Modeling and Optimal Control of Cocoa Black Pod Disease in Osun State ☆

Abstract

Cocoa black pod disease poses a significant threat to global cocoa production, affecting both yield and quality. This article presents a comprehensive study on the modeling and optimal control of cocoa black pod disease. Using the stability theory of differential equations, we develop, carefully examine, and numerically implement a mathematical model that takes into account the dynamics of black pod disease transmission in relation to the stage at which cocoa pods are developing as well as the disease's transmission pathways and its control mechanism, the study also explores the application of optimal control strategies to mitigate the impact of the disease on cocoa crops. Using the next generation matrix method, the basic reproduction number for Cocoa black pod disease (CBPD) is discovered. The prerequisites for CBPD-free and endemic equilibria's local and global stability are established. According to the sensitivity analysis carried out, the reproduction number is most susceptible to the rates at which diseased pods are removed, fungicides are applied, and healthy pods are harvested. To optimally control the disease, Pontryagin's Maximum Principle(PMP) was applied to determine the best strategy to optimally control the disease. The result of the numerical simulations reveals that the combination of two strategies is required to effectively control the menace of the disease. The proposed optimal control measures provide a foundation for the design of targeted interventions, aiding cocoa farmers, researchers, and policymakers in safeguarding global cocoa production against this detrimental disease. This research contributes to the ongoing efforts to ensure the resilience and sustainability of the cocoa industry in the face of emerging challenges.

Keywords: Cocoa, Differential equations, Basic reproductive number, Disease-free equilibrium, Endemic equilibrium, Asymptotic stability Optimal control.

Mathematics Subject Classification (2010) 92D30, 37M05, 34H05.

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1. Introduction

The cocoa industry is still faced with problems like crop damage from pests and diseases, decreasing output, and environmental and health issues. Diseases that affect cacao includes black pod, swollen shoot, and witches broom diseases, among others. The prevalence of pests and diseases on cultivated cacao farms contributes a large and substantial percentage to the loss of the total yield and quality of the final beans produced [8, 7]. The black pod disease poses a major problem and also concern to the global cacao world, It is caused by various species of *Phytophthora* (P), notably among them are *P. megakarya* and *P. palmivora*. Cocoa black pod disease caused by *P. megakarya* is a devastating fungal disease and is a soil-borne pathogen [13, 20]. The fungus infects the pods of the cocoa tree, which are the fruit that contains the cocoa beans. The infection can happen through wounds or cracks in the pods, or through the flowers and stems of the tree, once the fungus enters the pod [4], it starts to grow and develop, causing the pod to turn black and rot. The infected pods eventually fall off the tree, reducing the yield of cocoa beans. P. megakarya thrives in humid and warm environments, which makes cocoa-growing regions in West and Central Africa particularly vulnerable to the disease. Poor agricultural practices such as overcrowding and lack of proper sanitation can also contribute to the spread of the disease. The economic impact of cocoa black pod disease can be devastating for small holder farmers who depend on cocoa production for their livelihoods. It is estimated that the disease can reduce cocoa yields by up to 70 percent, leading to significant losses in income for farmers and affecting the global cocoa supply chain, [2, 6, 17, 24]. The disease can be transmitted directly or indirectly [17]. Direct transmission occurs when the pathogen spreads from tree to tree through infected plant parts such as leaves, stems, or pods. This can happen when rainwater washes the spores from an infected plant onto healthy plants growing nearby, or when tools or equipment used in pruning or harvesting infected plants are not properly disinfected. Indirect transmission occurs when the pathogen is carried by other organisms, such as insects or animals, that come into contact with infected plants. For example, some studies suggest that beetles may be responsible for spreading the pathogen between cocoa trees. Infected soil, water, and wind-blown debris can also spread the disease (Environment to pod transmission) [15].

Preventing the transmission of P. megakarya requires a combination of measures that include planting resistant varieties, practicing good sanitation, avoiding the movement of infected plant material, and using fungicides when necessary.

The bulk of epidermic models found in the literature were created using [11] groundbreaking research as their foundation. According to [23, 25], the differential equation used in the model give rise to the basic reproduction number needed to estimate the escalation of the epidemic. A study of the reproduction number's sensitivity identifies the factors that have the biggest effects on disease control and ought to be the main focus of prevention strategies [14, 18]. There are only a few studies in the literature on the mathematical modeling of cocoa disease [17, 18]. To control the spread of the disease, the study intends to develop, assess, and apply a new model that captures the dynamics and control of the disease coupled with direct and indirect modes of transmission.

The risk of the parasitic infection on cacao pods is of extreme uneasiness to cocoa growers and researchers, nevertheless the principles behind the rise of *P megakarya* on cacao are unresolved as a result, it is an increasing desire for fundamental understanding into the epidemiology of *P. megakarya* in order to develop practical and effective management strategies; [17]'s work has greatly contributed in this area, particularly in the area of transmission. In order to determine the best way to treat the disorder, we also added Removed compartment (R) and Treatment state (T) to our model. Additionally, we used sensitivity analysis of parameters to determine the most crucial parameters in deciding the seriousness of the disease.

Furthermore, the article explores the frontier of optimal control, aiming to identify strategies that not only curb the spread of black pod disease but do so with an eye toward sustainability, economic viability, and ecological balance. From targeted fungicide applications to precision pruning techniques, this research investigates a spectrum of control measures to ascertain the most efficient and resource-effective approaches. Employing optimization algorithms, the study seeks to uncover the optimal control parameters that strike a balance between disease containment and the broader considerations of economic feasibility and environmental impact. In the face of mounting challenges posed by cocoa black pod disease, this article endeavors to provide a comprehensive framework for optimal control, offering insights that extend beyond theoretical models to practical, actionable strategies. By doing so, we aspire to contribute to the resilience and sustainability of cocoa cultivation, safeguarding the global supply chain and ensuring the continued enjoyment of this indulgent treat for generations to come.

2. Cocoa Production in Osun State

Osun State is a significant player in Nigeria's cocoa production landscape, consistently ranking among the top producers in the country. The State is the second-largest cocoa producer in Nigeria, contributing substantially to the national cocoa output. The state boasts fertile soils and favorable climatic conditions, making it an ideal environment for cocoa cultivation. Cocoa production plays a vital role in the state's economy, providing livelihoods for numerous farmers and contributing to local and national revenue. The Osun State Government recognizes the importance of cocoa and has implemented various initiatives to promote its production, including: Subsidized seedlings and fertilizers, Agricultural extension services and Infrastructure development.

To investigate the impact of black pod disease, we surveyed selected cocoa farms across all local government areas in Osun State. We visited 20 farms per local government and, with the assistance of local farmers, recorded the number of cocoa pods showing signs of black pod disease.

The plot below display the finding regarding incidence of Cocoa black pod in Osun State.

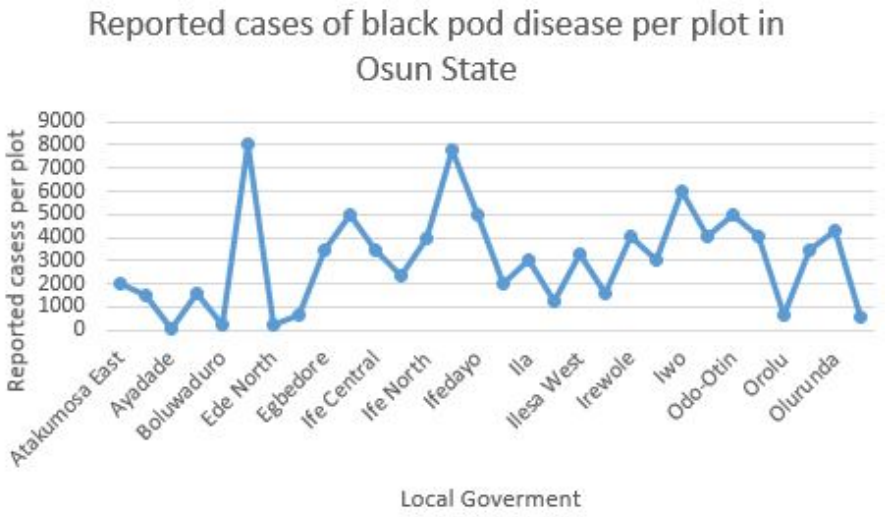

Figure 1: Line chart displaying incidence of Cocoa black pod disease in Osun State

3. MODEL DEVELOPMENT

In this part, we give a mathematical model that describe the transmission of infection among the infectious environment, infected pods and the healthy cocoa pods. The total population $N(t)$

at time $t > 0$ was sub-divided according to stages of development of cocoa pods, the first stage of cocoa pod development which is the floral and fruiting state is called Cherelles (S_c), next is the young and mature pod stage (S_p), lastly is the ripe pod stage (S_r).

