) + (B_2B_3 - B_1(B_2^2 - 2B_4))B_5 - B_5^2) > 0$*, then* E^* *of system* (2.1) *is locally asymptotically stable in* Ω.

Proof. The Jacobian matrix of system (2.1) at E^* is given by

$$J(E^*) = \begin{bmatrix} \frac{r\_r(I_c^* + 2S_c^*)}{K} - \beta_2 I_v^* - \theta & -(r-\theta) & 0 & -\beta_2 S_c^* & 0 \\ \beta_2 I_v^* & -\gamma - \eta & 0 & \beta_2 S_c^* & 0 \\ 0 & -\beta_3 S_v^* & -\beta_1 B^* - \beta_3 I_c^* - \delta & 0 & -\beta_1 S_v^* \\ 0 & \beta_3 S_v^* & \beta_1 B^* + \beta_3 I_c^* & -\delta & \beta_1 S_v^* \\ 0 & \eta & 0 & 0 & -m \end{bmatrix} \tag{3.27}$$

The eigenvalues of matrix (3.27) are computed from

$$|J(E^*) - \Lambda I_5| = 0 \tag{3.28}$$

Let us consider the non-zero entities of matrix (3.28) as:

$b_{11} = -\theta - r(I_c^* + 2S_c^*)/K - \beta_2 I_v^* + r$, $b_{12} = -(r-\theta)$, $b_{21} = \beta_2 I_v^*$, $b_{22} = -\gamma - \eta$,
$b_{32} = -\beta_3 S_v^*$, $b_{42} = \beta_3 S_v^*$, $b_{52} = \eta$, $b_{33} = -\beta_1 B^* - \beta_3 I_c^* - \delta$, $b_{43} = \beta_1 B^* + \beta_3 I_c^*$,
$b_{14} = -\beta_2 S_c^*$, $b_{24} = \beta_2 S_c^*$, $b_{44} = -\delta$, $b_{35} = -\beta_1 S_v^*$, $b_{45} = \beta_1 S_v^*$, $b_{55} = -m$.

Then, the characteristic polynomial of Eq (3.28) is given by

$$\Lambda^5 + B_1\Lambda^4 + B_2\Lambda^3 + B_3\Lambda^2 + B_4\Lambda + B_5 = 0, \tag{3.29}$$

where

$$\begin{aligned}
B_1 &= -(b_{11} + b_{22} + b_{33} + b_{44} + b_{55}),\\
B_2 &= b_{33}b_{44} + b_{22}(b_{33} + b_{44} + b_{55}) + (b_{33} + b_{44})b_{55} + b_{11}(b_{33} + b_{44} + b_{55}) + b_{24}b_{42}(b_{11} - b_{14})\\
&\quad - b_{11}((b_{44} + b_{55})b_{33} + b_{22} + b_{44}b_{55}),\\
B_3 &= b_{24}b_{33}b_{42} - b_{14}b_{21}b_{42} - b_{24}b_{32}(b_{43} - b_{24}b_{45}b_{52} - b_{22}b_{44}b_{55} - (b_{44} + b_{55})b_{22}b_{33} + (b_{24}b_{42} - b_{44})b_{55}\\
&\quad + b_{11}b_{24}b_{42} - (b_{44} + b_{55})b_{11}b_{33} - b_{11}b_{22} - b_{11}b_{44}b_{55},\\
B_4 &= b_{14}b_{21}(b_{33}b_{42} - b_{32}b_{43} - b_{45}b_{52}) + b_{24}(b_{33}b_{45} - b_{35}b_{43})b_{52} + (b_{22}b_{33}b_{44} + b_{24}b_{32}b_{43} + b_{14}b_{21}b_{42})b_{55}\\
&\quad + b_{11}b_{24}(b_{32}b_{43} + b_{45}b_{52} - b_{33}b_{42}) + b_{11}b_{33}b_{44}b_{55} + b_{11}b_{22}(b_{33} + b_{44}) - b_{11}b_{24}b_{42}b_{55},\\
B_5 &= b_{14}b_{21}((b_{33}b_{45} - b_{35}b_{43})b_{52} + (b_{32}b_{43} - b_{42})b_{55} + b_{11}b_{24}((b_{35}(b_{43} - b_{33}b_{45})b_{52}\\
&\quad + (b_{33}b_{42} - b_{32}b_{43})b_{55}) - b_{11}b_{22}b_{33}b_{44}.
\end{aligned}$$

Using the Routh Hurwitz criterion [39], the endemic equilibrium E^* is locally asymptotically stable for $R_0 > 1$ if $B_i > 0, i = 1, 2, ..., 5$ and

$$\begin{aligned}
&B_1B_2 - B_3 > 0, B_1(B_2B_3 + B_5) - B_3^2 - B_1^2B_4 > 0,\\
&(B_2B_3 - B_1(B_2^2 - 2B_4))B_5 - B_4^2(-B_1B_2B_3 + B_3^2 + B_1^2B_4) - B_5^2 > 0,\\
&B_5(-B_4(-B_1B_2B_3 + B_3^2 + B_1^2B_4) + (B_2B_3 - B_1(B_2^2 - 2B_4))B_5 - B_5^2) > 0.
\end{aligned}$$

Hence, all eigenvalues of Eq (3.29) evaluated at E^* have negative real parts whenever $R_0 > 1$ and the equilibrium E^* is locally asymptotically stable. □

3.1.9. Global stability of endemic equilibrium

Theorem 7. *If $R_0 > 1$, then E^* of the model* (2.1) *is globally asymptotically stable in* Ω.

