On the use of Mathematical Modelling to study the impact of phytosanitation on cocoa black pod disease caused by *Phytophthora megakarya*

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**Abstract**

Cocoa black pod rot, due to several *Phytophthora* spp., like *Phytophthora megakarya*, is the most important disease worldwide and the main cocoa disease in Africa, responsible for yield losses up to 50-80% in the absence of control measures. In this paper, we show how the use of Mathematical modelling and analysis can be helpful to better understand the mechanisms behind black pod rot epidemics and, also to identify gaps in our knowledge. Although black pod rot of cocoa is of major concern and much attention has been dedicated to it, there are still many questions regarding the factors that govern disease dynamics. For instance the relative importance of the two different sources of inoculum (primary and secondary) and spore dispersal dynamics are not well understood. In an attempt to provide answers to these (or some of these) questions, a temporal compartmental model has been developed and study, that allows to investigate the impact of phytosanitary pod removal on cocoa black pod epidemics. Using the model analysis, we highlight two thresholds, related to the model’s parameters, that drive all possible dynamics of the system, and show the relative importance of some compartments in the disease dynamics. Numerical simulations are also provided to illustrate our results and discuss the impact of sanitary harvest.

**Keywords:** Cocoa; Black pod rot; mathematical epidemiology; basic reproduction number; sanitary harvest; simulations.
Introduction

Control of cacao black pod disease

Cocoa (Theobroma cacao L.) is one of the most important perennial crops worldwide. World production was around 4.25 million tons in 2014/15 with an estimated value of around 12.6 billion US dollars [1] (ICCO, 2017). It is estimated however, that between 20 to 30% of annual yield is lost due to pests and diseases. At a global scale, the most important disease is cacao black pod rot, which is due to several species of *Phytophthora*. The most important one is *P. palmivora* (E. J. Butler) yet the most aggressive and damaging one is *P. megakarya* (Brasier & M.J. Griffin). *Phytophthora megakarya* can cause up to 60 to 100% crop loss if not managed [2]. *Phytophthora megakarya* only occurs in countries in West and Central Africa [3], yet since West and Central Africa produce around 70% of all cocoa beans worldwide, its economic impact is considerable. In Cameroon, cocoa black pod rot is only due to *P. megakarya* and losses can reach up to 80-90% when plantations are poorly managed [4, 5].

Cocoa black pod rot is generally controlled through fungicide applications, combined with the use of partially resistant or tolerant cocoa cultivars and appropriate cultural practices, such as phytosanitation [6]. Biological control seems to be promising but no commercial products are as yet available [7]. An increase in the effectiveness of control can be expected when these methods are rationally combined [8]. Phytosanitary pod removal is a preventive method which consists in cleaning trees at the beginning of the season by removing mummified fruits left from the previous season, and the subsequent regular removal of diseased pods which are the source of secondary inoculum [6]. However, phytosanitation is time-consuming and labour intensive and thus relatively expensive. Fungicide applications are economically speaking, effective [9]. Metalaxyl (either resolved or unresolved) is the main active ingredient of the most effective fungicides for preventing/controlling black pod disease. Yet, fungicide use has numerous negative externalities, such as human health problems, pollution, reductions in the populations of beneficial organisms, and the emergence of secondary diseases or pathogen resistance to pesticides [10]. Thus, there are compelling reasons to come up with alternative, innovative and more effective control strategies. However, in order to do so it is necessary to identify levers through which it is possible to exert control. This can only be done through a better understanding of disease epidemiology.

Mathematical Modelling can be a very helpful tool in such an undertaking. In particular, the analysis of the models can help to understand factors that govern temporal dynamics of epidemics and allow the identification of the main determinants for disease spread and evolution. Such modelling and analysis ultimately aim at developing sustainable strategies for tactical disease management [11]. Relatively few studies have focused on understanding the temporal evolution of *P. megakarya* epidemics, and there are still many unresolved questions regarding the factors that govern these dynamics. For instance, the relative importance of the two different sources of inoculum (primary and secondary) and spore dispersal dynamics are not well understood. Modelling can provide answers to these questions and help build efficient control strategies.