The infectious pod compartment is represented by I, I_s denotes the secondary infection(indirect transmission), likewise the primary infection(direct transmission) I_p are infection transmitted through the spores from infected pods to healthy pods. T denotes the treated pods and R represent the removed pod compartment.

Table 1: Description of model parameters.

Parameter	Descriptions	Unit	value/ range of value	Source
k	Cherelles recruitment rate	Day^{-1}	12	[3]
d_o	Natural death rate	Day^{-1}	0.05	[21]
β_1	Primary infection rate at Cherelles stage	Day^{-1}	0.05	[17]
β_2	Secondary infection rate at Cherelles stage	Day^{-1}	0.05	[17]
β_3	Primary infection rate at young and mature pod stage	Day^{-1}	0.02	[17]
β_4	Secondary infection rate at young and mature pod stage	Day^{-1}	0.02	[17]
γ_1	Transmission rate from S_c to S_p	Day^{-1}	0.05	[22]
γ_2	Transmission rate from S_p to S_r	Day^{-1}	0.027	[21]
γ_3	Rate of ripe infected pods	Day^{-1}	0.1	Estimated
ψ	Healthy pod harvesting rate	Day^{-1}	0.01	Estimated
θ_1	Rate of fungicide application	Day^{-1}	0.1	Estimated
θ_2	Rate of recovery of treated pods	Day^{-1}	0.09	Estimated
n	releasing rate of spore shedding	$SporesDay^{-1}$	0.4	[17]
ω	Speed rate of spore shedding	$SporesDay^{-1}$	0.4	[17]
δ	Rate of phytosanitary pod removal	Day^{-1}	[0-0.8]	[17]
N_o	Michaelis constant for infection transmission	No. od Spores	$[0 - 10^{10}]$	[17]

The inflow of new susceptible into the Cherelles compartment was considered at rate k (recruitment rate), this compartment reduces by (γ_1) which is the rate of transmission from Cherelles to young and mature stage, (d_o) the natural death rate at Cherelles stage and the forces of infection rate $((\sigma_1)\&(\sigma_2))$ denoted by

$$\sigma_1 = \frac{\beta_1 I_s}{N_o + I_s}, \tag{1}$$

$$\sigma_2 = \frac{\beta_2 I_p}{N_o + I_p}, \tag{2}$$

where N_o is the Michaelis constant for the disease transmission of pod infection, (β_1) & (β_2) denotes the primary and secondary infection rate at Cherelles stage respectively.

The young and mature susceptible compartment increases through transmission rate from Cherelles compartment (γ_1) and reduces due to ripening rate (γ_2) the ripening rate of young and mature pods , (d_o) the natural death rate at young and mature pod stage and forces of infection (σ_3) & (σ_4)

$$\sigma_3 = \frac{\beta_3 I_s}{N_o + I_s}, \tag{3}$$

$$\sigma_4 = \frac{\beta_4 I_p}{N_o + I_p}, \tag{4}$$

where (β_3) & (β_4) denotes the primary and secondary infection rate at mature pod stage respectively.

The ripening susceptible compartment increases by (γ_2) the ripening rate of young and mature pods and decreases by d_o the natural death rate at this stage and (ψ) is the pod harvesting rate

Infectious compartment increases due to the effective contact rates (σ_1) , (σ_2), (σ_3) , (σ_4) and reduces through the releasing rate of spores (n), (ω) & (θ_1), natural death (d_o), rate of infected pod removal (δ) and (γ_3) which is the ripe infected pod removal rate (the removed ripe pod here are infected but are still of economic importance).

The treated pods compartment increases due to the effective rate of fungicide spraying (θ_1) and reduces due to the infected pod recovery rate (θ_2) and natural death (d_o).

The recovery compartment increases due to the inflow (θ_2) from treatment compartment, ripe infected pod (γ_3) from infected class, healthy ripe pod harvested rate (ψ) and the compartment reduces due to the natural death (d_o).

The production rate of spore release rate n causes the secondary infection compartment to grow, and the natural death rate (d_o) causes it to reduce. The main infection compartment rises as a result of the spore release rate ω and reduces as a result of the natural death rate (d_o).

The set of first-order ordinary differential equations that are nonlinear governing the transmission in the figure 2 is provided as follows, under the aforementioned assumptions:

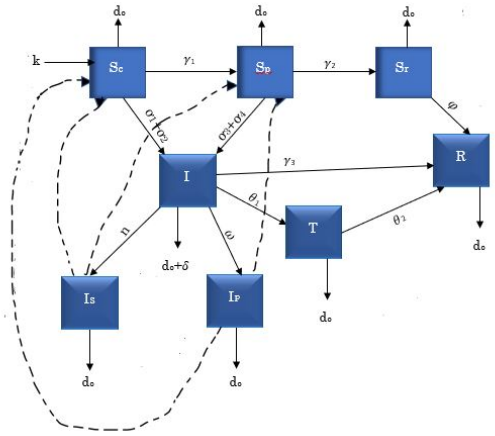

Figure 2: Compartmental diagram for the transmission dynamics of CBPD

$$
\begin{aligned}
\frac{dS_c}{dt} &= k - (d_o + \sigma_1 + \sigma_2 + \gamma_1)\, S_c, \\
\frac{dS_p}{dt} &= \gamma_1 S_c - (\sigma_3 + \sigma_4 + \gamma_2 + d_o)\, S_p, \\
\frac{dS_r}{dt} &= \gamma_2 S_p - (d_0 + \psi)\, S_r, \\
\frac{dI}{dt} &= (\sigma_1 + \sigma_2)\, S_c + (\sigma_3 + \sigma_4)\, S_p - (n + \omega + \theta_1 + \gamma_3 + d_o + \delta)\, I \\
\frac{dT}{dt} &= \theta_1 I - (\theta_2 + d_0)\, T, \\
\frac{dR}{dt} &= \psi S_r + \gamma_3 I + \theta_2 T - d_o R, \\
\frac{dI_s}{dt} &= nI - d_o I_s \\
\frac{dI_p}{dt} &= \omega I - d_o I_p.
\end{aligned}
\tag{5}
$$

Subject to the initial conditions

$$(S_c, S_p, S_r, I, T, R, I_s, I_p) \geq 0. \tag{6}$$

4. MODEL ANALYSIS

In this section, we considered the analysis of the model by considering its feasibility, positivity, disease free & endemic equilibrium and Stability of the developed model.

4.1. Boundedness(Feasibility) of the Model

Theorem 4.1. The domain $\Omega = \left\{(S_c, S_p, S_r, I, T, R, I_s, I_p) \in \Re^8_+; 0 \leq N(t) \leq \frac{k}{d_o}\right\}$ for the model system (5) with non negative starting conditions in $\Re^8_+$, is positively invariant and attracting.