Proof. Let us consider a Volterra-type Lyapunov function:

$$V = \zeta_1(S_c - S_c^* \ln S_c) + \zeta_2(S_c - S_c^* \ln S_c) + \zeta_3(S_v - S_v^* \ln S_v) + \zeta_4(I_c - I_c^* \ln I_c) + \zeta_5(B - B^* \ln B), \tag{3.30}$$

where $\zeta_1, \zeta_2, \zeta_3, \zeta_4$ and ζ_5 are non-negative constants. Then by taking the derivative of V with respect to t, we obtain

$$\begin{aligned}
\frac{dV}{dt} &= \zeta_1\left(1 - \frac{S_c^*}{S_c}\right)\frac{dS_c}{dt} + \zeta_2\left(1 - \frac{I_c^*}{I_c}\right)\frac{dI_c}{dt} + \zeta_3\left(1 - \frac{S_v^*}{S_v}\right)\frac{dS_v}{dt} + \zeta_4\left(1 - \frac{I_v^*}{I_v}\right)\frac{dI_v}{dt} + \zeta_5\left(1 - \frac{B^*}{B}\right)\frac{dB}{dt},\\
&= \zeta_1 S_c\left(1 - \frac{S_c^*}{S_c}\right)\left(r - \frac{rS_c}{K} - \frac{rI_c}{K} - \beta_2 I_v - \theta\right) + \zeta_2\left(1 - \frac{I_c^*}{I_c}\right)(\beta_2 S_c I_v - (\gamma + \eta)I_c)\\
&\quad + \zeta_3\left(1 - \frac{S_v^*}{S_v}\right)(\lambda - (\beta_1 B + \beta_3 I_c + \delta)S_v) + \zeta_4\left(1 - \frac{I_v^*}{I_v}\right)((\beta_1 B + \beta_3 I_c)S_v - \delta I_v) + \zeta_5\left(1 - \frac{B^*}{B}\right)(\eta I_c - mB).
\end{aligned} \tag{3.31}$$

Since E^* is an equilibrium point, Eq (3.24) can be rewritten as:

$$\begin{aligned} r - \frac{rS_c^*}{K} - \frac{rI_c^*}{K} - \beta_2 I_v^* &= \theta, \\ \beta_2 S_c^* I_v^* &= (\gamma + \eta) I_c^*, \\ \lambda &= (\beta_1 B^* + \beta_3 I_c^* + \delta) S_v^*, \\ (\beta_1 B^* + \beta_3 I_c^*) S_v^* &= \delta I_v^*, \\ \eta I_c^* &= m B^*. \end{aligned} \tag{3.32}$$

Substituting the relations in Eq (3.32) into Eq (3.31), we obtain

$$\begin{aligned} \frac{dV}{dt} &= \zeta_1 S_c \left(\frac{S_c - S_c^*}{S_c}\right)\left(-\frac{rS_c}{K} - \frac{rI_c}{K} - \beta_2 I_v + \frac{rS_c^*}{K} + \frac{rI_c^*}{K} + \beta_2 I_v^*\right) + \zeta_2 \left(1 - \frac{I_c^*}{I_c}\right)\left(\beta_2 S_c I_v - \beta_2 S_c^* I_v^* \frac{I_c}{I_c^*}\right) \\ &+ \zeta_3 \left(1 - \frac{S_v^*}{S_v}\right)((\beta_1 B^* + \beta_3 I_c^* + \delta) S_v^* - (\beta_1 B + \beta_3 I_c + \delta) S_v) \\ &+ \zeta_4 \left(1 - \frac{I_v^*}{I_v}\right)\left((\beta_1 B + \beta_3 I_c) S_v - (\beta_1 B^* + \beta_3 I_c^*) S_v^* \frac{I_v}{I_v^*}\right) + \zeta_5 \left(1 - \frac{B^*}{B}\right)\left(\eta I_c - \eta I_c^* \frac{B}{B^*}\right). \end{aligned} \tag{3.33}$$

Expanding and grouping Eq (3.33), we have

$$\begin{aligned} \frac{dV}{dt} &= -\zeta_1 \frac{r}{K}(s_c - S_c^*)^2 + \left[\zeta_1 \frac{rS_c^*}{K} - \zeta_2 \beta_2 \frac{S_c^* I_v^*}{I_c^*} + \zeta_3 \beta_3 S_v^* + \zeta_5 \eta\right] I_c + \left[\zeta_1 \beta_2 S_c^* - \zeta_4 (\beta_1 B^* + \beta_3 I_c^*) \frac{S_v^*}{I_v^*}\right] I_v \\ &+ \left[\zeta_3 \beta_1 S_v^* - \zeta_5 \eta \frac{I_c^*}{B^*}\right] B + \zeta_1 \left(\frac{rI_c^*}{K} + \beta_2 I_v^*\right) S_c - \zeta_1 \frac{rI_c S_c}{K} - \zeta_1 \frac{rI_c^* S_c^*}{K} + (-\zeta_1 + \zeta_2) \beta_2 S_c I_v \\ &+ (-\zeta_1 + \zeta_2) \beta_2 S_c^* I_v^* - \zeta_2 \frac{\beta_2 I_c^* S_c I_v}{I_c} + (-\zeta_3 + \zeta_4) \beta_1 B S_v + (-\zeta_3 + \zeta_4) \beta_3 I_c S_v + 2\zeta_3 \delta S_v^* - \zeta_3 \delta S_v \\ &- \zeta_3 (\beta_1 B^* + \beta_3 I_c^* + \delta) \frac{(S_v^*)^2}{S_v} + (\zeta_3 + \zeta_4)(\beta_1 B^* + \beta_3 I_c^*) S_v^* - \zeta_4 \beta_1 B^* - \zeta_5 \frac{\eta B^* I_c}{B} + \zeta_5 \eta I_c^*. \end{aligned} \tag{3.34}$$

Choose $\zeta_1, \zeta_2, \zeta_3, \zeta_4$ and ζ_5 such that the expressions in the brackets in Eq (3.34) vanish. That is

$$\begin{aligned} \zeta_1 \frac{rS_c^*}{K} - \zeta_2 \frac{\beta_2 S_c^* I_v^*}{I_c^*} + \zeta_3 \beta_3 S_v^* + \zeta_5 \eta &= 0, \\ \zeta_1 \beta_2 S_c^* - \zeta_4 (\beta_1 B^* + \beta_3 I_c^*) \frac{S_v^*}{I_v^*} &= 0, \\ \zeta_3 \beta_1 S_v^* - \zeta_5 \eta \frac{I_c^*}{B^*} &= 0. \end{aligned} \tag{3.35}$$

