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# Modelling of Infection Mildew of Taro (*Phytophthora colocasiae*)

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**Abstract:** Mildew taro caused by *Phytophthora colocasiae* affection is the most devastating of taro cultivation in Cameroon since 2010. It has been studied in leading the influence that can have a parameter considered favourable in the kinetics of the disease, and secondly, the interaction between plots through zoospores that can move from one field to another while estimating their dispersal throughout the plant. These models have allowed us to demonstrate that the duration of pathogen latency period, the number of sporangia produced on the surface of a lesion as well as the severity of the infection taken individually, are parameters to be taken into account in the development of a variety resistant to late blight taro. The dynamics of the fungus over time is represented by a matrix. The latter was used to establish a detailed estimate of the number of new infections caused by a sporangium placed in a landscape of healthy leaves. This number is known as the net rate of breeding base name ( $R_0$ ). The incidence and severity of disease are significantly reduced when the rate is less than or equal to one. So our approach can be used to guide research programs or evaluate the effectiveness of control strategies to design throughout the plant.

**Keywords:** *Phytophthora colocasiae*, Taro, Modelling, Simulation

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## 1. Introduction

Taro (*Colocasia esculenta* (L.) Schott) is a monocotyledonous plant belonging to the *Araceae* family [1]. This plant about two meters high is from Asia. Originally from Malaysia, its culture developed to Polynesia where does it taro name [2] and is now cultivated in all tropical regions. Its spread has been almost universal in tropical Asia and even Japan before reaching Arabia and the Mediterranean basin at the beginning of our era and Tropical Africa later [3]. Hence, this plant was introduced to the Caribbean with African slaves.

This is an important staple food and subsistence for millions people in the countries of Africa, Asia and Central America [4]. Its worldwide production is estimated at 12 million tons on a cultivated area of 2 million hectares [5, 6].

According to IITA [7], 77% of the world taro production comes from sub-Saharan Africa. This is the fourteenth most consumed vegetable in the world [7]. In 2012, global production was estimated at 10 million tons. Cameroon was the fourth largest producer of taro and the third largest producer in Africa after Nigeria and Ghana with a production of 1.6 million to 7 million tons for all of Africa [8, 9, 10, 5]. Taro is grown in all regions of Cameroon for its leaves and tubers that have good nutritional qualities (easily digestible starches, vitamin C). It occupies an important place in traditional ceremonies in some African and Asian populations [12, 13, 14]. Tubers and taro leaves also have medicinal properties against tuberculosis, ulcers, pulmonary congestion and fungal infections [15, 16]. The easily digestible starch tuber is an excellent food for diabetics [17]. A paradigm was observed in Japan and Egypt where taro has been used

satisfactorily as a first harvest in the rehabilitation of saline soils [18]. This certainly opens the possibility of using the taro to subtract some ecological constraints.

Like humans and animals, plants must cope with new diseases. These diseases are currently one of the major threats to public health, sustainable agriculture, and food and ecosystem conservation [19, 20]. The current environment is more conducive than ever to the appearance of new diseases: changes associated with globalization, especially increased trade and intercontinental economic climate modification, have important implications for the evolution of pathogens and dispersion of infectious agents [19, 21]. Thus in Cameroon since 2010, growing taro suffer of an epidemic disease; mildew caused by *P. colocasiae* [22]. This disease has reduced by over 70% taro production during the last three years in Cameroon (currently it is completely absent on the shelves in the markets) [23]. Although the impact of this disease on the population remains to be assessed, experience has shown that it can affect remarkably farmers' incomes and food security in Cameroon. Conventional modes of managements of this fungus that are far, widely used in agriculture require the use of phytosanitary products in large quantities. At the same time, measures are taken globally to encourage the farming community to limit its use. The avowed aim is to make all crops less dependent on fungicides to meet the ecological balance and preserve the environment. Thus, several measures have been taken as Ecophyto 2018 resulting from the Grenelle Environment Forum, the Framework Directive 2000 water or eco-conditionality of aid for the future CAP 2013 to comply with mandatory minimum standards. In addition, the massive and uncontrolled use of systemic fungicides has led to the birth *P. colocasiae* isolates resistant to these active ingredients. However, the prophylactic struggle and sanitation modes can represent popular among farmers because considered less effective. As for resistant cultivars, they are certainly as a durable solution in most taro production systems because the best control option is the spread and use of resistant cultivars [24]. Indeed, Cameroon, the fourth largest producer in 2010 with 1.6 million annual tons or about 13% of world production has since then losses ranging from 70 to 77%. This general observation demonstrates the need to provide solutions to farmers, firstly to better help they target their phytosanitary treatment in order to respect the environment and secondly to better explore the favourable traits for selection taro varieties; because according to Tansao [25], there is a high genetic variability of taro within and between countries. Any time dynamics of disease understanding and ability to evolve requires the study of the problem in its global dimension and must take into account the complex interactions between three key determinants: the characteristics of the pathogen, sensitivity of the host and the environment. It is in this light that we have created model taro blight in order to understand the evolution of this disease in one or more plots of taro to stem or stop this emerging epidemic in Central and West Africa.

In order to predict the attacks of *P. colocasiae*, we will

propose a temporal model that will allow for a clarification on the level of infectious lesions may appear on taro leaves before the development of tubers depending estimated parameters. The efficiency of infection of the zoospore and sporangium, defined as the probability of penetration of a zoospore or sporangium lying on a healthy leaf. Knowing of course that the success of infection depends on the number of sporangia produced by infectious lesion also called infectivity of the pathogen, the latency period, the number of gendered spores or oospores produced by infectious lesion. The aim of our study is to develop a mathematical model to use as strategic as planned order. A computer program then will simulate the state of evolution of the number of infectious lesions that represents the intensity of the disease in a secluded plot. From the scientific point of view, the construction of such a mathematical model in plant pathology will generate the information necessary to learn about the operation of the host-parasite relationship, calculate the evolution of the intensity of the disease on taro leaves. This study will also contribute to help, for laboratory experiments on the development of *P. colocasiae* by varying the parameters related to this pathogen.

## 2. Material and Methods

### 2.1. Material

#### 2.1.1. Biological Model

The term model has become very usual, both in research circles in crop protection, than in the agricultural council [26]. A model is to glue a mathematical theory on a piece of reality [27]. He tries to explain the functioning of a system through mathematical equations that relate the input and output components of the system [28]. This trend of modelling shows the determination of scientists trying to understand the dynamics of complex systems that involve the various processes. In plant pathology, models are built from key components of the development of the pathogen and the host-pathogen interaction [29]. Thus, for the parasitic plants fungi, the wetness is for example often paramount, because it affects spore germination and penetration of the pathogen into the host [30]. Accordingly, for epidemiological models in plant pathology, it is essential to integrate meteorological parameters in the data entering the system to predict the risk of infection [26].

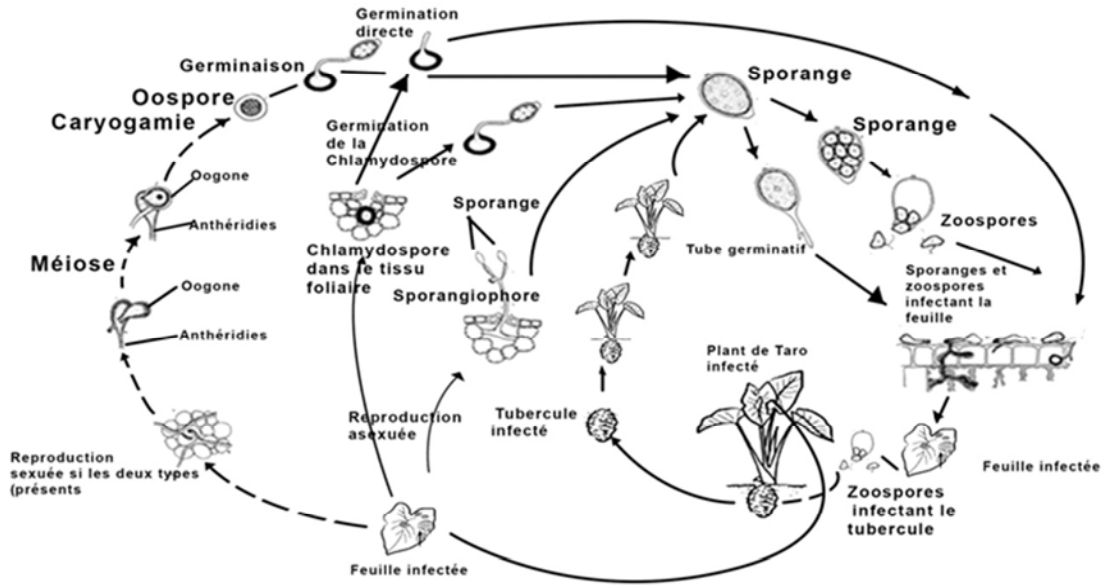
Epidemiology is for two interacting populations: the population of the host and the parasite [29]. Thus, in our case, are used simultaneously to two levels of integration: the leaves, representing the host, and lesions, representing the parasite. The model uses a series of variables that at a given time can in principle be measured to describe the dynamics of the system. It is here the number of healthy leaves (H), the number of latent lesions (L), the number of infectious lesions (I), the number of sporangia (Sp), and the number of zoospores (Zp) present in the system and the proportion of oospores (O) products in the system. Particular attention was paid to the flexibility of this new model so that it can easily

subject to change at the discretion of advanced research.

