



Mathematical modelling of Banana Black Sigatoka Disease with delay and Seasonality

Franklin Platini Agouanet, Israël Tankam Chedjou, Remy Etoua, Jean Jules Tewa

► To cite this version:

Franklin Platini Agouanet, Israël Tankam Chedjou, Remy Etoua, Jean Jules Tewa. Mathematical modelling of Banana Black Sigatoka Disease with delay and Seasonality. Applied Mathematical Modelling, 2021, 99, pp.380-399. 10.1016/j.apm.2021.06.030 . hal-03456840

HAL Id: hal-03456840

<https://inria.hal.science/hal-03456840>

Submitted on 2 Aug 2023

HAL is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.



Distributed under a Creative Commons Attribution - NonCommercial 4.0 International License

Mathematical modelling of Banana Black Sigatoka Disease with delay and Seasonality

Franklin Platini Agouanet^a, Israël Tankam-Chedjou^a, Remy M. Etoua^{d,c}, Jean Jules Tewa^{d,c,b,*}

^aDepartment of Mathematics, University of Yaoundé I, PO Box 812 Yaoundé, Cameroon

^bUMI 209 IRD - UPMC UMMISCO, Bondy, France

^cThe African Center of Excellence in Information and Communication Technologies (CETIC), University of Yaounde 1, Cameroon

^dNational Advanced School of Engineering, University of Yaoundé I, P.O. Box 8390, Yaoundé, Cameroon

Abstract

Black Sigatoka Disease, also called Black Leaf Streak Disease (BLSD), is caused by the fungus *Mycosphaerella fijiensis* and is arguably one of the most important pathogens affecting the banana and plantain industries. Theoretical results on its dynamics are rare, even though theoretical descriptions of epidemics of plant diseases are valuable steps toward their efficient management. In this paper, we propose a mathematical model describing the dynamics of BLSD on banana or plantain leaves within a whole field of plants. The model consists of a system of periodic non-autonomous differential equations with a time delay that accounts for the time of incubation of *M. fijiensis* spores. We compute the basic reproduction number of the disease and show that it does not depend on the time delay, meaning that the persistence of BLSD would not qualitatively change even if the incubation period of the pathogen is perturbed. We derive local and global long-term dynamics of the disease and provide numerical simulations to illustrate our results.

Keywords: black sigatoka, fungi, plant epidemics, delay differential equations, periodic systems, stability

*Corresponding author

Email address: tewajules@gmail.com (Jean Jules Tewa)

1. Introduction

Banana is grown in more than 120 countries around the world, primarily in the tropical, rain-fed regions of Africa, Asia, and Latin America, where it is very important in the food security of more than 400 million people and constitute an employment and income source for local populations [1]. It is also the fourth most produced agricultural product after rice, wheat, and corn and its cropping systems are very diverse around the world, with mixed objectives such as self-consumption, sales on local markets, export [1, 2]. About 34% of the annual world production of plantain is produced in Africa, among which around 16% is traded internationally and 84% is consumed locally (FAOSTAT, 2012). Its culture predominates in the lowlands of the humid tropics in central Africa, particularly in Cameroon, DRC, Congo, Gabon, and certain West African countries, notably, Nigeria, Ivory Coast, Ghana, Guinea, and Liberia. In Cameroon, plantain occupies the eighth rank in food productions (FAO, 2010), with an annual production of 1.4 million tons. This crop involves many stakeholders (more than 600,000 producers and around 40,000 tradesmen) and contributes to 2% of the gross domestic product [3].

Unfortunately, both banana and banana-plantain cultures are hampered by plant-parasitic nematodes, insect pests, and foliar fungi. Black sigatoka disease also called black leaf streak disease (BLSD) is caused by a pathogen fungus, *Mycosphaeralla fijiensis*, which is the most costly and damaging banana leaf disease worldwide [4]. *M. fijiensis* (Morelet) reproduces sexually and asexually and emits two kinds of spores that are responsible for the spread of the disease. The sexual spores (ascospores) are the product of sexual reproduction, while asexual spores (conidia) are produced through asexual reproduction. BLSD causes a more or less generalized drying of the foliar system leading to yield losses ranging from 20 to more than 50% [5]. BLSD also caused a 40% reduction in fruit weight. Fruits from plants with a high degree of BLSD undergo an altered maturation process leading to early ripening of fruit [6]. Therefore, the control of this disease relies on some cultural practices and extensive use of

fungicides [7, 4]. These methods have contributed to the eradication of BLSD from commercial plantations in Australia [8]. However, uses of fungicides are potentially detrimental, not only to the environment but also to those who live and work in the surrounding areas [4]. Besides, the use of fungicides can lead to the appearance of resistant strains of *M. Fijiensis* [5].

Mathematical modelling has proven to be valuable in understanding the dynamics of many plant disease[9]. In the 2000s and the early 2010s, a huge set of authors proposed more or less generic models to describe plant disease dynamics [10, 11, 12, 13, 14]. In the special case of banana diseases, not many models exist in the literature. Although, Tankam *et al.* recently build a model to describe and control the dynamics of a banana soilborne pest in a multi-seasonal framework [15], following a framework of Mailleret et al [16]. Concerning BLSD, Ravigné *et al.* proposed a SIR model using a system of ordinary differential equations (ODE) in which the effect of mate limitation on the dynamics of *M. Fijiensis* was studied[17]. In the same way, Hamelin *et al.* relied on a simple model with two coupled partial differential equations to evaluate the impact of mate limitation on the spreading speed of fungal plant parasites in which, only sexual spores diffuse [18]. These two models do not incorporate time-dependence of population parameters or intraguild predation among parasites infecting the same host [19].

The purpose of this work is to analyse the influence of the incubation duration of BLSD and seasonal changes in parameters on the dynamics of the interactions between banana/plantain plants and the fungus *M. Fijiensis*'s taking into account its two spore production means. In fact, several authors have proven the importance of some periodic climatic factors like relative humidity and temperature in the disease incidence [20, 5, 21], and there is no known study on the influence of the disease incubation period. Host-pathogen models being an effective tool to develop decision support for the control of airborne fungal diseases of major field crops [22], we propose a host-pathogen periodic model with time delay based on the epidemiological cycle of the disease with incubation periods.

The present work is organized as follows: Section 2 deals with the construction of our periodic model with time delay, based on our biological knowledge of the disease. In section 3, we analyze the global stability of our solutions with and without delay and illustrate our results in Section 4. We then discuss our results in Section 5 and summarize our results and outcomes in Section 6.

2. The model formulation

2.1. Biological background

The fungus *M. Fijiensis* performs its entire life cycle on the banana plant and has a cycle that consists of four distinct stages. These stages include the contamination stage (deposit and spore germination), the incubation (entering host), symptom development, and spore production (Figure1) [4, 21]. It has been noticed in the literature that each stage of this cycle is strongly influenced by climatic conditions such as temperature and relative humidity [20, 5, 21].

As for a large range of fungal plant diseases, healthy leave densities decrease over time so that most infections occur by spore deposition and germination on young leaves. When humidity and water are in a relatively high amount during the contamination stage, these spores take from 2 to 3 hours to germinate [7].

The incubation period begins with the fungus penetrating the leaf and ends with the appearance of first symptoms, namely: small specks that become streaks running parallel to the leaf veins and which aggregate to form larger compound streaks that vary in colour from rusty red to dark-brown to black, depending on the pathogen[4, 8]. It seems that the incubation period is inversely proportional to the quantity of inoculum that is laid on the leaf [23]. In favorable conditions, the fastest incubation duration ranges between 10 and 14 days but varies considerably depending on the host cultivar [7]. The disease symptoms keep growing until the final stage of lesion development, which corresponds to the conidial latency. The streaks eventually form fusiform or elliptical spots that coalesce, form a water-soaked border with variable degrees of a yellow halo, and eventually, merge to cause extensive leaf necrosis[4, 8].

The spore production stage begins with asexual spore production. Meanwhile, lesions keep on growing to form large necrotic areas. At some point the mycelia become sexually mature and sexual reproduction may occur between two adjacent mycelia in structures called pseudothecia, resulting in sexual spores.

Ascospores are the product of sexual reproduction, while conidia are produced through asexual reproduction and these spores are responsible for the spread of the disease. Conidia and ascospores can become wind-borne within infected plantations and travel up to several tens of kilometres away from disease sources, but ascospores play the most important epidemiological role in the spread of the disease through wind-borne dispersal [24, 25].

About banana leaves reproduction, approximately once a week, a young banana leaf deploys at the top of the stem and develops for approximately 12 weeks, progressively drooping down as new leaves are produced such as illustrated at the left of Figure 1. New leaves then dry and fall on the ground, where, depending on agricultural practices, they may or may not be removed. It is advisable to remove any leaf that shows necrotic lesions on its surface in order to reduce the fungus reservoir. Susceptibility to infection is maximal upon leaf emergence and decreases over time.

2.2. modelling

According to the previous biological background, we build a pathogen-host T-periodic delayed model describing the interaction between *M. fijiensis* spores and banana leaves. We consider a whole banana field and make the following additional modelling assumptions:

- We name N the total leaf biomass that is subdivided between healthy (or susceptible) leaves S and infected leaves X . In most banana plantations, bananas regularly produce healthy new leaves so that the density of plant tissue can be considered relatively constant at the scale of the pest [18]. N is therefore assumed constant.

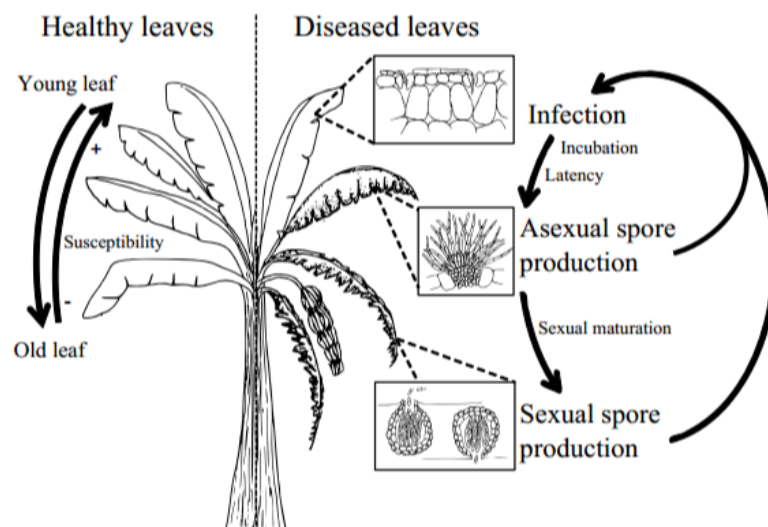


Figure 1: The biological cycle of Black Sigatoka diseases on a banana plant[17]. The infection occurs at spore deposits on leaves and germinates into stomata. Lesions grow up with mycelial growth. After a latent stage, they start producing asexual spores, called asexual spores (conidia), in specific structures called conidiophores (*M. fijiensis*) or sporodochia (*M. musicola*). Meanwhile, lesions keep on growing to form large necrotic areas. At some point these become sexually mature and sexual reproduction may occur between two adjacent mycelia, in structures called pseudothecia, resulting in sexual spores (ascospores). Both conidia and ascospores can initiate new lesions

- We assume that the rate of new leaf emission and leaf mortality is disease independent so that the leaf mortality is the same for infected leaves and healthy leaves. This because the fungus occupies leaf units without necessarily inducing additional mortality. We denote m the constant mortality rate of leaves.
- We term by the state variables $Y(t)$ and $Z(t)$ respectively the number of sexual and asexual spores of the pathogen.
- Sexual spores and asexual spores infect healthy leaves with respectively non-constant rates $\beta(t)$ and $\alpha(t)$. Both sexual and asexual spores succeed to infect a healthy leaf after contact with the same probability $0 < p < 1$ [20].
- The amount of spores produced by infected leaves per unit of time is non-constant and termed $\gamma(t)$ and $\sigma(t)$ respectively for sexual spores (conidia) and asexual spores(ascospores).
- The time of latency before the production of asexual spores after spore deposit is described by a time delay termed τ [20].