Fixing $\zeta_1 = \delta$, then Eq (3.35) leads to

$$\zeta_2 = \zeta_1, \zeta_3 = \zeta_1 \frac{\eta I_c^* S_c^*}{(\beta_1 + \beta_3 m) S_v^* B^*} \left(\frac{\beta_2 I_v^*}{I_c^*} - \frac{r}{K}\right), \zeta_4 = \zeta_1 \frac{\beta_2 S_c^*}{\delta}, \zeta_5 = \zeta_1 \frac{\beta_1 S_c^*}{\beta_1 + \beta_3 m} \left(\frac{\beta_2 I_v^*}{I_c^*} - \frac{r}{K}\right). \tag{3.36}$$

Replacing Eq (3.36) in Eq (3.34), we obtain

$$\begin{aligned}\frac{dV}{dt} =& -\zeta_1\frac{r}{K}(s_c - S_c^*)^2 + \zeta_1\left(\frac{rI_c^*}{K} + \beta_2 I_v^*\right)S_c - \zeta_1\frac{rI_cS_c}{K} - \zeta_1\frac{rI_c^*S_c^*}{K} - \zeta_1\frac{\beta_2 I_c^*S_cI_v}{I_c}\\ &+ \zeta_1\left(-\frac{\eta I_c^*S_c^*}{(\beta_1+\beta_3 m)S_v^*B^*}\left(\frac{\beta_2 I_v^*}{I_c^*} - \frac{r}{K}\right) + \frac{\beta_2 S_c^*}{\delta}\right)(\beta_1 B + \beta_3 I_c)S_v + \zeta_1\frac{2\eta\delta S_v^*I_c^*S_c^*}{(\beta_1+\beta_3 m)S_v^*B^*}\left(\frac{\beta_2 I_v^*}{I_c^*} - \frac{r}{K}\right)\\ &- \zeta_1\frac{\eta I_c^*S_c^*}{(\beta_1+\beta_3 m)S_v^*B^*}\left(\frac{\beta_2 I_v^*}{I_c^*} - \frac{r}{K}\right)\left(\delta S_v + \frac{\lambda S_v^*}{S_v}\right) + \zeta_1\left(\frac{\eta I_c^*S_c^*}{(\beta_1+\beta_3 m)S_v^*B^*}\left(\frac{\beta_2 I_v^*}{I_c^*} - \frac{r}{K}\right) + \frac{\beta_2 S_c^*}{\delta}\right)\lambda\\ &- \zeta_1\left(\frac{\eta I_c^*S_c^*}{(\beta_1+\beta_3 m)S_v^*B^*}\left(\frac{\beta_2 I_v^*}{I_c^*} - \frac{r}{K}\right) + \frac{\beta_2 S_c^*}{\delta}\right)\delta S_v^* - \zeta_1\frac{\beta_1\beta_2 B^*S_c^*}{\delta} - \zeta_1\frac{\beta_1 S_c^*}{\beta_1+\beta_3 m}\left(\frac{\beta_2 I_v^*}{I_c^*} - \frac{r}{K}\right)\frac{\eta B^* I_c}{B}\\ &+ \zeta_1\frac{\beta_1 S_c^*}{\beta_1+\beta_3 m}\left(\frac{\beta_2 I_v^*}{I_c^*} - \frac{r}{K}\right)\eta I_c^*,\end{aligned} \tag{3.37}$$

or equivalently

$$\begin{aligned}\frac{dV}{dt} =& -\zeta_1\frac{r}{K}(s_c - S_c^*)^2 - \zeta_1\frac{rI_cS_c}{K} - \zeta_1\frac{rI_c^*S_c^*}{K} - \zeta_1\frac{\beta_2 I_c^*S_cI_v}{I_c} - \zeta_1\frac{\eta I_c^*S_c^*(\beta_1 B + \beta_3 I_c)S_v}{(\beta_1+\beta_3 m)S_v^*B^*} - \zeta_1\frac{\beta_1 r\eta I_c^*S_c^*}{K(\beta_1+\beta_3 m)}\\ &- \zeta_1\frac{\eta I_c^*S_c^*}{(\beta_1+\beta_3 m)S_v^*B^*}\left(\frac{\beta_2 I_v^*}{I_c^*} - \frac{r}{K}\right)\left(\delta S_v + \frac{\lambda S_v^*}{S_v}\right) - \zeta_1\left(\frac{\eta I_c^*S_c^*}{(\beta_1+\beta_3 m)S_v^*B^*}\left(\frac{\beta_2 I_v^*}{I_c^*} - \frac{r}{K}\right) + \frac{\beta_2 S_c^*}{\delta}\right)\delta S_v^*\\ &- \zeta_1\frac{\beta_1\beta_2 B^*S_c^*}{\delta} - \zeta_1\frac{\beta_1 S_c^*}{\beta_1+\beta_3 m}\left(\frac{\beta_2 I_v^*}{I_c^*} - \frac{r}{K}\right)\frac{\eta B^* I_c}{B} - \zeta_1\frac{2r\eta\delta S_v^*I_c^*S_c^*}{K(\beta_1+\beta_3 m)S_v^*B^*} - \zeta_1\left(\frac{r\eta I_c^*S_c^*}{K(\beta_1+\beta_3 m)S_v^*B^*}\right)\lambda\\ &+ \zeta_1\frac{\delta(rI_c^* + K\beta_2 I_v^*)S_c + K\beta_2 S_c^*((\beta_1 B + \beta_3 I_c)S_v + \lambda)}{K\delta}\\ &+ \zeta_1\frac{K(2\delta + \beta_1 B^*)I_v^*S_v^*S_c^* + (K\beta_2\lambda I_v^* + rI_c^*(\beta_1 B + \beta_3 I_c)S_v)\eta S_c^*}{K(\beta_1+\beta_3 m)S_v^*B^*}.\end{aligned} \tag{3.38}$$