**2.1.2. Biological and Data-Processing Material**

The first mathematical model suggested in this work seek coupling the vegetative growth of the seedlings of taro and cycles it life of *P. colocasiae*, we have for this reason drawn using the software (illustrator) a cycle of probable life (Fig. 1) of this fungus, that of the taro being known the latter we

will be used as biological material. Computer equipment consists of a portable computer of mark DELL of 2Go of RAM, with triple processor functioning with an operating system Windows 10; virtual machine JAVA version 7.0.450. Software Latex for the seizure, of the compiler winEdit and the software Scilab version 2008 for the digital simulation.



*Fig. 1. Cycle life of P. colocasiae.*

**2.1.3. Inputs and Outputs of the Model**

Build a model requires understanding the conditions under which it can be used. The methods of measurement and observation of model inputs should be generalizable, easy to implement and especially inexpensive. The model runs on a daily time step, but it is important to note that the IT point of view, it was designed to accommodate any changes in no time calculation. It uses data measured: the latency period, lesion size, the efficiency of infection of zoospores and sporangia.

As output, the model gives the incidence or severity of the disease on the leaves. This data will be faced with field observations to validate the calculations. This will also help to develop the favourable traits for plant breeding in order to decide that range set up to advise farmers.

**2.2 Methods**

**2.2.1. Model Construction**

The dynamics of *P. colocasiae* is governed by the following set of biological assumptions: (i) units of dissemination of *P. colocasiae* is the sporangia and the zoospore; (ii) its support of infection is a healthy sheet of *C. esculenta*; (iii) the sporangia can be to infect directly that is to say to produce  $n_1$  zoospore to increase the infections; (iv) a lesion is latent during a certain period P then becomes infectious; (v) an infectious lesion produced  $n_2$  sporangia per day and can produce  $n_3$  oospores when the conditions are

unfavourable; (vi) daily, d number of infectious lesions stops the sporulation and (vii) m healthy or latent sheets dies under the blow of the bad weather. We consider six distinct population, according to their disease status: likely sheets that one notes H (sheets which never was in contact with a sporangia or a zoospore including the young sheets), the latent ones that one notes by L (healthy sheets which were in contact with the units of contaminations of fungus but do not develop the disease immediately because the units of contamination (sporangia and zoospore) inside the healthy sheet carry out a latent life), infectious noted the I (infected sheets which already started to produce sporangia and which is able to transmit the disease), the sporangia or contaminant primary educations which we note Sp (represent the first units of dissemination of fungus through the culture and are carried per thousands with stroke of an infectious lesion), the noted zoospore Zp which are produced by the sporangia when the conditions are favourable, the oospores or spores sexual that we noted by O are those produced when the living conditions are unfavourable and which are close initiating the first infections when these conditions become favourable. The sheets of taro are produced with an intrinsic growth rate  $\epsilon > 0$  of which the number of sheets will not exceed the capacity limits K that a foot of taro can have, i.e., the healthy sheets are produced according to the logistic law:

$$\epsilon \left(1 - \frac{H}{K}\right) H.$$

The contamination being air mainly, the units of contamination are transported by the wind and the splashes of rains, we modelled the contact of susceptible (healthy sheet) with infectious (sporangia and zoospore) by the law of action of standard mass for a population with not constant dynamics what satisfies:

$$\left( \frac{\beta_1 Z_P}{Z_P + D_1} + \frac{\beta_2 S_P}{S_P + D_2} \right).$$

Where:  $\beta_1 = e_1 g_1$  represents the force of infection of the zoospore;  $\beta_2 = e_2 g_2$  represents the force of infection of the

sporangia;  $D_1$  proportion of zoospore which gives a force of infection of 50% and  $D_2$  proportion of sporangia which gives a force of infection of 50%.

The natural mortalities occur at constant rate noted ( $m$ ), but the sick sheets have an additional mortality which one notes ( $d$ ) due to fungus.

A schematic model flowchart is depicted in (Fig. 2).

The dynamics of *P. colocasiae*, in a homogeneous population, is then described by the following system of nonlinear differential equations:

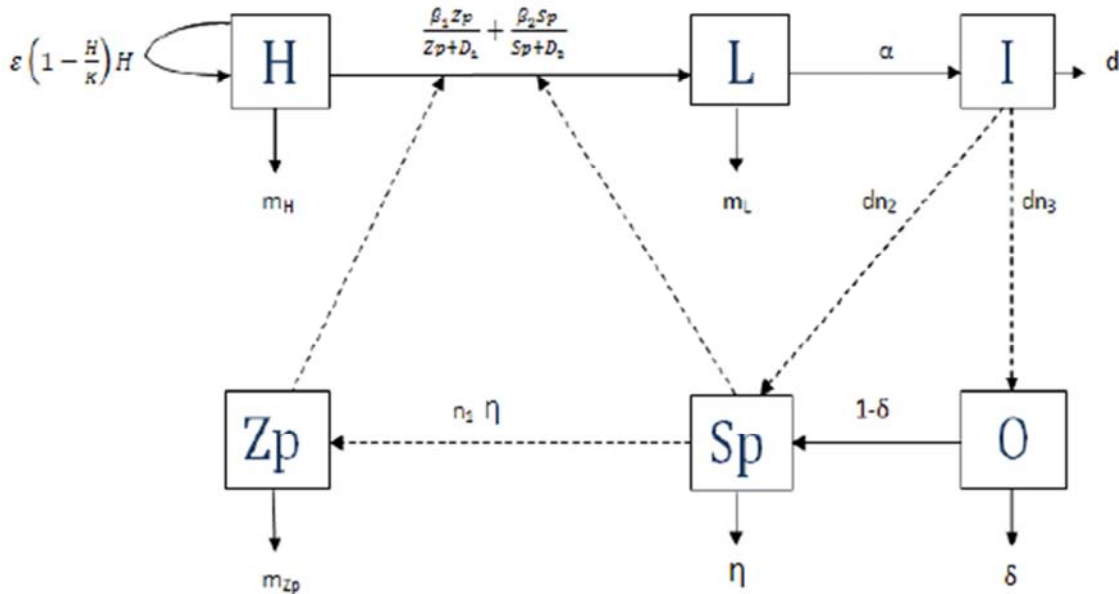


Fig. 2. Structure of the model.

$$\begin{cases} H' = \varepsilon \left(1 - \frac{H}{K}\right) H - \left(\frac{\beta_1 Z_P}{Z_P + D_1} + \frac{\beta_2 S_P}{S_P + D_2}\right) H - m_H H; \\ L' = \left(\frac{\beta_1 Z_P}{Z_P + D_1} + \frac{\beta_2 S_P}{S_P + D_2}\right) H - (\alpha + m_L) L; \\ I' = \alpha L - d I; \\ O' = d n_3 I - (\psi + \delta) O; \\ S_P' = \psi O + d n_2 I - \eta S_P; \\ Z_P' = n_1 \eta S_P - m_{Z_P} Z_P; \end{cases} \quad (1)$$

Where

$$\psi = (1 - \delta). \quad (2)$$

Table 1. Variables and parameters with units for model system (1).

Symbols	Description	Units
$g_1$	Proportion of zoospore able to germinate on a healthy sheet	
$g_2$	Proportion of sporangia able to germinate on a healthy sheet	
$e_1$	Effectiveness of infection of zoospore or severity	1/day
$e_2$	Effectiveness of infection of the sporangia	1/day
$n_1$	Quantity of zoospore released by a sporangia	50/day

Symbols	Description	Units
$n_2$	Quantity of sporangia produced by a lesion infectious	300000/day
$n_3$	Proportion of oospores produced by an infectious lesion	
$\eta$	Quantity of sporangia which releases from the zoospore	
$m_H$	Life expectancy (Longevity) of the healthy sheets	40 days approximately
$m_L$	Life expectancy (Longevity) of the sheets infected	10 to 20 days
$m_{Z_P}$	Rate of Zoospore washed on the sheets	
$d$	Quantity of infectious lesions who stop the sporulation per day	
$K$	Number of sheets which a seedling of taro can to have	6 to 7 sheets
$\alpha$	Duration of the latency period of fungus	24h to 18 days
$D_1$	Proportion of zoospore which gives a force of infection of 50%	
$D_2$	Proportion of sporangia which gives a force of infection of 50%.	
$\varepsilon$	Birth rate of the sheets	Day
$\delta$	Proportion of oospores washed on the sheets	

### 2.2.2 Basic Properties

Herein, we study the basic properties of the solutions of

model system (1), which are essential

in the proof of the stability result. We have the following result.