Figure 2 summarizes the previous assumption from which we derive the following dynamics.

- Taking a frequency dependent infestation $p [\beta(t)Y(t) + \alpha(t)Z(t)] \frac{S(t)}{N}$, the dynamics of healthy leaves and infected leaves are given respectively by:

$$\dot{S}(t) = mN - p [\beta(t)Y(t) + \alpha(t)Z(t)] \frac{S(t)}{N} - mS(t) \quad (1)$$

and

$$\dot{X}(t) = p [\beta(t)Y(t - \tau) + \alpha(t)Z(t - \tau)] \frac{S(t - \tau)}{N} - mX(t) \quad (2)$$

- The asexual spore dynamics are given by:

$$\dot{z}(t) = \sigma(t)X(t) - \alpha(t)Z(t), \quad (3)$$

where $\sigma(t)x(t)$ is the asexual spores production function.

- As the production of sexual spores results from the interaction between two individuals with compatible mating types (+ and -), it is conditioned by the local presence of mating partners. We assume that the sexual spore production function is given by $f(x, y) = \frac{2xy}{x+y}$ [26], where x and y are densities associated with both mating types. Indeed, if we consider X^+ and X^- the lesion densities of sexual types (+) and (-) respectively, then $f(X^+, X^-) = \frac{2X^+X^-}{X^+ + X^-}$. Hence the dynamic of sexual spores is given by $\dot{Y}(t) = \gamma(t)f(X^+, X^-) - \beta(t)Y(t)$. Hence, assuming a balanced mating-type ratio i.e. $X^+ = X^- = \frac{X}{2}$, we obtain the sexual spores equation :

$$\dot{Y}(t) = \frac{\gamma(t)}{2}X(t) - \beta(t)Y(t). \quad (4)$$

We summarize all the above dynamics in the following 4-dimensional delayed equation that describes the interaction between *M. fijiensis* spores and banana plant, considered as perennial, at the scale of a whole banana field:

$$\left\{ \begin{array}{l} \frac{dS}{dt} = mN - p[\beta(t)Y(t) + \alpha(t)Z(t)] \frac{S(t)}{N} - mS(t), \\ \frac{dX}{dt} = p[\beta(t)Y(t-\tau) + \alpha(t)Z(t-\tau)] \frac{S(t-\tau)}{N} - mX(t), \\ \frac{dY}{dt} = \frac{\gamma(t)}{2}X(t) - \beta(t)Y(t), \\ \frac{dZ}{dt} = \sigma(t)X(t) - \alpha(t)Z(t); \end{array} \right. \quad (5)$$

with initial conditions $S(\theta) = \varphi_1(\theta) \geq 0, X(\theta) = \varphi_2(\theta) \geq 0, Y(\theta) = \varphi_3(\theta) \geq 0$ and $Z(\theta) = \varphi_4(\theta) \geq 0$ for $\theta \in [-\tau, 0]$, where $\varphi \in \mathcal{C}([-\tau, 0], \mathbb{R}^4)$, the Banach space of continuous functions mapping the interval $[-\tau, 0]$ into \mathbb{R}^3 equipped with the norm $\|\varphi\| = \sup_{-\tau \leq t \leq 0} |\varphi(t)|$.

Since, the total leaf biomass N is constant, the biomass of healthy leaves can be deduced from the infected leaf biomass through the delayed equation

$S(t - \tau) = N - X(t - \tau)$. The dynamics of the infected leaves become :

$$\dot{X}(t) = p [\beta(t)Y(t - \tau) + \alpha(t)Z(t - \tau)] \left(1 - \frac{X(t - \tau)}{N} \right) - mx(t), \quad (6)$$

and we drop the dynamics of healthy leaves.

Considering the state variables $x = \frac{X}{N}$, $y = \frac{Y}{N}$ and $z = \frac{Z}{N}$ representing ratios, System 5 becomes:

$$\begin{cases} \frac{dx}{dt} &= p [\beta(t)y(t - \tau) + \alpha(t)z(t - \tau)] (1 - x(t - \tau)) - mx(t), \\ \frac{dy}{dt} &= \frac{\gamma(t)}{2} x(t) - \beta(t)y(t), \\ \frac{dz}{dt} &= \sigma(t)x(t) - \alpha(t)z(t), \end{cases} \quad (7)$$

with initial conditions $x(\theta) = \varphi_1(\theta) \geq 0$, $y(\theta) = \varphi_2(\theta) \geq 0$ and $z(\theta) = \varphi_3(\theta) \geq 0$ for $\theta \in [-\tau, 0]$, where $\varphi \in \mathcal{C}([-\tau, 0], \mathbb{R}^3)$.

The functions $\beta(t)$, $\alpha(t)$, $\gamma(t)$ and $\sigma(t)$ are continuous, positive and T-periodic such that the same dynamics occur over several cropping seasons.

Table 1 summarizes the parameters of System 7 and their dimensions.

3. Mathematical Analysis

3.1. Positivity and boundedness of solutions

Lemma 1. : *Let us consider System (7) with its initial conditions. Its trajectories are non-negative and bounded.*

Proof. : Let us consider the spaces $\mathcal{C} = \mathcal{C}([-\tau, 0], \mathbb{R}^3)$ and $\mathcal{C}^+ = \mathcal{C}([-\tau, 0], \mathbb{R}_+^3)$. Then, $(\mathcal{C}, \mathcal{C}^+)$ is an ordered Banach space equipped with the maximum norm and the positive cone \mathcal{C}^+ .

Let us consider $u_t : [-\tau, 0] \longrightarrow \mathbb{R}$ with $u_t(\theta) = u(t + \theta)$ for $t \geq 0$ and $\theta \in [-\tau, 0]$.

System(7) can take the following form

$$\dot{u}(t) = f(t, u_t), \quad (8)$$

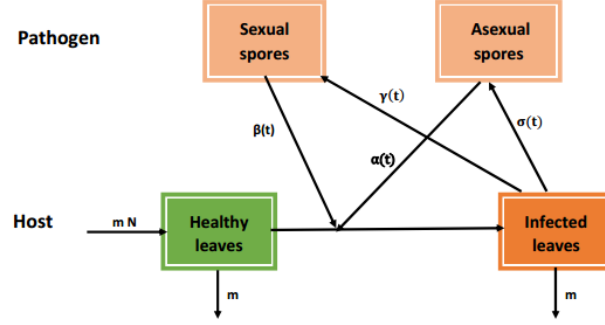


Figure 2: Schematic representation of the interaction between *M. fijiensis* spores and banana plant as described by System(5). The infection begins with the spore deposit on a healthy leaf with the rates $\beta(t)$ for the sexual spores and $\alpha(t)$ for the asexual spores. After germination, penetration on the leaf and incubation, there is an asexual spore production with the rate $\sigma(t)$ and a sexual spore production with the rate $\gamma(t)$. After their production, spores are dispersed to form a new focus.

Symbol	Meaning	Dimension
p	Sexual and asexual spores infectivity	none
γ	Sexual spore production rate	spores per day
σ	Asexual spore production rate	spores per day
β	Sexual spore dispersal rate	spores per day
α	Asexual spore dispersal rate	spores per day
m	Catabolism rate of plant leaves	days ⁻¹
τ	Asexual latency	days
T	Cropping season duration	days
x	Density of infected leaves	\
y	Density of sexual spores	Spores per unit of leaf biomass
z	Density of asexual spores	Spores per unit of leaf biomass

Table 1: Parameters of System 7 and their dimensions.

where for $\phi \in \mathcal{C}$,

$$f_1(t, \phi) = p [\beta(t)\phi_2(-\tau) + \alpha(t)\phi_3(-\tau)] (1 - \phi_1(-\tau)) - m\phi_1(0),$$

$$f_2(t, \phi) = \frac{\gamma(t)}{2} \phi_1(0) - \beta(t)\phi_2(0),$$

$$f_3(t, \phi) = \sigma(t)\phi_1(0) - \alpha(t)\phi_3(0).$$

It is well known that f is continuous for $(t, \phi) \in \mathbb{R}_+ \times \mathcal{C}([-\tau, 0], \mathbb{R}_+^3)$ and $f(t, \phi)$ is Lipschitz with respect to ϕ on each compact subset of $\mathcal{C}([-\tau, 0], \mathbb{R}_+^3)$. By the general theory of linear functional differential equations ([27], section 8.1), it follows that, for any $s \in \mathbb{R}$ and $\phi \in \mathcal{C}^+$, there is a unique solution $u = u(s, \phi)$ of System (7) defined on its maximal interval of existence $[s, \delta_s)$ with $u_s = \phi$.

Let us consider $\phi = (\phi_1, \phi_2, \phi_3) \in \mathcal{C}([-\tau, 0], \mathbb{R}_+^3)$. If $\phi_i(0) = 0$ for some $i = 1, 2, 3$, then $f_i(t, \phi) \geq 0$. It follows by [28] that for any $\phi \in \mathcal{C}([-\tau, 0], \mathbb{R}_+^3)$, and $s \in \mathbb{R}$, the unique solution $u = u(s, \phi)$ of System (7) with $u_s = \phi$ satisfies $u_t(\phi) \in \mathcal{C}^+$ for all $t \in [s, \delta_s)$.

Let us consider $s(t)$, the density of susceptible leaf. For all $t \in \mathbb{R}_+$, $s(t) + x(t) = 1$ and this implies $x(t) \leq 1$ for all $t \in \mathbb{R}_+$. Let

$$\beta_m = \min_{0 \leq t \leq T} \beta(t), \quad \alpha_m = \min_{0 \leq t \leq T} \alpha(t)$$

and

$$\sigma_M = \max_{0 \leq t \leq T} \sigma(t), \quad \gamma_M = \max_{0 \leq t \leq T} \gamma(t).$$

The second equation of System (7) implies $\dot{y}(t) \leq \frac{\gamma_M}{2} - \beta_m y(t)$ and finally $y(t) \leq \frac{\gamma_M}{2\beta_m}$. The third equation of System (7) implies $\dot{z}(t) \leq \frac{\sigma_M}{\alpha_m}$. Therefore, x, y and z are bounded and the set

$$\Gamma = \{0 \leq x(t) \leq 1; \quad 0 \leq y(t) \leq \frac{\gamma_M}{2\beta_m}; \quad 0 \leq z(t) \leq \frac{\sigma_M}{\alpha_m}, \quad \forall t \geq 0\}. \quad (9)$$

is a positively invariant set for system (7).

3.2. Existence and stability of solutions

System (7) always has $P(t) \equiv (0, 0, 0)$ as disease-free solution. In order to analyze the stability of this solution and the disease persistence, we consider the case without delay ($\tau = 0$) and the case with delay ($\tau > 0$).

Before the study, we briefly review the Floquet theorem. For more details of Floquet theory, we refer to [27, 29, 30]

Floquet exponents or characteristic multipliers are defined in terms of a fundamental matrix, solution of non-autonomous linear systems. The challenge here is to be able to determine the Floquet exponents without knowing explicit solutions. In fact, for many non-autonomous linear systems, analytical solutions in an explicit form are hard to obtain.

Let us consider the following homogeneous linear periodic system

$$\dot{X} = A(t)X, \quad (10)$$

where $A(t + T) = A(t)$, $T > 0$, $X \in \mathbb{R}^n$ and $A(t)$ is a $n \times n$ real matrix-valued function of t .

The stability of a periodic system is given by the following theorem.

Theorem 3.1. (*Theorem 7.1 Hale's book [31]*).

