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A Continuum Mathematical Modeling of Diseases Caused by Fusarium Oxysporum

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Abstract

In this work, we propose a continuum mathematical model that describes the disease caused by the fungus Fusarium oxysporum, which is spread by contact between plant roots and soil fungi Fusarium oxysporum; two types of susceptible plants S^a and S^b, infected palm (I), recovered palm (R), Fusarium oxysporum fungi (C). We also focus on the importance of treatments in order to find optimal strategies to minimize the number of infected and Fusarium oxysporum fungi and maximize the number of recovered plants under treatment. The results of this optimal system are solved numerically by Matlab. Therefore, the results obtained confirm the performance of the optimization strategy.

Keywords: continuum mathematical model; Fusarium oxysporum; optimal control; Optimization strategy .

1. INTRODUCTION

Fusarium oxysporum is the causal agent of Fusariosis, of Fusarium wilt and of root in many crop plants. 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 [54, 8, 6]. *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 [32]. 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 but not 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 which 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 [13] [36]. 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 [22] . 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.

During the first half of the 20th century, the disease decimated banana plantations in Central Africa and the Caribbean. It is considered to be the most important disease on bananas. The world banana market has only been saved by the introduction of the Cavendish cultivar, which is resistant to the pathogen [67]. Similarly, the development of *F. oxysporum F.* sp. vanillae in vanilla plantations is the main factor limiting world production [71]. 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*.

Currently, only pathogenicity tests on plants and verification of Koch's postulates allow the identification of the special form to which a strain belongs [40]. 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). [48]. 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 section, we consider a system of $C - SIRS$ with five compartments to describe the spatial spread of two types of plants infected by the fungus *Fusarium oxysporum*.

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

Table 1: Description of compartments used in model 1

C	Fungus Fusaruim oxyporum.
S^a	Plant trees belonging to the area that are not sick but likely to become sick
.	(young, resistant, good quality, ...).
S^b	Plant trees belonging to the area that are not sick but likely to become sick
.	(old, infected, poorly watered, rocky soil, ...).
I	Plant trees infected with Fusaruim oxyporum.
R	Plant trees that have already had the disease and are now immune to Fusaruim oxyporum.

Table 2: Description of parameters used in model 1

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.

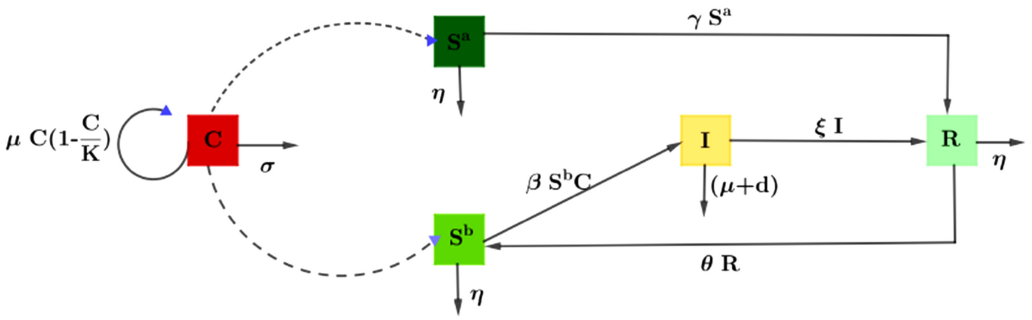

Figure 1: The C-SIRS model

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

$$\begin{cases} \dfrac{dC}{dt} = \mu C\left(1-\frac{C}{K}\right) - \sigma C, \\ \dfrac{dS^a}{dt} = -\eta S^a - \gamma S^a, \\ \dfrac{dS^b}{dt} = -\beta S^b C - \eta S^b + \theta R, \\ \dfrac{dI}{dt} = \beta S^b C - (\eta + d) I - \xi I, \\ \dfrac{dR}{dt} = \gamma S^a - (\eta + \theta) R + \xi I. \end{cases} \tag{1}$$

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 $-\gamma S_k^b C_k$. 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".