Theorem 1: Model system (3) is a dynamical system on the biologically feasible compact domain:

Proof: The proof is provided in two steps.

Step 1: We show that the solution  $(H(t); L(t); I(t); Sp(t); Zp(t); O(t))$  of model system (1)

corresponding to initial conditions such that  $H(0) > 0$ ;  $L(0) > 0$ ;  $I(0) > 0$ ;  $Sp(0) > 0$ ;  $Zp(0) > 0$  and  $O(0) > 0$  are not negative. Define

$$\tau = \sup \{s > 0, /H(s) > 0, L(s) > 0, I(s) > 0, O(s) > 0, Sp(s) > 0, Zp(s) > 0\} \in [0, t].$$

If  $\tau = \infty$ , then, all the solutions of model system (1) are positive.

Let us apply the formula of Duhamel to prove the positivity of each equation of model system

(1):

$$\begin{aligned} H(\tau) \geq & H(0) \exp \left[ - \int_0^\tau \frac{\beta_1 Z_P(s)}{Z_P(s) + D_1} + \frac{\beta_2 S_P(s)}{S_P(s) + D_2} + m_H \right. \\ & \left. - \varepsilon \left( 1 - \frac{H(s)}{K} \right) \right] d_s \exp \left[ \int_0^\tau \left( \frac{\beta_1 Z_P(v)}{Z_P(v) + D_1} \right. \right. \\ & \left. \left. + \frac{\beta_2 S_P(v)}{S_P(v) + D_2} + m_H \right. \right. \\ & \left. \left. - \varepsilon \left( 1 - \frac{H(v)}{K} \right) d_v \right) \right] d_s > 0; \end{aligned}$$

I.e.  $H(t) > 0$  for any  $t > 0$ .

In the same way, it is shown that

$$\begin{aligned} L(\tau) \geq & L(0) \exp \left( - \int_0^\tau \alpha + m_L \right) d_s \\ & + \left( \int_0^\tau \frac{\beta_1 Z_P(s)}{Z_P(s) + D_1} \right. \\ & \left. + \frac{\beta_2 S_P(s)}{S_P(s) + D_2} \right) \exp \left( \int_0^s (\alpha + m_L) d_v \right) d_s \\ & > 0; \end{aligned}$$

Thus,  $L(t) > 0$  for any  $t > 0$ .

Third equation of the model system (2), it comes that

$$\begin{aligned} I(\tau) \geq & I(0) \exp \left( - \int_0^\tau d \right) d_s + \int_0^\tau \alpha L(s) \exp \left( \int_0^s d \right) d_v d_s \\ & > 0; \end{aligned}$$

From there, one has  $I(t) > 0$  for any  $t > 0$ .

$$\begin{aligned} O(\tau) \geq & O(0) \exp \left( - \int_0^\tau \psi + \delta \right) d_s \\ & + \int_0^\tau dn_3 I(s) \exp \left( \int_0^s \psi \right) d_v d_s > 0; \end{aligned}$$

It comes whereas,  $O(t) > 0$  for any  $t > 0$ .

Fifth equation of the model system (2) one has

$$\begin{aligned} S_P(\tau) \geq & S_P(0) \exp \left( - \int_0^\tau \eta \right) d_s + \int_0^\tau \psi O(s) \\ & + dn_2 I(s) \exp \left( \int_0^s \eta d_v \right) d_s > 0; \end{aligned}$$

One can then conclude that  $S_P(t) > 0$  for any  $t > 0$ .

Lastly, of the last equation of the model system (2), one has

$$\begin{aligned} Z_P(\tau) \geq & Z_P(0) \exp \left( - \int_0^\tau m_{Z_P} \right) d_s \\ & + \int_0^\tau \eta n_1 S_P(s) \exp \left( \int_0^s m_{Z_P} d_v \right) d_s > 0; \end{aligned}$$

I.e.  $Z_P(t) > 0$  for any  $t > 0$ ; this completes the proof.

Step 2: It is proven that the model system (1) admits a single maximum solution.

Let us pose  $X(t) = (H(t); L(t); I(t); O(t); Sp(t); Zp(t))$  and  $f$  a function of class  $C^\infty$  of  $\mathbb{R}_+^6$  in  $\mathbb{R}^6$  defined by:

$$\begin{aligned} & f(X(t)) \\ = & \begin{pmatrix} \varepsilon \left( 1 - \frac{H}{K} \right) H - \left( \frac{\beta_1 Z_P}{Z_P + D_1} + \frac{\beta_2 S_P}{S_P + D_2} \right) H - m_H H \\ \left( \frac{\beta_1 Z_P}{Z_P + D_1} + \frac{\beta_2 S_P}{S_P + D_2} \right) H - (\alpha + m_L) L \\ \alpha L - dI \\ dn_3 I - (\psi + \delta) O \\ \psi O + dn_2 I - \eta S_P \\ n_1 \eta S_P - m_{Z_P} Z_P \end{pmatrix} \end{aligned}$$

The function  $f(X(t))$  being infinitely differentiable on  $\mathbb{R}_+^6$ , it is consequently locally Lipschitzienne.

One considers the problem of following Cauchy:

$$\begin{cases} X'(t) = f(X(t)); \\ X(t_0) = X_0 \in \mathbb{R}_+ \times \mathbb{R}_+^6 \end{cases}$$

Since  $f$  is of class  $C^\infty$  then; it admits a single maximum solution. Combining Step 1 and Step 2, Theorem 1 follows from the classical theory of dynamical systems. This concludes the proof.

### 2.2.3. The Disease-Free Equilibrium (DFE) and Its Stability

The disease-free equilibrium for model system (1) is given by

$$X_0 = (H_0, 0, 0, 0, 0). \tag{3}$$

Where

$$H_0 = \frac{K(\epsilon - m_H)}{\epsilon}$$

Then that  $H_0 > 0$ , this point exist if and only if  $\epsilon > m_H$ .

In order to investigate the stability properties of the disease-free equilibrium  $X_0$ , we need to compute the basic reproduction or threshold number  $R_0$  of model system (1). To this end, we define the following Jacobins matrix:

$$J(X_0) = \begin{pmatrix} \epsilon - \frac{2H^*}{K} - m_H & 0 & 0 & 0 & -\frac{\beta_2 H^*}{D_2} & -\frac{\beta_1 H^*}{D_1} \\ 0 & -(\alpha + m_L) & 0 & 0 & \frac{\beta_2 H^*}{D_2} & \frac{\beta_1 H^*}{D_1} \\ 0 & \alpha & -d & 0 & 0 & 0 \\ 0 & 0 & dn_3 & -(\psi + \delta) & 0 & 0 \\ 0 & 0 & dn_2 & \psi & 0 & 0 \\ 0 & 0 & 0 & 0 & -\eta & 0 \\ 0 & 0 & 0 & 0 & \eta n_1 & -m_{z_p} \end{pmatrix}$$

The matrix Jacobienne  $J(X_0)$  having a triangular structure. So that this commonplace point is stable, it is necessary that the various eigenvalues of this Jacobienne matrix are strictly negative real parts. Thus, the eigenvalues of the matrix  $J(X_0)$  are  $\lambda_1 = \epsilon - \frac{2H^*}{K} - m_H < 0$  and the eigenvalues of the new following matrix:

$$J(X_0) = \begin{pmatrix} -(\alpha + m_L) & 0 & 0 & \frac{\beta_2 H^*}{D_2} & \frac{\beta_1 H^*}{D_1} \\ \alpha & -d & 0 & 0 & 0 \\ 0 & dn_3 & -(\psi + \delta) & 0 & 0 \\ 0 & dn_2 & \psi & -\eta & 0 \\ 0 & 0 & 0 & \eta n_1 & -m_{z_p} \end{pmatrix}$$

This new matrix  $J(X_0)$  can cut out as it follows:

$$A = \begin{pmatrix} -(\alpha + m_L) & 0 & 0 \\ \alpha & -d & 0 \\ 0 & dn_3 & -(\psi + \delta) \end{pmatrix}$$

$$B = \begin{pmatrix} \frac{\beta_2 H^*}{D_2} & \frac{\beta_1 H^*}{D_1} \\ 0 & 0 \\ 0 & 0 \end{pmatrix}$$

$$C = \begin{pmatrix} 0 & dn_2 & \psi \\ 0 & 0 & 0 \end{pmatrix}$$

$$D = \begin{pmatrix} -\eta & 0 \\ \eta n_1 & -m_{z_p} \end{pmatrix}$$

Thus, according to Kamgang and Sallet (2008), the new matrix  $J(X_0)$  is stable if and only if matrix  $(D - CA^{-1}B)$  is stable. A simple calculation gives

$$A^{-1} = \begin{pmatrix} -\frac{1}{(\alpha + m_L)} & 0 & 0 \\ -\frac{\alpha}{d(\alpha + m_L)} & -\frac{1}{d} & 0 \\ -\frac{\alpha n_3}{(\alpha + m_L)(\psi + \delta)} & -\frac{n_3}{(\psi + \delta)} & -\frac{1}{(\psi + \delta)} \end{pmatrix}$$