1. *A necessary and sufficient condition that System (10) is uniformly stable is that the modulus of characteristic multipliers are less or equal to 1 (Floquet exponents have negative real parts) and the multiplicity of those with modulus equal to 1 (Floquet exponents with real parts equal to 0) is equal to 1.*
2. *A necessary and sufficient condition that System(10) is uniformly asymptotically stable is that the modulus of all characteristic multipliers are less than 1 (all Floquet exponents have negative real parts).*

Let $(\mathbb{R}^n, \mathbb{R}_+^n)$, be the standard ordered n -dimensional Euclidean space with a norm $\|\cdot\|$. For $u, v \in \mathbb{R}^n$, we write $u \geq v$ provided $u - v \in \mathbb{R}_+^n$, $u > v$ provided $u - v \in \mathbb{R}_+^n \setminus \{0\}$ and $u \gg v$ provided $u - v \in \text{int}(\mathbb{R}_+^n)$, where $\text{int}(\mathbb{R}_+^n)$ is the

interior of \mathbb{R}_+^n .

Recall that a $n \times n$ matrix (a_{ij}) is said to be cooperative if all of its off-diagonal entries are nonnegative; irreducible if its index set $\{1, 2, \dots, n\}$ cannot be split into two complementary sets (without common indices) $\{m_1, m_2, \dots, m_p\}$ and $\{k_1, k_2, \dots, k_q\}$ ($p + q = n$) such that $a_{m_i k_j} = 0, \forall 1 \leq i \leq p, 1 \leq j \leq q$.

Let $A(t)$ be a continuous, cooperative, irreducible, and T -periodic $n \times n$ matrix function, $\Phi(t)$ be the fundamental solution matrix of the linear ordinary differential system $\dot{x} = A(t)x$ and $r(\Phi(T))$ be the spectral radius of $\Phi(T)$. It then follows from [32] that $\Phi(t)$ is a matrix with all entries positive for each $t > 0$. By the Perron-Frobenius theorem, $r(\Phi(T))$ is the principal eigenvalue of $\Phi(T)$ in the sense that it is simple and admits an eigenvector $v^* \gg 0$. The following result is useful for our subsequent comparison arguments.

Lemma 2. ([32], Lemma 2.1) *Let $\mu = \frac{1}{T} \ln r(\Phi(T))$ the Floquet exponent associated to $r(\Phi(T))$. Then there exists a positive, T -periodic function $v(t)$ such that $e^{\mu t} v(t)$ is a solution of system $\dot{x} = A(t)x$.*

3.2.1. Global stability of disease free solution without delay

When $\tau = 0$, System 7 is resumed to the following:

$$\begin{cases} \dot{x} &= p(\beta(t)y(t) + \alpha(t)z(t))(1 - x(t)) - mx(t), \\ \dot{y} &= \frac{\gamma(t)}{2}x(t) - \beta(t)y(t), \\ \dot{z} &= \sigma(t)x(t) - \alpha(t)z(t), \end{cases} \quad (11)$$

with initial conditions $x(0) = x_0 \geq 0$, $y(0) = y_0 \geq 0$ and $z(0) = z_0 \geq 0$.

We assume that the parameters of System 11 are non-negative and

$$\int_0^T \beta(t)dt > 0, \int_0^T \alpha(t)dt > 0, \int_0^T \gamma(t)dt > 0 \quad \text{and} \quad \int_0^T \sigma(t)dt > 0.$$

System 11 linearized around the DFE is written:

$$\begin{cases} \dot{x} &= -mx(t) + p\beta(t)y(t) + p\alpha(t)z(t), \\ \dot{y} &= \frac{\gamma(t)}{2}x(t) - \beta(t)y(t), \\ \dot{z} &= \sigma(t)x(t) - \alpha(t)z(t), \end{cases} \quad (12)$$

Or in the following compact form:

$$\dot{X}(t) = A(t)X(t), \quad (13)$$

where $X = (x, y, z)^T$ and

$$A(t) = D_X F(t, 0) \begin{pmatrix} -m & \beta(t)p & \alpha(t)p \\ \frac{\gamma(t)}{2} & -\beta(t) & 0 \\ \sigma(t) & 0 & -\alpha(t) \end{pmatrix},$$

where $F(t, X)$ is the right hand side of System 11.

If ρ_1 and ρ_2 are the Floquet multipliers of System 12, then the DFE is asymptotically stable if $\rho_i < 1$, $i = 1, 2$, and unstable if $\rho_1 > 1$ or $\rho_2 > 1$. Notice that if one Floquet multiplier has a modulus that is equal to 1, then System 12 has a periodic solution.

The main difficulty in trying to apply Floquet's theory to our three-dimensional model (11) is the explicit indeterminacy of the Floquet multipliers that are the eigenvalues of the monodromy matrix, whereas Floquet's theory consists of studying these eigenvalues. We notice that for dimension three, the averaging theory gives in some cases a condition of asymptotic stability of the trivial solution that is stronger than that obtained with the Lyapunov function. This is because if the origin of the averaged system is stable then the origin of the initial system is also stable. The averaging method is therefore effective for studying the stability of homogeneous and T-periodic systems. The complexity of the model is overcome by studying the properties of the averaged system which is made autonomous.

We reduce our system to an autonomous system by replacing time-dependent parameters with their long-term averages. We can instead characterize the Floquet's multipliers by some thresholds. This method has been used by many authors to compute the basic reproduction number of periodic systems [33, 34, 35, 36].

The long-term average of a function h , is given by:

$$\langle h \rangle = \lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t h(s) ds \quad (14)$$

and if h is T -periodic, then its long-term average becomes $\langle h \rangle = \frac{1}{T} \int_0^T h(t) dt$ [37].

Theorem 3.2. *Considering System 11 and terming:*

$$\mathcal{R}_1 = \frac{p\langle\sigma\rangle}{m}, \quad \mathcal{R}_2 = \frac{p\langle\gamma\rangle}{2m}, \quad \text{and} \quad \mathcal{R}_0 = \mathcal{R}_1 + \mathcal{R}_2,$$

we have the following results:

1. *If $\mathcal{R}_0 < 1$, then, the disease free solution $P(t) \equiv 0$ is globally asymptotically stable.*
2. *if $\mathcal{R}_0 > 1$, then, the disease free solution is unstable.*

Proof. : Dropping the symbol $\langle \rangle$ for convenience, the $A(t)$ matrix is constant and the Floquet exponents are the eigenvalues of $\frac{1}{T} \int_0^T A(t) dt = A$. We aim to determine the conditions under which the real parts of all Floquet exponents are positive. As we have

$$A = \begin{pmatrix} -m & \beta p & \alpha p \\ \frac{\gamma}{2} & -\beta & 0 \\ \sigma & 0 & -\alpha \end{pmatrix},$$

the characteristic equation associated with A is given by

$$X^3 + (\alpha + \beta + m)X^2 + [\alpha\beta + \alpha m(1 - \mathcal{R}_1) + \beta m(1 - \mathcal{R}_2)]X + m\alpha\beta(1 - \mathcal{R}_0). \quad (15)$$

Using the Descartes rule of signs, we figure that if $\mathcal{R}_0 > 1$, equation 15 has at most one positive real solution, that is a positive real Floquet exponent and $P(t) \equiv 0$ is unstable.

If $\mathcal{R}_0 < 1$, then we can rewrite 15 in the form $X^3 + AX^2 + BX + C = 0$ with

$$A = \alpha + \beta + m > 0, B = (\alpha\beta + \alpha m(1 - \mathcal{R}_1) + \beta m(1 - \mathcal{R}_2)) > 0 \quad \text{and} \quad C = m\alpha\beta(1 - \mathcal{R}_0) > 0.$$

We also have

$$AB - C = \alpha\beta(\alpha + \beta + 2m) + m\alpha(1 - \mathcal{R}_1)(m + \alpha) + m\beta(1 - \mathcal{R}_2)(m + \beta) > 0.$$

Hence, according to Routh-Hurwitz stability criterion, each solution of Equation 15 has a negative real part, and from Theorem 3.1 the DFE is locally asymptotically stable.

We then show the global attractivity of the disease-free solution when $\mathcal{R}_0 < 1$.

The following inequality system holds for System 11:

$$\begin{cases} \dot{x} & \leq -mx(t) + p\beta(t)y(t) + p\alpha(t)z(t), \\ \dot{y} & = \frac{\gamma(t)}{2}x(t) - \beta(t)y(t), \\ \dot{z} & = \sigma(t)x(t) - \alpha(t)z(t). \end{cases} \quad (16)$$

Let us consider the following system:

$$\begin{cases} \dot{x} & = -mx(t) + p\beta(t)y(t) + p\alpha(t)z(t), \\ \dot{y} & = \frac{\gamma(t)}{2}x(t) - \beta(t)y(t), \\ \dot{z} & = \sigma(t)x(t) - \alpha(t)z(t). \end{cases} \quad (17)$$

If $\Phi(t)$ is the fundamental matrix associated with System 17, then $r(\Phi(T)) < 1$. By Lemma 2 and the standard comparison principle, there exists a positive T -periodic function $v(t)$ such that $J(t) \leq v(t)e^{\lambda t}$ is a positive solution of 11 with $J(t) = (x(t), y(t), z(t))^T$ and $\lambda = \frac{1}{T} \ln r(\Phi(T)) < 0$. Then we see that $\lim_{t \rightarrow +\infty} x(t) = \lim_{t \rightarrow +\infty} y(t) = \lim_{t \rightarrow +\infty} z(t) = 0$. Hence, the DFE $(0, 0, 0)$ is globally attractive.

Remark 3.1. (*Thresholds interpretation*)

- As $p\langle\sigma\rangle$ and $p\langle\frac{\gamma}{2}\rangle$ respectively represent the average number of asexual spores and sexual spores produced by an infected leaf per day, and $\frac{1}{m}$ is the infected leaf duration, \mathcal{R}_1 and \mathcal{R}_2 respectively denote the average number of asexual spores and sexual spores produced by an infected leaf during its entire infection period.
- \mathcal{R}_0 denotes the average number of spores (sexual and asexual) produced by an infected leaf during its entire infection period.

- The threshold \mathcal{R}_0 is related to both asexual and sexual reproduction. In absence of sexual spore production, \mathcal{R}_0 is resumed to \mathcal{R}_1 and In absence of asexual spore production, it is resumed to \mathcal{R}_2 .

3.2.2. Disease persistence without delay.

Uniform persistence is an important concept in population dynamics since it characterizes the long-term survival of some or all interacting species in an ecosystem. In this, section we analyze the dynamics of the periodic model when $\mathcal{R}_0 > 1$.

We show that \mathcal{R}_0 is a threshold parameter for the extinction and the uniform persistence of the disease.

Let $P : \mathbb{R}_+^3 \longrightarrow \mathbb{R}_+^3$ be the Poincaré map associated with System 11. Namely:

$$\begin{aligned} P(\phi) &= u(T, \phi) \quad \text{for } \phi = (x_0, y_0, z_0)^T \in \mathbb{R}_+^3, \\ P^m(\phi) &= u(mT, \phi) \quad \forall m \geq 0, \end{aligned}$$

where $u(t, \phi)$ is the unique solution of System 11 with initial condition $u(0, \phi) = \phi$.

We define:

$$\Omega = \{(x, y, z) \in \mathbb{R}_+^3\}, \quad \Omega_0 = \{(x, y, z) \in \text{int}(\mathbb{R}_+^3)\} \quad \text{and} \quad \partial\Omega_0 = \Omega \setminus \Omega_0.$$

Both Ω and Ω_0 are positively invariant for System 11. We set:

$$M_\partial = \{(x_0, y_0, z_0) \in \partial\Omega_0 : P^m(x_0, y_0, z_0) \in \partial\Omega_0, \forall m \geq 0\}.$$

To use the theory of uniform persistence developed in [38], we show that:

$$M_\partial = \{(0, 0, 0)\}.$$

Note that $\{(0, 0, 0)\} \subseteq M_\partial$. To show that $M_\partial \setminus \{(0, 0, 0)\} = \emptyset$, we consider a $u_0 \in M_\partial \setminus \{(0, 0, 0)\}$. Thus, u_0 is also in $\partial\Omega_0$; that is $x_0 > 0$ or $y_0 > 0$ or $z_0 > 0$.