Proof. Let

$$N(t) = S_c(t) + S_p(t) + S_r(t) + I(t) + T(t) + R(t) + I_s(t) + I_p(t). \tag{7}$$

Then, we have

$$\frac{dN(t)}{dt} = \frac{dS_c(t)}{dt} + \frac{dS_p(t)}{dt} + \frac{dS_r(t)}{dt} + \frac{dI(t)}{dt} + \frac{dT(t)}{dt} + \frac{dR(t)}{dt} + \frac{dI_s(t)}{dt} + \frac{dI_p(t)}{dt}, \tag{8}$$

$$\frac{dN}{dt} = k - d_o N(t) - \delta I(t), \tag{9}$$

Equation (9) is known as model population dynamics. In the absence of infection $\delta = 0$, equation (9) is reduced to a differential inequality,

$$\frac{dN}{dt} \leq k - d_o N(t), \tag{10}$$

on integrating the differential inequality (10), we have

$$-\frac{1}{d_o} \ln(k - d_o N(t)) \leq t + C_o, \quad \text{(where } C_o \text{ is constant of integration)} \tag{11}$$

$$k - d_o N(t) \leq A e^{-d_o t}, \quad \text{where A is a constant, } A = e^{-d_o C_o}, \tag{12}$$

at $t = 0$

$$k - d_o N(0) \leq A, \tag{13}$$

substituting equation (13) into (12) yields

$$N(t) \leq \frac{k}{d_o}(1 - e^{-dt}) + N(0)e^{-d_o t},$$

as $t \to \infty$

$$N(t) \leq \frac{k}{d_o}, \tag{14}$$

hence, the feasible(boundedness) solution for the system (5)is given by

$$\Omega = \left\{ (S_c, S_p, S_r, I, T, R, I_s, I_p) \in \Re_+^8; 0 \leq N(t) \leq \frac{k}{d_o} \right\}$$

is a compact forward invariant set.

Every solution with a starting condition of $\Re_+^8$ thus stays in the Ω region for $t > 0$. As such, the model system (5) is both mathematically and epidemiologically well-posed. □

4.2. Positivity of solutions

The following theorem, which needs to be confirmed, will establish that all solutions of the system with positive starting data will remain positive for all time $t > 0$.

Theorem 4.2. System (5) maintains the positivity of the solutions, which means that the system's initial conditions for the state variables are always greater than zero for all time t.

Proof. Considering the first equation in system (5),

$$\frac{dS_c}{dt} = k - (d_o + \sigma_1 + \sigma_2 + \gamma_1)\, S_c$$

$$\frac{dS_c}{dt} \geq -(d_o + \sigma_1 + \sigma_2 + \gamma_1)S_c, \tag{15}$$

Integrating the inequality in (15) gives

$$S_c(t) \geq Be^{-(d_o+\sigma_1+\sigma_2+\gamma_1)t}, \tag{16}$$

where C_1 is constant of integration and $B = e^{C_1}$,

at $t = 0$, equation (16) becomes

$$S_c(0) \geq B, \tag{17}$$

inserting (17) into (16) gives

$$S_c(t) \geq S_c(0)e^{-(d_o+\sigma_1+\sigma_2+\gamma_1)t} \geq 0, \tag{18}$$

since

$$(d_o + \sigma_1 + \sigma_2 + \gamma_1)t > 0, \tag{19}$$

hence $S_c(t)$ is positive for $t > 0$. Similar proof can be established for the positivity of the other solutions in equation (5). Thus the solution of the model in equation(5) are positive for all time $t > 0$. □

4.3. Disease Free Equilibrium (DFE)

When there are no infections among the pods population, it is said to be in a condition of DFE, all DFE classes will be denoted by (o). In the absence of disease, we assume that I^o=$I_p^o = I_s^o$, see [10] for details.

let

$$E^o = \left(S_c^o, S_p^o, S_r^o, I^o, T^o, R^o, I_s^o, I_p^o, \right) \tag{20}$$

be the DFE state for the population, therefore the DFE point is given by

$$\begin{aligned} S_c^o &= \frac{k}{d_o + \gamma_1} \\ S_p^o &= \frac{\gamma_1 k}{(d_o + \gamma_1)(d_o + \gamma_2)} \\ S_r^o &= \frac{\gamma_2 \gamma_1 k}{(d_o + \gamma_1)(d_o + \gamma_2)(d_o + \psi)} \\ R^o &= \frac{\psi \gamma_2 \gamma_1 k}{d_o (d_o + \gamma_1)(d_o + \gamma_2)(d_o + \psi)} \end{aligned} \tag{21}$$

hence, the DFE of the model is

$$E^o = \left(\frac{k}{d_o + \gamma_1}, \frac{\gamma_1 k}{(d_o + \gamma_1)(d_o + \gamma_2)}, \frac{\gamma_2 \gamma_1 k}{(d_o + \gamma_1)(d_o + \gamma_2)(d_o + \psi)}, 0, 0, \frac{\psi \gamma_2 \gamma_1 k}{d_o (d_o + \gamma_1)(d_o + \gamma_2)(d_o + \psi)}, 0, 0 \right).$$

4.4. Endemic Equilibrium State(EES)

EES (E^+) is the state where the disease persist in the pod population, let (see [10] for details.)

$$E^+ = \left(S_c^+, S_p^+, S_r^+, I^+, T^+, R^+, I_s^+, I_p^+, \right) \tag{22}$$

where

$$\begin{aligned} S_c^+ &= \frac{k}{(d_o + \sigma_1 + \sigma_2 + \gamma_1)}, \\ S_p^+ &= \frac{\gamma_1 k}{(\sigma_3 + \sigma_4 + d_o + \gamma_2)(d_o + \sigma_1 + \sigma_2 + \gamma_1)}, \end{aligned}$$

$$S_r^+ = \frac{\gamma_1\gamma_2 k}{(d_o+\psi)(\sigma_3+\sigma_4+d_o+\gamma_2)(d_o+\sigma_1+\sigma_2+\gamma_1)},$$

$$I^+ = \frac{\phi_1 k}{\phi_2},$$

$$T^+ = \frac{\theta_1 k\phi_1}{(\theta_2+d_o)\phi_2},$$

$$I_s^+ = \frac{nk\phi_1}{\phi_2},$$

$$I_p^+ = \frac{\omega k\phi_1}{\phi_2},$$

$$R^+ = (\phi_2\gamma_1\gamma_2 k\psi(\theta_2+d_o) + (d_o+\psi)(\sigma_3+\sigma_4+d_o+\gamma_2)(\sigma_1+\sigma_2+d_o+\gamma_1)[\gamma_3(\theta_2+d_o)+\theta_1\theta_3]\phi_1)$$

$$/\,(\phi_2 d_o(d_o+\psi)(\theta_2+d_o)(\sigma_3+\sigma_4+d_o+\gamma_2)(\sigma_1+\sigma_2+d_o+\gamma_1))$$

where

$$\phi_1 = [(\sigma_1+\sigma_2)(\sigma_3+\sigma_4+d_o+\gamma_2)+\gamma_1(\sigma_3+\sigma_4),]$$

$$\phi_2 = (n+\omega+\theta_1+\gamma_3+d+\delta)(\sigma_3+\sigma_4+d_o+\gamma_2)(\sigma_1+\sigma_2+d_o+\gamma_1).$$

5. BASIC REPRODUCTION NUMBER

The overall number of illnesses caused by one freshly contaminated pods brought into a healthy community is known as the basic reproduction number ($\Re_o$). Computation of $\Re_o$ is carried out using the next generation matrix as laid out in [23]. $\Re_o$ is obtained using

$$\Re_o = \rho\left(FV^{-1}\right), \tag{23}$$

where ρ is the spectral radius of the matrix FV^{-1} Differential equations associated with I, T, I_s & I_s compartment are the infective classes and will be used in the computation of $\Re_o$.

$$\begin{aligned}
\frac{dI}{dt} &= (\sigma_1+\sigma_2)S_c + (\sigma_3+\sigma_4)S_p,\\
&-(n+\omega+\theta_1+\gamma_3+d_o+\delta)I,\\
\frac{dT}{dt} &= \theta_1 I - (\theta_2+d_0)T,\\
\frac{dI_s}{dt} &= nI - d_o I_s,\\
\frac{dI_p}{dt} &= \omega I - d_o I_p,
\end{aligned} \tag{24}$$

Operator F_i is the rate at which new infection arises and V_i is the rate which compartments corresponding to the infection are exited.

