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Assessing the Effect of Fungicide Treatment on Cocoa Black Pod Disease in Ghana: Insight from Mathematical Modeling

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Abstract Black pod disease is caused by fungi of the species *Phytophthora palmivora* or *Phytophthora megakarya*. The disease causes darkening of affected areas of cocoa trees and/or fruits and leads to significant reduction in crop yields and decreases lifespan of the plant. This study presents a simple S_1S_2IT-type model with variable population size to assess the impact of fungicide treatment on the dynamics of the black pod disease. We do both theoretical studies and numerical simulations of the model. In particular, we analyze the existence of equilibrium points and their stability, simulate the model using data on reported black pod cases from Ghana. In addition, we perform sensitivity analysis of the basic reproduction number with respect to the model parameters. The results show that the top three parameters that govern the dynamics of the black pod disease are the treatment rate, transmission rate, and planting rate of new trees.

Keywords Black pod disease, fungicide treatment, sensitivity analysis, basic reproduction number.

AMS 2010 subject classifications 34C60, 49M05, 92D30

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1. Introduction

Cocoa is the most economically important species in the Phytophthora genus [1, 2]. It can be grown only within the tropical belt [1, 3] with the largest growing region being the west coast of Africa. Ivory Coast and Ghana contributes about 75% [1] of universal cocoa exports. However, there have been a sturdy decline in production due to increased incidences of the black pod disease. Black pod disease is caused by fungi known as Phytophthora palmivora or Phytophthora megakarya [11, 12, 13], these are dangerous parasites which mainly affect cocoa [14, 15, 16]. Phytophthora palmivora was the most common causal agent for Phytophthora pod rot (black pod) disease in Ghana until in 1973 when the emergence of Phytophthora megakarya brought a new dimension to cocoa disease in the country [7, 8, 9, 10]. The impact of the effect of the disease incidence on the cocoa varies from one farm to the other depending on the changes of weather within a season [17, 18, 19, 20]. Infection of the pods leads to significant reduction in crop yields and decreases lifespan of the plant[4, 5, 6]. Cocoa beans (inside cocoa pods) are used in production of chocolates and cocoa beverages. Mathematical models of disease dynamics can provide a framework to understand disease transmission [23], and to derive effective intervention and prevention measures. There are few research studies on modeling of black pod diseases mathematically [21, 22]. None of these published papers is an SIR-type[27, 28, 29, 30]. Moreover, to the best of our knowledge, there are no prospectively designed studies to

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assess the impact of fungicide on black pod disease, and using the combination of Bayesian approach [24, 25, 26] for parameter estimations. In this paper, we develop and analyze a system of differential equations model describing the black pod transmission with variable population size and fungicide spraying of cocoa trees. Our model is an SIR-type[27, 28, 29, 30] where trees are classified as susceptible, infected, and recovery. A schematic diagram is shown in Figure 1, see Table 1 for the description of the model parameters. The main focus of this study is to assess the potential impact of fungicide treatment on the black pod disease that has been a burden for cocoa farmers in the west coast of Africa, over the years. The paper is organized as follows: The model is formulated in Section 2, the existence of equilibria and their stability is presented in Section 3, numerical simulations and sensitivity analyzes of the reproduction number of the model are performed in Section 4, and Section 5 discusses the results from the analysis of the model.

2. Model formulation

In this section, we present S_1S_2IT model where hosts are classified as: Susceptible, infected, and treated. Hosts are cocoa trees. We sub-divide the susceptible trees into two compartments S_1 and S_2. We assume that a cocoa tree is infected if at least one fruit on it has been affected by the black pod disease or if the fungus is found on the tree itself.

Let $S_1(t)$ be the number of cocoa trees without fungicide treatment and are susceptible to the disease, $I(t)$ be the number of infectious cocoa trees, $S_2(t)$ be the number of susceptible trees that have been sprayed with fungicide or trees where the disease has only affected baby fruit(s) on the tree (such fruit(s) cannot be matured); we assume that such trees can be treated and the infected baby fruit(s) be removed, and $T(t)$ be the number of trees infected with the disease but have been treated with fungicide and are temporarily immunized at time t. We further assume that:

- Susceptible trees in S_1 become infected when there is a contact between a susceptible tree in S_1 and an infectious tree at rate β. The contact can be translated by movement of rodents and insects that feed on the cocoa fruits.
- Trees (farms) are treated continuously with fungicide at rate k. That is, farmers treat their farms as soon as new infections are detected.
- Susceptible trees in S_2 are temporarily immunized and do not get the infection unless they move to the S_1 compartment.
- The fungicide is applied to almost all trees and is meant for both treatment and prevention measures.
- Due to the treatment, a proportion p of infected trees, pkI, returns to the susceptible but treated compartment S_2; the remaining $(1-p)kI$ enters the $T-$compartment.
- Since the effectiveness of the fungicide wears out, trees in the S_2 and $T-$compartments loose immunity at rate α. See Figure 1 for an illustration of the model.

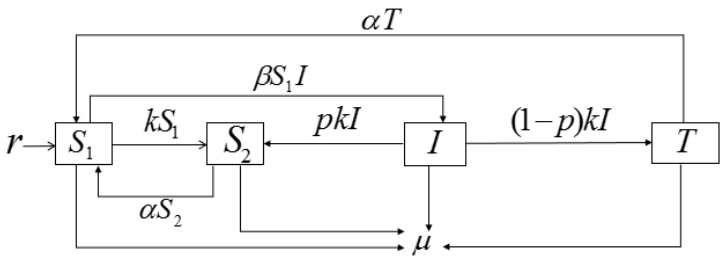

Figure 1. Flow diagram of an S_1S_2IT model with fungicide.

A model under the above transmission assumptions is given by

$$\begin{aligned}
\frac{dS_1}{dt} &= r - \beta S_1 I + \alpha(S_2 + T) - (\mu + k)S_1 \\
\frac{dS_2}{dt} &= kS_1 + pkI - (\alpha + \mu)S_2 \\
\frac{dI}{dt} &= \beta S_1 I - (k + \mu)I \\
\frac{dT}{dt} &= (1 - p)kI - (\alpha + \mu)T,
\end{aligned} \tag{1}$$

with $N = S_1 + S_2 + I + T$, where μ is the natural death rate of cocoa trees. This means that

$$\frac{dN}{dt} = r - \mu N$$

.

Let $S_2 = N - S_1 - I - T$, then model (1) can be reduced to

$$\begin{aligned}
\frac{dS_1}{dt} &= r - \beta S_1 I + \alpha(N - I) - (\alpha + \mu + k)S_1 \\
\frac{dI}{dt} &= \beta I S_1 - (k + \mu)I \\
\frac{dT}{dt} &= (1 - p)kI - (\alpha + \mu)T \\
\frac{dN}{dt} &= r - \mu N.
\end{aligned} \tag{2}$$

The biologically feasible region of model (2) is defined as

$$\Gamma_1 = \left\{ S_1, I, T \in \mathbb{R}^3_+ : S_1, I, T \geq 0,\ S_1 + I + T \leq N \quad \text{and} \quad N \leq \frac{r}{\mu} \right\}.$$

3. Analysis of the model

3.1. Analysis of the basic reproduction number

The basic reproduction number, $\mathcal{R}_0$, of the model is given by

$$\mathcal{R}_0 = \frac{\beta r(\alpha + \mu)}{\mu(k + \mu)(\alpha + \mu + k)}.$$

Since the fungicide spraying rate, k, is our control parameter, let us consider the effect of this parameter on the basic reproduction number.

The partial derivative w.r.t k is

$$\frac{\partial \mathcal{R}_0}{\partial k} = -\frac{\beta r(\alpha + \mu)\left[\mu\alpha + 2\mu(k + \mu)\right]}{\left(\mu(k + \mu)(\alpha + \mu + k)\right)^2} < 0. \tag{3}$$

Thus, the $\mathcal{R}_0$ is always decreasing with respect to k. Also

$$\lim_{k \to \infty} \mathcal{R}_0 = 0.$$

This suggests that eradication will be achieved with sufficiently high spraying rate, the basic reproduction number decreases substantially as the spraying rate increases.

3.2. *Existence and stability of equilibria*

3.2.1. *Existence and local stability of equilibria*

We summarize existence and local stability of equilibria as follows.

Theorem 1
Consider model (2).