In the case $x_0 > 0$, it comes from Lemma 1 that $x(t) > 0$ and:

$$y(t) = \exp\left(-\int_0^t \beta(s)ds\right) \left[y_0 + \int_0^t \frac{\gamma(s)}{2} x(s) \exp\left(\int_0^s \beta(r)dr\right) ds \right] > 0 \quad (18)$$

$$z(t) = \exp\left(-\int_0^t \alpha(s)ds\right) \left[z_0 + \int_0^t \sigma(s)x(s) \exp\left(\int_0^s \alpha(r)dr\right) ds \right] > 0 \quad (19)$$

for any $t > 0$. It follows that $(x(t), y(t), z(t)) \notin \partial\Omega_0$. This is a contradiction.

In the case $x_0 = 0$,

$$\frac{dx}{dt}\bigg|_{t=0} = p(\beta(0)y(0) + \alpha(0)z(0)) > 0.$$

By continuity and the sign of derivative of $x(t)$, we have that for small $0 < t \ll 1$, $x(t) > 0$. This implies in (18) and (19) that $y(t) > 0$ and $z(t) > 0$. So for, $0 < t \ll 1$, $(x(t), y(t), z(t)) \in \Omega_0 \Rightarrow (x(t), y(t), z(t)) \in \Omega_0$ for all $t > 0$ since Ω_0 is positively invariant. It follows that $(x(t), y(t), z(t)) \notin \partial\Omega_0$. This is a contradiction. Therefore we have $M_\partial = \{(0, 0, 0)\}$.

Now we can introduce the following result of uniform persistence of the disease.

Theorem 3.3. *When $\mathcal{R}_0 > 1$, System 11 admits at least one positive periodic solution and there exists a positive constant ε such that for all initial condition $(x_0, y_0, z_0) \in \Omega_0$,*

$$\liminf_{t \rightarrow +\infty} (x(t), y(t), z(t)) \geq (\varepsilon, \varepsilon, \varepsilon)$$

.

Proof. : We first prove that P is weakly uniformly persistent (see definition 1.3.2 from [38]) with respect to $(\Omega_0, \partial\Omega_0)$, because this implies that the solution of System 11 is uniformly persistent with respect to $(\Omega_0, \partial\Omega_0)$ ([38], theorem 3.1.1).

Since $\mathcal{R}_0 > 1$, we have $r(P) > 1$, where $r(P)$ is the spectral radius of Poincaré map P . Let P_η be the Poincaré map of the following perturbed linear periodic system:

$$\begin{cases} \frac{dx}{dt} = -mx(t) + p\beta(t)(1-\eta)y(t) + p\alpha(t)(1-\eta)z(t), \\ \frac{dy}{dt} = \frac{\gamma(t)}{2}x(t) - \beta(t)y(t), \\ \frac{dz}{dt} = \sigma x(t) - \beta(t)z(t). \end{cases} \quad (20)$$

We have $\lim_{\eta \rightarrow 0} r(P_\eta) = r(P)$. We can choose a small number $\eta > 0$ such that $r(P_\eta) > 1$.

Let us define

$$W^s(M_0) = \{\phi \in \Omega_0 : \lim_{m \rightarrow +\infty} \|P^m(\phi) - M_0\|_1 \leq 0\},$$

the stable set of M_0 where $M_0 = (0, 0, 0)$. We will show that $W^s(M_0) \cap \Omega_0 = \emptyset$.

By the continuity of solutions in $[0, T]$ with respect to initial values, for a small real number $\eta > 0$, there exist $\delta > 0$ such that for all $\phi \in \Omega_0$, with $\|\phi - M_0\|_1 \leq \delta$, we have

$$\|u(t, \phi) - u(t, M_0)\|_1 < \eta, \forall t \in [0, T], \text{ with } u \text{ solution of Equation 11.}$$

We now claim that:

$$\limsup_{m \rightarrow +\infty} \|P^m(\phi) - M_0\|_1 \geq \delta, \forall \phi \in \Omega_0.$$

By contradiction, we suppose that $\limsup_{m \rightarrow +\infty} \|P^m(\phi) - M_0\|_1 < \delta$ for some $\phi \in \Omega_0$. Without lost generality, we can assume that $\|P^m(\phi) - M_0\|_1 < \delta$ for all $m \geq 0$ (see [39], Appendix c). Then from the the above discussion, it follows that

$$\|u(t, P^m(\phi)) - u(t, M_0)\|_1 < \eta,$$

for any $m > 0$ and $t \in [0, T]$.

For any $t \geq 0$, let $t = mT + t_1$, where $t_1 \in [0, T]$ and $m = \lfloor \frac{t}{T} \rfloor$ is the greatest integer less than or equal to $\frac{t}{T}$. We then have:

$$\begin{aligned} \|u(t, \phi) - u(t, M_0)\|_1 &= \|u(t_1 + mT, \phi) - u(t_1 + mT, M_0)\|_1 \\ &= \|u(t_1, \phi) \circ u(mT, \phi) - u(t_1, M_0) \circ u(mT, M_0)\|_1 \\ &= \|u(t_1, P^m(\phi)) - u(t_1, P^m(M_0))\|_1 \\ &= \|u(t_1, P^m(\phi)) - u(t_1, M_0)\|_1 < \eta. \end{aligned}$$

If we set $u(t, \phi) = (x(t), y(t), z(t))$, then we have:

$$0 \leq x(t) \leq \eta, \quad 0 \leq y(t) \leq \eta, \quad 0 \leq z(t) \leq \eta, \quad \forall t \geq 0.$$

And from Equation 11,

$$\begin{cases} \frac{dx}{dt} & \geq -mx(t) + p\beta(t)(1-\eta)y(t) + p\alpha(t)(1-\eta)z(t), \\ \frac{dy}{dt} & = \frac{\gamma(t)}{2}x(t) - \beta(t)y(t), \\ \frac{dz}{dt} & = \sigma x(t) - \beta(t)z(t), \end{cases} \quad (21)$$

and $r(P_\eta) > 1$. By lemme 2 and the comparison principle, there exist a positive T-periodic function $v_1(t)$ solution of system 20 such that $J(t) \geq e^{rt}v(t)$, where $r = \frac{1}{T} \ln(r(p_\eta)) > 0$ and $J(t) = (x(t), y(t), z(t))^t$. Therefore, $\lim_{t \rightarrow +\infty} (x(t), y(t), z(t)) = (+\infty, +\infty, +\infty)$, this leads to a contradiction and our claim holds.

This claim shows that P is weakly uniformly persistent with respect to $(\Omega_0, \partial\Omega_0)$. From Equation 9, Γ is a compact set which attracts all positive orbits in Ω . This implies that the discrete-time System $P : \Omega \rightarrow \Omega$ is point-dissipative. Moreover, $\forall n \geq 1, P^n$ is compact. It then follows that P admits a global attractor in Ω . It follows that M_0 is an isolated invariant set in Ω and $W^s(M_0) \cap \Omega_0 = \emptyset$. Any solution in M_∂ then converges to M_0 and M_0 is acyclic in M_∂ . By the acyclicity theorem on uniform persistence for maps ([38] Theorem 1.3.1 and Remark 1.3.1), it follows that P is uniformly persistent with respect to $(\Omega_0, \partial\Omega_0)$. This means that there exists $\varepsilon > 0$ such that any solution of System 11 satisfies:

$$\liminf_{t \rightarrow +\infty} (x(t), y(t), z(t)) \geq (\varepsilon, \varepsilon, \varepsilon).$$

Moreover, P has a fixed point $(\tilde{x}_0, \tilde{y}_0, \tilde{z}_0) \in \Omega_0$ that is a positive T-periodic solution for System 11 ([38], Theorem 1.3.6). This ends our proof.

3.2.3. Stability of disease free solution with delay

We now analyse the stability of System 7 when $\tau > 0$.

We firstly determine the basic reproduction number for this model.

Let us define the evolution operator $U(t, s)$ on \mathcal{C} associated to System 7 as

$$U(t, s)\phi = u_t(s, \phi), \quad \text{for } t \geq s, s \in \mathbb{R},$$

where

$$u_t(s, \phi)(\theta) = u(t + \theta, s, \phi), \quad \forall \theta \in [-\tau, 0].$$

$U(t, s)$ is continuous and

$$U(s, s) = I, \quad U(t, s)U(s, r) = U(t, r) \quad \text{and} \quad U(t+T, s+T) = U(t, s) \quad \text{for} \quad r \leq s \leq t.$$

The monodromy operator is introduced as the evolution operator evaluated at the period $U(T, 0)$. The characteristic multipliers or Floquet multipliers of System 7 are the eigenvalues of the operator $U(T, 0)$. It is well known that for most non autonomous systems, analytical solutions in explicit form are difficult to obtain. We will analyse the stability of the free solution of System 7 by introducing the basic reproduction number \mathcal{R}_0 .

In order to analyse the stability of the free solution of System 7, we make the following assumption.

Assumption : The parameters of System 7 are non-negative and continuous functions with $\int_0^T \alpha(t)dt > 0$, $\int_0^T \beta(t)dt > 0$, $\int_0^T \gamma(t)dt > 0$, and $\int_0^T \sigma(t)dt > 0$.

In order to linearize System 7 at $p(t) \equiv 0$, we put it in the following form

$$\frac{du}{dt} = F(t)u_t - V(t)u(t), \quad (22)$$

where $F : \mathbb{R} \rightarrow \mathcal{L}(\mathcal{C}, \mathbb{R}^3)$ is a map and $V(t)$ is a continuous 3×3 matrix function on \mathbb{R} , $F(t)$ and $V(t)$ are T -periodic functions. Let us refer to ([40, 38]).

The Linearization at $(0, 0, 0)$ gives the following linear periodic system :

$$\begin{cases} \dot{x} &= p\beta(t)y(t-\tau) + p\alpha(t)z(t-\tau) - mx(t), \\ \dot{y} &= \frac{\gamma(t)}{2}x(t) - \beta(t)y(t), \\ \dot{z} &= \sigma x(t) - \alpha(t)z(t). \end{cases} \quad (23)$$

If we let

$$F(t) \begin{pmatrix} \phi_1 \\ \phi_2 \\ \phi_3 \end{pmatrix} = \begin{pmatrix} p\beta(t)\phi_2(-\tau) + p\alpha(t)\phi_3(-\tau) \\ \frac{\gamma(t)}{2}\phi_1(0) \\ \sigma(t)\phi_1(0) \end{pmatrix} \quad \text{and} \quad V(t) = \begin{pmatrix} m & 0 & 0 \\ 0 & \beta(t) & 0 \\ 0 & 0 & \alpha(t) \end{pmatrix}.$$

Then, System 23 can be written in the matrix form as follows:

$$\frac{du(t)}{dt} = F(t)u_t - V(t)u(t), \quad \forall t \geq 0 \quad \text{where} \quad u = (x, y, z)^T. \quad (24)$$

Note that $F(t)$ and $V(t)$ are T -periodic functions in t and the newly infection is represented by $F(t)$ while the growth of the pathogen is described by the system

$$\frac{du(t)}{dt} = -V(t)u(t). \quad (25)$$

Let $\Phi(t, s), t \geq s$ be the evolution matrix of System 25, that is, for each $s \in \mathbb{R}$, the 3×3 matrix solution of

$$\frac{\partial \Phi(t, s)}{\partial t} = -V(t)\Phi(t, s), \quad t \geq s, \quad \Phi(s, s) = I_3.$$

Then,

$$\Phi(t, s) = \begin{pmatrix} \exp(-m(t-s)) & 0 & 0 \\ 0 & \exp \int_s^t -\beta(r)dr & 0 \\ 0 & 0 & \exp \int_s^t -\alpha(r)dr \end{pmatrix}.$$

The exponential growth bound of $\Phi(t, s)$ is defined as

$$w(\Phi) = \inf \{ \tilde{w} : \exists M \geq 1 : \|\Phi(t+s, s)\| \leq M e^{\tilde{w}t}, \quad \forall s \in \mathbb{R}, t \geq 0, s \leq t \}.$$

Hence,

$$\begin{aligned} \|\Phi(t+s, s)\| &= \max \left\{ e^{-mt}; \quad e^{-\int_s^{t+s} \beta(r)dr}; \quad e^{-\int_s^{t+s} \alpha(r)dr} \right\} \\ &\leq \max \{ e^{-mt}; \quad e^{-at}; \quad e^{-bt} \} \end{aligned} \quad (26)$$

where

$$a = \min_{0 \leq t \leq T} \alpha(t), \quad b = \min_{0 \leq t \leq T} \beta(t).$$

It follows that $w(\Phi) \leq -\min\{m, a, b\}$. Therefore, $F(t)$ and $V(t)$ satisfy the following assumptions:

Assumption : $F(t) : \mathcal{C} \longrightarrow \mathbb{R}^3$ is positive in the sense that $F(t)\mathcal{C}^+ \subseteq \mathbb{R}_+^3$.

