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A DISCRETE MATHEMATICAL MODELING AND OPTIMAL CONTROL OF DISEASES CAUSED BY *FUSARIUM OXYSPORUM*

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Abstract. *Fusarium Oxysporum* is a pathogenic plant causes two distinct types of symptoms: vascular wilts and root and crown rots. A wide variety of plants can be affected by *Fusarium* wilt caused by *Fusarium Oxysporum*. In this work, we propose a discrete mathematical model that describes the diseases caused by *Fusarium Oxysporum*, which has serious economic consequences and a negative impact on public health. We examine the optimal control problem concerning the number of plants infected by the fungus *Fusarium Oxysporum*. To show the important of the controls, we have made analytical estimates that are validated using simulations.

Keywords: discrete mathematical model; fusarium oxysporum; optimal control; optimization strategy.
2010 AMS Subject Classification: 39A05, 39A14, 93C35, 93C55.

1. INTRODUCTION

Fusarium oxysporum is an Ascomycete fungus that has never been observed as a teleomorph (Leslie and Summerell [24]). The species has been isolated from soils around the world (cultivated and uncultivated) and in all climates (arctic, tropical, desert) [13, 4, 26]. Although the species is classically found in soils, it is also isolated from more unusual places: hospital water circuit, sea water, river water, tap water, dishwashers (water, detergents), contact lenses or food

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[11, 27, 20, 18, 3, 2]. *F. oxysporum* is capable of growing as a saprophyte in soil and surviving for several years as a storage structure. When the fungus grows, it produces two types of conidia that ensure its dissemination.

The plant pathogenic *F. oxysporum* causes two distinct types of symptoms: vascular wilts and root and/or crown rots [10]. A wide variety of plants can be affected by *Fusarium* wilt caused by *F. oxysporum*. The species has been ranked among the "top 10" plant pathogenic fungi, based on its economic importance and scientific interest. However, if the fungus is polyphagous at the species level, it is not so at the strain level. Indeed, each strain of phytopathogenic *F. oxysporum* shows a high host specificity. Strains are classified into special forms according to the plant species infected. The special forms are sometimes subdivided into races, named for the resistance genes they are able to bypass. The number of races per special form varies from two, as in the special form conglutinans, to six as in the special form vasinfectum [6] [12]. However, for some special forms, notably *F. oxysporum F.* sp. cyclaminis, no race has been identified, although differences in aggressiveness between strains have been noted [9] . Currently, more than 150 special forms and races are described within the species *F. oxysporum* . However, some pathogenic strains are described in the literature without a special form name being systematically associated with them. The number of special forms and races of *F. oxysporum* is therefore certainly much greater. The economic impact of *F. oxysporum* is important because of the diversity of its hosts. Indeed, crops as important as tomato, cotton, vanilla, flax or cucumber can be attacked. One of the most notable examples is certainly the *Fusarium* disease of banana (Panama disease) caused by *F. oxysporum F.* sp. cubense. During the first half of the 20th century, the disease decimated banana plantations in Central Africa and the Caribbean and is considered to be the most important disease on banana.The world banana market has only been saved by the introduction of the Cavendish cultivar, which is resistant to the pathogen [23]. Similarly, the development of *F. oxysporum F.* sp. vanillae in vanilla plantations is the main factor limiting world production [25]. While a trained eye can differentiate *F. oxysporum* from other *Fusarium* species under the microscope, it is not possible to do the same with two strains of *F. oxysporum*. Thus, strains sharing the same morphological characteristics can be pathogenic to tomato, a biological control agent or an opportunistic pathogen of humans. The

differentiation of *F. oxysporum* strains and the identification of special forms are very important to understand the ecology of pathogen populations and the epidemiology of diseases. This identification will also determine the implementation of appropriate control measures. For the moment, only pathogenicity tests on plants and verification of Koch's postulates allow the identification of the special form to which a strain belongs [14]. However, pathogenicity tests are very time consuming. For example, such a test takes 4-6 months on Canary Island date palm (*F. oxysporum F.* sp. canariensis) and 8-12 months on oil plant (*F. oxysporum F.* sp. canariensis). [16]. Although molecular tools can identify taxon-specific genetic markers, few special forms of *F. oxysporum* have such a detection marker available. Their detection would offer a faster, more sensitive, more reproducible and less expensive way to specifically detect a special form, but requires prior knowledge of the genetic diversity of the special form considered.