From there, it comes that

$$= \begin{pmatrix} D - CA^{-1}B \\ \left( \frac{\alpha(n_2(\psi + \delta) + \psi n_3)\beta_2 H^*}{(\alpha + m_L)(\psi + \delta)D_2} \right) - \eta & \left( \frac{\alpha(n_2(\psi + \delta) + \psi n_3)\beta_1 H^*}{(\alpha + m_L)(\psi + \delta)D_1} \right) \\ \eta n_1 & -m_{z_p} \end{pmatrix}$$

The matrix  $(D - CA^{-1}B)$  being of order 2, it is stable if and only if its trace is negative and its determinant is positive, i.e.  $tr(D - CA^{-1}B) < 0$  and  $det(D - CA^{-1}B) \geq 0$ . From of the matrix  $(D - CA^{-1}B)$ , one has:

$$tr(D - CA^{-1}B) = \left[ \frac{\alpha(n_2(\psi + \delta) + \psi n_3)\beta_2 H^*}{(\alpha + m_L)(\psi + \delta)D_2} \right] - (\eta + m_{z_p})$$

and

$$det(D - CA^{-1}B) = \left[ -\frac{\alpha(n_2(\psi + \delta) + \psi n_3)\beta_2 H^*}{(\alpha + m_L)(\psi + \delta)D_2} + \eta \right] m_{z_p} - \left[ \frac{\alpha(n_2(\psi + \delta) + \psi n_3)\beta_1 H^*}{(\alpha + m_L)(\psi + \delta)D_1} \right] \eta n_1$$

It is noticed that  $det(D - CA^{-1}B) > 0$  involve  $tr(D - CA^{-1}B) < 0$

Proof:

Indeed,  $det(D - CA^{-1}B) > 0$  involve

$$\left( \frac{\alpha(n_2(\psi + \delta) + \psi n_3)\beta_2 H^*}{(\alpha + m_L)(\psi + \delta)D_2} \right) - (\eta + m_{z_p}) < -\frac{\eta n_1 \left( \frac{\alpha(n_2(\psi + \delta) + \psi n_3)\beta_1 H^*}{(\alpha + m_L)(\psi + \delta)D_1} \right)}{m_{z_p}} - m_{z_p}$$

Donc  $tr(D - CA^{-1}B) < 0$ .

This completes de proof.

Thus the matrix  $(D - CA^{-1}B)$  is stable if and only if  $det(D - CA^{-1}B) > 0$  i.e.

$$\left[ -\frac{\alpha(n_2(\psi + \delta) + \psi n_3)\beta_2 H^*}{(\alpha + m_L)(\psi + \delta)D_2} + \eta \right] m_{z_p} \geq \left[ \frac{\alpha(n_2(\psi + \delta) + \psi n_3)\beta_1 H^*}{(\alpha + m_L)(\psi + \delta)D_1} \right] \eta n_1$$

$$\begin{aligned} \Rightarrow \eta m_{Z_P} &\geq \left[ \frac{\alpha(n_2(\psi + \delta) + \psi n_3)\beta_1 H^*}{(\alpha + m_L)(\psi + \delta)D_1} \right] \eta n_1 \\ &+ \frac{\alpha(n_2(\psi + \delta) + \psi n_3)\beta_2 H^*}{(\alpha + m_L)(\psi + \delta)D_2} m_{Z_P} \\ \Rightarrow \left( \frac{\beta_1 n_1}{D_1 m_{Z_P}} + \frac{\beta_2}{D_2 \eta} \right) \left[ \frac{H^* \alpha(n_2(\psi + \delta) + \psi n_3)}{(\alpha + m_L)(\psi + \delta)} \right] &\leq 1 \end{aligned}$$

Thus, the basic net reproduction rate of model system (1) is

$$R_0 = \left( \frac{\beta_1 n_1}{D_1 m_{Z_P}} + \frac{\beta_2}{D_2 \eta} \right) \left[ \frac{H^* \alpha(n_2(\psi + \delta) + \psi n_3)}{(\alpha + m_L)(\psi + \delta)} \right]. \quad (4)$$

It measures the number of new infectious lesions generated by an infectious starting lesion in a population entirely made up of healthy sheets. Consequently, the DFE of our model is locally asymptotically stable for  $R_0 \leq 1$  and unstable for  $R_0 > 1$ . Therefore, any fight planned to make disappear the mildew should seek to lower this rate below 1. We then showed the following result.

When

1.  $R_0 \leq 1$  the DFE is locally asymptotically stable.
2.  $R_0 > 1$  the DFE is unstable.

#### 2.2.4. The Endemic Point of Balance and Its Stability

That is to say  $X = (H^*, L^*, I^*, O^*, Sp^*, Zp^*)$  a point of endemic balance unspecified of the model system (1). This point is obtained by posing the entire derivative equal to 0, which gives us.

$$\left\{ \begin{aligned} A_4 &= \frac{\varepsilon x^4 \alpha^4 n_1^2}{(\alpha + m_L)(\psi + \delta)^2 D_1 \eta D_2 m_{Z_P}}; \\ A_3 &= \frac{(\beta_1^2 - \beta_2^2 - 2\beta_1 \beta_2) x^4 \alpha^4 n_1^2 K + \varepsilon (\alpha + m_L)(\psi + \delta) (D_2 \eta n_1 + D_1 m_{Z_P}) 2x^3 \alpha^3 n_1 - (\beta_1 + \beta_2) \varepsilon H^* x^4 \alpha^4 n_1^2}{(\alpha + m_L)^2 (\psi + \delta)^2 D_1 \eta D_2 m_{Z_P}}; \\ A_2 &= \left[ \left( (m_{Z_P} D_1 + n_1 \eta D_2)(\psi + \delta) \beta_1 x^3 \alpha^3 n_1 + (\beta_1 + \beta_2)(\psi + \delta) x^3 \alpha^3 n_1^2 D_2 \eta + \right. \right. \\ &\quad \left. \left. 2\beta_2 x^3 \alpha^3 n_1 m_{Z_P} D_1 (\psi + \delta) \right) \varepsilon H^* + \right. \\ &\quad \left. 2K \left( \beta_2^2 D_1 m_{Z_P} x^3 \alpha^3 n_1 (\psi + \delta) + (n_1 D_2 \eta + D_1 m_{Z_P}) \beta_1 \beta_2 x^3 \alpha^3 n_1 (\psi + \delta) \right) \right. \\ &\quad \left. - \varepsilon (\alpha + m_{Z_P}) \left( 4x^2 \alpha^2 n_1 D_1 D_2 \eta m_{Z_P} (\psi + \delta)^2 + D_1^2 x^2 \alpha^2 m_{Z_P}^2 (\psi + \delta)^2 \right) \right] \frac{1}{(\alpha + m_L)^2 (\psi + \delta)^2 D_1 \eta D_2 m_{Z_P}}; \\ A_1 &= \left[ \left( (\beta_1 + \beta_2) D_1 D_2 x^2 \alpha^2 n_1 m_{Z_P} \eta (\psi + \delta)^2 + (D_2 \beta_1 n_1 \eta + \beta_2 D_1 m_{Z_P}) x^2 \alpha^2 n_1 \eta D_2 (\psi + \delta)^2 + \right. \right. \\ &\quad \left. \left. (\beta_1 n_1 D_2 \eta + \beta_2 D_1 m_{Z_P}) x^2 \alpha^2 D_1 m_{Z_P} (\psi + \delta)^2 \right) \varepsilon H^* \right. \\ &\quad \left. + \left( (\beta_1^2 n_1^2 \eta^2 + \beta_2^2 m_{Z_P}^2) x^2 \alpha^2 D_1^2 (\psi + \delta)^2 + 2\beta_1 \beta_2 x^2 \alpha^2 n_1 D_1 D_2 \eta m_{Z_P} (\psi + \delta)^2 \right) K - \right. \\ &\quad \left. \varepsilon (\alpha + m_L) (m_{Z_P} D_1 + D_2 n_1 \eta) 2x \alpha D_2 D_1 m_{Z_P} \eta (\psi + \delta)^3 \right] \frac{1}{(\alpha + m_L)^2 (\psi + \delta)^2 D_1 \eta D_2 m_{Z_P}}; \\ A_0 &= \sigma (1 - R_0); \end{aligned} \right. \quad (9)$$

and

$$\sigma = \frac{\varepsilon D_2 D_1 \eta m_{Z_P} (\psi + \delta)^2}{\alpha + m_L} \quad (10)$$