From Eq (3.38), since $\frac{\eta I_c^*S_c^*}{(\beta_1+\beta_3 m)S_v^*B^*}\left(\frac{\beta_2 I_v^*}{I_c^*} - \frac{r}{K}\right) = \zeta_3 \geq 0$, we have

$$-\zeta_1\frac{\eta I_c^*S_c^*}{(\beta_1+\beta_3 m)S_v^*B^*}\left(\frac{\beta_2 I_v^*}{I_c^*} - \frac{r}{K}\right)\left(\delta S_v + \frac{\lambda S_v^*}{S_v}\right) \leq 0.$$

With this in mind, and if we let

$$\begin{aligned}Z =& -\zeta_1\frac{r}{K}(s_c - S_c^*)^2 - \zeta_1\frac{rI_cS_c}{K} - \zeta_1\frac{rI_c^*S_c^*}{K} - \zeta_1\frac{\beta_2 I_c^*S_cI_v}{I_c} - \zeta_1\frac{\eta I_c^*S_c^*(\beta_1 B + \beta_3 I_c)S_v}{(\beta_1+\beta_3 m)S_v^*B^*} - \zeta_1\frac{\beta_1 r\eta I_c^*S_c^*}{K(\beta_1+\beta_3 m)}\\ &- \zeta_1\frac{\eta I_c^*S_c^*}{(\beta_1+\beta_3 m)S_v^*B^*}\left(\frac{\beta_2 I_v^*}{I_c^*} - \frac{r}{K}\right)\left(\delta S_v + \frac{\lambda S_v^*}{S_v}\right) - \zeta_1\left(\frac{\eta I_c^*S_c^*}{(\beta_1+\beta_3 m)S_v^*B^*}\left(\frac{\beta_2 I_v^*}{I_c^*} - \frac{r}{K}\right) + \frac{\beta_2 S_c^*}{\delta}\right)\delta S_v^*\\ &- \zeta_1\frac{\beta_1\beta_2 B^*S_c^*}{\delta} - \zeta_1\frac{\beta_1 S_c^*}{\beta_1+\beta_3 m}\left(\frac{\beta_2 I_v^*}{I_c^*} - \frac{r}{K}\right)\frac{\eta B^* I_c}{B} - \zeta_1\frac{2r\eta\delta S_v^*I_c^*S_c^*}{K(\beta_1+\beta_3 m)S_v^*B^*} - \zeta_1\left(\frac{r\eta I_c^*S_c^*}{K(\beta_1+\beta_3 m)S_v^*B^*}\right)\lambda \leq 0,\end{aligned}$$

$$\begin{aligned}W =& \zeta_1\frac{\delta(rI_c^* + K\beta_2 I_v^*)S_c + K\beta_2 S_c^*((\beta_1 B + \beta_3 I_c)S_v + \lambda)}{K\delta}\\ &+ \zeta_1\frac{K(2\delta + \beta_1 B^*)I_v^*S_v^*S_c^* + (K\beta_2\lambda I_v^* + rI_c^*(\beta_1 B + \beta_3 I_c)S_v)\eta S_c^*}{K(\beta_1+\beta_3 m)S_v^*B^*} \geq 0,\end{aligned}$$

then $(dV/dt)(S_c,\ I_c,\ S_v,\ I_v,\ B) \leq 0$ if and only if $S_c = S_c^*,\ I_c = I_c^*,\ S_v = S_v^*,\ I_v = I_v^*,\ B = B^*$ and $Z + (-W) \leq 0$.

Therefore, the largest compact invariant set in $\{(S_c,\ I_c,\ S_v,\ I_v,\ B) \in \Omega : dV/dt = 0\}$ is the singleton $\{E^*\}$. By LaSalle [39], it then implies that E^* is globally asymptotically stable in Ω. □

3.1.10. Bifurcation analysis

To investigate the possibility of existence of the bifurcation analysis of the system (2.1) at $R_0 = 1$, we use the center manifold theory [40]. So using this approach, the following theorem can be established.

Theorem 8. *If $R_0 < 1$, then the system* (2.1) *exhibits forward bifurcation at $R_0 = 1$.*

Proof. Using center manifold theory [40], we carry out bifurcation analysis of system (2.1) at $R_0 = 1$. Let us consider the change of variables $S_c = x_1,\ I_c = x_2,\ S_v = x_3,\ I_v = x_4,\ B = x_5$. Furthermore, using the vector notation: $X = (x_1, x_2, x_3, x_4, x_5)^T$. Then the system (2.1) can be written in the form $dX/dt = (g_1,\ g_2,\ g_3,\ g_4,\ g_5)^T$ as:

$$\begin{cases} \dfrac{dx_1}{dt} = rx_1\left(1 - \dfrac{x_1 + x_2}{K}\right) - \beta_2 x_1 x_4 - \theta x_1, \\ \dfrac{dx_2}{dt} = \beta_2 x_1 x_4 - (\gamma + \eta)x_2, \\ \dfrac{dx_3}{dt} = \lambda - (\beta_1 x_5 + \beta_3 x_2)x_3 - \delta x_3, \\ \dfrac{dx_4}{dt} = (\beta_1 x_5 + \beta_3 x_2)x_3 - \delta x_4, \\ \dfrac{dx_5}{dt} = \eta x_2 - m x_5. \end{cases} \tag{3.39}$$

We consider β_2 as bifurcation parameter so that $R_0 = 1$ if

$$\beta_2 = \beta_2^* = \frac{r(\gamma + \eta)\delta^2 m}{K\lambda(r - \theta)\,(m\beta_3 + \beta_1\eta)}. \tag{3.40}$$