2. Formulation of the Mathematical Model

In this paper, we consider a system of $C-SIRS$ with four compartments to describe the spatial spread of two types of plants infected by the fungus *Fusarium oxysporum*.

- **The compartment** C: Density of the fungus *Fusaruim oxyporum*.
- **The compartment** S^a: Number of plant trees belonging to the area that are not sick but likely to become sick (e.g. young, resistant, good quality, ...).
- **The compartment** S^b: Number of plant trees belonging to the area that are not sick but likely to become sick (e.g. old, infected, poorly watered, rocky soil, ...).
- **The compartment** I: Number of plant trees infected with *Fusaruim oxyporum*.
- **The compartment** R: Number of plant trees that have already had the disease and are now immune to *Fusaruim oxyporum*.

The following diagram shows the flow directions of the plant between the compartments.

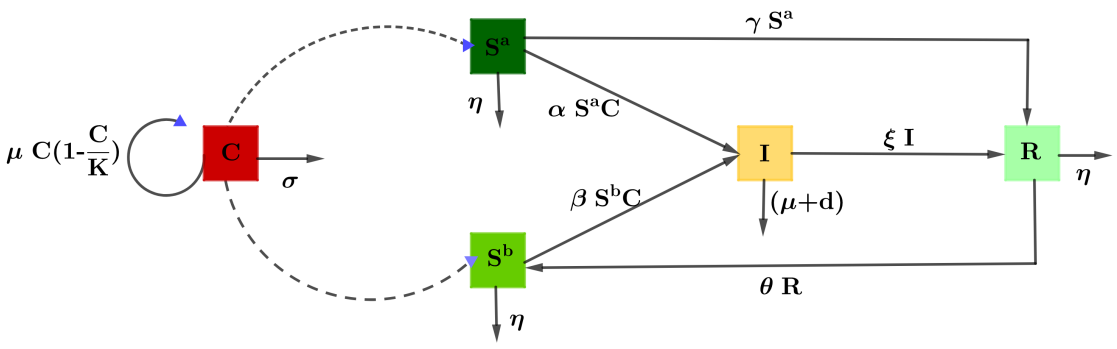

FIGURE 1. The C-SIRS model

μ	Intrinsic growth rate of fungus.
K	Carrying capacity of the environment.
η	Mortality rate of fungus.
α	Rate of fungus encounters with S^a plant.
β	Rate of fungus encounters with S^b plant.
γ	Rate of plant healing by self resistance.
d	Rate of fungus mortality .
ξ	Rate of fungus encounters with plant.
α	Rate of plant tree deaths due to infection.
θ	Rate of cured lant that becomes susceptible due to loss of immunity.

TABLE 1. Description of parameters used in model 1

In discrete time, the predefined model is given by the following system of equations:

$$
(1)\qquad \begin{cases} C_{k+1} = C_k + \mu C_k \left(1 - \frac{C_k}{K}\right) - \sigma C_k, \\ S^a_{k+1} = S^a_k - \alpha S^a_k C_k - \eta S^a_k - \gamma S^a_k, \\ S^b_{k+1} = S^b_k - \beta S^b_k C_k - \eta S^b_k + \theta R_k, \\ I_{k+1} = I_k + \alpha S^a_k C_k + \beta S^b_k C_k - (\eta + d) I_k - \xi I_k, \\ R_{k+1} = R_k + \gamma S^a_i - (\eta + \theta) R_k + \xi I_k. \end{cases}
$$

The fact that the *Fusarium oxysporum* fungi population is increasing exponentially is not biologically satisfactory, because even if a population arrives in an environment containing all the necessary resources, which is the case for invasive species, a population cannot increase exponentially to infinity. Self-regulation phenomena will therefore take place. These phenomena are taken into account in Verhulst's model (1838), also known as the logistic growth model.
The susceptible plant S^b becomes infected at rate γ when they come in contact with the *Fusarium oxysporum* . That is, the change in population is equal to $-\gamma S^b_k C_k$. In addition, individuals from the recovered group become susceptible again at a certain rate " η " to give ηR_k,.
The infected plant begins with adding what was just removed from the susceptible population, $\gamma S_k C_k$ and then a reduction in two ways i.e. plant can either recover or they are killed by the virus. They recovered from the virus at rate " θ" and are killed at rate " α ".
The recovered plant R_i is increased by those that recovered from the virus and reduced by the number of plant that join the susceptible group at rate "λ".