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. EQUILIBRIUM POINTS

The equilibrium points are defined by resuloving the system:

$$\begin{cases} \mu C\left(1-\frac{C}{K}\right)-\sigma C=0, \\ -\eta S^a-\gamma S^a=0, \\ -\beta S^b C-\eta S^b+\theta R=0, \\ \beta S^b C-(\eta+d)I-\xi I=0, \\ \gamma S^a-(\eta+\theta)R+\xi I=0. \end{cases} \tag{2}$$

Theorem 3.1. $E_0=(0,0,0,0)$,

The Jacobian matrix associated with an equilibrium point (C, S^a, S^b, I, R) is given by:

$$J(C,S^a,S^b,I,R)=\begin{pmatrix} \mu-\mu C/K-\sigma & 0 & 0 & 0 & 0 \\ 0 & -\eta-\gamma & 0 & 0 & 0 \\ -\beta S^b & 0 & -\beta C-\eta & 0 & \theta \\ \beta S^b & 0 & \beta C & -\eta-d-\xi & 0 \\ 0 & \gamma & 0 & \xi & -\eta-\theta \end{pmatrix}$$

3.1 Analysis stability

Theorem 3.2. *The system admit the following equilibrium point E_0 is stable if $\mu<\sigma$.*

Proof.

$$J(E_0)=\begin{pmatrix} \mu-\sigma & 0 & 0 & 0 & 0 \\ 0 & -\eta-\gamma & 0 & 0 & 0 \\ 0 & 0 & -\eta & 0 & \theta \\ 0 & 0 & 0 & -\eta-d-\xi & 0 \\ 0 & \gamma & 0 & \xi & -\eta-\theta \end{pmatrix}$$

Table 3: Parameters values

Parameter	Values
γ	0.01
β	0.00001
θ	0.00002
μ	0.09
η	0.01

the eigenvalues of the matrix $J(E_0)$ are

$\lambda_1 = \mu - \sigma, \;\; \lambda_2 = \eta - \gamma < 0, \;\; \lambda_3 = -\eta <, \;\; \lambda_4 = -\eta - d - \xi < 0, \;\; \lambda_5 = -\eta - \theta < 0.$

So the point $E_0 = (0, 0, 0, 0, 0)$ is an stable point if $\mu < \sigma$. □

3.2 Numerical simulation without control

We present in this section, 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 $\gamma = 0.01, \beta = 0.00001, \mu = 0.09$, and $\eta = 0.01$. We 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.

4. 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:

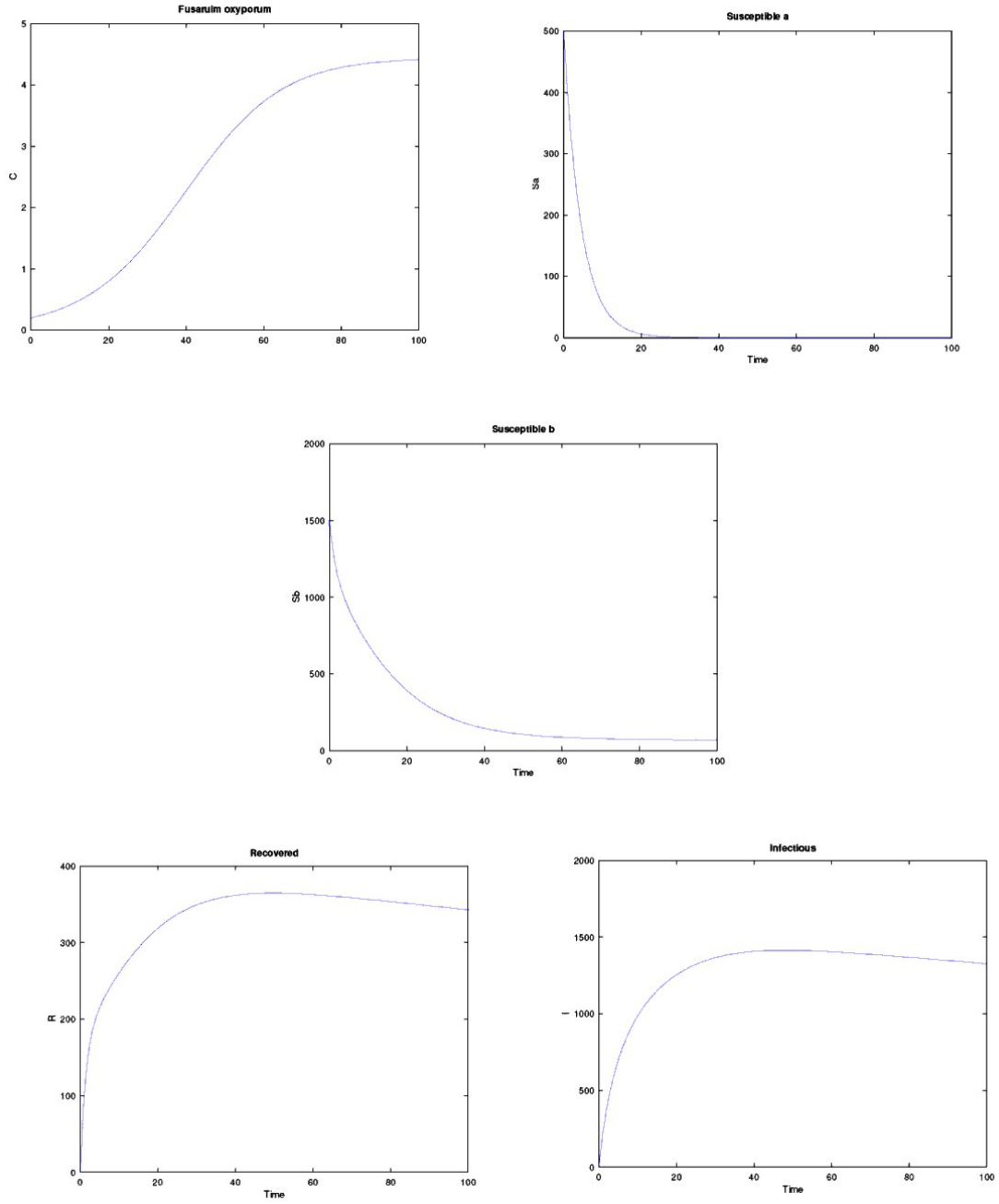

Figure 2: Without control

$$\begin{cases} \dfrac{dC}{dt} = \mu C\left(1 - \dfrac{C}{K}\right) - \varepsilon_1 u_1(t) C, \\ \dfrac{dS^a}{dt} = -\eta S^a - \gamma S^a, \\ \dfrac{dS^b}{dt} = -\beta S^b C - \eta S^b + \theta R, \\ \dfrac{dI}{dt} = \beta S^b C - (\eta + d) I - \varepsilon_2 v I - \xi I, \\ \dfrac{dR}{dt} = \gamma S^a - (\eta + \theta) R + \xi I + \varepsilon_2 u_2(t) I. \end{cases} \tag{3}$$

with

$$\epsilon_i = \begin{cases} 1 \\ 0 \end{cases} \quad \text{for } i = 1, 2$$

For exemple $\epsilon_1 = 1$ and $\epsilon_2 = 1$ means that we apply only a single control; $\epsilon_1 = 1$ and $\epsilon_2 \neq 1$ means that we apply two controls; $\epsilon_1 \neq 1$ and $\epsilon_2 \neq 1$ means that we apply three controls.