The Jacobian matrix of Eq (3.39) at disease-free equilibrium E_0 is given by

$$J(E_0) = \begin{bmatrix} \theta - r & -(r - \theta) & 0 & -\dfrac{K(r - \theta)\beta_2^*}{r} & 0 \\ 0 & -\gamma - \eta & 0 & \dfrac{K(r - \theta)\beta_2^*}{r} & 0 \\ 0 & -\dfrac{\beta_3\lambda}{\delta} & -\delta & 0 & -\dfrac{\beta_1\lambda}{\delta} \\ 0 & \dfrac{\beta_3\lambda}{\delta} & 0 & -\delta & \dfrac{\beta_1\lambda}{\delta} \\ 0 & \eta & 0 & 0 & -m \end{bmatrix}. \tag{3.41}$$

The right eigenvector, $u = (u_1,\ u_2,\ u_3,\ u_4,\ u_5)^T$ is computed from $J(E_0)u = 0$ so that

$$u_1 = -\frac{K\beta_2^*}{r}\left(\frac{r - \theta}{\gamma + \eta} + 1\right)u_4,\ \ u_2 = \frac{K(r - \theta)\beta_2^*}{r(\gamma + \eta)}u_4,\ \ u_3 = -u_4,\ \ u_5 = \frac{\eta K(r - \theta)\beta_2^*}{r(\gamma + \eta)m}u_4,$$

where $u_4 = u_4 > 0$. Similarly, the left eigenvector, $z = (z_1,\ z_2,\ z_3,\ z_4,\ z_5)$ is computed from $zJ(E_0) = 0$ so that $z_1 = z_3 = 0,\ z_2 = \dfrac{\delta r}{K(r-\theta)\beta_2^*}z_4,\ z_5 = \dfrac{\beta_1\lambda}{\delta m}z_4;\quad z_4 = z_4 > 0$. The nonzero second partial derivatives of $g_1,\ g_2,\ g_3$ and g_4 are given as follows:

$$\frac{\partial^2 g_1}{\partial x_1\partial x_1} = \frac{\partial^2 g_1}{\partial x_1\partial x_1} = -\frac{2r}{K},\quad \frac{\partial^2 g_1}{\partial x_1\partial x_2} = \frac{\partial^2 g_1}{\partial x_2\partial x_1} = -\frac{r}{K},\quad \frac{\partial^2 g_1}{\partial x_1\partial x_4} = \frac{\partial^2 g_1}{\partial x_4\partial x_1} = -\beta_2^*,$$
$$\frac{\partial^2 g_2}{\partial x_1\partial x_2} = \frac{\partial^2 g_2}{\partial x_2\partial x_1} = \beta_2^*,\quad \frac{\partial^2 g_3}{\partial x_2\partial x_3} = \frac{\partial^2 g_3}{\partial x_3\partial x_2} = -\beta_3,\quad \frac{\partial^2 g_3}{\partial x_3\partial x_5} = \frac{\partial^2 g_3}{\partial x_5\partial x_3} = -\beta_1,$$
$$\frac{\partial^2 g_4}{\partial x_2\partial x_3} = \frac{\partial^2 g_4}{\partial x_3\partial x_2} = \beta_3,\quad \frac{\partial^2 g_4}{\partial x_3\partial x_5} = \frac{\partial^2 g_4}{\partial x_5\partial x_3} = \beta_1,\quad \frac{\partial^2 g_1}{\partial x_4\partial \beta_2^*} = \frac{\partial^2 g_1}{\partial \beta_2^*\partial x_4} = -x_1^*,$$
$$\frac{\partial^2 g_2}{\partial x_4\partial \beta_2^*} = \frac{\partial^2 g_2}{\partial \beta_2^*\partial x_4} = x_1^*.$$

All the others second partial derivatives of $g_i,\ i = 1, ..., 5$ are zero. Based on center manifold theorem [40], the coefficients a and b are given by

$$\begin{aligned} a &= \sum_{i,j,k=1}^{5} z_k u_i u_j \frac{\partial^2 g_k}{\partial x_i \partial x_j}(E_0) \\ &= -\left[\frac{2\delta^3 r(\gamma+\eta)m}{K\lambda(r-\theta)^2(m\beta_3+\beta_1\eta)}\left(\frac{r-\theta}{\gamma+\eta}+1\right) + \frac{2\delta\beta_3}{\gamma+\eta} + 2\eta K(r-\theta)\delta^2\right] z_4 u_4^2 < 0, \\ b &= \sum_{i,k=1}^{5} z_k u_i \frac{\partial^2 g_k}{\partial x_i \partial \beta_2^*}(E_0) = \frac{K(r-\theta)(m\beta_3+\beta_1\eta)}{r(\gamma+\eta)\delta m} z_4 u_4 > 0. \end{aligned}$$

Since a is negative and b is positive, then the system (2.1) exhibits forward bifurcation at $R_0 = 1$ and there exists at least one stable endemic equilibrium when $R_0 > 1$ (See Figure 4). □

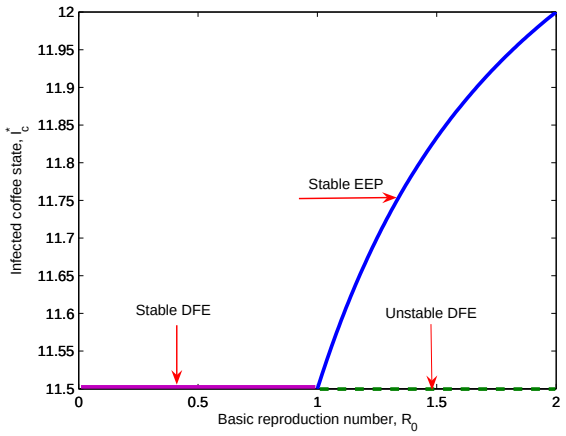


Figure 4. Forward bifurcation diagram. Parameter values are taken from Table 1 (except $\beta_2 = 0.5,\ \gamma = 0.99,\ \eta = 0.09,\ \lambda = 0.08,\ \delta = 0.099,\ \theta = 0.016,\ m = 0.900982$).