(a.) When $R_0 \leq 1$, there exists a unique disease-free equilibrium $E_0 = \left(S_{1_o}^*, I_o^*, T_o^*, N_o^*\right) = \left(\frac{r(\alpha+\mu)}{\mu(\alpha+\mu+k)}, 0, 0, \frac{r}{\mu}\right)$ in Γ_1. It is locally asymptotically stable on Γ_1.

(b.) When $R_0 > 1$, there exists a disease-free equilibrium $E_0 = \left(S_{1_o}^*, I_o^*, T_o^*, N_o^*\right) = \left(\frac{r(\alpha+\mu)}{\mu(\alpha+\mu+k)}, 0, 0, \frac{r}{\mu}\right)$ and an endemic equilibrium $E_1 = (S_1^*, I^*, T^*, N^*)$, where

$$
\begin{aligned}
S_1^* &= \frac{k+\mu}{\beta} \\
I^* &= \frac{\mu+k}{\beta}\left(\frac{\beta r(\alpha+\mu)}{\mu(\alpha+\mu+k)(k+\mu)} - 1\right) = \frac{\mu+k}{\beta}\left(\mathcal{R}_0 - 1\right) \\
T^* &= \frac{(1-p)k(\mu+k)}{\beta(\alpha+\mu)}\left(\mathcal{R}_0 - 1\right) \\
N^* &= \frac{r}{\mu}
\end{aligned}
$$

(i.) The endemic equilibrium E_1 is locally asymptotically stable and will be approached by all trajectories that start in Γ_1 except when $I(0) + T(0) \neq 0$.

(ii.) The disease-free equilibrium E_0 is unstable.

Proof
For the existence of equilibria, see Appendix 5. Consider the Jacobian matrix at the disease-free equilibrium, $\mathcal{J}(E_0) = \mathcal{J}\left(S_{1_o}^*, I_o^*, T_o^*, N_o^*\right)$, given by:

$$
\mathcal{J} = \begin{pmatrix}
-(\alpha+k+\mu) & -(\alpha+\beta S_1^*) & 0 & \alpha \\
0 & (k+\mu)\left(\mathcal{R}_0 - 1\right) & 0 & 0 \\
0 & (1-p)k & -(\alpha+\mu) & 0 \\
0 & 0 & 0 & -\mu
\end{pmatrix}.
$$

The stability of the disease-free equilibrium is determined using the eigenvalues of the characteristic equation of the Jacobian matrix; the equation corresponding to $\mathcal{J}(E_0)$ is given by

$$
f(\lambda) = -\left(\lambda+\alpha+k+\mu\right)\left(\lambda - (k+\mu)(\mathcal{R}_0 - 1)\right)\left(\lambda+\alpha+\mu\right)\left(\lambda+\mu\right). \tag{4}
$$

When $\mathcal{R}_0 < 1$, all the roots of the characteristic equation (4) are negative and the disease-free equilibrium is locally asymptotically stable. However when $\mathcal{R}_0 > 1$, the disease-free equilibrium becomes unstable.

For $\mathcal{R}_0 > 1$, the endemic equilibrium exists, in addition to the disease-free equilibrium. Similarly, the local stability of the endemic equilibrium is determined using the eigenvalues of the characteristic equation corresponding to the Jacobian matrix at E_1, see Appendix 5 for details. The characteristic equation is given by

$$
f(\lambda) = -\left(\alpha+\mu+\lambda\right)\left(\mu+\lambda\right)\left(\alpha+k+\mu+\lambda\right)\left((k+\mu)(\mathcal{R}_0 - 1) + \lambda\right). \tag{5}
$$

When $\mathcal{R}_0 > 1$, all the roots of (5) are negative. We conclude that the endemic equilibrium of the system is locally asymptotically stable. □

3.2.2. Global stability

Lemma 1
The inequality $S_1 \leq S_{1_o}^*$ holds.

Proof
By the first line of system (2)

$$\frac{dS_1}{dt} \leq r + \alpha N - (\alpha + \mu + k)S_1 \leq \frac{r(\alpha+\mu)}{\mu} - (\alpha + \mu + k)S_1. \tag{6}$$

It follows from comparison theorem that

$$S_1 \leq \frac{r(\alpha+\mu)}{\mu(\alpha+\mu+k)} = S_{1_o}^*.$$

□

Theorem 2
For $\mathcal{R}_0 \leq 1$, then the disease-free equilibrium E_0 is globally asymptotically stable on Γ.