Assumption : The periodic matrix $-V(t)$ is cooperative, and $w(\Phi) < 0$.

Let us assume that hypothesis 1, 2 and 3 hold.

Let us consider C_T , the Banach space of all T -periodic functions from \mathbb{R} to \mathbb{R}^3 equipped with the maximum norm and the positive cone $C_T^+ = \{u \in C_T : u(t) \geq 0, \forall t \in \mathbb{R}\}$. According to ([40], section 2), we assume that $v \in C_T$ is the initial distribution of infectious individuals. Then, for $s \geq 0$, $F(t-s)v_{t-s}$ is the distribution of newly infected individuals at time $t-s$, which was produced by the infectious individuals who were introduced over the time interval $[t-s-\tau, t-s]$. Then $\Phi(t, t-s)F(t-s)v_{t-s}$ is the distribution of those infected individuals who were newly infected at time $t-s$ and remain in the infected compartment at time t for $t \geq s$. Hence,

$$\int_0^\infty \Phi(t, t-s)F(t-s)v_{t-s}ds = \int_0^\infty \Phi(t, t-s)F(t-s)v(t-s+\cdot)ds$$

is the distribution of cumulative new infections at time t , produced by all those infectious individuals introduced at all previous times to t .

We define the next generation operator $L : C_T \longrightarrow C_T$ by

$$[Lv](t) = \int_0^\infty \Phi(t, t-s)F(t-s)v(t-s-\tau)ds, \quad \forall t \in \mathbb{R}, v \in C_T.$$

The basic reproduction ratio, $\mathcal{R}_0 = \rho(L)$, is the spectral radius of L . It stands that $\mathcal{R}_0 - 1$ is a threshold value for the stability of the zero solution of System 23 ([40], Theorem 2.1). Theorem 3.4 gives the stability of the zero solution of System 23.

Theorem 3.4. *[40] Since $\mathcal{R}_0 - 1$ has the same sign as $r(U(T, 0)) - 1$ and the following results hold:*

1. *If $\mathcal{R}_0 < 1$, then the disease free solution $P(t) \equiv 0$ is asymptotically stable.*
2. *If $\mathcal{R}_0 > 1$, then free solution is unstable and the disease persists.*

This result shows that the sign of $\mathcal{R}_0 - 1$ determines the stability of the zero solution of System 23, and hence, \mathcal{R}_0 is a threshold value for the local invasion

of the disease.

In order to compute numerically the basic reproduction ratio defined by $\mathcal{R}_0 = \rho(L)$, we can use the results of [41]. The expression obtained by the latter method is often difficult to exploit. From the perspective of applying to a real-world problem, as is the case here, then it may be unnecessary.

Once again we rely on long-term averaging to compute the basic reproduction number. The system is then reduced to an autonomous delayed system, and the method exposed by Xiao. *et al*[40] can be used. The basic reproduction number of the autonomous system is still calculated as the spectral radius of the matrix FV^{-1} . Many different models in the literature are analyzed using long-time averaging method [33, 34, 35, 36] and for most of them, the calculated basic reproduction number turned out to be correct and equivalent to that found using the linear operator method [42].

since all parameters values are T -periodic, then for some parameter $\delta(t)$, the averages value is given by

$$\langle \delta \rangle = \frac{1}{T} \int_0^T \delta(t) dt.$$

Let us refer in this case to [40]. For any given $F \in \mathcal{L}(\mathcal{C}, \mathbb{R}^m)$, we define $\hat{F} \in \mathcal{C}(\mathbb{R}^m, \mathbb{R}^m)$ by

$$\hat{F}u = F(\hat{u}), \forall u \in \mathbb{R}^m,$$

where $\hat{u}(\theta) = u, \forall \theta \in [-\tau, 0]$. Then, \hat{F} can be looked as a $m \times m$ matrix and the following results hold.

Theorem 3.5. *Let us consider $F(t) \equiv F \in \mathcal{L}(\mathcal{C}, \mathbb{R}^m)$ and $V(t) \equiv V$. Then, the basic reproduction number is given by*

$$\mathcal{R}_0 = r(V^{-1}\hat{F}) = r(\hat{F}V^{-1}).$$

Therefore, the following proposition holds.

Theorem 3.6. *Let us consider $\mathcal{R}_0 = \frac{p\langle \sigma \rangle}{m} + \frac{p\langle \gamma \rangle}{2m}$. The following results hold:*

1. If $\mathcal{R}_0 < 1$, then, disease free solution $p(t) \equiv 0$ is asymptotically stable.
2. If $\mathcal{R}_0 > 1$, then, solution 0 is unstable and the disease persists.

Proof. : In this case, dropping the symbol $\langle \rangle$ for convenience, linear System 23 can be written as follows,

$$\begin{cases} \dot{x} &= p\beta y(t - \tau) + p\alpha z(t - \tau) - mx(t), \\ \dot{y} &= \frac{\gamma}{2} - \beta y(t), \\ \dot{z} &= \sigma x(t) - \alpha z(t). \end{cases} \quad (27)$$

Let us consider

$$F \begin{pmatrix} \phi_1 \\ \phi_2 \\ \phi_3 \end{pmatrix} = \begin{pmatrix} p\beta\phi_2(-\tau) + p\alpha\phi_3(-\tau) \\ 0 \\ 0 \end{pmatrix} \quad \text{and} \quad V = \begin{pmatrix} m & 0 & 0 \\ -\frac{\gamma}{2} & \beta & 0 \\ -\sigma & 0 & \alpha \end{pmatrix}.$$

Then, System 27 can be written as

$$\frac{du(t)}{dt} = Fu_t - Vu(t), \quad \forall t \geq 0 \quad \text{where} \quad u = (x, y, z)^T. \quad (28)$$

We have

$$F \begin{pmatrix} \phi_1 \\ \phi_2 \\ \phi_3 \end{pmatrix} = \begin{pmatrix} 0 & p\beta & p\alpha \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \begin{pmatrix} \phi_1(-\tau) \\ \phi_2(-\tau) \\ \phi_3(-\tau) \end{pmatrix}.$$

Therefore, using Theorem 3.5, the basic reproduction number is given by $\mathcal{R}_0 = r(\hat{F}V^{-1})$. We have,

$$\hat{F} = \begin{pmatrix} 0 & p\beta & p\alpha \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}, \quad V^{-1} = \begin{pmatrix} \frac{1}{m} & 0 & 0 \\ \frac{\gamma}{2m\beta} & \frac{1}{\beta} & 0 \\ \frac{\sigma}{m\alpha} & 0 & \frac{1}{\alpha} \end{pmatrix}$$

and

$$\mathcal{R}_0 = \frac{p\sigma}{m} + \frac{p\gamma}{2m}.$$

The stability follows by Theorem 3.1 and 3.4. Hence, disease-free solution is asymptotically stable if $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$.

We can observe that, in this case, the threshold \mathcal{R}_0 for the model without delay is equal to \mathcal{R}_0 for the model with delay using time-average parameters. Hence, when the model has constant parameters, the incubation period has no influence on the disease extinction. Therefore, this increases the importance of taking into account the seasonality in the disease incidence.

3.2.4. Disease persistence with time delay

In this section, we show that if $\mathcal{R}_0 < 1$, then the disease free equilibrium $(0, 0, 0)$ is globally asymptotically stable and hence, the disease declines. Next, we show that if $\mathcal{R}_0 > 1$, then $x(t)$, $y(t)$ and $z(t)$ are uniform persistent, and hence the disease persists. Consequently, the basic reproduction number \mathcal{R}_0 is the threshold parameter between the extinction and the uniform persistence of the disease.

Let

$$\Omega = \mathcal{C}([-\tau, 0], \mathbb{R}_+^3), \quad \Omega_0 = \{\phi = (\phi_1, \phi_2, \phi_3) \in \Omega : \phi_i(0) > 0, i = 1, 2, 3\}$$

and

$$\partial\Omega_0 = \Omega/\Omega_0.$$

Let $P(t)$ be the solution maps of System 23 on the space $\mathbb{Y} = \mathcal{C}([-\tau, 0], \mathbb{R}^+)^3$. This is $P(t)\phi = u(t, \phi)$, $t \geq 0$, where $u(t, \phi)$ is the unique solution of System 23 satisfying $u_0 = \phi \in \mathbb{Y}$. Then $P \equiv P(T)$ is the Poincaré map of System 23. Let $r(P)$ be the spectral radius of P . Then we have the following threshold-type result for the persistence of system 7.

Theorem 3.7. 1. *If $\mathcal{R}_0 < 1$, then the disease free equilibrium $(0, 0, 0)$ is globally attractive for system 7 in Ω ,*
2. *If $\mathcal{R}_0 > 1$, then System 7 admits a least one positive T -periodic solution and there exist a positive constant ε such that for all initial condition $(x_0, y_0, z_0) \in \Omega_0$,*

$$\liminf_{t \rightarrow +\infty} (x(t), y(t), z(t)) \geq (\varepsilon, \varepsilon, \varepsilon).$$

Proof. : From Theorem 3.4, we have that $\text{sign}(\mathcal{R}_0 - 1) = \text{sign}(r(P) - 1)$. It follows that for each $t \geq 2\tau$, the linear operator $P(t)$ is compact and strongly positive on \mathbb{Y} ([27], Theorem 3.6.1), [28], Lemma 5.3.2). Let us consider an integer $n_0 > 0$ such that $n_0 T \geq 2\tau$. Since $P^{n_0} = P(n_0 T)$, then $r(P)$ is a simple eigenvalue of P having a strongly positive eigenvector, and the modulus of any other eigenvalue is less than $r(P)$ ([43], Lemma 3.1). Let now $\mu = \frac{1}{T} \ln r(P)$. It follows, from Lemma 1 in [44], that there is positive T -periodic function $v(t)$ such that $u(t) = v(t)e^{\mu t}$ is a positive solution of 23.

In the case $\mathcal{R}_0 < 1$, we have $r(P) < 1$. Since $x(t), y(t), z(t)$ verified the system

$$\begin{cases} \dot{x} & \leq -mx(t) + p\beta(t)y(t - \tau) + p\alpha(t - \tau)z(t), \\ \dot{y} & = \frac{\gamma(t)}{2}x(t) - \beta(t)y(t), \\ \dot{z} & = \sigma(t)x(t) - \alpha(t)z(t), \end{cases} \quad (29)$$

it then by the standard comparison principle, there exists a positive T -periodic function $v(t)$ such that $J(t) \leq v(t)e^{\mu t}$ is a positive solution of 7 with $J(t) = (x(t), y(t), z(t))^T$. Then we see that

$$\lim_{t \rightarrow +\infty} x(t) = \lim_{t \rightarrow +\infty} y(t) = \lim_{t \rightarrow +\infty} z(t) = 0.$$

Hence, the DFE $(0, 0, 0)$ is globally attractive.

For $\mathcal{R}_0 > 1$ then $r(P) > 1$. We rely on the persistence theory for periodic semi-flows.

Let $Q(t)$ be the solution maps of system 7 on Ω . Then $Q := Q(T)$ is the Poincaré map associated with System 7.