*Phytophthora megakarya*: life cycle and epidemiology

An overview of the life cycle of *Phytophthora megakarya* is presented in Fig. 1. Infection by *P. megakarya* takes place either from an environmental reservoir containing propagules of *P. megakarya* to a healthy cacao pod or from an infected to a healthy pod. Primary inoculum in the soil is activated under wet and humid conditions and develops sporangia. Through rain-splash or by certain insects such as ants, sporangia or the motile biflagellate zoospores they contain, are then transported onto pods where they can establish an infection [12]. Successful infection subsequently results in the generation of secondary inoculum, which gets dispersed, again primarily through rain-splash, which can cause secondary infections. Under favorable conditions, a single pod may produce several million motile zoospores [13]. According to [13] *P. megakarya* inoculum can survive up to 18 months in the soil of cocoa plantations.
Figure 1. The life cycle of Phytophthora megakarya (modified from [14]).

Although P. megakarya can infect cocoa pods at all developmental stages, studies from Cameroon showed that susceptibility and the risk of attack depends among others on the developmental stage of cocoa pods [15, 16]. All current knowledge on disease dynamics was used to build the following temporal model describing the black pod disease evolution in a cocoa plot.

The epidemiological model

In order to develop our mathematical model [17], we first described the biological system through a compartmental diagram that is depicted in Fig. 2: Cocoa pods (hosts) are divided in two epidemiological states: susceptible (S) and infectious (I). Transmissions from pod to pod and from environment to pod are the only ways where black pod rot disease can spread. Healthy (susceptible) pods are classified in three classes according to their developmental stage: Cherelles (S1), young and mature pods (S2), and ripe pods (S3), ripe pods are the pods used for commercialization and consumption. The infectious pod class (I) has two sub-classes according to infection pathway: through spores produced by infected pods and released into the environment, responsible for primary infections ($S_{p,e}$) and spores produced by infected pods and directly responsible for pod to pod infections ($S_{p,i}$). The relative importance of these two inoculum sources is investigated in the model study. According to the maturity stage $k$, fruits in contact with spores, produced either from the environment or from infected pods, are contaminated may be contaminated or not.

Figure 2. A compartmental diagram of the black pod rot epidemic
New Cherelles enter in the compartment S1 with the recruitment rate $\Lambda$. Cherelles can either appear continuously throughout the year, or periodically. Both cases are considered. Subsequently cherelles become either young pods, infected or wilted. Young pods can become either ripe pods, infected or die naturally due to other causes. Ripe pods can be harvested or die naturally by excessive ripening or mummification. Altogether, based on the compartmental diagram in Fig. 2, we have developed a mathematical model [17]. For convenience, it is given in the Appendix. The Model parameters are also summarized in Table 2 (Appendix). In the next section, results obtained through a detailed analysis of the mathematical model are presented. Details on the equations and the theoretical analysis are given in [17].

2- Mathematical Modelling

The use of mathematical modelling and the subsequent model analyses allow to highlight the existence of a Disease Free Equilibrium (DFE) and, at least, one Endemic Equilibrium (EE). The two equilibria define respectively the healthy state and the endemic state of our system. The dynamics of the system depend on threshold parameters and the basic reproduction number, estimated through the parameters of the systems. We show that control of black pod epidemics is strongly linked to the stability of the system’s disease free equilibrium which allows discussion of either an outbreak or the extinction of the disease. Black pod disease dynamic was investigated under two specific scenarios.

- **Constant cherelle recruitment rate $\Lambda$**

Considering the cherelle birth rate as constant (continuous during the year) and investigating the impact of phytosanitary pod removal on disease dynamic for two phytosanitary pod removal intensities: without ($\gamma_I = 0$) and with ($\gamma_I = 0.4$).

- **Periodic cherelle recruitment rate $\Lambda(t)$**

Given the fact that the recruitment of cherelles is strongly linked to rainy periods, the cherelle recruitment rate $\Lambda(t)$ was also estimated as a T-periodic function where $T = 365$ days. Based on data from the literature [18], we define the periodic function $\Lambda(t)$ as shown in Fig. 3. Then, we assess the impact of phytosanitary pod removal intensity and frequency for three different time intervals (days).