$$F_i = \begin{pmatrix} \left(\frac{\beta_1 I_s}{N_o+I_s} + \frac{\beta_2 I_p}{N_o+I_p}\right) S_c + \left(\frac{\beta_3 I_s}{N_o+I_s} + \frac{\beta_4 I_p}{N_o+I_p}\right) S_p \\ 0 \\ 0 \\ 0 \end{pmatrix}, V_i = \begin{pmatrix} (n+\omega+\theta_1+\gamma_3+d_o+\delta)\, I \\ (\theta_2+d_0)\, T - \theta_1 I \\ d_o I_s - nI \\ d_o I_p - \omega I \end{pmatrix}.$$

Obtaining the partial derivative of F_i and V_i with respect to I, T, I_s, I_p, and later substituting the value of S_c, S_p, I_s and I_p at DFE, we obtain F and V as

$$F = \begin{pmatrix} 0 & 0 & \frac{\beta_1 k}{N_o(d_o+\gamma_1)} + \frac{\beta_3\gamma_1 k}{N_o(d_o+\gamma_1)(d_o+\gamma_2)} & \frac{\beta_2 k}{N_o(d_o+\gamma_1)} + \frac{\beta_4\gamma_1 k}{N_o(d_o+\gamma_1)(d_o+\gamma_2)} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

$$V = \begin{pmatrix} (n+\omega+\theta_1+\gamma_3+d_o+\delta) & 0 & 0 & 0 \\ -\theta_1 & (\theta_2+d_o) & 0 & 0 \\ -n & 0 & d_o & 0 \\ -\omega & 0 & 0 & d_o \end{pmatrix}.$$

$\Re_o$, which is the dominant eigenvalue of equation (22) is expressed as

$$\Re_o = \frac{k\left(n\beta_1 d_o + n\beta_1\gamma_2 + n\beta_3\gamma_1 + \omega\,\beta_2 d_o + \omega\,\beta_2\gamma_2 + \omega\,\beta_4\gamma_1\right)}{N_o\left(d_o+\gamma_1\right) d_o\left(\delta\, d_o + \delta\,\gamma_2 + n d_o + n\gamma_2 + \omega\, d_o + \omega\,\gamma_2 + {d_o}^2 + d_o\gamma_2 + d_o\gamma_3 + \gamma_2\gamma_3\right)}. \quad (25)$$

Remark 5.1. (i) If $\Re_o < 1$, the infection on the pods will be decreasing.

(ii) If $\Re_o = 0$, the pod infection will be constant(remain the same).

(iii) If $\Re_o > 1$, The infection on the pods will appear more frequently and last longer.

6. Stability Analysis

6.1. Local Stability of DFE State

Theorem 6.1. If $\Re_o < 1$, the DFE point of the equation (5) is locally asymptotically stable; otherwise, it is unstable.

Proof. : Based on the eigenvalue λ of the Jacobian at E^o, the local asymptotic stability is obtained.If the real parts of λ are all negative, the E^o is locally asymptotically stable, and the Jacobian matrix at E^o is given as

$$J(E^o) = \begin{pmatrix} -(d_o+\gamma_1) & 0 & 0 & 0 & 0 & 0 & -\frac{\beta_1 S_c}{N_g} & -\frac{\beta_2 S_c}{N_g} \\ 0 & -(d_o+\gamma_2) & 0 & 0 & 0 & 0 & -\frac{\beta_3 S_p}{N_o} & -\frac{\beta_4 S_p}{N_o} \\ 0 & \gamma_2 & (d_o+\psi) & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -(n+\omega+\theta_1+\gamma_3+d_o+\delta) & 0 & 0 & \frac{\beta_1 S_c+\beta_3 S_p}{N} & \frac{\beta_2 S_c+\beta_4 S_p}{N_o} \\ 0 & 0 & 0 & \theta_1 & -(d_o+\theta_2) & 0 & 0 & 0 \\ 0 & 0 & \psi & \gamma_3 & \theta_2 & -d_o & 0 & 0 \\ 0 & 0 & 0 & \omega & 0 & 0 & -d_o & 0 \\ 0 & 0 & 0 & \omega & 0 & 0 & 0 & -d_o \end{pmatrix} \tag{26}$$

The characteristic equation is given by

$$\lambda^2 + (n+\omega+\theta_1+\gamma_3+2d_o+\delta)\,\lambda + d_o\,(n+\omega+\theta_1+\gamma_3+d_o+\delta)\,(1-\Re_o) = 0 \tag{27}$$

Clearly the eigenvalues of the system are negative, thus E^o will be locally asymptotically stable if $\Re_o < 1$ and when ever $\Re_o > 1$ is unstable and disease will persist. □

6.2. Global Stability Analysis

The globally stability of the disease-free and endemic equilibrium is established using the Lyapunov method and Lasalle's invariance principle to identify the control condition under which the disease can be eradicated.

Theorem 6.2. (i) The disease-free equilibrium point E^o tends to remain asymptotically stable in Ω if $\Re_0 \leq 1$,

(ii) The endemic equilibrium point E^+ is usually asymptotically stable in Ω if $\Re_0 > 1$.

Proof. Theorem 5.2 (1) and 5.2 (2) can be proved using a already constructed Lyapunov function, we adopt the quadratic Lyapunov function used in [9].
Consider the Lyapunov candidate

$$L = \frac{1}{2}\left[(S_c - S_c^o) + \left(S_p - S_p^o\right) + (S_r - S_r^o) + (I - I^o) + (T - T^o) + (R - R^o) + (I_s - I_s^o) + \left(I_p - I_p^o\right)\right]^2,$$

on differentiating the function L with respect to time yields

$$\frac{dL}{dt} = \frac{1}{2}\left[(S_c - S_c^o) + \left(S_p - S_p^o\right) + (S_r - S_r^o) + (I - I^o) + (T - T^o) + (R - R^o)\,(I_s - I_s^o) + \left(I_p - I_p^o\right)\right]\frac{dN}{dt}, \tag{28}$$

substituting the value of S_c^o, S_p^o, S_r^o, I^o, T^o, R^o, I_s^o, I_p^o and $\frac{dN}{dt}$ into equation (28) yields

$$\frac{dL}{dt} = \left[N - \frac{k\left[\left(d_o+\gamma_1\right)\left(d_o+\gamma_2\right)\right]}{d_o\left(d_o+\gamma_1\right)\left(d_o+\gamma_2\right)}\right]\left(k - d_oN - \delta I\right)$$

for $I = 0$ at DFE

$$\frac{dL}{dt} = -\frac{1}{d_o}\left(k - d_oN\right)^2$$

$$\frac{dL}{dt} \leq 0. \tag{29}$$

hence , $L \leq 0$, if k, d_o and N are positive, this implies that the function L is strictly Lyapunuv function which indicate the disease free equilibrium point (E^o) is globally asymptotically stable. Next, we establish prove for Theorem 5.2 (2). Let V define the Lyapunuv function

$$V : \{(S_c, S_p, S_r, I, T, R, I_s, I_p) \in \Omega | S_c, S_p, S_r, I, T, R, I_s, I_p > 0\} \longrightarrow \Re_8^+$$

(see [12, 19] for details).