The rule of the signs of Descartes will thus enable us to

$$\left\{ \begin{aligned} \varepsilon \left( 1 - \frac{H^*}{K} \right) H^* - \left( \frac{\beta_1 Z_P^*}{Z_P^* + D_1} + \frac{\beta_2 S_P^*}{S_P^* + D_2} \right) H^* - m_H H^* &= 0; \\ \left( \frac{\beta_1 Z_P^*}{Z_P^* + D_1} + \frac{\beta_2 S_P^*}{S_P^* + D_2} \right) H^* - (\alpha + m_L) L^* &= 0; \\ \alpha L^* - d I^* &= 0; \\ d n_3 I^* - (\psi + \delta) O^* &= 0; \\ \psi O^* + d n_2 I^* - \eta S_P^* &= 0; \\ n_1 \eta S_P^* - m_{Z_P} Z_P^* &= 0; \end{aligned} \right. \quad (5)$$

By using the first, third, fourth and fifth equation of the model system (5);  $(I^*, (O^*, (S_P^*, (Z_P^*)$  and  $(H^*)$  express themselves according to  $(L^*)$  as follows.

$$\left\{ \begin{aligned} I^* &= \frac{\alpha L^*}{d}; \\ O^* &= \frac{\alpha n_3 L^*}{\psi + \delta}; \\ S_P^* &= \frac{x \alpha L^*}{\eta (\psi + \delta)}; \\ Z_P^* &= \frac{x \alpha n_1 L^*}{m_{Z_P} (\psi + \delta)}; \\ H^* &= \frac{K}{\varepsilon} \left[ \varepsilon - m_H - \frac{\beta_1 x \alpha n_1 L^*}{x \alpha n_1 L^* + D_1 m_{Z_P} (\psi + \delta)} - \frac{\beta_2 x \alpha L^*}{x \alpha L^* + D_2 \eta (\psi + \delta)} \right]; \end{aligned} \right. \quad (6)$$

Where

$$x = \psi n_3 + n_2 (\psi + \delta). \quad (7)$$

By integrating the expressions of  $(I^*, (O^*, (S_P^*, (Z_P^*)$  and  $(H^*)$  given to the equation (6) in the second equation of the system (5), one obtains the following equation of degree four in  $(L^*)$ :

$$A_4 L^4 + A_3 L^3 + A_2 L^2 + A_1 L^* + A_0 = 0. \quad (8)$$

discuss on the existence the possible solutions this equation.

Theorem When  $R_0 > 1$  and  $R_0$  close to 1, the model of the system (1) admit locally a point of endemic balance asymptotically stable.



Table 2. Solutions of the equation (8).

$A_4$	$A_3$	$A_2$	$A_1$	$A_0$	$R_0$	Number of solutions
+	+	+	+	+	$R_0 < 1$	0 solution
+	+	+	+	-	$R_0 > 1$	1 solution
+	+	+	-	+	$R_0 < 1$	2 solutions
+	+	+	-	-	$R_0 > 1$	1 solution
+	+	-	+	+	$R_0 < 1$	2 solutions
+	+	-	+	-	$R_0 > 1$	3 solutions
+	+	-	-	+	$R_0 < 1$	2 solutions
+	+	-	-	-	$R_0 > 1$	1 solution
+	-	+	+	+	$R_0 < 1$	2 solutions
+	-	+	+	-	$R_0 > 1$	3 solutions
+	-	+	-	+	$R_0 < 1$	4 solutions
+	-	+	-	-	$R_0 > 1$	3 solutions
+	-	-	+	+	$R_0 < 1$	2 solutions
+	-	-	+	-	$R_0 > 1$	3 solutions
+	-	-	-	+	$R_0 < 1$	2 solutions
+	-	-	-	-	$R_0 > 1$	1 solution

2.3. Numerical Simulations

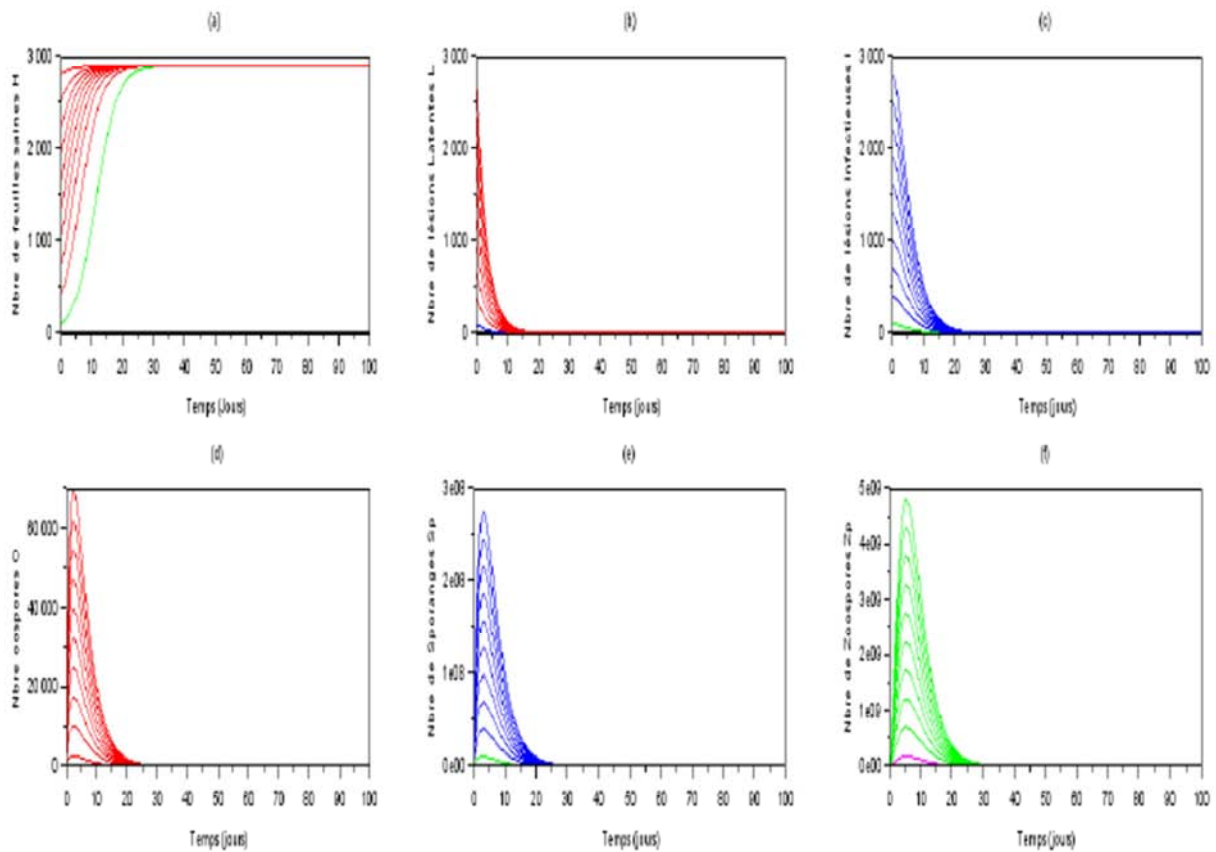
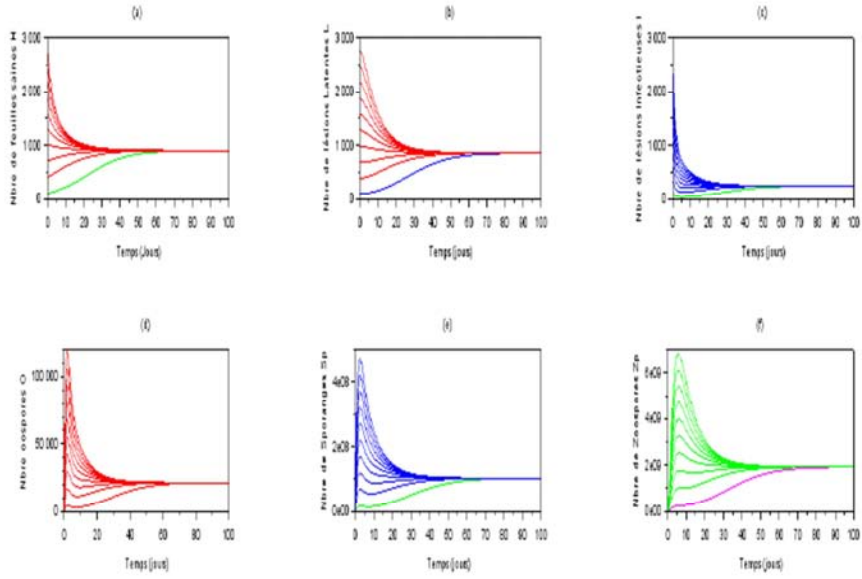


Fig. 3. Simulation of model system (1) when  $(\epsilon = 0.3; K = 3000; \beta = 10^6; D_1 = 45000; D_2 = 45000; m_H = 0.01; \alpha = 0.3; m_L = 0.01; \delta = 0.4; n_1 = 15; d = 0.3; n_3 = 100; n_2 = 300000; \eta = 0.7; mZp = 0.5; \psi = 0.6; (R_0 = 0.153) < 1)$  (a) healthy sheets; (b) infected sheets; (c) infectious sheets; (d) oospore numbers; (e) sporangia numbers; (f) zoospore numbers.