3. THE OPTIMAL CONTROL PROBLEM

In the absence of effective treatment available for the disease caused by the fungus *Fusarium oxysporum*, infected plants are not controlled, they can serve the disease, and the fungus can spread. So our aim is to minimise the number of plants infected by *Fusarium oxysporum*:

$$
(2)\qquad \begin{cases} C_{k+1} = C_k + \mu C_k \left(1 - \dfrac{C_k}{K}\right) - \varepsilon_1 u C_k, \\ S^a_{k+1} = S^a_k - \alpha S^a_k C_k - \eta S^a_k - \gamma S^a_k, \\ S^b_{k+1} = S^b_k - \beta S^b_k C_k - \eta S^b_k + \theta R_k, \\ I_{k+1} = I_k + \alpha S^a_k C_k + \beta S^b_k C_k - (\eta + d) I_k - \varepsilon_2 v I_k - \xi I_k, \\ R_{k+1} = R_k + \gamma S^a_i - (\eta + \theta) R_k + \xi I_k + \varepsilon_2 v I_k. \end{cases}
$$

with

$$
\varepsilon_i = \begin{cases} 1 \\ 0 \end{cases} \qquad \text{for } i = 1, 2
$$

For exemple $\varepsilon_1 = 1$ and $\varepsilon_2 = 1$ means that we apply only a single control; $\varepsilon_1 = 1$ and $\varepsilon_2 \neq 1$ means that we apply two controls; $\varepsilon_1 \neq 1$ and $\varepsilon_2 \neq 1$ means that we apply three controls.

We consider the following objective functional:

$$
(3)\qquad J(u_k, v_k) = \sum_{k=0}^{N} \left(a_k C_k + b_k S^a_k + c_k S^b_k + d_k I_k - e_k R_k\right) + \sum_{k=0}^{N-1} \left(A^k_1 \frac{u_k^2}{2} + A^k_2 \frac{v_k^2}{2}\right).
$$

The parameters $a_k > 0$, $b_k > 0$, $c_k > 0$, $d_k > 0$ and $e_k > 0$ are the cost coefficients; they are selected to weigh the relative importance of $C^t_k, S^a_k, S^b_k, I_k, R^t_k, u_k$ and v_k, at time k. N is the final time.

In other words, we search to determine the optimal controls u_k and v_k, such that

$$
(4)\qquad J(u^*_k, v^*_k) = \min_{(u_k, v_k) \in U_{ad}} J(u_k, v_k),
$$

where U_{ad} is the set of allowable controls defined by:

$$
(5)\qquad U_{ad} = \{(u_k, v_k) \quad 0 \leq u_k \leq 1, \quad 0 \leq v_k \leq 1, \quad k = 0, 1 \ldots N-1\}
$$

The sufficient condition for the existence of optimal controls (u_k, v_k) for problems (2) and (3) comes from the following theorem:

Theorem 3.1. *There exists an optimal control* $\left(u_k^*, v_k^*\right)$ *such that*

$$J\left(u_k^*, v_k^*\right) = \min_{(u_k, v_k) \in U_{ad}} J\left(u_k, v_k\right).$$

subjet to the control system (2).

Proof. Since the coefficients of the state equations are bounded and there are a finite number of time steps C, S^a, S^b, I and R are uniformly bounded for all (u_k, v_k) in the control set U_{ad}; thus $J\left(u_k, v_k\right)$ is bounded for all $(u_k, v_k) \in U_{ad}$.
Since $J\left(u_k, v_k\right)$ is bounded,

$$\inf_{(u_k, v_k) \in U_{ad}} J\left(u_k, v_k\right).$$

is finite, and there exists a sequence $\left(u_k^n, v_k^n\right) \in U_{ad}$ such that

$$\lim_{n \to +\infty} \left(u_k^n, v_k^n\right) = \inf_{(u_k, v_k) \in U_{ad}} J\left(u_k, v_k\right),$$