Proof
Recall that when $\mathcal{R}_0 \leq 1$, the disease-free equilibrium is $DFE = \left(S_{1_o}^*, I_o^*, T_o^*, N_o^*\right) = \left(\frac{r(\alpha+\mu)}{\mu(\alpha+\mu+k)}, 0, 0, \frac{r}{\mu}\right)$. We construct the Lyapunov function as $V = (S_1, I, T) : \mathbb{R}_+^3$ defined as $V = \omega I$ for some constant ω. We show that

$$\begin{aligned}
\frac{dV}{dt} =& \omega \frac{dI}{dt} \\
=& \omega \left(\beta I S_1 - (k+\mu)I\right) \\
=& \omega \left(\beta S_1 - (k+\mu)\right) I \\
\leq& \omega \left(\beta S_{1_o}^* - (k+\mu)\right) I, \quad \text{since} \quad S_1 \leq S_{1_o}^* \\
=& \omega \left(\frac{\beta r(\alpha+\mu)}{\mu(\alpha+\mu+k)} - (k+\mu)\right) I \\
=& \omega(k+\mu)\left(\frac{\beta r(\alpha+\mu)}{\mu(\alpha+\mu+k)(k+\mu)} - 1\right) I \\
=& \omega(k+\mu)\left(\mathcal{R}_0 - 1\right) I \\
\leq& 0
\end{aligned} \tag{7}$$

This implies that $V' = 0$, when $I = 0$. And $V' < 0$ when $I > 0$ provided that $\mathcal{R}_0 < 1$. Therefore, by LaSalles invariance principle [39], we can conclude that the disease-free equilibrium E_0 is globally asymptotically stable. □

Theorem 3
The endemic equilibrium E_1 is globally asymptotically stable in Γ_1.

Proof
Let us consider the following positive definite function about E_1:

$$L = (S_1 - S_1^*)^2 + \frac{2(\alpha + \beta S_1^*)}{\beta}\left(I - I^* - I^* \ln \frac{I}{I^*}\right) + \frac{\alpha}{2\mu}(N - N^*)^2. \tag{8}$$

The derivative of L along the solution curve of the equation (8) yields

$$\frac{dL}{dt} = 2(S_1 - S_1^*)\frac{dS_1}{dt} + \frac{2(\alpha + \beta S_1^*)}{\beta}\left(\frac{I - I^*}{I}\right)\frac{dI}{dt} + \frac{\alpha}{\mu}(N - N^*)\frac{dN}{dt} \tag{9}$$

Substituting the model (2) into the equation (9) gives

$$\begin{aligned}\frac{dL}{dt} =& 2(S_1 - S_1^*)\left(r - \beta S_1 I + \alpha(N - I) - (\alpha + \mu + k)S_1\right) \\ &+ \frac{2(\alpha + \beta S_1^*)}{\beta}\left(\frac{I - I^*}{I}\right)(\beta I S_1 - (k + \mu)I) + \frac{\alpha}{\mu}(N - N^*)(r - \mu N) \\ =& 2(S_1 - S_1^*)\left(r - \beta S_1 I + \alpha(N - I) - (\alpha + \mu + k)S_1\right) \\ &+ \frac{2(\alpha + \beta S_1^*)}{\beta}(I - I^*)(\beta S_1 - (k + \mu)) + \frac{\alpha}{\mu}(N - N^*)(r - \mu N)\end{aligned} \tag{10}$$

Recall that at the endemic equilibrium, we have

$$\begin{aligned} r =& \beta S_1^* I^* - \alpha N^* + \alpha I^* + (\alpha + \mu + k)S_1^* = \mu N^*, \\ \beta I^* S_1^* =& (k + \mu)I^* \quad \Rightarrow \beta S_1^* = (k + \mu).\end{aligned} \tag{11}$$