**Fig. 4.** Simulation of model system (1) when  $(\epsilon = 0.3; K = 3000; \beta = 10^{-1}; D_1 = 450000; D_2 = 450000; m_H = 0.01; \alpha = 0.2; m_L = 0.01; \delta = 0.5; n_1 = 15; d = 0.7; n_3 = 100; n_2 = 300000; \eta = 0.5; m_{Zp} = 0.4; \psi = 0.3; (R_0 = 7.66) > 1)$  (a) healthy sheets; (b) infected sheets; (c) infectious sheets; (d) oospores numbers; (e) sporangia numbers; (f) zoospore numbers.

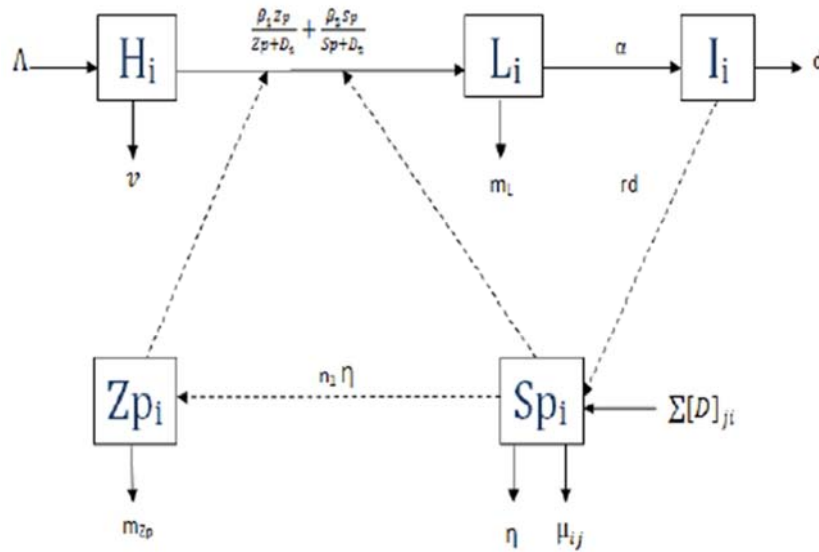
**2.4 Model Multi-patches**

**2.4.1. Biological Facts**

It is supposed that at one moment  $t$ , a piece is in one of the

following states: healthy sites (H), latent sites (L), infectious sites (I).

The structure of the model is given to the Fig. 5.



**Fig. 5.** Structure of the model.

If one takes  $v(i)$  variety of taro in the piece  $i$ . An infectious lesion produced  $r_{v(i)}$  sporangia per day. The sporangia are dispersed on another piece  $j$  with a probability  $\mu_{ij}$ . Thus, at time  $t$ , the number of sporangia dispersed starting from the piece  $i$  on other pieces follows a distribution multinomial

$$Multi(r_{v(i)} \cdot d \cdot I_i(t - 1), [\mu_{i_1}, \mu_{i_2}]). \quad (11)$$

The number of sporangia arriving in the piece  $j$  coming from the piece  $i$  at time  $t$  is.

$$S_{P_i}(t) = \sum_i [D(t)]_{ij}. \quad (12)$$

Infection of the healthy sites: a healthy sheet in an unspecified piece receives a sporangia or a zoospore then becomes infected with the probability  $\beta_{v(i)}$ . In the same way, a piece can undergo harvests with a force of  $v(i)$

When a sheet is infected, the mushroom multiplies inside fabric during this time one speaks about latent lesion. The appearance of a new latent lesion follows the following binomial distribution:

$$\langle H \rightarrow L \rangle_i(t) \rightsquigarrow \text{Bin}(H_i(t-1), \beta_{v(i)}). \quad (13)$$

There is the following recurring equation.

$$H_i(t) = H_i(t-1) + \langle \Lambda \rangle(t) - \langle vH \rangle_i(t) - \langle H \rightarrow L \rangle_i(t). \quad (14)$$

Sporangia and the zoospore which do not manage to infect are withdrawn from the system with the respective probabilities  $\mu$  and  $m_{Zp}$ .

Transition from the sites of latent to the infectious one: After an infection, the healthy sites become latent lasting  $P_{v(i)}$  days (latency period) before producing new sporangia

Of this fact, the probability so that a new infectious lesion appears in a piece  $i$  between time

$t-1$  and time  $t$  is  $\frac{1}{P_{v(i)}}$ . This appearance follows a binomial distribution then such as:

$$\langle L \rightarrow I \rangle_i(t) \rightsquigarrow \text{Bin} \left( L_i(t-1), \frac{1}{P_{v(i)}} \right). \quad (15)$$

One obtains the following recurring equation:

$$L_i(t) = L_i(t-1) + \langle H \rightarrow L \rangle_i(t) - \langle L \rightarrow I \rangle_i(t) - \langle m_{L_i} L \rangle_i(t). \quad (16)$$

Passage of the infectious sites to the sites whose lesion ceases the sporulation, after  $t_{v(i)}$  days of sporulation (period of infection) the site is regarded as withdrawn of the system studied

There is the following recurring equation:

$$I_i(t) = I_i(t-1) + \langle L \rightarrow I \rangle_i(t) - \langle \frac{1}{T_{v(i)}} I \rangle_i(t). \quad (17)$$

Dispersion: We admit that the sporangia move at long distance by the wind. The density of spores emitted by a source  $z$  located at the point  $(x; y)$  and which are received at the point  $z'$  located in the zone  $(x'; y')$  is given by (Klein *et al.*, 2006):

$$g(\|z - z'\|) = \frac{(a-2)(a-1)}{2\pi b^2} \left( 1 + \frac{\|z-z'\|}{b} \right)^{-a}. \quad (18)$$

Where  $\|z - z'\|$  the Euclidiene distance is enters  $z$  and  $z'$ ,  $b > 0$  are a scalar parameter and  $a > 2$  determine the distance from dispersion.

The average distance is only defined when  $a > 3$  then that  $\mu_0 = \frac{2b}{a-3}$ .

The probability for a spore of dispersing of a piece  $i$  a piece  $j$  is

$$\mu_{ij} = \left( \int_{A_i} \int_{A_j} g(\|z - z'\|) d_z d'_z \right) \frac{1}{A_i}. \quad (19)$$

Where,  $A_i$  and  $A_j$  are respective surfaces of the pieces  $i$  and  $j$ .

### 2.4.2. Model

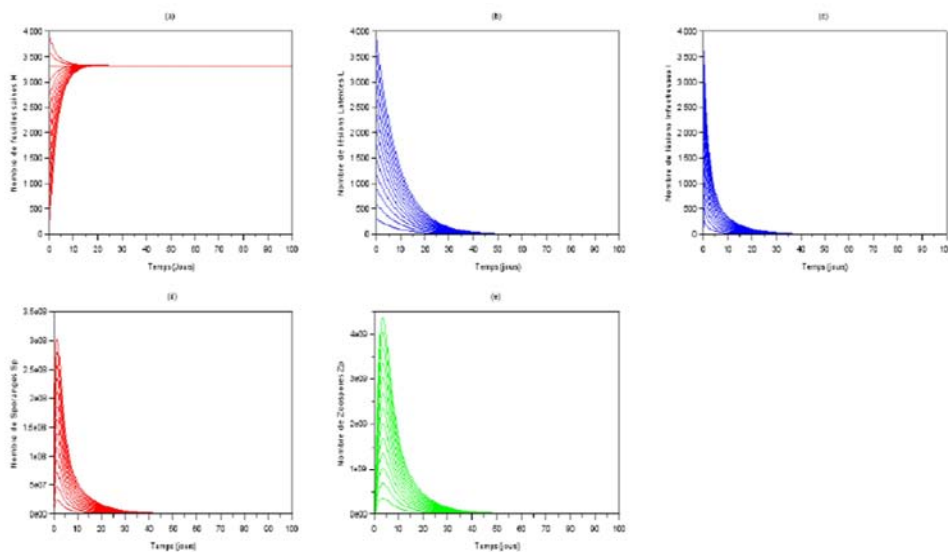
$$\begin{cases} \dot{H}_i = \Lambda - \left( \frac{\beta_1 Z_{P_i}}{Z_{P_i} + D_1} + \frac{\beta_2 S_{P_i}}{S_{P_i} + D_2} \right) H_i - v H_i \\ \dot{L}_i = \left( \frac{\beta_1 Z_{P_i}}{Z_{P_i} + D_1} + \frac{\beta_2 S_{P_i}}{S_{P_i} + D_2} \right) H_i - (\alpha + m_{L_i}) L_i; \\ \dot{I}_i = \alpha L_i - d I_i; \\ \dot{S}_{P_i} = D_i S_{P_i} + r d I_i - (\eta_i + \mu_i) S_{P_i}; \\ \dot{Z}_{P_i} = n_1 \eta_i S_{P_i} - m_{ZP} Z_{P_i}; \end{cases} \quad (20)$$

Where  $\alpha = \frac{1}{P_{v(i)}}$  et  $d = \frac{1}{T_{v(i)}}$ .