and corresponding sequences of states

$$C^n \to C,$$

$$S^{a,n} \to S^a,$$

$$S^{b,n} \to S^b,$$

$$I^n \to I,$$

$$R^n \to R,$$

Since there is a finite number of uniformly bounded sequences, there exist $\left(u_k^*, v_k^*\right) \in U_{ad}$ and $\left(C^*, S^{a,*}, S^{b,*}, I^*, R^*\right) \in \mathbb{R}^{t_{\text{end}}+1}$ such that, on a subsequence,

$$\left(u_k, v_k\right) \to \left(u_k^*, v_k^*\right).$$

Finally, due to the finite dimensional structure of system (2) and the objective function $J(u_k, v_k)$, $\left(u_k^*, v_k^*\right)$ is an optimal control with corresponding states $\left(C^*, S^{a,*}, S^{b,*}, I^*, R^*\right)$. Therefore

$$\min_{(u_k, v_k) \in U_{ad}} J(u_k, v_k).$$

□

In order to derive the necessary condition for optimal control, the pontryagins maximum principle in discrete time. This principle converts into a problem of minimizing a Hamiltonian H at time step k defined by:

$$H_k = L\left(C_k, S_k^a, S_k^b, I_k, R_k\right) + \sum_{i=1}^{5} \lambda_{i,k+1} f_{i,k+1}, \tag{6}$$

where $f_{i,k+1}$ is the right side of the system of difference equations (2) of the i^{th} state variable at time step $k+1$

$$L\left(C_k, S_k^a, S_k^b, I_k, R_k\right) = a_k C_k + b_k S_k^a + c_k S_k^b + d_k I_k - e_k R_k + A_1^k \frac{u_k^2}{2} + A_2^k \frac{v_k^2}{2}. \tag{7}$$

Theorem 3.2. *Given an optimal control $\left(u_k^*, v_k^*\right)$ and the solutions C_k^*, $S_k^{a,*}$,$S_k^{b,*}$, I_k^* and R_k^* of the corresponding state system (2), there exist adjoint functions $\lambda_{1,k}, \lambda_{2,k}, \lambda_{3,k}$, and $\lambda_{4,k}$ satisfying*

$$\lambda_{1,k} = a_k + \lambda_{1,k+1}\left[1 + \mu - 2\mu\frac{C_k}{K} - \varepsilon_1 u_k\right] + \lambda_{2,k+1}\alpha S_k^a - \lambda_{3,k+1}\beta S_k^b + \lambda_{4,k+1}\left(\alpha S_k^a + \beta S_k^b\right),$$

$$\lambda_{2,k} = b_k + \lambda_{2,k+1}\left[1 - \alpha C_k - \mu - \gamma\right] + \lambda_{4,k+1}\alpha C_k + \lambda_{5,k+1}\gamma,$$

$$\lambda_{3,k} = -c_k + \lambda_{3,k+1}\left[1 - \beta C_k - \mu\right] + \lambda_{4,k+1}\beta C_k,$$

$$\lambda_{4,k} = d_k + \lambda_{4,k+1}\left[1 - (\mu + d) - \xi - \varepsilon_2 v_k\right] + \lambda_{5,k+1}\left[\xi + \varepsilon_2 v_k\right],$$

$$\lambda_{5,k} = -e_k + \lambda_{3,k+1}\theta + \lambda_{5,k+1}\left[1 - \theta - \eta\right],$$

With the transversability conditions at time $N, \lambda_{1,N} = \lambda_{5,N} = 0, \lambda_{2,N} = B_N, \lambda_{3,N} = A_N, \lambda_{4,N} = I_N$ and $\lambda_{5,N} = R_N$.