Using the equilibrium condition (11) above, equation (10) becomes

$$\begin{aligned}\frac{dL}{dt} =& 2(S_1 - S_1^*)\left(\beta S_1^* I^* - \alpha N^* + \alpha I^* + (\alpha + \mu + k)S_1^* - \beta S_1 I + \alpha N - \alpha I - (\alpha + \mu + k)S_1\right) \\ &+ \frac{2(\alpha + \beta S_1^*)}{\beta}(I - I^*)(\beta S_1 - \beta S_1^*) + \frac{\alpha}{\mu}(N - N^*)(\mu N^* - \mu N) \\ =& 2\alpha(S_1 - S_1^*)(N - N^*) - 2(\alpha + \mu + k)(S_1 - S_1^*)^2 - 2\alpha(S_1 - S_1^*)(I - I^*) \\ &+ 2\beta(S_1 - S_1^*)(S_1^* I^* - S_1 I) + 2(\alpha + \beta S_1^*)(I - I^*)(S_1 - S_1^*) - \alpha(N - N^*)^2.\end{aligned}$$

Add and subtract the term $S_1^* I$.

$$\begin{aligned}\frac{dL}{dt} =& -\alpha(N - N^*)^2 + 2\alpha(S_1 - S_1^*)(N - N^*) - \alpha(S_1 - S_1^*)^2 \\ &- 2(\frac{\alpha}{2} + \mu + k)(S_1 - S_1^*)^2 - 2\alpha(S_1 - S_1^*)(I - I^*) \\ &+ 2\beta(S_1 - S_1^*)(S_1^* I^* - S_1^* I + S_1^* I - S_1 I) + 2(\alpha + \beta S_1^*)(I - I^*)(S_1 - S_1^*) \\ =& -\alpha\left[(N - N^*) - (S_1 - S_1^*)\right]^2 - 2(\frac{\alpha}{2} + \mu + k)(S_1 - S_1^*)^2 - 2\alpha(S_1 - S_1^*)(I - I^*) \\ &- 2\beta S_1^*(S_1 - S_1^*)(I - I^*) - 2\beta I(S_1 - S_1^*)^2 + 2(\alpha + \beta S_1^*)(I - I^*)(S_1 - S_1^*) \\ =& -\alpha\left[(N - N^*) - (S_1 - S_1^*)\right]^2 - 2(\frac{\alpha}{2} + \mu + k)(S_1 - S_1^*)^2 - 2\beta I(S_1 - S_1^*)^2 \\ &- 2(\alpha + \beta S_1^*)(S_1 - S_1^*)(I - I^*) + 2(\alpha + \beta S_1^*)(I - I^*)(S_1 - S_1^*) \\ =& -\alpha\left[(N - N^*) - (S_1 - S_1^*)\right]^2 - 2(\frac{\alpha}{2} + \mu + k + \beta I)(S_1 - S_1^*)^2 \\ \leq& 0.\end{aligned}$$

Thus, $\dot{L} = 0$ holds only at E_1. Hence, L is a Lyapunov function for model (2) and by the Lyapunov asymptotic stability theorem [37] and LaSalle invariance principle [39], we can conclude that the endemic equilibrium is globally asymptotically stable. □

4. Model validation

In order to validate the model, we use data on reported black pod cases from April 2008 - March 2010 obtained from the Cocoa Research Institute of Ghana [32], to test the model. There are five main parameters present in the proposed model and the values of these parameters will directly affect the dynamics of the black pod disease.

4.1. Simulation and parameter estimations

Table 1 shows the five estimated parameters involved in the proposed model. The transmission coefficient of susceptible and infected cocoa trees, β, is 0.3927, the rate at which cocoa trees get treated with fungicide, k, was estimated to be 6.548. See Table 1 for all the estimated parameter values. The fraction of infected cocoa trees that get successfully treated and moved to S_2-class, p, was assumed to be 0.2.

Table 1. Model parameters definitions and their estimated parameters

Parameter	Description	Value	Source
β	Contact rate between S_1 and I	0.7303	Estimated
α	Loss of immunity rate	1.8331	Estimated
k	Fungicide spraying rate	8.1161	Estimated
r	Planting/recruitment rate of new trees	4267	Estimated
μ	Natural death of cocoa trees	0.7919	Estimated
p	Proportion of infected trees that move to S_2	0.2000	Assumed

Figure 2 depicts an overlay of monthly reported black pod cases and simulation with fitted parameters during the 12-months (April 2008-March 2010) calibration period. The black line incorporates the estimated parameters. The highest peak of black pod cases during this period occurred in May 2008, where approximately 6486 cases were reported. Though we did not obtain a strong fit, the basic reproduction number was computed to be 95.0347 and makes the disease-free equilibrium unstable. Eradication could be achieved with a given spraying rate only when the equilibrium is asymptotically stable. Thus, based on this result, it is not possible to permanently eradicate the disease with fungicide spraying. Combination of various control measures (including fungicide spraying) will be needed to eradicate the disease or at least greatly reduce the prevalence level.