$$V = S_c - S_c^+ \ln S_c + S_p - S_p^+ \ln S_p + S_r - S_r^+ \ln S_r + b_1\left(I - I^+ \ln I + +b_2\right)\left(T - T^+ \ln T\right) + b_3\left(R - R^+ \ln R\right)$$

$$+b_4\left(I_s - I_s^+ \ln I_s\right) + b_5\left(I_p - I_p^+ \ln I_p\right)$$

on differentiating with respect to time

$$\frac{dV}{dt} = \left(1 - \frac{S_c^+}{S_c}\right)\frac{dS_c}{dt} + \left(1 - \frac{S_p^+}{S_p}\right)\frac{dS_p}{dt} + \left(1 - \frac{S_r^+}{S_r}\right)\frac{dS_r}{dt} + b_1\left(1 - \frac{I^+}{I}\right)\frac{dI}{dt} + b_2\left(1 - \frac{T^+}{T}\right)\frac{dT}{dt}$$

$$+b_3\left(1 - \frac{R^+}{R}\right)\frac{dR}{dt} + b_4\left(1 - \frac{I_s^+}{I_s}\right)\frac{dI_s}{dt} + b_5\left(1 - \frac{I_p^+}{I_p}\right)\frac{dI_p}{dt}$$

$$= \left(1 - \frac{S_c^+}{S_c}\right)\left[k - \left(d_o + \gamma_1\right)S_c - \left(\sigma_1 + \sigma_1\right)S_c\right] + \left(1 - \frac{S_p^+}{S_p}\right)\left[\gamma_1 S_c - \left(\sigma_3 + \sigma_4\right)S_p - \left(\gamma_2 + d_o\right)S_p\right] +$$

$$\left(1 - \frac{S_r^+}{S_r}\right)\left[\gamma_2 S_p - \left(d_o + \psi\right)S_r\right] + b_1\left(1 - \frac{I^+}{I}\right)\left[\left(\sigma_1 + \sigma_2\right)S_c + \left(\sigma_3 + \sigma_4\right)S_p - \left(n + \omega + \theta_1 + \gamma_3 + d_o + \delta\right)I\right]$$

$$+b_2\left(1 - \frac{T^+}{T}\right)\left[\theta_1 I - \left(\theta_2 + d_o\right)T\right] + b_3\left(1 - \frac{R^+}{R}\right)\left[\psi S_r + \gamma_3 I + \theta_2 T - d_o R\right] + b_4\left(1 - \frac{I_s^+}{I_s}\right)\left[nI - d_o I_s\right] +$$

$$b_5\left(1 - \frac{I_p^+}{I_p}\right)\left[\omega I - d_o I_p\right] \tag{30}$$

Substitute for the value of from equation5 and simplification gives

$$= -[(1 - \frac{S_c^+}{S_c})(d_o + \sigma_1 + \sigma_2 + \gamma_1)(S_c - S_c^+) + \gamma_1(\frac{S_p^+}{S_p} - 1)S_c + (\sigma_3 + \sigma_2 + d_o + \gamma_2)[S_p - S_p^+] + \gamma_2)(S_r - S_r^+)S_p$$

$$+(d_o+\psi)[S_r-S_r^+]+b_1(\sigma_1+\sigma_2)(\frac{I^+}{I}-1)S_c+b_1(\sigma_3+\sigma_4)(\frac{I^+}{I}-1)S_p+$$

$$b_1(n+\omega+\theta_1+\gamma_3+d_o+\delta)(I-I^+)+b_2\theta_1(\frac{I^+}{I}-1)I+b_2(\theta_2+d_o)(T-T^+)+$$

$$b_3\psi(\frac{R^+}{R}-1)S_r+b_3\gamma_3(\frac{R^+}{R}-1)I+b_3\theta_2(\frac{R^+}{R}-1)T+b_3d_o(R-R^+)+b_4n(\frac{I^+}{I}-1)+$$

$$b_4d_o(I_s-I_s^+)+b_5\omega(\frac{I_p^+}{I_p}-1)-b_5d_o(I_p-I_p^+)] \tag{31}$$

Hence $V<0$, if and only if b_1, b_2, b_3, b_4,$b_5>0$. If $V=0$ if and only if $S_c=S_c^+$, $S_p=S_p^+$, $S_r=S_r^+$, $I=I^+$, $T=T^+$, $R=R^+$, $I_s=I_s^+$, $I_p=I_p^+$. Therefore E^+ is globally asymptotically stable in the interior of Ω.

We have demonstrated that the disease can be controlled as long as the threshold $\Re_o \leq 1$ and the DFE point E^o are globally asymptotically steady. The endemic equilibrium point is globally asymptotically stable and the disease will continue if $\Re_o > 1$. From epidemiological perspective, the objective is to limit the spread of the illness by lowering $\Re_o$ to less than one and optimizing economic profits. To accomplish this, it is necessary to determine the variables that have the biggest effects on the stability of $\Re_o$. □

7. SENSITIVITY ANALYSIS

$\Re_o$ is the epidemic threshold that controls spread, understanding of various disease transmission, the variables is helpful in determining the most effective control approach. Predicting the sensitivities of each component involved in $\Re_o$ is crucial. Sensitivity analysis measures how much a variable has changed in relation to how much the factors have changed.

Definition 7.1. The following formula is used to describe the standardized forward sensitivity index of a variable B that differently relies on a parameter m: [1, 5].

$$Z_m^B=\frac{\partial B}{\partial m}\times\frac{m}{B} \tag{32}$$

The sensitivity indices of reproduction number $\Re_o$ corresponding to our model parameters is given as

$$Z_k^{R_o}=\frac{\partial R_o}{\partial k}\times\frac{k}{R_o}=+1.0000 \tag{33}$$

In a similar approach, we obtain the remaining indices for the model parameters as displayed in Table 2.

Table 2: sensitivity indices of $\Re_o$.

Parameters	Sensitivity indices
k	+ve
n	+ve
d_o	-ve
ω	+ve
δ	-ve
ψ	-ve
β_1	+ve
β_2	+ve
β_3	+ve
β_4	+ve
θ_1	-ve
γ_1	-ve
γ_2	-ve
γ_3	-ve
N_o	-ve

The value of $\Re_o$ increases when the index with a positive indication are increased, also the value of $\Re_o$ reduces when the index with a negative indication are increased, thus making the value with negative indices a importance parameter used in the control of the disease.

The most sensitive parameters of our model are θ_1, δ, γ_3, γ_1 and d_o, since it is not practical to increase d_o, γ_3, γ_2, γ_1, ψ and d_o, thus reducing $\Re_o$ is a function of θ_1 and δ. The result suggested that intervention strategy should be targeted at fungicide spraying rate θ_1, and rate of infected pod removal δ.

8. OPTIMAL CONTROL OF COCOA BLACK POD DISEASE

8.1. Mathematical Formulation

The system in equation (5) was extended by incorporating time dependent control $u(t)$.

The optimal control in this article focuses on:

(i) u_1 : Prevention against pod infection (practicing good sanitation, avoiding the movement of infected plant material, pod removal),

(ii) u_2 : Treatment effort on the infected pods, they measure includes using an appropriate fungicides application.

The optimal control variables incorporated into equation 5 is given below:

$$\begin{aligned}
\frac{dS_c}{dt} &= k - (d_o + \sigma_1 + \sigma_2 + \gamma_1 + u_1) S_c \\
\frac{dS_p}{dt} &= \gamma_1 S_c - (\sigma_3 + \sigma_4 + \gamma_2 + d_o) S_p \\
\frac{dS_r}{dt} &= \gamma_2 S_p - (d_0 + \psi) S_r \\
\frac{dI}{dt} &= (\sigma_1 + \sigma_2 + u_1) S_c + (\sigma_3 + \sigma_4) S_p - (n + \omega + \theta_1 + \gamma_3 + d_o + \delta + u_2) I \\
\frac{dT}{dt} &= (\theta_1 + u_2) I - (\theta_2 + d_0) T \\
\frac{dR}{dt} &= \psi S_r + \gamma_3 I + \theta_2 T - d_o R \\
\frac{dI_s}{dt} &= nI - d_o I_s \\
\frac{dI_p}{dt} &= \omega I - d_o I_p
\end{aligned} \tag{34}$$

where $0 \leq u(t) \leq 1$ is the control on the pod to reduce the disease. The objective function J is defined over a feasible set of control $u(t)$ applied over the finite time interval $[t_o, T]$, which is given in the equation below

$$J(\cup) = \min_{\cup} \int_{t_o}^{T} \left[W_1 S_c(t) + W_2 I(t) + \frac{1}{2} \left(A_1 u_1^2 + A_2 u_2^2 \right) \right] dt \tag{35}$$