Furthermore, for $k = 0, 1, 2 \ldots N-1$ the optimal controls u_k^*, v_k^*, and w_k^* are given by

$$u_k^* = \min\left\{\max\left\{\frac{1}{A_1^k}\lambda_{1,k+1}\lambda\varepsilon_1 C_k^*, 1\right\}, 0\right\}. \tag{8}$$

$$v_k^* = \min\left\{\max\left\{\frac{1}{A_2^k}(\lambda_{4,k+1} - \lambda_{5,k+1})\varepsilon_1 I_k^*, 1\right\}, 0\right\}. \tag{9}$$

Proof. The Hamiltonian at time step k is given by

$$\begin{aligned} H_k = \quad & L(S_k I_k, R_k, C_k) + \lambda_{1,k+1}\left(C_k + \mu C_k\left(1-\frac{C_k}{K}\right) - \varepsilon_1 u_k C_k\right) \\ & + \lambda_{2,k+1}\left(S_k^a - \alpha S_k^a C_k - \eta S_k^a - \gamma S_k^a\right) \\ & + \lambda_{3,k+1}\left(S_k^b - \beta S_k^b C_k - \eta S_k^b + \theta R_k\right) \\ & + \lambda_{4,k+1}\left(I_k + \alpha S_k^a C_k + \beta S_k^b C_k - (\eta + d) I_k - \varepsilon_2 v_k I_k - \xi I_k\right) \\ & + \lambda_{5,k+1}\left(R_k + \gamma S_i^a - (\eta + \theta) R_k + \xi I_k + \varepsilon_2 v_k I_k\right) \end{aligned}$$

For $k = 0, 1 \ldots N-1$ the optimal controls u_k, v_k, w_k can be solved from the optimality condition,

$$\begin{aligned} \frac{\partial H_k}{\partial u_k} &= 0, \\ \frac{\partial H_k}{\partial v_k} &= 0, \end{aligned} \tag{10}$$

that are

$$\begin{aligned} \left.\frac{\partial H_k}{\partial u_k}\right|_{u_k = u_k^*} &= A_1^k u_k^* - \lambda_{1,k+1}\varepsilon_1 C_k = 0, \\ \left.\frac{\partial H_k}{\partial v_k}\right|_{v_k = v_k^*} &= A_2^k v_k^* + \left(\lambda_{5,k+1} - \lambda_{4,k+1}\right)\varepsilon_2 I_k^* = 0, \end{aligned}$$

we get:

$$\begin{aligned} u_k^* &= \frac{1}{A_1^k}\lambda_{1,k+1}\lambda\,\varepsilon_1 C_k^*, \\ v_k^* &= \frac{1}{A_2}\left(\lambda_{4,k+1} - \lambda_{5,k+1}\right)\varepsilon_1 I_k^*. \end{aligned} \tag{11}$$

□

4. NUMERICAL SIMULATION

4.1. Numerical simulation without control. In this section, we present the results obtained by solving numerically the optimality system. This system consists of the state system, adjoint system, initial and final time conditions, and the controls characterization. So, the optimality system is given by the following. We firstly consider the numerical simulation of the C-SIRS model when $\gamma = 0.01, \beta = 0.00001, \alpha = 0.00001, \mu = 0.09$, and $\eta = 0.01$. In our simulation,

we also assume that population size is constant and the natural mortality rate of individuals is equal to the birth rate. The figure 2 represent the evolution of the five populations (susceptible a,susceptible b, influenced, recovered, *Fusarium oxysporum* fungi) in the uncontrolled situation. Following this figure, we can clearly shows that when the level of the *Fusarium oxysporum* fungi population increases the level of infected population increases too, and for the recovered it decreases. So, we can deduce the importance of controlling these populations.