We now prove that Q is uniformly persistent with respect to Ω_0 .

Let $M_1 = (0, 0, 0)$. Then $Q(T)M_1 = M_1$ because, M_1 is an equilibrium of System 7. Since $\lim_{\phi \rightarrow M_1} \|Q(t)\phi - M_1\|_1 = 0$ uniformly for $t \in [0, T]$, then for $\eta > 0$, there exists $\eta_1 > 0$ such that for any $\phi \in \Omega_0$ with $\|\phi - M_1\|_1 < \eta_1$, we have

$$\|Q(t)\phi - M_1\|_1 < \eta, \forall t \in [0, T].$$

By a contradiction argument similar to that in proof of Theorem 3.3, we can

further prove the following claim:

$$\limsup_{n \rightarrow \infty} \|Q^n(\phi) - M_1\|_1 \geq \eta_1, \forall \phi \in \Omega_0.$$

This implies that M_1 is an isolated invariant set for Q in Ω and $W^s(M_1) \cap \Omega_0 = \emptyset$ where $W^s(M_1)$ is the stable set of M_1 for Q (see proof of Theorem 3.3).

Let us set

$$M_\partial = \{\phi \in \partial\Omega_0 : Q^n(\phi) \in \partial\Omega_0, \forall n \geq 0\}.$$

We have $M_\partial = M_1$, and M_1 cannot form a cycle for Q in $\partial\Omega_0$. By the acyclicity theorem on uniform persistence for maps ([38], Theorem 1.3.1 and Remark 1.3.1), $Q : \Omega \rightarrow \Omega$ is uniformly persistent with respect to $(\Omega_0, \partial\Omega_0)$. Note that for any integer n with $nT \geq \tau$, $Q^n = Q(nT)$ is compact (see [27], Theorem 3.6.1). Moreover, $Q(t)$ is an α -contraction with respect to an equivalent norm in $\mathcal{C}([-\tau, 0], \mathbb{R}^3)$ for any $t > 0$, where α is the Kuratowski measure of non-compactness ([38], Theorem 3.5.1, [27], Theorem 3.6.1). It then follows from ([45], Theorem 4.5) that there exists a global attractor A_0 for $Q : \Omega_0 \rightarrow \Omega_0$ and Q has a fixed point $\phi^* \in A_0$. Hence, $u(t, \phi^*) = (s^*(t), y^*(t), z^*(t))$ is a T -periodic solution of system 8 with $s^*(t) > 0$, $y^*(t) > 0$ and $z^*(t) > 0$. Since $A_0 = Q(A_0) = Q(T)A_0$, we have $\phi_i(0) > 0, i = 1, 2, 3$.

Let

$$B = \bigcup_{t \in [0, T]} Q(t)A_0.$$

Then $B \subset \Omega_0$, which implies that $\lim_{t \rightarrow +\infty} d(Q(t)\phi, B) = 0, \forall \phi \in \Omega_0$ ([38], Theorem 3.1.1).

Let us define a continuous function $p : \Omega \rightarrow \mathbb{R}^+$ by

$$p(\phi) = \min\{\phi_1(0), \phi_2(0), \phi_3(0)\}, \forall \phi \in \Omega.$$

Since B is a compact subset of Ω_0 , it follows that $\inf_{\phi \in B} p(\phi) = \min_{\phi \in B} p(\phi) > 0$. Consequently, there exists $\varepsilon > 0$ such that

$$\liminf(\min\{x(t, \phi), y(t, \phi), z(t, \phi)\}) = \liminf_{t \rightarrow +\infty} p(Q(t)\phi) > \varepsilon.$$

This ends our proof.

4. Numerical simulations

Most of the parameters are set to realistic values after previous biological research and modelling works in the literature.

We assume the same infection efficiency for both ascospores and conidia: $0.0004 \leq p \leq 0.01853$ [46]. Each week, a single banana plant deploys new leaves that keep their freshness for about 12 weeks without control [17]. The old leaves which fall on the ground after senescence may keep hosting sexual spores while they are not completely disintegrated [47], i.e. for several weeks, giving a range for parameter m . The delay that is the time between infection and the appearance of spots on leaves vary from 11 to 36 days [48, 46] and the cropping season duration vary from 300 to 360 days [49].

Since there are not reliable results in the literature about the relationship between the disease incidence and the temperature, the following T-periodic forms of $\alpha(t)$, $\beta(t)$, $\sigma(t)$ and $\gamma(t)$ are assumed:

$$\begin{aligned}\alpha(t) &= \bar{\alpha}(1 + 0.5 \sin(\frac{2\pi t}{T})), \\ \beta(t) &= \bar{\beta}(1 + 0.5 \sin(\frac{2\pi t}{T})), \\ \sigma(t) &= \bar{\sigma}(1 + 0.9 \sin(\frac{2\pi t}{T})), \\ \gamma(t) &= \bar{\gamma}(1 + 0.75 \sin(\frac{2\pi t}{T})).\end{aligned}$$

We consider the constant initial condition $\varphi(\theta) = (0.2, 0.3, 0.3)$ for all $\theta \in [-\tau, 0]$. Table 2 summarizes the parameter values for the simulation of System 7.

We numerically illustrate the results of theorems 3.2, 3.4 and 3.7, over four cropping seasons. With a constant value of parameter p , we play on the values of parameters $\bar{\sigma}$ and m within their ranges in order to obtain different values of the basic reproduction number around the threshold values $\mathcal{R}_0 = 1$.

- If we set $m = 0.6$ and $\bar{\sigma} = 5$, then the basic reproduction number takes the value $\mathcal{R}_0 = 0.2$. According to Theorem 3.2, the DFE of System 7 is globally asymptotically stable when there is no time delay (Figure 3 (1)).

Parameter	Range	Selected Value	References
p	[0.0004, 0.018]	0.005	[46]
m	[0.002, 0.01]	variable	[17], [47]
$\bar{\gamma}$	[46.4, 350.4]	40	[46], [48] [17], [47]
$\bar{\sigma}$	[2, 120]	variable	[21], [46], [17], [47], [48]
$\bar{\alpha}$	assumed	5	/
$\bar{\beta}$	assumed	20	/
T	[300, 360]	360	[49]
τ	[11, 36]	variable	[48], [46]

Table 2: Parameter values for system 7 simulation

Also, from Theorem 3.7, it remains stable with a time delay; this result is illustrated in Figure 3 (2).

- If we set $m = 0.185$ and $\bar{\sigma} = 10$, then the basic reproduction number takes the value $\mathcal{R}_0 = 0.8$ that is still below 1. The DFE remains stable but the convergence to the DFE is slower since the basic reproduction number is higher (Figure 4).
- If we set $m = 0.125$ and $\bar{\sigma} = 10$, then the basic reproduction number takes the value $\mathcal{R}_0 = 1.2$. According to Theorems 3.2 and 3.3, when there is no time delay, the DFE is unstable and System 7 admits a periodic solution endemic equilibrium that is stable (Figure 5 (1)). Also, from Theorem 3.7, the stable periodic endemic equilibrium still appears with non null values of the time delay. We illustrate this result for two different values $\tau = 15$ (see Figure 5 (2)) and $\tau = 25$ (see Figure 5 (3)) of the time delay τ .
- If we set $m = 0.025$ and $\bar{\sigma} = 20$, then the basic reproduction number takes the value $\mathcal{R}_0 = 8$ that is much higher than 1. According to the same theorems, than previously, the DFE is unstable, and an endemic equilibrium appears and persist with strong oscillations (Figure 6).

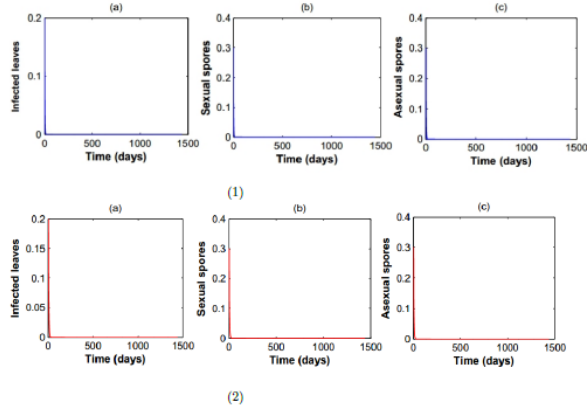


Figure 3: BLS dynamics over 4 cropping seasons: trajectories of Model 7 when the basic reproduction number takes the value $\mathcal{R}_0 = 0.2$. Blocks (a), (b) and (c) describe respectively the densities Infected leaves $x(t)$, densities of Sexual spores $y(t)$ and densities of asexual spores $z(t)$. The sub-figure (1) stands for trajectories without the delay ($\tau = 0$) and the sub-figure (2) describes the trajectories when $\tau = 15$ days. Whether the value of the time delay is $\tau = 0$ or $\tau = 15$, the disease quickly disappears as the basic reproduction number value is far below 1.

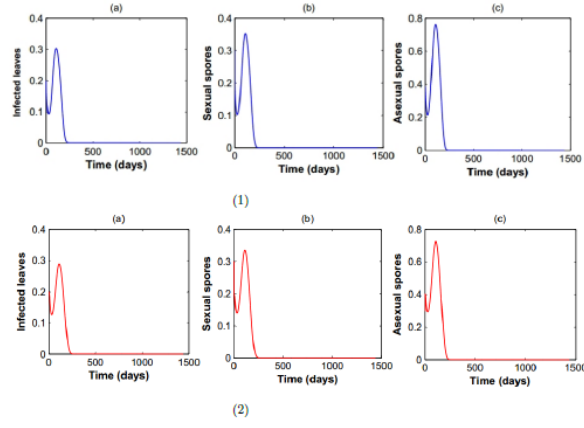


Figure 4: BLS dynamics over 4 cropping seasons: trajectories of Model 7 when the basic reproduction number takes the value $\mathcal{R}_0 = 0.8$. Blocks (a), (b) and (c) describe respectively the densities Infected leaves $x(t)$, densities of Sexual spores $y(t)$ and densities of asexual spores $z(t)$. The sub-figure (1) stands for trajectories without the delay ($\tau = 0$) and the sub-figure (2) describes the trajectories when $\tau = 15$ days. Whether the value of the time delay is $\tau = 0$ or $\tau = 15$, the disease disappears less quickly than in Figure 3 as the basic reproduction number is higher but still below 1.

The figures 3 and 4 illustrate each a scenario in which the basic reproduction number is less than 1. In the case of Figure 3, as \mathcal{R}_0 is far smaller than 1, the disease disappears quickly. In Figure 4, as \mathcal{R}_0 is closer to 1, it takes much time to disappears, and we can observe that the density of infected leaves even increases at a moment before declining to 0.

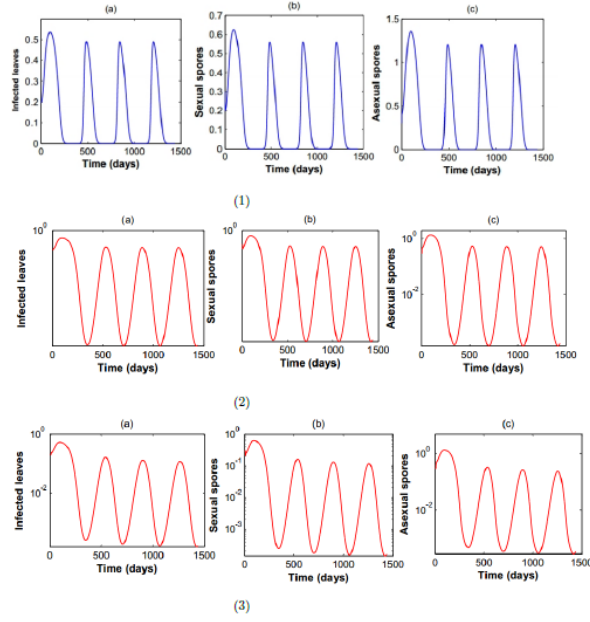


Figure 5: BLSD dynamics over 4 cropping seasons: trajectories of Model 7 when the basic reproduction number takes the value $\mathcal{R}_0 = 1.2$. Blocks (a), (b) and (c) describe respectively the densities Infected leaves $x(t)$, densities of Sexual spores $y(t)$ and densities of asexual spores $z(t)$. The sub-figure (1) stands for trajectories without the delay ($\tau = 0$). The sub-figures (2) and (3) describe the trajectories when $\tau = 15$ days and $\tau = 25$ days respectively, and their y-axis are set to a logarithmic scale to figure out the trajectories since the densities are close to zero but non-zero. Whether the value of the time delay is $\tau = 0$, $\tau = 15$ or $\tau = 25$, the disease persists with periodic oscillation of its trajectories.