where t_o, T are called initial and final time respectively. W_1 and W_2 are weight constraints factors corresponding to S_c and I respectively, while A_1 and A_2 are coefficient for each control measure. The term $A_1 u_1^2$ & $A_2 u_2^2$ represent the cost associated with the prevention of infection in cherelles and treated pods respectively. The square of the control variables is taken to remove the severity of the control applied and the side effects of the fungicides. The integrand is in quadratic form because we assume that cost are non-linear in nature [16]. Where $A_1 u_1^2$ and $A_2 u_2^2$ represent the cost associated with the prevention of infection in cherelles and treated pods respectively, W_1 and W_2 are weight constraints factors corresponding to S_c and I. The goal of the research is to minimize the cost of the control $u(t)$ and the number of infected pods, thus we seek an optimal control such that

$$J(u^*) = \text{Min } \{J(u)\}. \tag{36}$$

Pontrayagin's maximum principle gives the necessary conditions that an optimal control problem must satisfy. The principle convert equation (34) and (35) to a problem of minimizing point wise Hamiltonian **H**, with respect to $u(t)$ defined by

$$H(S_c, S_p, S_r, I, T, R, I_s, I_p, u_1, u_2, t) = W_1 S_c(t) + W_1 I(t) + \frac{A_1}{2} u_1^2 + \frac{A_2}{2} u_2^2 + \sum_{i=1}^{8} \lambda_i q_i(t, x, u_1, u_2), \tag{37}$$

where λ_i are adjoint variables. Considering

$$\frac{\partial H}{\partial y} = -\frac{\partial \lambda_i}{\partial t}, \tag{38}$$

we have

$$\begin{aligned}
-\frac{\partial H}{\partial S_c} &= -W_1 + \lambda_1\left[d + \tfrac{\beta_1}{N_o+I_s}I_s + \tfrac{\beta_2}{N_o+I_p}I_p + \gamma_1 + u_1\right] - \lambda_2\gamma_1 - \lambda_4\left[\tfrac{\beta_1}{N_o+I_s}I_s + \tfrac{\beta_2}{N_o+I_p}I_p + u_1\right] \\
-\frac{\partial H}{\partial S_p} &= \lambda_2\left[\beta_3 N_o + I_s I_s + \tfrac{\beta_4}{N_o+I_p}I_p + \gamma_2 + d\right] - \lambda_3\gamma_2 - \lambda_4\left[\tfrac{\beta_3}{N_o+I_s}I_s + \tfrac{\beta_4}{N_o+I_p}I_p\right] \\
-\frac{\partial H}{\partial S_r} &= \lambda_3\left[d + \psi\right] + \lambda_6\psi \\
-\frac{\partial H}{\partial I} &= -W_2 + \lambda_4\left(n + \omega + \theta_1 + \gamma_3 + d + \delta + u_2\right) - \lambda_5\left(\theta_1 + u_2\right) - \lambda_6\gamma_3 - \lambda_7 n - \lambda_8\omega \\
-\frac{\partial H}{\partial R} &= \lambda_6 d \\
-\frac{\partial H}{\partial I_s} &= \lambda_1\left[\tfrac{\beta_1}{(N_o+I_s)^2}S_c N_o\right] + \lambda_2\left[\tfrac{\beta_3}{(N_o+I_s)^2}S_p N_o\right] - \lambda_4\left[\tfrac{\beta_1}{(N_o+I_s)^2}S_c N_o\right] - \left[\tfrac{\beta_3}{(N_o+I_s)^2}S_p N_o\right] + \lambda_7 d \\
-\frac{\partial H}{\partial I_p} &= +\lambda_1\left[\tfrac{\beta_1}{(N_o+I_p)^2}S_c N_o\right] + \lambda_2\left[\tfrac{\beta_4}{(N_o+I_p)^2}S_p N_o\right] - \lambda_4\left[\tfrac{\beta_2}{(N_o+I_p)^2}S_c N_o\right] - \left[\tfrac{\beta_4}{(N_o+I_p)^2}S_p N_o\right] + \lambda_8 d
\end{aligned} \tag{39}$$

Therefore, the adjoint equation in (34) is obtained as

$$\begin{aligned}
\frac{d\lambda_1}{dt} &= -W_1 + \left(\lambda_1 - \lambda_4\right)\left[\tfrac{\beta_1}{N_o+I_s}I_s + \tfrac{\beta_2}{N_o+I_p}I_p + u_1\right] + \lambda_1 d + \left(\lambda_1 - \lambda_2\right)\gamma_1 \\
\frac{d\lambda_2}{dt} &= \left(\lambda_2 - \lambda_4\right)\left[\tfrac{\beta_3}{N_o+I_s}I_s + \tfrac{\beta_4}{N_o+I_p}I_p\right] + \left(\lambda_2 - \lambda_3\right)\gamma_2 + \lambda_2 d \\
\frac{d\lambda_3}{dt} &= \left(\lambda_3 - \lambda_6\right)\psi + \lambda_3 d \\
\frac{d\lambda_4}{dt} &= -W_2 + \left(\lambda_4 - \lambda_7\right)n + \left(\lambda_4 - \lambda_8\right)\omega + \left(\lambda_4 - \lambda_6\right)\gamma + \left(\lambda_4 - \lambda_5\right)\left(\theta_1 + u_2 + \lambda_4 d + \lambda_4\delta\right) \\
\frac{d\lambda_5}{dt} &= \left(\lambda_5 - \lambda_6\right)\theta_2 + \lambda_5 d \\
\frac{d\lambda_6}{dt} &= \lambda_6 d \\
\frac{d\lambda_7}{dt} &= \left(\lambda_1 - \lambda_4\right)\tfrac{\beta_1}{(N_o+I_s)^2}S_c N_o + \left(\lambda_2 - \lambda_4\right)\tfrac{\beta_3}{(N_o+I_s)^2}S_c N_o + \lambda_7 d \\
\frac{d\lambda_8}{dt} &= \left(\lambda_1 - \lambda_4\right)\tfrac{\beta_2}{(N_o+I_p)^2}S_c N_o + \left(\lambda_2 - \lambda_4\right)\tfrac{\beta_4}{(N_o+I_p)^2}S_p N_o + \lambda_8 d
\end{aligned} \tag{40}$$

the transversality conditions

$$\lambda_1(tf) = \lambda_2(tf) = \lambda_3(tf) = \lambda_4(tf) = \lambda_5(tf) = \lambda_6(tf) = \lambda_7(tf) = \lambda_8(tf) = 0$$

We maximize the Hamitonian with respect to the control u_1, u_2, thus we differentiate with respect to u_1, u_2 to obtain the following

$$\begin{aligned}\frac{\partial H}{\partial u_1} &= A_1 u_1 + (\lambda_4 - \lambda_1) S_c \\ \frac{\partial H}{\partial u_2} &= A_2 u_2 + (\lambda_5 - \lambda_4) I\end{aligned} \tag{41}$$

Solving equation (41) by letting $\frac{\partial H}{\partial u_1} = \frac{\partial H}{\partial u_2} = 0$, we obtain

$$\begin{aligned}u_1 &= \frac{\lambda_1 - \lambda_4}{A_1} S_c \\ u_2 &= \frac{\lambda_4 - \lambda_5}{A_2} I\end{aligned} \tag{42}$$

Imposing the bounds $0 \leq u_1 \leq u_{1max}$ and $0 \leq u_2 \leq u_{2max}$ on the controls.Then the optimal control are characterized as:

$$u_1^*(t) = \min\left\{\max(0, \frac{(\lambda_1 - \lambda_4)}{A_1} S_c), u_{1,max}\right\}$$
$$u_2^*(t) = \min\left\{\max(0, \frac{(\lambda_4 - \lambda_5)}{A_2} I), u_{1,max}\right\}$$

where $u_{1max}(t) = u_{2max}(t) = 1$

$$\begin{aligned}u_1(t) &= \min\left\{\max(0, \tfrac{(\lambda_1 - \lambda_4)}{A_1} S_c), 1\right\} \\ u_2(t) &= \min\left\{\max(0, \tfrac{(\lambda_4 - \lambda_5)}{A_2} I), 1\right\}\end{aligned} \tag{43}$$