**Figure 3.** Cherelle recruitment rate function for a year
3- Results

We first assume that the recruitment of cherelles is constant, i.e. \( \Lambda(t) = \Lambda \). Then theoretical analysis of our mathematical model [17], leads to the following results:

- The existence of a Disease Free equilibrium (DFE), and at least one Endemic Equilibrium under some conditions.
- Two basic reproduction number \( R_{0,\ell} \) and \( R_{0,g} \) were computed based on the model’s parameters (given in the Appendix). In our case, these threshold parameters are defined as the number of secondary infections that an infectious pod might produce when introduced in a population of healthy pods of any maturity stage. These threshold summarize the dynamics of the models. Indeed according to their values (less or greater than 1), the disease dies out (convergence to DFE) or maintain (convergence to an EE).
- The stability and unstability of the disease free equilibrium of the model was investigated.

3-1 Impact of phytosanitary pod removal intensity

In the forthcoming simulations, we consider two cases: without and with sanitary harvest. In [17], we have studied the effects of the sanitary control strategy, focusing on the evolution of the number of healthy and infected pods (S and I) and the evolution of the two type of spores (Spe and Spi).

- Constant cherelle recruitment rate \( \Lambda \)

\( \Lambda \) is equal to the mean value of the periodic \( \Lambda(t) \) (≈14 pods per day). The simulation below (Fig. 4), when one infectious pod is introduced in the system, illustrates clearly the impact of phytosanitary pod removal on disease dynamics. Without phytosanitation \( (t < 500 \text{ days}) \), i.e. \( \gamma_I = 0 \), we clearly see that infected pods and spores are lasting in the system, causing an epidemic \( (R_{0,\ell} = 6.04) \). The application of sanitary harvesting \( (t > 500 \text{ days}) \), i.e. \( \gamma_I = 0.4 \), drives \( R_{0,\ell} \) from a value greater than one to a value lower than 1 \( (R_{0,\ell} = 0.81) \), such that the infection dies out. This result confirms one of our theoretical results: when \( R_{0,\ell} < 1 \), the Disease Free Equilibrium is globally asymptotically stable, that is, whatever the initial conditions the system will converge to the DFE. From the epidemiological point of view, this means that an outbreak can occur but will not maintain.

![Figure 4](image)

**Figure 4.** Time evolution of the system without and with phytosanitary pod removal, applied at \( t=500 \). (S = healthy susceptible pods, I = infected/infectious pods, Spe = environmental spores, Spi = spores produced by infected pods)
• Periodic cherelle recruitment rate $\Lambda(t)$

The simulations with a periodic $\Lambda(t)$, and for different sanitary harvest rates, $\gamma_I = 0$, $\gamma_I = 0.4$ and $\gamma_I = 0.6$ are shown in Fig 5, below. Using the same parameter values and the same initial conditions, we show that phytosanitary pod removal, with $\gamma_I = 0.4$, is relatively efficient in reducing the number of infected pods and the amount of spores. However, $R_{0,per}$ (the periodic reproduction number, that is estimated numerically) only decays from 5.63 to 1.18, and, thus, remains greater than 1. Thus, contrary to the constant case, the disease does not stop. This is more in line with observed dynamics in the field and clearly shows that considering $\Lambda(t)$ as a constant may give poor results in terms of control strategies. Finally, as expected, with a greater sanitary harvest intensity $\gamma_I = 0.6$, $R_{0,per}$ only decays below 1, such that the disease dies out (no infected pods and spores in the system). We summarize the values computed for the basic reproduction numbers, both for constant and periodic recruitment rates, in Table 1.

### Table 1. $R_0$ Estimates for different values of phytosanitation rate $\gamma_I$

<table>
<thead>
<tr>
<th>$\gamma_I$</th>
<th>$R_{0,per}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>6.04</td>
</tr>
<tr>
<td>0.4</td>
<td>0.81</td>
</tr>
<tr>
<td>0.6</td>
<td>0.87</td>
</tr>
</tbody>
</table>

Fig. 5 shows clearly that an increase in the sanitary harvest intensity can reduce the epidemiological risk. According to the second picture in Fig. 5, it seems that environmental spores ($Spe$) are more difficult to control than spores produced on pods, and, thus, seem to be more responsible for diseases outbreaks.