To minimize the number of infected individuals We consider the following objective functional:

$$J(u_1, u_2) = \int_{t_0}^{t_f} \left[I(t) + C(t) + R(t) + \frac{B_1}{2} u_1^2(t) + \frac{B_2}{2} u_2^2(t) \right] dt \tag{4}$$

here t_0 and t_f denote the initial and final time respectively. Suppose the cost being a quadratic nonlinear function of $u_i(t)(i = 1, 2)$, and B_1, B_2 being weight coefficients that reflect the value and importance of the cost. By the convexity of integrand in (...) with respect to $u_i(i = 1, 2)$, the boundedness and Lipschitz property of system $(...)$, it follows that the optimal control u_1^* and u_2^* exists such that. In other words, we search to determine the optimal controls u_k and v_k, such that

$$J(u_1^*, u_2^*) = \min_{U_{ad}} J(u_1, u_2)$$

where $U_{ad} = \{(u_1, u_2) \in L^1(t_0, t_f) \mid 0 \leq u_i \leq 1, i = 1, 2\}$. By the method of Pontryagin Maximum Principle, an adjoint system of differential equations with terminal boundary is generally appended to the original model, thus the optimality system consists of the differential equations of the original model, which is regarded as the state system, along with the adjoint equations, which are characterized by the

same number of equations as in the state system. In order to find the optimal solution, we give the Lagrange function of (...) as

$$L\left(C, S^a, S^b, I, R\right) = I(t) + C(t) + R(t) + \frac{B_1}{2}u_1^2(t) + \frac{B_2}{2}u_2^2(t)$$

and define the Hamilton function relating our control as

$$\begin{aligned} H =& L\left(C, S^a, S^b, I, R\right) + \sum_{i=1}^{5} \lambda_i f_i \\ =& L\left(C, S^a, S^b, I, R\right) + \lambda_1 \left[\mu C\left(1 - \frac{C}{K}\right) - \varepsilon_1 u_1(t) C\right] \\ &+ \lambda_2 \left[-\eta S^a - \gamma S^a\right] \\ &+ \lambda_3 \left[-\beta S^b C - \eta S^b + \theta R\right] + \lambda_4 \left[\beta S^b C - (\eta + d) I - \varepsilon_2 u_2(t) I - \xi I\right] \\ &+ \lambda_5 \left[\gamma S^a - (\eta + \theta) R + \xi I + \varepsilon_2 u_2(t) I\right], \end{aligned}$$

here $f_i (i = 1, 2, 3, 4)$ denoting the right hand side of $(...)$, and $\lambda_i (i = 1, 2, 3, 4)$ being the adjoint variables satisfying

$$\begin{aligned} \frac{d\lambda_1(t)}{dt} &= -\frac{\partial H}{\partial C} = -1 - \lambda_1 \left[\mu\left(1 - 2\frac{C}{K}\right) - \varepsilon_1 u_1(t)\right] + \lambda_3 \beta S^b - \lambda_4 \left[\beta S^b\right] \\ \frac{d\lambda_2(t)}{dt} &= -\frac{\partial H}{\partial S^a} = -\lambda_2 \left[-\eta - \gamma\right] - \lambda_5 \gamma \\ \frac{d\lambda_3(t)}{dt} &= -\frac{\partial H}{\partial S^b} = +\lambda_3 \beta C - \lambda_4 \beta C \\ \frac{d\lambda_4(t)}{dt} &= -\frac{\partial H}{\partial I} = -1 + \lambda_4 \left[\eta + d + \epsilon_2 u_2 + \xi\right] - \lambda_5 \xi \\ \frac{d\lambda_5(t)}{dt} &= -\frac{\partial H}{\partial R} = -1 - \lambda_3 \theta + \lambda_5 (\eta + \theta). \end{aligned}$$

Let the transversality conditions be $\lambda_i\left(t_f\right) = 0, i = 1, 2, 3, 4, 5$. The adjoint variables maximize or minimize the state variables with respect to the desired objective function. By the conditions of optimal control, we have

$$\begin{aligned} \left.\frac{\partial H}{\partial u_1}\right|_{u_1 = u_1^*} &= B_1 u_1^* - \lambda_1 \epsilon_1 C^* = 0, \Rightarrow u_1^* = \frac{\epsilon_1}{B_1} \lambda_1 C^* \\ \left.\frac{\partial H}{\partial u_2}\right|_{u_2 = u_2^*} &= B_2 u_2^* - \lambda_4 \epsilon_2 I^* + \lambda_5 \epsilon_2 I^* = 0, \Rightarrow u_2^* = \frac{\epsilon_2}{B_2} \left(\lambda_4 - \lambda_5\right) I^*. \end{aligned}$$