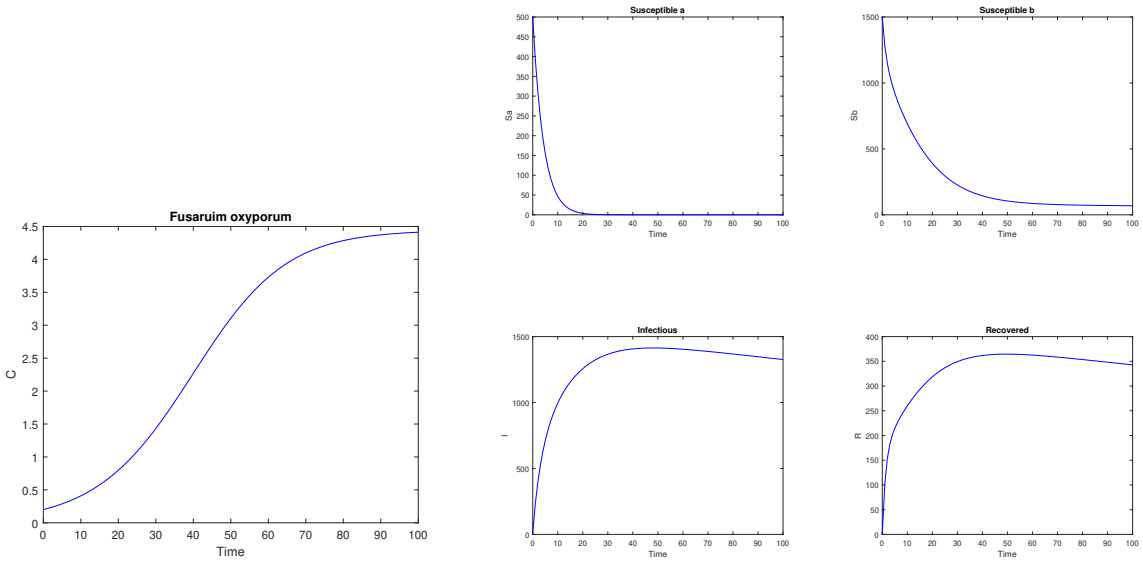

FIGURE 2. Evolution of the five populations in the uncontrolled situation

4.2. Numerical simulation with control. In this part of numerical simulation, we deal with three cases. Firstly, we apply a single control. Secondly, two controls. In the case of a single control, we start with the optimal control v that is applied to the infected populationin order to minimizing this population. Next, we discuss the results obtained by applying control w that has as purpose the minimization of the *Fusarium oxysporum* fungi population. Then, we move on to show the importance of increasing the number of controls in the study.

Resistant plants to *Fusarium oxysporum* and the genetic control:

The first strategy to control *Fusarium oxysporum* is to select disease-resistant material of good plant quality and to use the resistance of certain plants varieties to *Fusarium oxysporum* [7]. The search for more resistant plants is also the general direction taken to control vascular

Fusarium of the different cultivated plants. This method has the disadvantage of being long to implement, not allowing the conservation of certain sensitive varieties that are highly appreciated for their yield and the quality of their fruit [24].

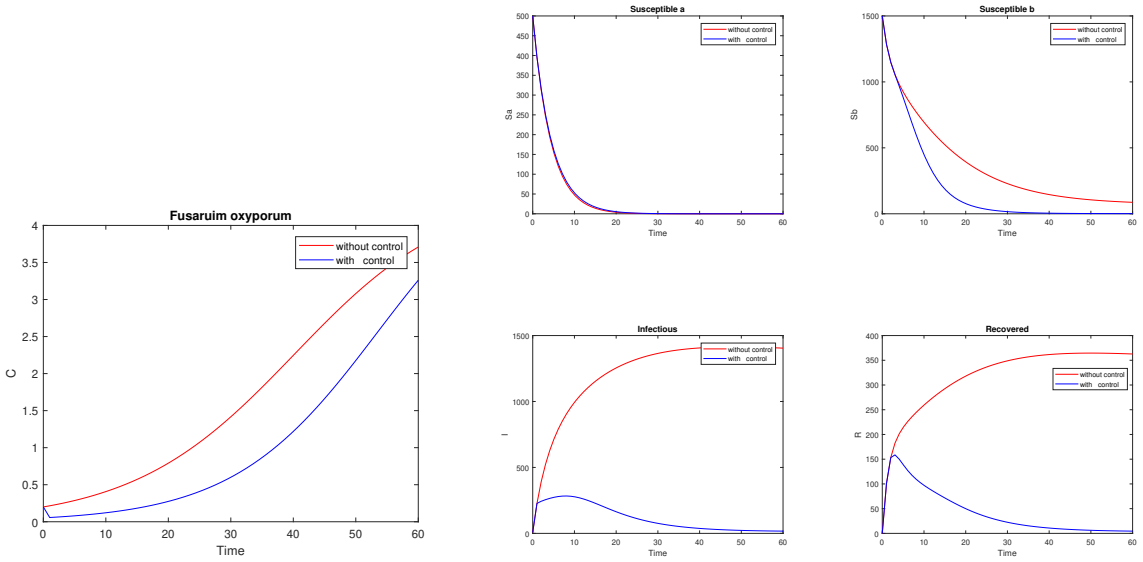

FIGURE 3. C-SIRS model with control u.