Since eradication is not possible with the estimated parameter settings, we investigate the dual-rate effect derived in [31, 35], where the cost function is defined as:

Definition 1 (Dual-rate effect)
Let $I^*(k)$ be the numbers of infected trees at endemic equilibrium and let $C(k) = kI^*(k)$. A *dual-rate effect* occurs if there exist two different spraying rates k_1 and k_2 such that $C(k_1) = C(k_2)$ and $I^*(k_2) < I^*(k_1)$.

This effect occurs in our model, see Figure 3. When such an effect is present, it would then be more cost effective in the long run to spray at the higher rate, k_2, as long as it is feasible to pay a higher cost over an initial period. This will give a lower level of prevalence in the long run than for the lower rate, k_1, at the same long-term cost.

4.2. Sensitivity analysis of the model parameters

In this section, we carry out a sensitivity analysis to assess the relative impact of each parameter on the transmission and prevalence of the disease. We compute the sensitivity indices of the basic reproduction number, $\mathcal{R}_0$, in terms of the model parameters. The index measures the relative change in $\mathcal{R}_0$ with respective to the relative change in the parameters [33, 34, 36]. The analysis could help identify which parameter causes the most reduction in $\mathcal{R}_0$, and such parameter could be targeted at keeping the prevalence level sufficiently low that might be considered tolerable.

Definition: The normalized forward sensitivity index of a variable, L, that depends differentially on a parameter, y, is defined as:

$$\xi_y = \frac{y}{L}\frac{\partial L}{\partial y}.$$

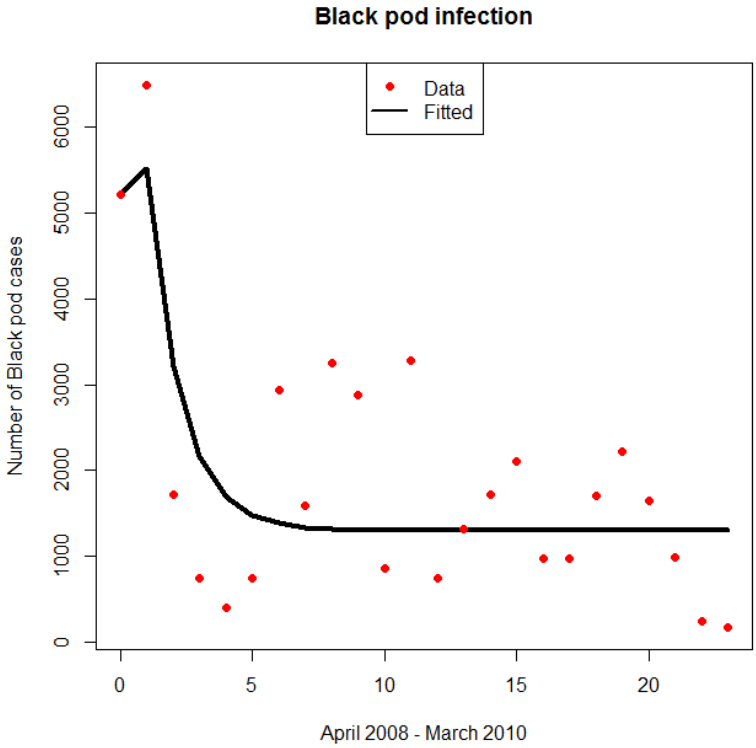

Figure 2. The fitted growth to the incidence values of infected cocoa trees from April 2008 to March 2010.

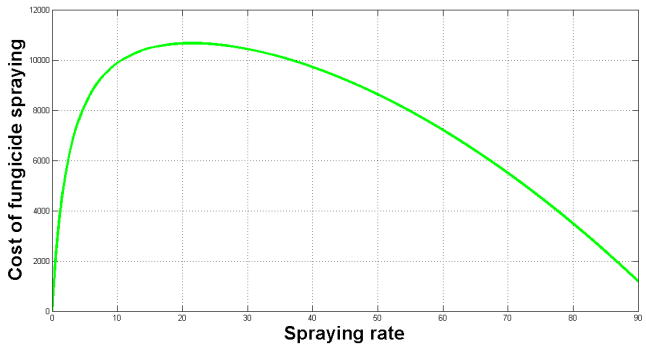

Figure 3. Existence of dual-rate effect with $\beta = 0.7303$, $r = 4267$, $\mu = 0.7919$, $\alpha = 1.8331$. The graph shows that two different spraying rates can carry identical cost.