$$
\begin{aligned}
u_1^* &= \max\left\{\min\left\{\frac{\epsilon_1}{B_1}\lambda_1 C^*, 1\right\}, 0\right\} \\
u_2^* &= \max\left\{\min\left\{\frac{\epsilon_2}{B_2}\left(\lambda_4 - \lambda_5\right) I^*, 1\right\}, 0\right\}
\end{aligned} \tag{5}
$$

5. NUMERICAL SIMULATION WITH CONTROL

In this part of numerical simulation, we deal with three cases. 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 [17]. 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 [70].

As another solution, the world has rallied around a genetic fight against the disease[4][12]. A second strategy in the field has been to induce the plant's defense reactions using salicylic acid (SA)[53].

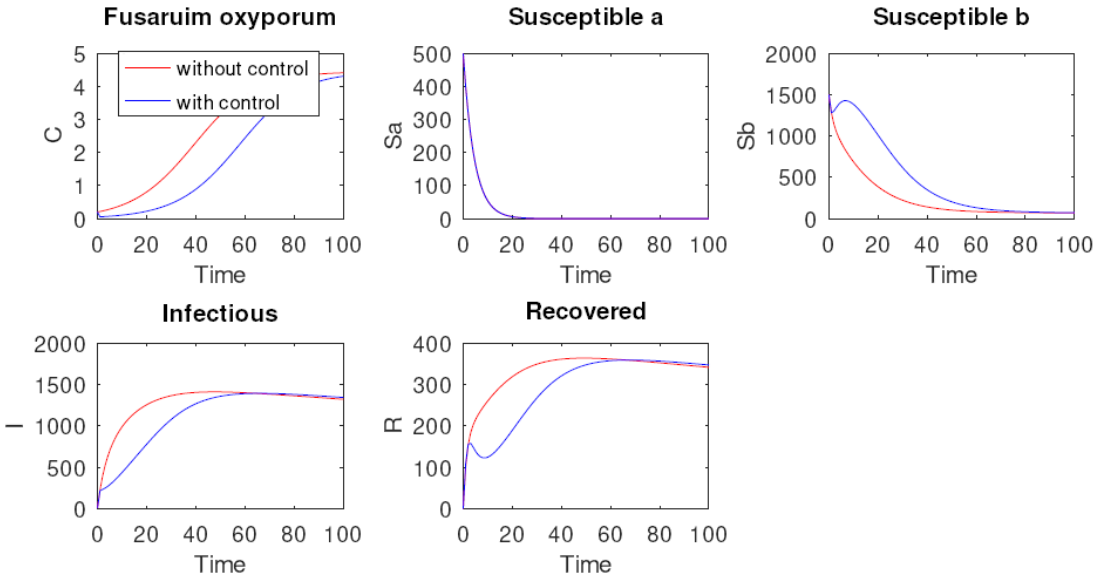

Figure 3: With and without control

In the figure 3, we illustrate the results obtained after applying the control on the Fusarium oxyporum 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 but in the case with control the number of infected increases over time, after a certain time (after 50) it becomes equal to the number of infected in the case without control (almost 1500).
The recovered increases and after a certain time it decreases to 100, but after 20 it increases again without exceeding the number of recovered in the case without control.

6. CONCLUSION

Despite their non-application in infected areas on a large scale in this work, the plants affected by Fusarium oxysporum are many and occupy a large area in the world, our study show that the reproduction of plants resistant to the disease of Fusarium oxysporum remains the most adequate means for the technical management of infected plants in areas of expansion. The early detection of plants infected by the pathogen, destroy or control them, are also factors that contribute to this control strategy. This could help protect plants from the effects of the pest disease to protect investments in the long term.

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