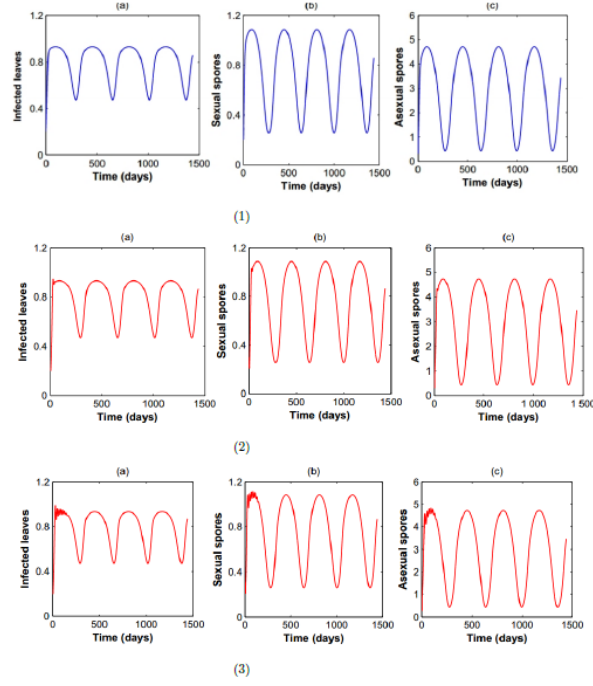


Figure 6: BLS dynamics over 4 cropping seasons: trajectories of Model 7 when the basic reproduction number takes the value $\mathcal{R}_0 = 8$. Blocks (a), (b) and (c) describe respectively the densities Infected leaves $x(t)$, densities of Sexual spores $y(t)$ and densities of asexual spores $z(t)$. The sub-figure (1) stands for trajectories without the delay ($\tau = 0$). The sub-figures (2) and (3) describe the trajectories when $\tau = 15$ days and $\tau = 25$ days respectively. Whether the value of the time delay is $\tau = 0$, $\tau = 15$ or $\tau = 25$, the disease persists with periodic oscillation of its trajectories and the infection reaches higher levels than in Figure 5 as the basic reproduction number value is much larger than 1.

5. Discussion

There are not a lot of studies on the dynamics of BLSD in the literature. Two previous works studied the effect of mate limitation on the dynamics of the disease though [18, 17]. While the first study [18] deals with the spread of the fungus and the second specifically focuses on mate limitation, both have the same outcome: the two reproduction means of *M. Fijiensis* are relevant in the survival and dissemination of the disease. Moreover, the second study especially showed that sexually produced spores could cause sustained oscillations of disease prevalence [17]. Our present study does not contradict these results as, on one hand, our values of \mathcal{R}_0 , whether with or without delay, depend on parameters related to both reproduction means (Theorem 3.2, Proposition 3.6). On the second hand, sustained oscillations also occur in our model because of periodic parameters, namely the sexual and asexual spore production and dispersal rates. These concordances are even more interesting as our study does not take into account either the mate limitation or spatial diffusion, showing the various range of biological mechanisms that can drive oscillations in nature ([50], Chapter 15).

The main focus of the present study was to study the effect of the seasonality of spore production and dispersal as described in the literature [20] and the influence of the disease incubation [20, 23] on the long-term dynamics of BLSD. We showed in Theorems 3.3 and 3.7 that the BLSD may persist with regular oscillations because of the periodicity of some parameters, and depending on the values of the basic reproduction number around the threshold value $\mathcal{R}_0 = 1$. This basic reproduction number, which is the average number of spores produced by an infected leaf during its infection duration, appears to depend on the production rates of spores, and not on their dispersal rates. This is probably because only the previously produced spores spread. Even more surprising, the basic reproduction number of the disease does not depend on the incubation period of the disease, namely the time delay τ we introduced in this work. We indeed proved it and illustrated it in Figures 3–6. This behavior is curious

enough not to appear often in the literature, whether in population dynamics, in human and animal epidemiology, or in plant epidemiology. Though, it may appear in a prey-predator model with infection in the prey population in the case where the infection rate of preys is low [51]. For higher infestation rates the delay, representing the incubation period of the disease, plays a significant role in the stability of the system. The latter behavior appears more commonly in plant epidemics, where for example in vector-borne viral plant diseases, the basic reproduction number depends either on the incubation of the disease in the vector and the incubation of the disease in the plant [52]. In population dynamics, the incubation period of a disease or parasite in a susceptible individual often influence the dynamics [53, 54], or in human epidemiology in AIDS [55] or malaria [56] dynamics for example. Therefore, even if it is uncommon, there is not a great relevance in the case of BLSD to try to control the incubation periods. Figures 3–4 and 5–6 however show that even if the stability of the disease is not influenced by the delay, its quantitative behavior depends on the delay. It is hard to give an analytical description of the oscillations of the periodic solutions in those figures, but a further sensitivity analysis of the system according to the delay may help confirm a very slight but not neglectful influence of the incubation duration [57].

The sexual reproduction function of our model is somehow simplistic. Our findings on the basic reproduction number could be more or less different if we take into account different reproduction functions. Indeed, as *M. fijiensis* reproduces predominantly in a sexual manner, a model taking into account a mate limitation in sexual spore reproduction would make a better job than a model without mate limitation in estimating disease spreading levels [18]. Moreover, taking into account spatial diffusion in dynamical models shows that disease spreads are often correlated with the basic reproduction number. For instance, in multi-patch environments, the basic reproduction number depends on mobility parameters [58]. Also, spatial heterogeneity may produce larger basic reproduction numbers [59]. Hence there are chances that taking into account mate limitation in sexual reproduction may yield a lower basic reproduction

number.

More biological realities need to be incorporated in our model to have a full simulation and comparison with available surveillance data if applicable. Our model, for example, ignores the effects of the spatial distribution of hosts. However, the main prospect of this study was to show that the incubation duration, assumed constant, was not relevant enough. So, future modelling works on the control of the disease should look in a different direction. Though, in realistic environments, the incubation duration also depends on climatic conditions. The time delay should therefore depend on time, which certainly explains why in its current form it does not influence the stability of our dynamics.

While controlling the disease spread, the pathogenic fungus *M. fijiensis* has long been considered as relying mostly on sexual reproduction [47, 48]. Management practices used to rely on the removal of tissues that produce sexual spores. Our present study, as same as previous modelling works in the literature [18, 17], suggests that hampering sexual reproduction is likely not sufficient to fight BLSD. Also, despite its deleterious consequences on plant yields, the removal of wounded tissues should be considered as a complement to achieve the disease's disappearance.

To more rigorously prove the relevance of this control strategy, we plan in future work to build an optimal control model upon the model discussed in this study. Future works may also include spatial availability of hosts to address an issue discussed below, multiple and alternate hosts, and linking the epidemiological model to the crop yield.

6. Conclusion

In this work, we proposed a mathematical pathogen-host model with a time delay for the dynamics of the banana black leaf streak disease. Our model accounted for the two reproduction means of the pathogen spores, seasonality, and a delay to describe the incubation of the disease. We studied the global stability of the disease-free periodic solution and disease persistence. We first studied

the case without delay in which we computed the basic reproduction number \mathcal{R}_0 . We showed that \mathcal{R}_0 is related to both sexual and asexual spore production. We also proved that the disease-free solution was globally asymptotically stable when $\mathcal{R}_0 < 1$ and the disease dies out. Conversely, the disease appears to persist with oscillations when $\mathcal{R}_0 > 1$. Our results proved that the control of sexual spore production is not sufficient. Secondly, we relied on the theory of basic reproduction number for periodic systems, described by Xiao [40], to study the global stability of the disease-free solution and the persistence of the disease with time delay. We showed that stability of the disease-free solution is related to the sign of $\mathcal{R}_0 - 1$ and hence on the relative position around the threshold 1 of the same \mathcal{R}_0 than previously. Thus, the stability of the system was shown to not depend on the delay, i.e. on the duration of the incubation period. We finally provided some numerical simulations to illustrate our results.

References

References

- [1] L. Lassois, J.-P. Busogoro, H. Jijakli, La banane : de son origine à sa commercialisation, *Biotechnol. Agron. Soc. Environ* 13 (4) (2009) 575–586.
- [2] A. Lassoudière, Le bananier et sa culture, Editions Qu, 2007.
- [3] C. Efanden, M. Kwa, L. Temple, D. Foundjem, Production de plantain dans la zone périurbaine de Yaoundé : identification des contraintes et incidences sur la localisation de la zone d’approvisionnement et stratégie d’intervention, in: L. Parrot, A. Njoya, L. Temple, F. Assogba-Komlan, R. Kahane, M. Ba Diao, M. Havard (Eds.), *Actes de l’atelier international “Agricultures et développement urbain en Afrique de l’ouest et du centre”*, Yaoundé, Cameroun, 2005, pp. 109–113.
- [4] A. C. Churchill, *Mycosphaerella fijiensis*, the black leaf streak pathogen of banana: progress towards understanding pathogen biology and detection,

- disease development, and the challenges of control, *Molecular Plant Pathology* 12 (4) (2011) 307–328. doi:10.1111/j.1364-3703.2010.00672.x.
- [5] D. Onautshu Odimba, A. Legrève, B. Dhed'A Djailo, Caractérisation des populations de *Mycosphaerella fijiensis* et épidémiologie de la cercosporiose noire du bananier dans la région de Kisangani, RDC. Sciences du vivant [q-bio], Ph.D. thesis, Université Catholique de Louvain, Français (2013).
- [6] F. P. Castelan, L. A. Saraiva, F. Lange, L. d. L. de Bellaire, B. R. Corde-nunsi, M. Chillet, Effects of black leaf streak disease and sigatoka disease on fruit quality and maturation process of bananas produced in the sub-tropical conditions of southern Brazil, *Crop Protection* 35 (2012) 127–131. doi:10.1016/j.cropro.2011.08.002.
- [7] X. Mourichon, Analyse du risque phytosanitaire (arp), CIRAD:http://agriculture.gouv.fr/IMG/pdf/BAN/-c2.pdf.
- [8] J. Henderson, J. Pattemore, S. Porchun, H. Hayden, S. Van Brunshot, K. Grice, R. Peterson, S. Thomas Hall, E. Aitken, Black sigatoka disease: new technologies to strengthen eradication strategies in Australia, *Australasian Plant Pathology* 35 (2) (2006) 181–193. doi:10.1071/AP06017.
- [9] N. J. Cunniffe, B. Koskella, C. J. E. Metcalf, S. Parnell, T. R. Gottwald, C. A. Gilligan, Thirteen challenges in modelling plant diseases, *Epidemics* 10 (2015) 6–10. doi:10.1016/j.epidem.2014.06.002.
- [10] L. V. Madden, F. Van Den Bosch, A population dynamics approach to assess the threat of plant pathogens as biological weapons against annual crops: Using a couple differential-equation model , we show the conditions necessary for long-term persistence of a plant disease after a pathogenic microorganism is introduced into a susceptible annual crop, *BioScience* 52 (1) (2002) 65–74. doi:10.1641/0006-3568(2002)052[0065:APDATA]2.0.CO;2.