It is important to note that the characterization of the above controls can be written in a simpler piecewise form given below

$$u_1 = \begin{cases} 0, & \text{if } \frac{(\lambda_1 - \lambda_4)}{A_1} S_c \leq 0 \\ \frac{(\lambda_1 - \lambda_4)}{A_1} S_c, & \text{if } 0 < \frac{(\lambda_1 - \lambda_4)}{A_1} S_c < 1, \\ 1, & \text{if } \frac{(\lambda_1 - \lambda_4)}{A_1} S_c \geq 1 \end{cases} \tag{44}$$

$$u_2 = \begin{cases} 0, & \text{if } \frac{(\lambda_4 - \lambda_5)}{A_2} I \leq 0 \\ \frac{(\lambda_4 - \lambda_5)}{A_2} I, & \text{if } 0 < \frac{(\lambda_4 - \lambda_5)}{A_2} I < 1, \\ 1, & \text{if } \frac{(\lambda_4 - \lambda_5)}{A_2} I \geq 1 \end{cases} \tag{45}$$

9. NUMERICAL SIMULATION

In this section, numerical simulation was carried out to approximate the solution of the ordinary differential equation formulated for the model. The Runge-Kutta scheme of order four together with forward- backward sweep method, which is a accurate method was used to solve the formulated optimal control model. The forward Runge-Kutta technique is used to solve the state variables, while the backward fourth order technique is used to solve the adjoint variables with the consideration of the initial and final condition.

The numerical simulation was carried out with the aid of help of MATLAB software, in order the see the impact of different combinations of the two controls intervention strategies on the Cocoa pod infection. The initial values for the model state variables and weight constraints are:

$$S_c(0) = 1500,\ S_p(0) = 0,\ S_r(0) = 0,\ I(0) = 0,\ T(0) = 0,\ R(0) = 0,\ I_s(0) = 800,\ I_p(0) = 0,$$

$$A_2 = 20,\ W_1 = 20,\ W_2 = 20,\ A_1 = 20.$$

The following four control strategies for the numerical simulations are considered.