3.1.11. Sensitivity of the basic reproduction number

In this section, we compute the sensitivity index of basic reproduction number, R_0, with respect to the main parameters [41]. This help us to check and identify parameters which highly affect R_0 on the eradication or spread of CBD. For example, sensitivity index of R_0 with respect to K is given by

$$\Delta_K^{R_0} = \frac{\partial R_0}{\partial K} \times \frac{K}{R_0} = 0.5.$$

In the same way, the sensitivity index of R_0 are also computed and the corresponding sensitivity indeces are given in Table 2.

Table 2. Sensitivity indices.

Parameter	**Sensitivity indices of R_0**	**Parameter**	**Sensitivity indices of R_0**
K	+0.5	θ	–0.0454545
β_1	+0.0616258	λ	+0.5
β_2	+0.5	δ	–1
β_3	+0.438374	γ	–0.178571
η	–0.259803	m	–0.0616258
r	+0.0454545		

3.1.12. Interpretation of the sensitivity indices

The parameters: K, β_1, β_2, β_3, r and λ have positive sensitivity indices show that the more impact on expanding the disease in the population if their values are increasing since R_0 increases. Besides, the parameters: θ, η, δ, γ and m have negative sensitivity indices show that less impact on expanding the disease as their values increase. The bar diagram of the sensitivity indices of R_0 in Table 2 is depicted in Figure 5. Hence, the parameters: K, β_2 and λ are the most in influential in expanding the disease.

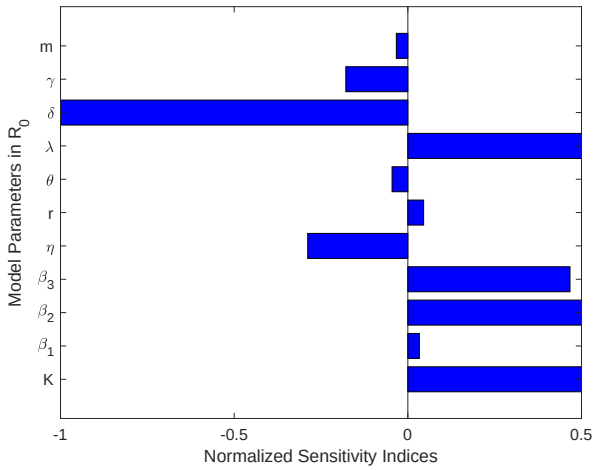
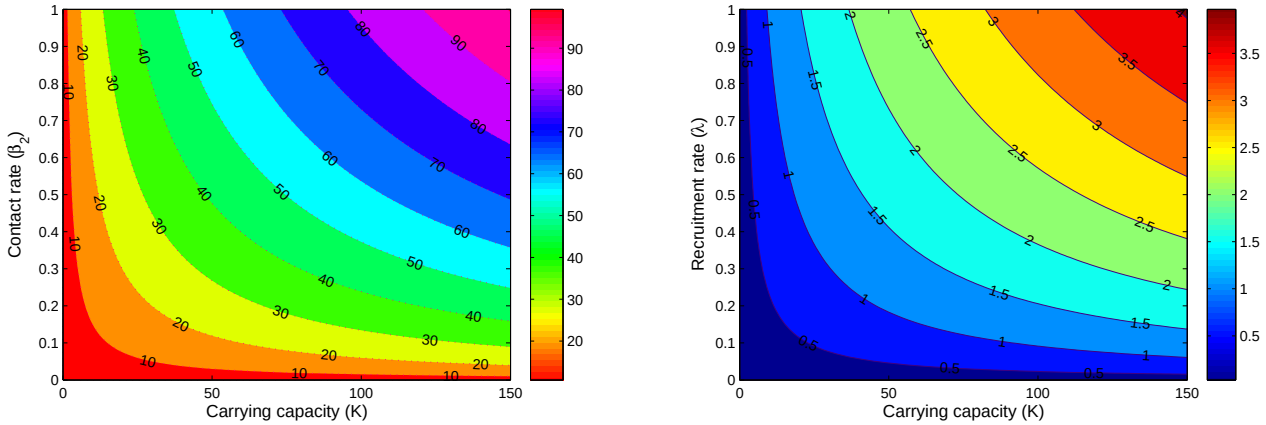


Figure 5. Normalized sensitivity indices of R_0 with respect to parameters of the system (2.1). Parameter values are taken from Table 1.

Furthermore, in Figure 6, we have seen that the contour plot of basic reproduction number R_0 with respect to the parameters K, β_2 and K, λ, respectively. It can be observed from Figure 6(a) that for decreasing value of K and β_2, R_0 decreases. For increasing K and λ the basic reproduction number R_0 increases, which has been shown in Figure 6(b).



(a) **(b)**

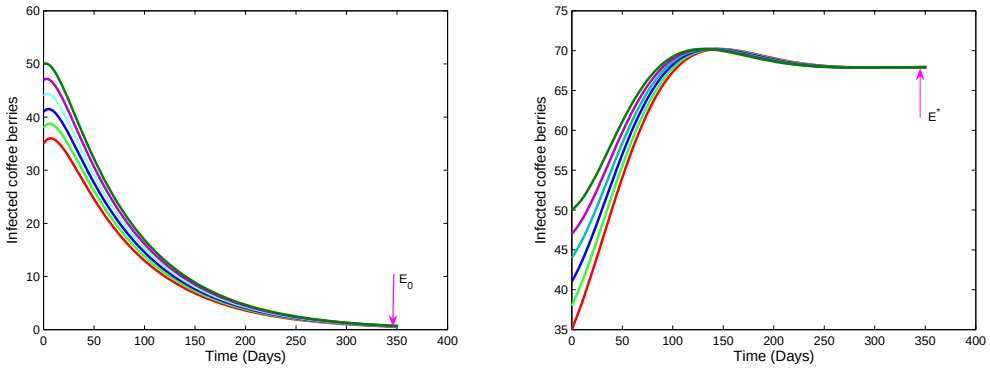
Figure 6. Contour plots of R_0 versus (a) (K, β_2)-plane and (b) (K, λ)-plane. Parameter values are taken from Table 1.