As another solution, the world has rallied around a genetic fight against the disease[1][5]. A second strategy in the field has been to induce the plant's defense reactions using salicylic acid (SA)[17].

This led to a significant reduction in mortality rates of plants inoculated . This result was obtained in correlation with a marked increase in the content of phenolic compounds, H2O2 and malondialdehyde on the one hand and phenylalanine ammonia-lyase and peroxidase activities on the other. In addition, we noted that activation of these components of plant resistance by SA was greater after inoculation of the pathogen [19]. Also, a localized necrosis, reminiscent of the necrosis formed during the establishment of the hypersensitive reaction (HR), was formed at the site of infection. It was positively correlated with the accumulation of H2O2 and malonyldialdehyde and with the establishment of resistance. Furthermore, analysis of phenolic compounds by HPLC, led to the identification of caffeoylshikimic acid isomers constitutively

present in plant roots. When these roots are treated by salicylic acid and inoculated, new phenolic compounds identified as hydroxycinnamic acid derivatives with strong antifungal activity were induced. Their accumulation is thought to be responsible for the improvement in the resistance of plant, obtained following treatment with SA. The histochemical revelation of phenolic compounds in the plant root tissues revealed flavonoids in the cell walls of the vascular parenchyma of the xylem [15]. These flavonoids are only expressed in the roots which are both treated by the SA and inoculated by the pathogen, and would be at the origin of the cytological alterations observed in the Foa mycelium, in electronog raphe of plant roots.

According to Figure 3, the control of the infected population leads to the following results: A decrease in the susceptible population that converges to 0. A significant increase in the infected population that exceeds the level reached in the situation without control. An increase, in an interval with a small amplitude, of the recovered, followed by their decrease, but they always remain higher than the number of recovered in the case without control. An increase in *Fusarium oxysporum* level.

Chemical control:

Fungicides are pesticides that kill or inhibit the fungi responsible for certain diseases. On the other hand, not all fungal diseases can be controlled with a fungicide, we can think of vascular diseases such as *Fusarium*. So the second strategy is the use of fungicides with systemic or endotherapeutic action.

This method is ruled out, as the practical possibilities for the use of systematic fungicides for tracheomycoses are very limited [21]. In addition, these products are not very stable in the soil and may favor the selection of resistant strains [22]. If used repeatedly over many years, these chemicals can harm the environment [28].

In the figure 4 , we illustrate the results obtained after applying the control v on the *Fusarium oxysporum* fungi population. Here, the alarming result is that even if we apply this control for minimizing the *Fusarium oxysporum* fungi population over time, this last one still increasing. However, the number of susceptible is minimized, it decreases to zero. The number of infected becomes lower than that in the case without control, it does not exceed 200. The recovered