The sensitivity indices of $\mathcal{R}_0$ in terms of the model parameters are computed below.

$$\begin{aligned}
\xi_k &= \frac{k}{\mathcal{R}_0}\frac{\partial \mathcal{R}_0}{\partial k} = \frac{-k\left(\alpha + 2(k+\mu)\right)}{(k+\mu)(\alpha+\mu+k)} \\
\xi_r &= \frac{r}{\mathcal{R}_0}\frac{\partial \mathcal{R}_0}{\partial r} = 1 \\
\xi_\beta &= \frac{\beta}{\mathcal{R}_0}\frac{\partial \mathcal{R}_0}{\partial \beta} = 1 \\
\xi_\alpha &= \frac{\alpha}{\mathcal{R}_0}\frac{\partial \mathcal{R}_0}{\partial \alpha} = \frac{\alpha k}{(\alpha+\mu)(\alpha+\mu+k)} \\
\xi_\mu &= \frac{\mu}{\mathcal{R}_0}\frac{\partial \mathcal{R}_0}{\partial \mu} = \frac{\mu k(k+\mu) - (\alpha+\mu)(k+2\mu)(\alpha+\mu+k)}{(\alpha+\mu)(k+\mu)(\alpha+\mu+k)}.
\end{aligned} \tag{12}$$

Based on equation (12) and the numerical results displayed in Table 2, the most sensitive parameter is the fungicide spraying rate k, followed by the planting/recruitment rate of new trees r and transmission coefficient β, whereas the rate at which temporarily treated trees become entirely susceptible again as the effect of the fungicide wears out, α, is the least sensitivity parameter.

Table 2. Sensitivity indices of the five parameters involved in the model

Parameter	Parameter Description	Sensitivity index
k	Fungicide spraying rate	-1.6451
β	Contact rate between S_1 and I	1
r	Planting/recruitment rate of new trees	1
μ	Natural death of cocoa trees	-0.8523
α	Loss of immunity rate	0.4974

These results suggest that intervention strategies that focus on increasing the fungicide spraying and decreasing the transmission coefficient could be effective in controlling the black pod disease. Surprising, the results also show that increasing the planting rate of new trees increases the transmission of the disease, since a key strategy to control the transmission of the disease is to reduce the basic reproductive so that it is less than unity.

5. Discussion

In this paper, we have demonstrated how to model black pod disease that affects cocoa. We incorporated a control parameter in a form of fungicide treatment of cocoa trees. It is observed that when the $\mathcal{R}_0 \leq 1$, a unique disease-free equilibrium state (DFE) exists. This equilibrium is both locally and globally asymptotically stable. Whereas when $\mathcal{R}_0 > 1$, the black pod disease persists and the endemic equilibrium is globally asymptotically stable. The challenge in controlling the black pod disease, with the estimated parameter settings, would be very difficult for cocoa farmers. This is because the basic reproduction number is more than unity. The $\mathcal{R}_0$ obtained from this study indicates that the disease will continue to show an epidemic pattern in cocoa farms. This, therefore, calls for combination of various control measures (including fungicide spraying) to eradicate the disease or at least greatly reduce the prevalence to a tolerable level. The study has some limitations. The estimated parameter values used to compute the basic reproduction number were based on available data and these estimates could change subject to the quality of the data from the start of the epidemic. A lot of factors in the data collection could affect parameter estimation results. Nevertheless, our theoretical and simulations results are the first significant step in quantifying the magnitude of the black pod epidemic in Ghana. The study lays a foundation for follow-up research to further investigate intervention strategies that could eliminate or reduce the persistence of the disease substantially.