3-2 Impact of phytosanitary pod removal frequency

Here, we consider sanitary harvest, with a frequency of 4, 7, and 14 days respectively. In other words, we investigate what happens when we assume that a proportion of infected pods ($I$), are removed instantaneously every 4 days, weekly and fortnightly. In Fig. 7a, b and c, we show, that when we consider a phytosanitary harvest with a daily removal efficiency of 30% (that is 30% of the infected fruits removed) and with an appropriate frequency (here every 4 days) the disease dies out (Fig. 7a). Yet, if we consider a
harvest frequency of 7 or 14 days (weekly or fortnightly), then disease incidence, although reduced remains present in the plot (Figs. 7b and c). This shows that frequency and intensity of phytosanitary pod removal impacts the efficiency of this control method. We also note that the following figures highlight the fact that environmental spores are in general more lasting in the system than spores produced by infected pods when phytosanitation is applied. Thus phytosanitary pod removal seems to have much more impact on secondary P megakarya inoculum.
Figure 7c

Figure 7a,b and c. Periodic time evolution of Susceptible pods, Infected pods, and spores, when sanitary harvest starts at $t = 500$, with a frequency of 4, 7 and 14 days.

4-Discussion and Conclusion

We have considered an epidemiological model [17] which incorporates a periodic recruitment rate function for cocoa pods and assessed the impact of phytosanitation on black pod disease dynamic in a plot. The results obtained seem to be in agreement with the literature. In [6], the authors show that application of sanitary harvest could lead to reduced black pod incidence and [19] found that weekly removal, of $P. palmivora$ infected pods reduced the incidence of epidemics significantly in comparison with fortnightly removal. In another modelling study, yet focusing on phytosanitation as a means of frosty pod rot (caused by $Moniliophthora roreri$) control [20] also demonstrate the need for frequent stripping of infected pods to prevent sporulating pods accumulating in the field. Yet, $P. megakarya$ being polycyclic (multiple cycles of infection and spore production during a production season) in contrast to $Moniliophthora roreri$ which is in reality more monocyclic given the very long latent period required for sporulation, makes black pod disease more difficult to control unless a very rigorous and efficient phytosanitation program is put into place.

The analysis and simulations seem to indicate that intense and regular phytosanitary pod removal could significantly reduce disease incidence and in some cases (every four days) with an appropriate efficiency, could practically reduce the number of infected pods in the plot to nothing. It is important to mention that the good qualitative properties of the system can be explained partially by the fact that the model considers an isolated cocoa plot. Thus we are neglecting external factors such as external inoculum and neighbouring plots which can provide other sources of infections. It is also important to notice that in reality the application of phytosanitary pod removal is often difficult to realize because it is time consuming and, in general, only a certain percentage of infected/diseased pods will be removed, such that inoculum remains in the system causing secondary infections. That is why it is important to combine this cultural practice with other alternative control strategies such as planting resistant material and the rational use of (bio)-fungicides, as a means to establish an integrated control system against the disease.

In this paper, we investigated the relative importance of the two different inoculum sources ($S_{p,e}$) and ($S_{p,i}$). According to our results it seems that the environmental reservoir plays a more important role in disease dynamics than previously thought. However, from the literature, it is clear that little attention has been given to this specific source of inoculum. The model highlights the fact that disease outbreaks and spores lasting in the environment are strongly linked to the environmental spores ($S_{p,e}$). These results are also in agreement with [21] who hypothesized that primary inoculum ($S_{p,e}$) is the main determinant for the spatial and temporal development of an epidemic at the plantation level and that secondary inoculum ($S_{p,i}$) is mainly responsible for the within-tree temporal development of black pod. This could also explain why sanitary harvesting seems to have more effect on the secondary inoculum. Our study also indicates the need to focus and engage new experimentations for a more reliable and better estimation of model parameters. This can help broaden our knowledge on the role of the environmental spore reservoir in disease dynamic and establishment of efficient control strategies.
The next steps will be to recommend new experiments to improve our knowledge on this pathosystem. In particular we may investigate:
- the infectious potential of the environmental reservoir of spores
- the factors governing the environmental infection dynamic.
- the spatial nature of the disease.
- simulations versus empirical data.