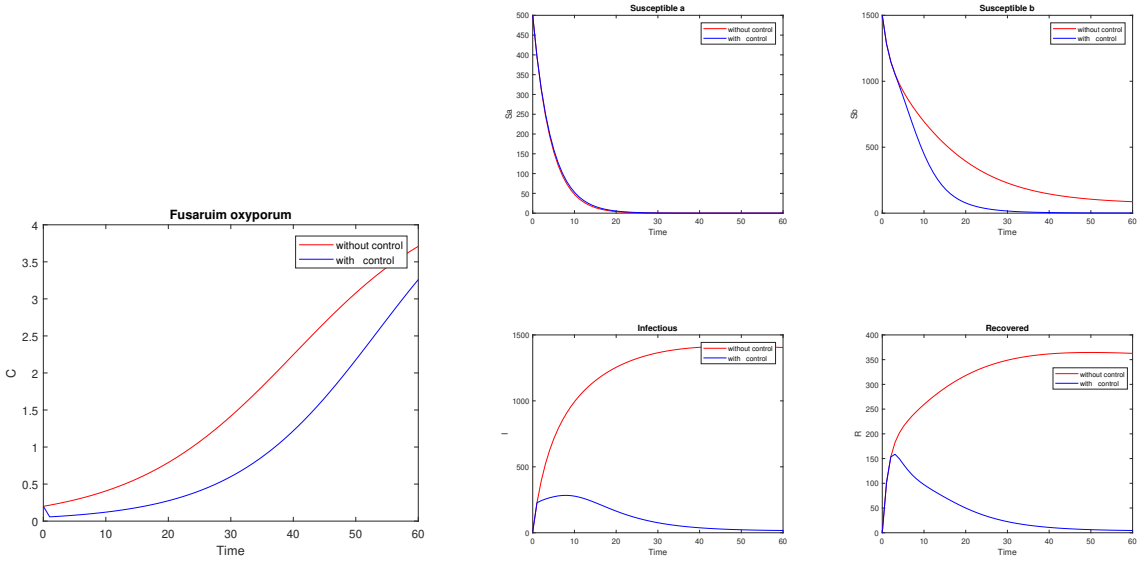

FIGURE 4. C-SIRS model with control v.

increases and after a certain time it decreases so as not to exceed the number of recovered in the case without control.

Resistant plants to *Fusarium oxysporum*, the genetic control and chemical control:

Now we move to the case of the two controls u and v. By figure 5, we show that, in this case, the susceptible population is minimized and converges to zero. The infected population is minimized too and it not exceeds the threshold of 200. The recovered population decreases by staying lower than the number of recovered in the uncontrolled situation. The *Fusarium oxysporum* fungi population is minimized by attaining values less than in the uncontrolled situation.

We can deduce that the results obtained in this case are more convincing than those of the case of a single control v. Because, the control w alone leads to an increase in the number of the *Fusarium oxysporum* fungi population, however, when a second control is added, we can minimize the number of individuals of this population. And the same goes for the susceptible and the infected controls.

5. CONCLUSION

This study evaluated the performance of combination of use the resistance of certain plants varieties, the genetic and chemical control remains the most effective way to fight *Fusarium*

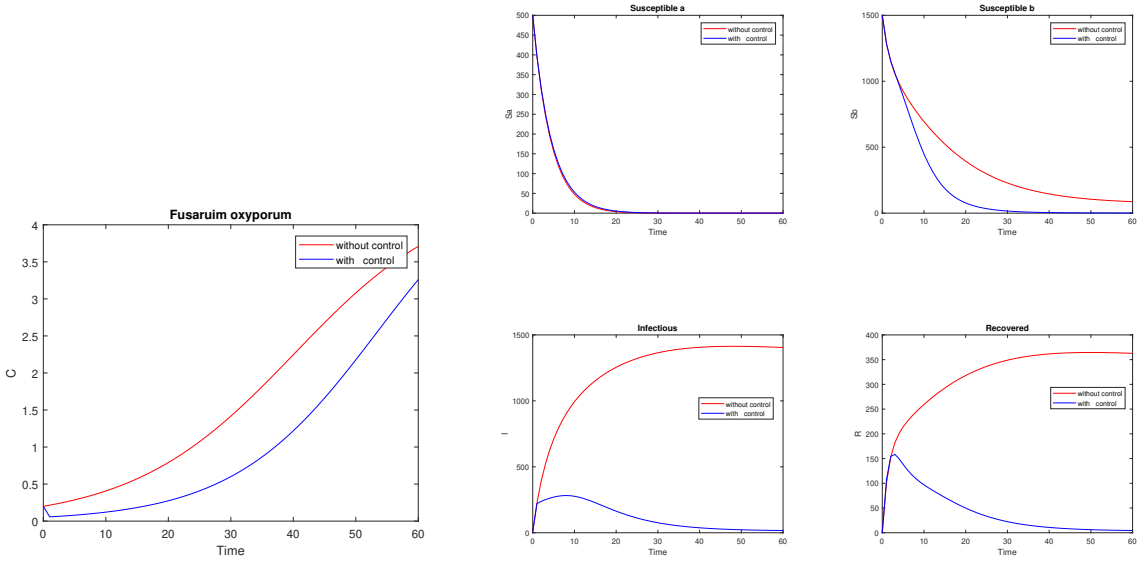

FIGURE 5. C-SIRS model with controls u and v.

oxysporum disease. We plan to apply the approach developed in this paper to *Fusarium oxysporum* fungi models in continuous time taking into account optimal variables.

CONFLICT OF INTERESTS

The authors declare that there is no conflict of interests.

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