(i) Strategy 1: Using $u_1 = 0$, $u_2 = 0$

(ii) Strategy 2: Using $u_1 = 0$, $u_2 \neq 0$

(iii) Strategy 3: Using $u_1 \neq 0$, $u_2 = 0$

(iv) Strategy 4: Using $u_1 \neq 0$, $u_2 \neq 0$

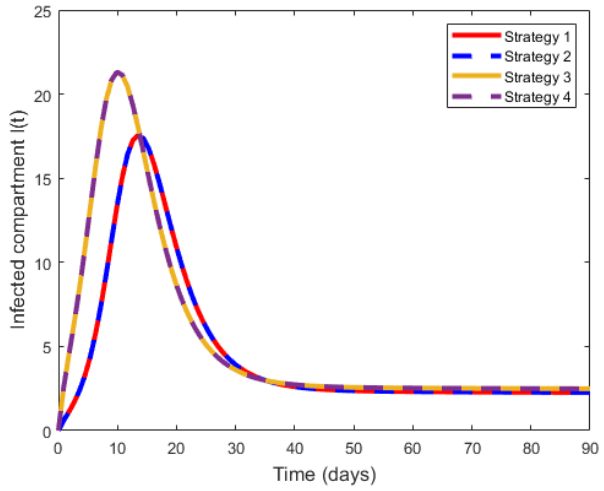

Figure 3: control strategies for model

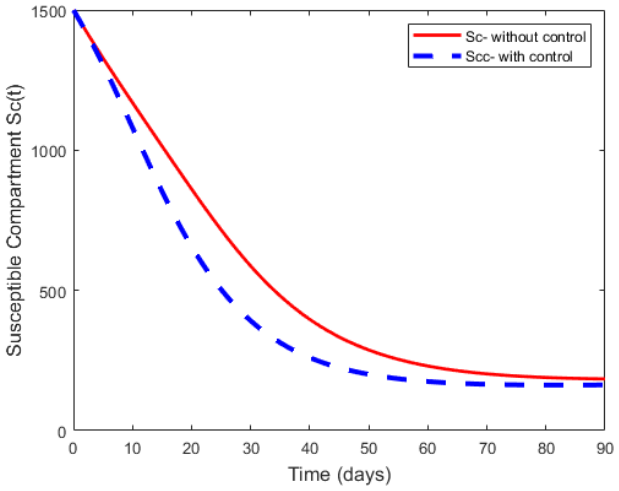

Figure 4: plot of Cherelles compartment against time for Model

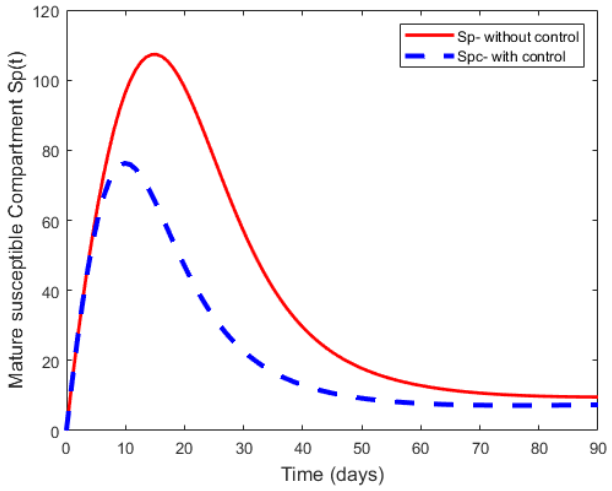

Figure 5: plot of young and mature pod against time Compartment for Model

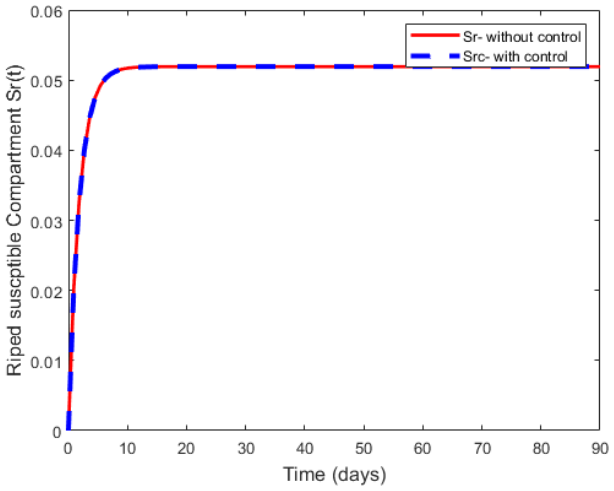

Figure 6: showing the graph of ripe pod Compartment for Model

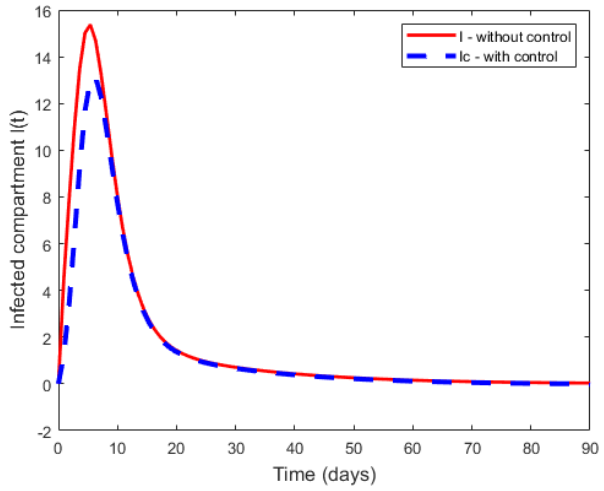

Figure 7: showing the graph of Infected pod Compartment for Model

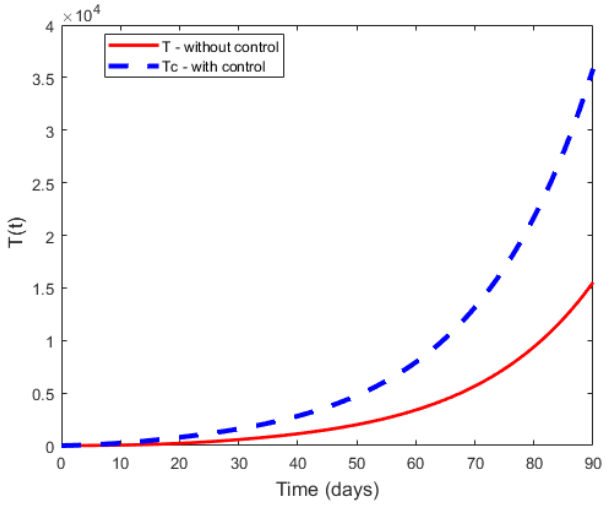

Figure 8: showing the graph of treated pod Compartment for Model

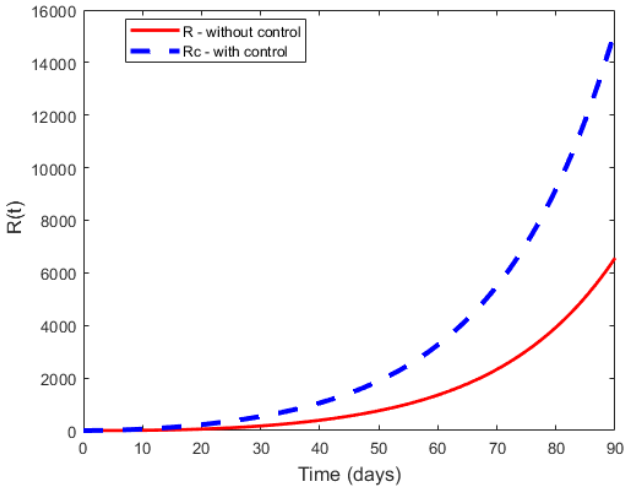

Figure 9: showing the graph of secondary pod Compartment for Model

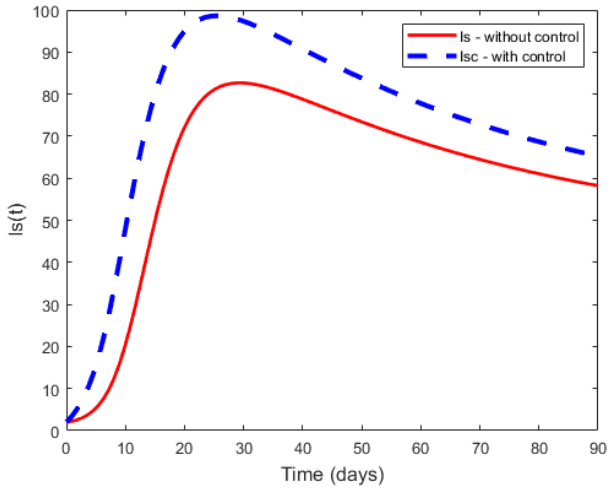

Figure 10: showing the secondary infection compartment for model

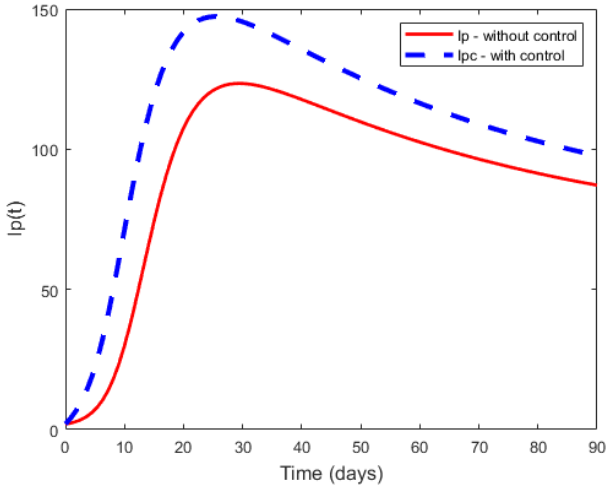

Figure 11: showing primary infection Compartment for Model

10. DISCUSSION OF RESULTS

In this section, we will delve into the key results presented in Figure 2 to Figure 10 and provide a comprehensive discussion of their significance. The research intends to determine the impact of optimal control on the transmission of cocoa black pod disease, i e. to control the rate of transmission from the infected pods to the susceptible pods. The control is applied in a period of 90 days which implies that the final time ($T = 90$). The simulations were carried out using the values taken from [3], [20] and [21], as presented in Table 1 and some estimation made from ecological observations.

Figure 2 represents the effect of the control strategies on the infected pods, strategy 3 and strategy 4 has a reduced number of infected pods when compared with strategy 1 and strategy 2 as reveal by the plot, thus this signifies that strategy 3 and strategy 4 are more effective in the control of the disease. Thus, either of the 2 strategies can be applied in the control of the disease.

Figure 3 presents the plot of Cherelles compartment (S_c) with and without control measures, the susceptible Cherelles compartment reduces with respect to time, but with the application of control measure, it reduces at faster.

Figure 4 presents the plot of young and mature pod compartment (S_p) with and without control measures, the graph of young and mature pod compartment without control increases till it reach its peak at day 20, before it gradually slopes downward till day 63 , when it remains constants for the rest days considered. Likewise, the graph of young and mature pod compartment with control measure applied increases till it reach its peak at day 12, before it gradually slopes downward till day 63 , when it remains constants for the rest days considered.

Figure 5 presents the plot of ripe pod compartment (S_r) with and without control measures, the graph of ripe pod compartment with and without control increases gradually till it reach its peak at day 10 and it remains constants for the rest days considered.

Figure 6 presents the plot of infected pod compartment (I) with and without control measures, graph of infected pod compartment without control increases gradually till it reach its peak at day 10, likewise also for the infected pods with control but with a reduced number of infected pods, after day 10, there is no significant difference between the infected pods with and without control.

Figure 7 presents the plot of treated pod compartment (T) with and without control measures, graph of treated pod compartment increases gradually with respect to time.

Figure 8 presents the plot of removed pod compartment (R) with and without control measures, graph of removed pod compartment increases gradually with respect to time. but it take the removed pods compartment with control lesser days to gradually slopes upward.

Figure 9 presents the plot of secondary infection compartment (I_s) with and without control measures, graph of secondary infection pod compartment increases gradually with respect to time until it reach a period whereby it will slope downward.

Figure 10 presents the plot of primary infection compartment (I_p) with and without control measures, graph of primary infection pod compartment increases gradually with respect to time until it reach a period whereby it will slope downward.

11. CONCLUSION

In conclusion, our mathematical modeling study of black pod disease has provided valuable insights into the dynamics and spread of the disease, as well as the effectiveness of various control strategies. Our analysis suggests that the model is epidemiologically and mathematically well-posed. The model's associated equilibrium's stability (both Local and Global) was analyzed qualitatively and well established. The reproduction number was subjected to a sensitivity analysis to ascertain the relative significance of the various variables responsible for disease transmission and control, and this reveals that rate of fungicide application and infected pod remover are two factors that can be altered to reduce the spread of the disease and for potential long time control, regular harvest ripe pods and maintaining a healthy environment plays a significant factor in the eradicating the disease.

The incorporation of optimal control strategies into our analysis has yielded actionable recommendations for cocoa farmers and policymakers alike. By identifying parameters that minimize disease prevalence while considering economic constraints and ecological sustainability, we have strived to bridge the gap between theoretical modeling and practical application. This research underscores the importance of adaptive and sustainable approaches, recognizing that the battle against cocoa black pod disease requires a dynamic and evolving strategy.

However, it is important to note that our model is based on certain assumptions and limitations, and there is still much that is not fully understood about the disease. Future research could incorporate effect of environmental factors into the model development. Overall, the amalgamation of mathematical modeling and optimal control strategies offers a promising pathway towards a more

robust and sustainable cocoa industry. The ongoing collaboration between researchers, farmers, and policymakers is essential for translating these findings into on-the-ground practices that effectively combat cocoa black pod disease, thereby preserving the integrity and longevity of cocoa cultivation worldwide.

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