3.2. Numerical simulations

In order to illustrate the analytical results, the numerical simulations of the system (2.1) were performed. Some parameter values were assumed and some of them were obtained from literature. In addition to parameter values in Table 1, we assumed the initial data: $S_c(0) = 100$, $I_c(0) = 50$, $S_v(0) = 50$, $I_v(0) = 10$, $B(0) = 2$.

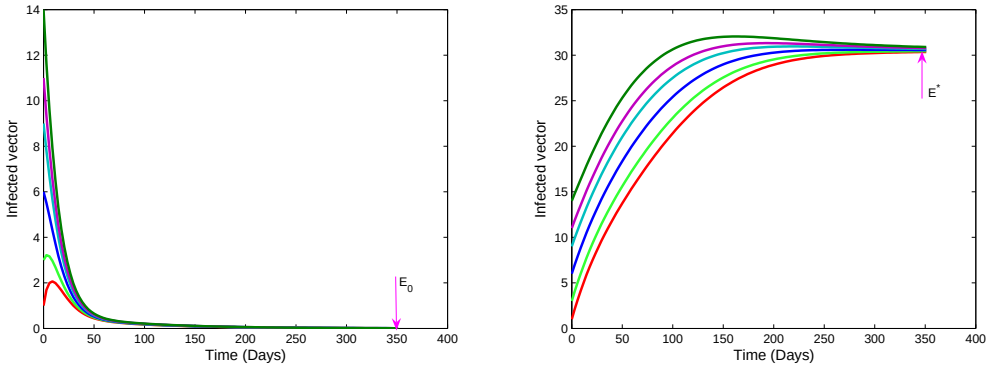
Figures 7(a) and 8(a) depict the global asymptotic behaviour of the infected coffee and infected vector at the disease-free equilibrium E_0 when $R_0 < 1$ where the disease dies out regardless of large initial values of the infectious population. On the other hand, Figures 7(b) and 8(b) show the global asymptotic behaviour of the infected coffee and infected vector at the endemic equilibrium E^* when $R_0 > 1$ where the disease persists regardless of the initial sizes of the infectious population.

From the Figure 9(a) susceptible coffee berries decreases while the infected coffee berries increases with $R_0 > 1$ due to there is very favorable condition for breeding of pathogen and vector that transmit the disease. The susceptible vector population in the Figure 9(c) decreases while infected vector population increases. The CBD pathogen population in the Figure 9(b) increases to a maximum point and then proceed decrease with minimum at unfavorable climate.



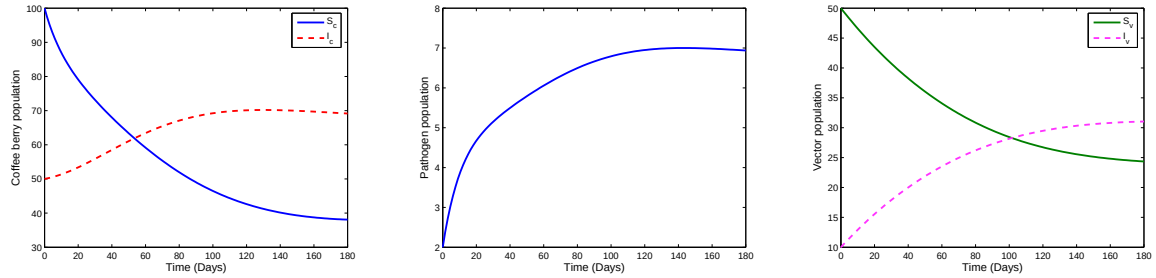
(a) (b)

Figure 7. Numerical simulation of the convergence of the infected coffee berries to the disease-free and endemic equilibrium points.



(a) (b)

Figure 8. Numerical simulation of the convergence of the infected vectors to the disease-free and endemic equilibrium points.



(a) (b) (c)

Figure 9. Numerical simulation of coffee berries, vector and pathogen population when $R_0 = 2.83$.

In the Figure 10(a),(b), the effects of the modification factor for asymptomatic coefficient at different values: $K = 150$ and $\beta_2 = 0.000795455$ are displayed. The projection shows that the cumulative number of individuals becoming infected coffee berries is high when K and β_2 become high and becoming decreasing with low values of K and β_2, which minimize also the values of basic reproduction number less one. As shown in Figure 10(c),(d), when the rates: K and β_2 increase, the number of individuals becoming infected vector is high and decreasing the value of decreases the number of individuals becoming infected. As shown in Figure 11(a),(b), when the rates: K and β_2 increase, the concentration of the pathogen becomes high, and decreasing the value of decreases the pathogen.