- [11] L. V. Madden, M. J. Jeger, F. van den Bosch, A theoretical assessment of the effects of vector-virus transmission mechanism on plant virus disease epidemics, *Phytopathology* 90 (6) (2000) 576–594. doi:10.1094/PHYTO.2000.90.6.576.
- [12] R. Shi, H. Zhao, S. Tang, Global dynamic analysis of a vector-borne plant disease model, *Difference Equations* 2014 (1) (2014) 59. doi:10.1186/1687-1847-2014-59.
- [13] J. Jeger, M. P. Jeffries, Y. Elad, M. Xu, X. A generic theoretical model for biological control of foliar plant diseases, *Journal of Theoretical Biology* 56 (2) (2009) 201–14. doi:10.1016/j.jtbi.2008.09.036.
- [14] R. Anguelov, J. Lubuma, Y. Dumont, Mathematical analysis of vector-borne diseases on plants, in: 2012 IEEE 4th International Symposium on Plant Growth Modeling, Simulation, Visualization and Application, IEEE, 2012, pp. 22–29. doi:10.1109/PMA.2012.6524808.
- [15] I. Tankam-Chedjou, S. Touzeau, L. Mailleret, J. J. Tewa, G. Frédéric, Modelling and control of a banana soilborne pest in a multi-seasonal framework, *Mathematical Biosciences* 322 (2020) 108324. doi:10.1016/j.mbs.2020.108324.
- [16] L. Mailleret, M. Castel, J. Montarry, F. M. Hamelin, From elaborate to compact seasonal plant epidemic models and back: is competitive exclusion in the details?, *Theoretical ecology* 5 (3) (2012) 311–324. doi:10.1007/s12080-011-0126-0.
- [17] V. Ravigné, V. Lemesle, A. Walter, L. Mailleret, F. Hamelin, Mate limitation in fungal plant parasites can lead to cyclic epidemics in perennial host populations, *Bulletin of Mathematical Biology* 79 (3) (2017) 430–447. doi:10.1007/s11538-016-0240-7.
- [18] F. M. Hamelin, F. Castella, V. Doli, B. Marçais, R. Virginie, A. Lewis, Mark, Mate finding, sexual spore production, and the spread of fungal

- plant parasites, *Society for Mathematical Biology* 78 (4) (2016) 695–712. doi:10.1007/s11538-016-0157-1.
- [19] M. E. Hochberg, R. D. Holt, The coexistence of competing parasites. I. The role of cross-species infection, *American Naturalist*, 1990, pp. 517–541.
- [20] J. Ngando Essoh Otto, Sélection et évolution de la rsistance aux fongicides systémiques chez *mycosphaerella fijiensis* agent causal de la maladie des raies noires des bananiers. thèse de doctorat, Ph.D. thesis, Centre international d’études supérieures en sciences agronomiques; Montpellier (2014).
- [21] S. Robert, Emergence mondiale de la maladie des raies noires du bananier: histoire de l’invasion et stratégie de vie du champignon phytopathogène *Mycosphaerella fijiensis*, Ph.D. thesis, Université de Montpellier 2 ((2012)).
- [22] M. Jeger, Analysis of disease progress as a basis for evaluating disease management practice, *Ann. Rev. Phytopathol.* 42 (2004) 61–82. doi:10.1146/annurev.phyto.42.040803.140427.
- [23] E. Fouré, A. Moreau, Contibution l’tude pidmiologique de la cercosporiose noire dans la zone bananiaire du mungo de 1987 1989, *Fruits* 47 (1) (1992) 3–16.
- [24] A. F. Amil, S. P. Heaney, C. Stanger, M. W. Shaw, Dynamics of QoI sensitivity in *Mycosphaerella fijiensis* in Costa Rica during 2000 to 2003, *Phytopathology* 97 (11) (2007) 1451–1457. doi:10.1094/phyto-97-11-1451.
- [25] F. Gauhl, C. Pasberg-Gauhl, D. R. Jones, Black leaf streak: disease cycle and epidemiology, in: *Diseases of Banana, Abac and Enset*, New York, NY:CABI, 2000, pp. 56–62.
- [26] K. Hadeler, Pair formation, *Journal of Mathematical Biology* 64 (4) (2012) 613–645. doi:10.1007/s00285-011-0454-0.
- [27] J. K. Hale, S. M. Verduyn Lunel, *Introduction to Functional Differential Equations*, Springer, New York, 1993.

- [28] H. L. Smith, Monotone dynamical systems: An introduction to the theory of competitive and cooperative systems, *Bulletin of the American Mathematical Society* 33 (1996) 203–209.
- [29] C. Chicone, Ordinary differential equations with applications, Vol. 34, Springer, 1999.
- [30] P. Hartman, Ordinary differential equations, Vol. 38, SIAM. Wiley, New York (NY), 2002.
- [31] J. K. Hale, Ordinary differential equations, Vol. XXI, Wiley-Interscience, New York(NY), 1969.
- [32] F. Zhang, X.-Q. Zhao, A periodic epidemic model in a patchy environment, *J. Math. Anal. Appl* 325 (1) (2007) 496–516. doi:10.1016/j.jmaa.2006.01.085.
- [33] S. Gao, Y. Liu, J. Nieto, H. Andrade, Seasonality and mixed vaccination strategy in an epidemic model with vertical transmission, *Mathematics and Computers in Simulation* 81 (9) (2011) 1855–1868. doi:10.1016/j.matcom.2010.10.032.
- [34] N. C. Grassly, C. Fraser, Seasonal infectious epidemiology, *Proceedings of the Royal Society B* 273 (1600) (2006) 2541–2550. doi:10.1098/rspb.2006.3604.
- [35] M. J. Keeling, P. Rohani, B. T. Grenfell, Seasonally forced disease dynamics explored as switching between attractors, *Physica* 148 (3–4) (2001) 317–335. doi:10.1016/s0167-2789(00)00187-1.
- [36] J. Ma, Z. Ma, Epidemic threshold conditions for seasonally forced seir models, *Mathematical Biosciences & Engineering* 3 (1) (2006) 161, doi:10.3934/mbe.2006.3.161.
- [37] C. Mitchell, C. Kribs, A comparison of methods for calculating the basic reproductive number for periodic epidemic systems, *Bulletin of Mathematical Biology* 79 (2017) 1846–1869. doi:10.1007/s11538-017-0309-y.

- [38] X.-Q. Zhao, *Dynamical Systems in Population Biology*, second edition Edition, Springer, 2017. doi:10.1007/978-3-319-56433-3.
- [39] E. Ávila-Vales, E. Rivero-Esquivel, G. E. García-Almeida, Global dynamics of a periodic seirs model with general incidence rate, *International Journal of Differential Equations* 2017 (2017) 14 pages. doi:10.1155/2017/5796958.
- [40] X.-Q. Zhao, Basic reproduction ratios for periodic compartmental models with time delay, *J. Dyn. Diff. Equat.* 29 (1) (2017) 67–82. doi:10.1007/s10884-015-9425-2.
- [41] D. Posny, J. Wang, Computing the basic reproductive numbers for epidemiological models in nonhomogeneous environments, *Appl. Math. Comput* 242 (2014) 473–490. doi:10.1016/j.amc.2014.05.079.
- [42] N. Bacaër, S. Guernaoui, The epidemic threshold of vector-borne diseases with seasonality, *Journal of mathematical biology* 53 (3) (2006) 421–436. doi:10.1007/s00285-006-0015-0.
- [43] X. Liang, X.-Q. Zhao, Asymptotic speeds of spread and traveling waves for monotone semiflows with applications, *Communications on Pure and Applied Mathematics* 60 (1) (2007) 1–40. doi:10.1002/cpa.20154.
- [44] W. Xiunan, Z. Xiao-Qiang, Dynamics of a time-delayed lyme disease model with seasonality, *Journal of Applied Dynamical Systems* 15 (2) (2017) 853–881. doi:10.1137/16M1087916.
- [45] P. Magal, X.-Q. Zhao, Global attractors and steady states for uniformly persistent dynamical systems, *SIAM journal on mathematical analysis* 37 (1) (2005) 251–275. doi:10.1137/S0036141003439173.
- [46] L. Clara, *Modélisation des dynamiques de maladies foliaires de cultures pérennes tropicales différentes échelles spatiales : cas de la cercosporiose noire du bananier*. phd thesis, Ph.D. thesis, Université des Antilles (2015).

- [47] R. Stover, Sigatoka leaf spots of bananas, *Plant Disease* 64 (8) (1980) 751.
- [48] E. Fouré, Les cercosporioses du bananier et leurs traitements. comportement des varités. 1: Incubation et évolution de la maladie, *Fruits* 37 (12) (1982) 749–766.
- [49] Banana cultivation guide, accessed 23 March 2018 (2018).
URL http://mahaprison.gov.in/Uploads/Dockets_Files/635259935664912504Banana_Cultivation_Guide_%C2%AB_Banana_Planters.pdf
- [50] P. Turchin, *Complex population dynamics: a theoretical/empirical synthesis*, Vol. 35, Princeton university press, 2003.
- [51] N. Bairagi, R. R. Sarkar, J. Chattopadhyay, Impacts of incubation delay on the dynamics of an eco-epidemiological system: a theoretical study, *Bulletin of mathematical biology* 70 (7) (2008) 2017. doi:10.1007/s11538-008-9337-y.
- [52] F. Al Basir, S. Adhurya, M. Banerjee, E. Venturino, S. Ray, Modelling the effect of incubation and latent periods on the dynamics of vector-borne plant viral diseases, *Bulletin of Mathematical Biology* 82 (7) (2020) 1–22. doi:10.1007/s11538-020-00767-2.
- [53] C. Liu, W. Yue, P. Liu, Dynamical behavior and stability analysis in a hybrid epidemiological-economic model with incubation, in: *Abstract and Applied Analysis*, Vol. 2014, Hindawi, 2014, p. 22. doi:10.1155/2014/639405.
- [54] B. G. Sampath Aruna Pradeep, W. Ma, Z. Jiang, Mathematical analysis of the effect of cuckoo birds incubation period in population dynamics, *Applied Mathematical Modelling* 40 (23) (2016) 10167 – 10180. doi:10.1016/j.apm.2016.06.048.
- [55] Z. Mukandavire, W. Garira, J. M. Tchuente, Modelling effects of public health educational campaigns on hiv/aids transmission dynamics, *Applied*

- Mathematical Modelling 33 (4) (2009) 2084 – 2095. doi:10.1016/j.apm.2008.05.017.
- [56] K. Nah, Y. Nakata, G. Rst, Malaria dynamics with long incubation period in hosts, Computers & Mathematics with Applications 68 (9) (2014) 915 – 930, bIOMATH 2013. doi:10.1016/j.camwa.2014.05.001.
- [57] M. Koda, Sensitivity analysis of time-delay systems, International Journal of Systems Science 12 (11) (1981) 1389–1397. doi:10.1080/00207728108963825.
- [58] J. Arino, J. R. Davis, D. Hartley, R. Jordan, J. M. Miller, P. van den Driessche, A multi-species epidemic model with spatial dynamics, Mathematical Medicine and Biology: A Journal of the IMA 22 (2) (2005) 129–142. arXiv:<https://academic.oup.com/imammb/article-pdf/22/2/129/2312764/dqi003.pdf>, doi:10.1093/imammb/dqi003.
- [59] W. Wang, X.-Q. Zhao, Basic reproduction numbers for reaction-diffusion epidemic models, SIAM Journal on Applied Dynamical Systems 11 (4) (2012) 1652–1673. arXiv:<https://doi.org/10.1137/120872942>, doi:10.1137/120872942.

Highlights

- We proposed a mathematical pathogen-host model with a time delay for the dynamics of the banana black leaf streak disease
- Model accounted for the two reproduction means of the pathogen spores, seasonality and time delay to describe the incubation
- The basic reproduction number R_0 does not depend on the time delay and is related to both sexual and asexual spore production
- The stability of the system was shown to not depend on the time delay, i.e. on the duration of the incubation period
- Results proved that the control of sexual spore production is not sufficient. We provide numerical simulations