Thus, the basic net reproduction rate of the model (20) is

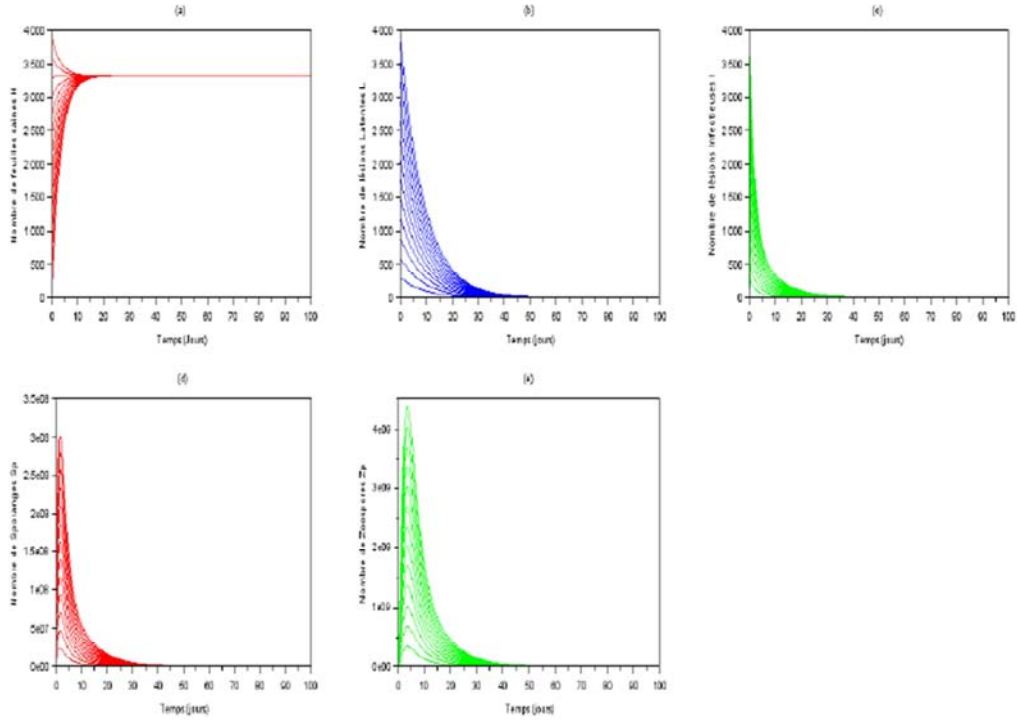
$$R_{0i} = \left( \frac{\beta_1 n_1 \eta_i}{D_1 m_{ZP_i}} + \frac{\beta_2}{D_2} \right) \left[ \frac{\alpha_i r_i \Lambda}{(\alpha + m_{L_i})(\eta_i + \mu_i - D_i) v_i} \right]. \quad (21)$$

### 2.4.3. Numerical Simulations



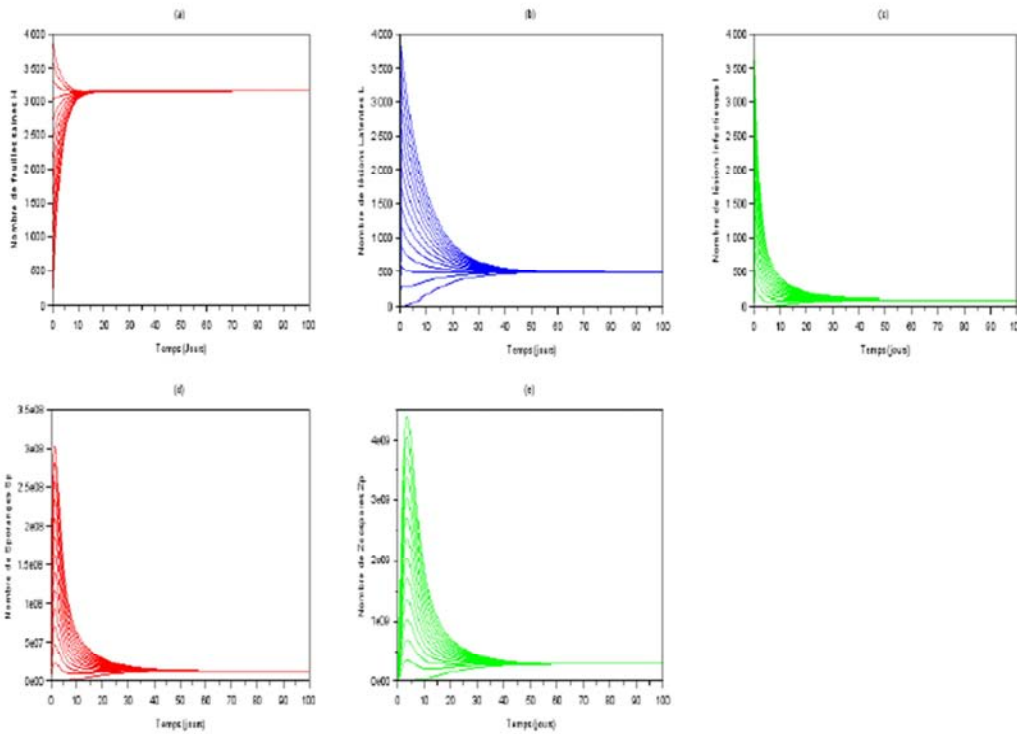
(a) healthy sheets; (b) infected sheets; (c) infectious sheets; (d) sporangia numbers; (e) zoospore numbers.

**Fig. 6.** Simulation of model system (20) when  $(i = 1; \lambda = 1000; \beta = 10^9; v = 0.3; \alpha = 0.1; \mu = 0.5; m_L = 0.01; d = 0.5; D = 0.1; r = 300000; \eta = 0.7; n_1 = 15; m_{Zp} = 0.5; D_1 = 45000; D_2 = 45000; (R_{0i} = 0.00017) < 1)$ .



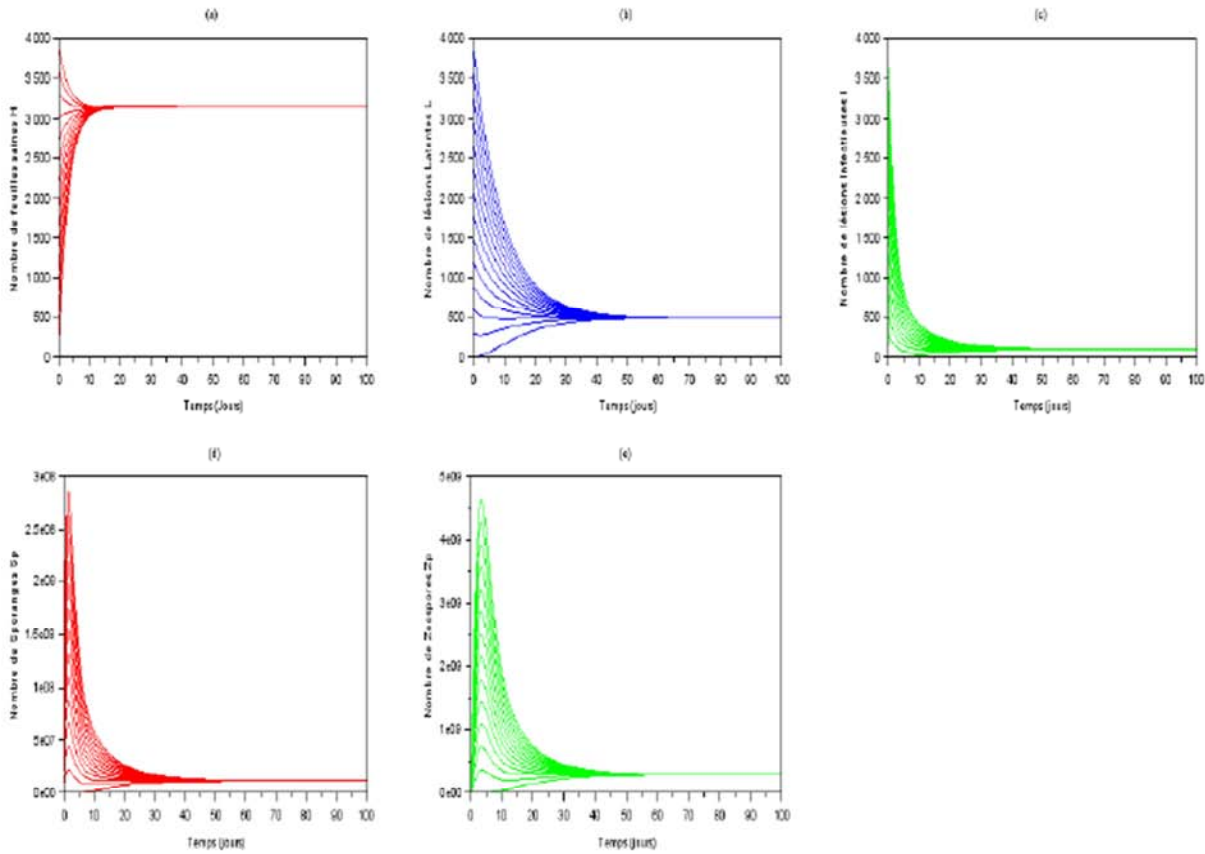
(a) healthy sheets; (b) infected sheets; (c) infectious sheets; (d) sporangia numbers; (e) zoospore numbers.