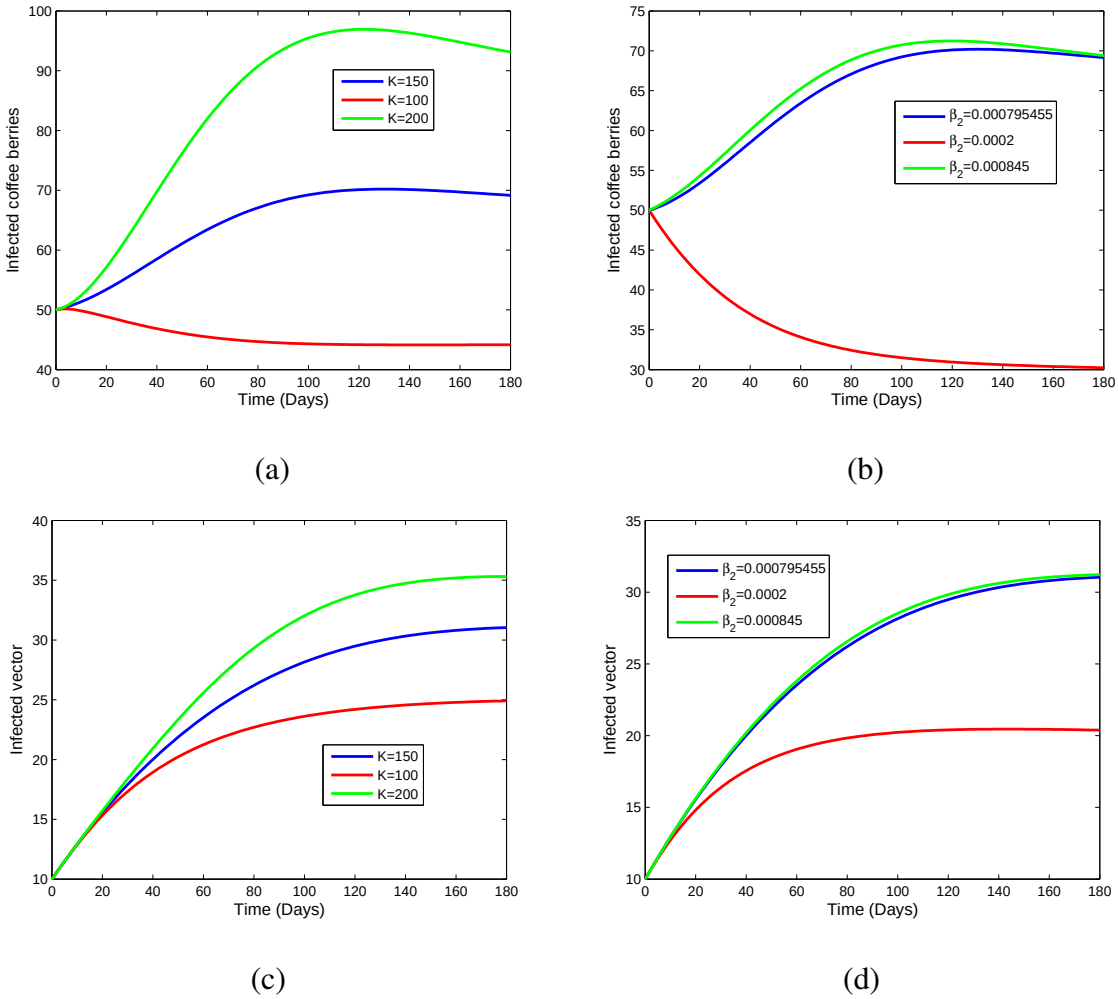
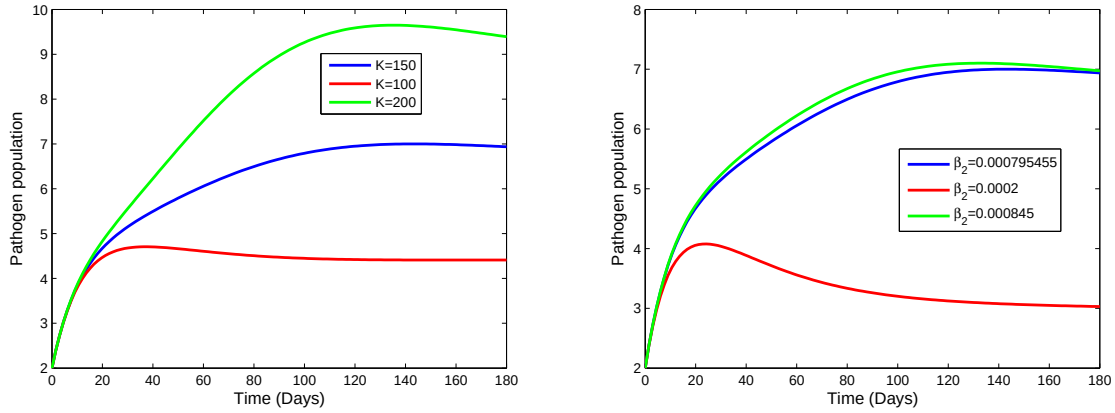


Figure 10. Projections of infected coffee and infected vector population with varying effect of parameters at values of $K = 100, K = 150, K = 200; \beta_2 = 0.0002, \beta_2 = 0.000795455, \beta_2 = 0.000845$.



(a) (b)

Figure 11. Projections of pathogen population with varying effect of parameters at values of $K = 100, K = 150, K = 200; \beta_2 = 0.0002, \beta_2 = 0.000795455, \beta_2 = 0.000845$.

4. Discussion and conclusions

In this paper, we formulated and analyzed a nonlinear deterministic mathematical model for CBD transmission dynamics in a coffee farm. A compartmental mathematical model by including coffee plants and vectors of disease with the interaction of fungal pathogen was investigated. We obtained the feasible region where the model is epidemiologically and mathematically meaningful. The positivity, existence and uniqueness of model solutions are also demonstrated. The equilibria (disease-free and endemic equilibrium) of the model were computed. We calculated the basic reproduction number at the disease-free equilibrium point by using the next-generation matrix method. The local stability of disease-free and endemic equilibria is shown using the Routh-Hurwitz criteria. Besides, the global stability of equilibria is proved by using the Lyapunov function. Moreover, bifurcation analysis is proved by the Center Manifold theory. The sensitivity indices of basic reproduction number with respect to the main parameters are computed. Further, sensitivity indices are graphically shown and the most influential parameters in expanding the disease are identified. Despite coffee playing a dominant role in the social, cultural, and national economy, the country's coffee industry is potentially at risk due to climate changes. We discussed the impact of *Colletotrichum kahawae* fungus and vectors of disease in relation to climatic variables. More vectors and CBD pathogens are produced at the favourable climate which indirectly increases the rate of infected coffee berries. Thus, the frequency and severity of climate extremes are increasing and making adaptation an absolute imperative necessity through using current information on climate variability to develop long-term plans for managing CBD. Since CBD is highly dependent upon climatic factors, we are doing for the future with a paper applying the climatic variability in this model. Numerical simulations of the model suggested we apply effective control interventions on some parameters to control the disease. This will be done using chemical, cultural, and biological control strategies. We conclude that according to our mathematical model we have an endemic equilibrium point and the coffee disease remains endemic provided that the basic reproduction number is greater than one. Thus, we are developing the model with optimal control for

future work to eradicate the disease.

Declaration of Competing Interest

The authors declare that there is no conflict of interests.

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