Appendix

Existence of equilibria

At equilibrium of (2), we have

$$\begin{aligned} r - \beta S_1^* I^* + \alpha(N^* - I^*) - (\alpha + \mu + k)S_1^* &= 0 \\ \beta I^* S_1^* - (k + \mu)I^* &= 0 \\ (1-p)kI^* - (\alpha + \mu)T^* &= 0 \\ r - \mu N &= 0. \end{aligned} \tag{13}$$

By the second line, we get $I^* = 0$ or $S_1^* = \frac{k+\mu}{\beta}$. For $I^* = 0$, it follows from the third line that $T^* = 0$. Substituting $I^*, T^* = 0$ in the first yields

$$r + \alpha N^* - (\alpha + \mu + k)S_1^* = 0 \quad \Rightarrow \quad S_1^* = \frac{r + \alpha N^*}{\alpha + \mu + k} = \frac{r(\alpha + \mu)}{\mu(\alpha + \mu + k)}. \tag{14}$$

It implies that model (2) has a disease-free equilibrium given by

$$E_0 = \left(S_{1_o}^*, I_o^*, T_o^*, N_o^*\right) = \left(\frac{r(\alpha + \mu)}{\mu(\alpha + \mu + k)}, 0, 0, \frac{r}{\mu}\right).$$

Now, substitute $S_1^* = \frac{k+\mu}{\beta}$ into the first of (13) to have

$$r - \beta\frac{k + \mu}{\beta}I^* + \alpha(N^* - I^*) - (\alpha + \mu + k)\frac{k + \mu}{\beta} = 0. \tag{15}$$

Solving for I^* result

$$I^* = \frac{\mu + k}{\beta}\left(\frac{\beta r(\alpha + \mu)}{\mu(\alpha + \mu + k)(k + \mu)} - 1\right) = \frac{\mu + k}{\beta}\left(\mathcal{R}_0 - 1\right). \tag{16}$$

By the third line of (13),

$$T^* = \frac{(1 - p)kI^*}{\alpha + \mu} = \frac{(1 - p)k}{\alpha + \mu}\frac{\mu + k}{\beta}\left(\mathcal{R}_0 - 1\right) = \frac{(1 - p)k(\mu + k)}{\beta(\alpha + \mu)}\left(\mathcal{R}_0 - 1\right). \tag{17}$$

This shows that model (2) has an endemic equilibrium given by $E_1 = (S_1^*, I^*, T^*, N^*)$, where

$$\begin{aligned} S_1^* &= \frac{k + \mu}{\beta} \\ I^* &= \frac{\mu + k}{\beta}\left(\frac{\beta r(\alpha + \mu)}{\mu(\alpha + \mu + k)(k + \mu)} - 1\right) = \frac{\mu + k}{\beta}\left(\mathcal{R}_0 - 1\right) \\ T^* &= \frac{(1 - p)k(\mu + k)}{\beta(\alpha + \mu)}\left(\mathcal{R}_0 - 1\right) \\ N^* &= \frac{r}{\mu}. \end{aligned} \tag{18}$$

Local stability of the endemic equilibrium:

$$\begin{aligned} \mathcal{J}(E_1) &= \begin{pmatrix} -(\beta I^* + \alpha + k + \mu) & -(\alpha + \beta S_1^*) & 0 & \alpha \\ \beta I^* & \beta S_1^* - (k + \mu) & 0 & 0 \\ 0 & (1 - p)k & -(\alpha + \mu) & 0 \\ 0 & 0 & 0 & -\mu \end{pmatrix} \\ &= \begin{pmatrix} -(k + \mu)\mathcal{R}_0 - \alpha & -(\alpha + k + \mu) & 0 & \alpha \\ (k + \mu)(\mathcal{R}_0 - 1) & 0 & 0 & 0 \\ 0 & (1 - p)k & -(\alpha + \mu) & 0 \\ 0 & 0 & 0 & -\mu \end{pmatrix} \end{aligned}$$

This has a characteristic equation given by

$$\begin{aligned} f(\lambda) &= -\left(\alpha + \mu + \lambda\right)\left(\mu + \lambda\right)\left[\lambda^2 + \left(\alpha + (k + \mu)\mathcal{R}_0\right)\lambda + (\alpha + k + \mu)(k + \mu)(\mathcal{R}_0 - 1)\right] \\ &= -\left(\alpha + \mu + \lambda\right)\left(\mu + \lambda\right)\left(\alpha + k + \mu + \lambda\right)\left((k + \mu)(\mathcal{R}_0 - 1) + \lambda\right). \end{aligned}$$

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