**Fig. 7.** Simulation of model system (20) when  $(i = 2; \lambda = 1000; \beta = 10^{-5}; \nu = 0.3; \alpha = 0.1; \mu = 0.5; m_L = 0.01; d = 0.5; D = 0.1; r = 300000; \eta = 0.7; n_1 = 15; m_{Zp} = 0.5; D_1 = 45000; D_2 = 45000; (R_{02} = 0.17) < 1)$ .



(a) healthy sheets; (b) infected sheets; (c) infectious sheets; (d) sporangia numbers; (e) zoospore numbers.

**Fig. 8.** Simulation of model system (20) when  $(i = 1; \lambda = 1000; \beta = 10^{-2}; \nu = 0.3; \alpha = 0.1; \mu = 0.5; m_L = 0.01; d = 0.5; D = 0.1; r = 300000; \eta = 0.7; n_1 = 15; m_{Zp} = 0.5; D_1 = 4500000; D_2 = 4500000; (R_{01} = 1.33) > 1)$ .



(a) healthy sheets; (b) infected sheets; (c) infectious sheets; (d) sporangia numbers; (e) zoospore numbers.

**Fig. 9.** Simulation of model system (20) when  $(i = 2; \lambda = 1000; \beta = 10^{-2}; v = 0.3; \alpha = 0.1; \mu = 0.5; m_L = 0.01; d = 0.5; D = 0.1; r = 300000; \eta = 0.8; n_1 = 15; mZp = 0.5; D_1 = 4500000; D_2 = 4500000; (R_{02} = 1.7) > 1)$ .

### 3. Results and Discussion

The model is not necessarily based on a complex mathematical formulation. It is built from information that has been acquired on the system studied and allows to simulate the operation [33]. To explain the behaviour of the epidemic, we make numerical simulations that explore the epidemic by varying the conditions of development of the epidemic. Hen t is expressed in days,  $R_0$  the multiplying daily factor, that is to say the number of girls lesions initiated daily by a parent injury.

The model (Fig. 2) shows that the latency period, lesion size, the efficiency of infection of sporangia and zoospores have significant effects on both the speed of the epidemic on the maximum level.

#### 3.1. Biological Interpretation of the Fig. 3

Fig. 3 presents six compartments: (a), (b), (c), (d), (e), (f).

1) The compartment (a) represents the evolution of the number of healthy leaves in a plot in the absence of disease because ( $R_0 < 1$ ). To follow this development we set the maximum number of healthy leaves that may have a plot in 3000 (this is an example) that said, whatever the number of healthy leaves we at the beginning of the experiment, should expect that number to reach and does not exceed 3,000 sheets

taking into account the constant renewals. However, the compartment (a) shows that 40 days after planting taro bulbs, the maximum number of sheets is reached, but it is not equal to 3,000 sheets as expected because healthy leaves if not infected die naturally. So we can have a complete defoliation of the field if weather is severe.

2) The compartment (b) is in direct communication with the compartment (a). This means that, if there is no infection in the compartment (a), and of this compartment there is no appearance even if we were to introduce into the plot of sick leaves in order to initiate infections, it will be observed that after 20 days the leaves die without even initiate infections.

3) (c) which represents the evolution of the number of sheets capable of transmitting the disease, there is no sheet capable of transmitting the disease because of (a) there is no infection.

4) (d) represents the number of oospores the oospores is fungal survival unit into the ground. So compartment (d) reports that if the previous crop due to leave millions of oospores in soil, these oospores transported and deposited on healthy leaves, due to being able to initiate damage, they will start die from the 20th day to the point of completely disappearing the 25th day.

5) Compartments (e) and (f) represent the number of sporangia and zoospores, describing the situation that these

cannot be produced by the experimental culture, may come from the surrounding plots. Any time when placed on healthy leaves, due to not being able to initiate infection, they will start dying from the 20th day as oospores.

### 3.2. Biological Interpretation of (Fig. 4)

In Fig. 4,  $R_0 > 1$ , so the illness persists. But this disease cannot grow indefinitely. It will stabilize at a point that we call endemic disease.

But what is the biological information that we give these curves?

1) In the compartment (a), with the same lines as before; this time the number of healthy leaves is rather stable in 1000 still from the 40th day. This means that 2000 leaves are dead under the influence of weather and largely under the influence of the fungus. It appears here that there is for any culture of taro, a number of sheets for which although the variety of taro is sensitive, if this number is not reached the disease does not develop.

2) The compartment (b) is in communion with the compartment (a). So we should expect that 2000 healthy leaves infected (a) find themselves in (b) not. The block (b) presents the appearance rather than 1000 infected leaves only; therefore, infected leaves can also die naturally without any time to cause new infections.

It appears here that whatever the speed of disease progression, it should stabilize from the 45th day. In other words, whatever the number of infected leaves, the disease will cease to grow of the 45th day. So in all taro crops whatever the speed of evolution of the disease, there are a limited number of infected leaves that may appear during the disease. This number is in this case 1000.

3) The compartment (c) and (d) are similar since they are in direct communication. So what comes out of (b) is found in (c) without any time to forget that an infectious sheet can naturally die without having had time to infect another.

4) (d), (e), (f), there are also biological stabilities thresholds. So, whatever the level of disease, each culture can only produce a given number of oospores, sporangia and zoospores. Knowledge of the day when that number is reached is very useful for plant pathologists; in our case this value is reached the 45th day.

The values given in this simulation can be used as a scale for clarification on the field.

We say that as there is a very close relationship between the development of the disease and micro-meteorological variables such as temperature humidity and precipitation [32] as it exists between the evolution of the disease, the number of sheets, oospores, sporangia and zoospores. This relationship has helped to build predictive models of epidemics and thus identify the periods of maximum risk [33, 34, 35, 36, 37, 38, 39, 40, 41, 42, 43]. The ultimate goal of these models is to target periods or intervention is necessary and thus reduce the number of fungicide application.

Despite these models, it is still difficult to predict the contamination dates for two main reasons: the first is the heterogeneous distribution of primary sources of inoculum;

the second is that these models do not incorporate conservation factors or germination and infectivity or oospores [44]. According Andrivon and Lebreton [44], the development of a reliable and realistic model requires first to fill the lack of information about the biology of these organs.

## 4. Conclusion and Perspectives

In this work, we studied in leading the influence that can have a favourable trial setting in the kinetics of the disease, the end of which we believe that the duration of the pathogen latency period, the number of sporangia products on the surface of a lesion as the efficiency of infection of the zoospore or sporangia individual parameters are taken into account in the development of a variety of taro appropriate level of resistance. But these parameters are exacerbated when taken together.

The model is then used to compare the impact of different resistance components on the evolution of the disease, and therefore to optimize varietal selection: choice of selection goals based on their ability to practice and assessment their epidemiological effects.

In the background, we addressed the question of the benefit of a diversified operation taro plant in the mushroom control *P. colocasiae* so the idea is to grow in the same plot several varieties of taro to minimize the severity of the disease. In this second model, the interaction between plots will be made only via sporangia, in perspective, it would be interesting for a complete model that strongly closer to reality also take into account the interactions through zoospores that can move from one plot to another; while considering the dispersion of these through culture, because this probability has only been a mere guess. Parameter estimation, analysis of the model parameters of sensitivity as well as field evaluation for model validation will be made. In developing countries, where climate data acquisitions are difficult or inputs and pesticides and fungicides are in certain circumstances prohibitively expensive, models that look for help in plant breeding are of great importance.

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