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References
The compartimental model [17]:

\[
\begin{align*}
\frac{dS_1}{dt} & = \Lambda - \theta_1 S_1 - \left[ \frac{g_1 S_{p,e}}{K_1 + S_{p,e}} + \frac{g_1 S_{p,i}}{K_1 + S_{p,i}} \right] S_1 - \mu_1 S_1 - wS_1 \\
\frac{dS_2}{dt} & = \theta_1 S_1 - \left[ \frac{g_2 S_{p,e}}{K_2 + S_{p,e}} + \frac{g_2 S_{p,i}}{K_2 + S_{p,i}} \right] S_2 - \mu_2 S_2 \\
\frac{dI}{dt} & = \left[ \frac{g_1 S_{p,e}}{K_1 + S_{p,e}} + \frac{g_1 S_{p,i}}{K_1 + S_{p,i}} \right] S_1 + \left[ \frac{g_2 S_{p,e}}{K_2 + S_{p,e}} + \frac{g_2 S_{p,i}}{K_2 + S_{p,i}} \right] S_2 - \gamma I - \mu I \\
\frac{dS_{p,e}}{dt} & = r_2 I - d_2 S_{p,e} \\
\frac{dS_{p,i}}{dt} & = r_1 I - d_1 S_{p,i}.
\end{align*}
\]

APPENDIX

The compartimental model [17]

Figure 2. A compartimental diagram of the black pod rot epidemic
Table 1: An overview of model parameters and their units

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Description</th>
<th>Unit</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \Lambda )</td>
<td>Cherelle birth rate</td>
<td>days</td>
</tr>
<tr>
<td>( \theta_1 )</td>
<td>Growth rate from cherelle to young pod</td>
<td>days</td>
</tr>
<tr>
<td>( \theta_2 )</td>
<td>Ripening rate</td>
<td>days</td>
</tr>
<tr>
<td>( \gamma_r )</td>
<td>Harvesting rate of ripe pods</td>
<td>days</td>
</tr>
<tr>
<td>( \gamma_f )</td>
<td>Rate of phytosanitary pod removal</td>
<td>days</td>
</tr>
<tr>
<td>( \lambda_1 )</td>
<td>Cherelle Contamination rate</td>
<td>days</td>
</tr>
<tr>
<td>( \lambda_2 )</td>
<td>Pods Contamination rate</td>
<td>days</td>
</tr>
<tr>
<td>( w )</td>
<td>Attack rate by wilt</td>
<td>days</td>
</tr>
<tr>
<td>( \mu_1 )</td>
<td>Natural death rate of cherelle</td>
<td>days</td>
</tr>
<tr>
<td>( \mu_2 )</td>
<td>Natural death rate of pods</td>
<td>days</td>
</tr>
<tr>
<td>( \mu_3 )</td>
<td>Natural death rate of ripe pods</td>
<td>days</td>
</tr>
<tr>
<td>( \mu_l )</td>
<td>Natural death rate of infected pods</td>
<td>days</td>
</tr>
<tr>
<td>( \sigma )</td>
<td>Production rate of spores by infected pods</td>
<td>spores/day</td>
</tr>
<tr>
<td>( d_1 )</td>
<td>Natural decay of spores in the environment</td>
<td>days</td>
</tr>
<tr>
<td>( d_2 )</td>
<td>Inactivation speed of spores</td>
<td>days</td>
</tr>
<tr>
<td>( r_1 )</td>
<td>Shedding rate of spores in the environment</td>
<td>spores/day</td>
</tr>
<tr>
<td>( r_2 )</td>
<td>Releasing speed of spores</td>
<td>spores/day</td>
</tr>
<tr>
<td>( K_1 )</td>
<td>Mickaelis-menten parameter for environmental transmission</td>
<td></td>
</tr>
<tr>
<td>( K_2 )</td>
<td>Mickaelis-menten parameter for pod to pod transmission</td>
<td></td>
</tr>
</tbody>
</table>

The expressions of the two thresholds parameters \( R_{0,\ell} \) and \( R_{0,\varrho} \) are:

\[
R_{0,\ell} = \frac{1}{2} \left( R_{0,d} + \sqrt{(R_{0,d})^2 + 4R_{0,i}} \right) \quad \text{and} \quad R_{0,\varrho} = R_{0,d} + R_{0,i},
\]

where

\[
R_{0,d} = \frac{r_2 \sigma (\beta_2 S_1^{dfe} + \beta_2 S_2^{dfe})}{K_2 d_2 (\gamma_l + \mu_l)} \quad \text{and} \quad R_{0,i} = \frac{r_1 \sigma (\beta_1 S_1^{dfe} + \beta_1 S_2^{dfe})}{K_1 d_1 (\gamma_l + \mu_l)}.
\]

See [17] for more details about the computations of these thresholds.