A DELAY MATHEMATICAL MODEL FOR THE DYNAMICS OF OLIVE TREES INFECTED BY XYLELLA FASTIDIOSA

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In this work, we study the dynamics of a mathematical model de-scribing the spread of Xylella fastidiosa disease in an olive orchard. We begin by in-troducing an age-structured model composed of two equations: an ordinary differential equation (ODE) describing the evolution of healthy olive trees, and a partial differential equation (PDE) modeling infected olive trees according to their age of infection. Refer-ring to the work of P. Magal ([1]), this system is reduced to a delay differential equations model, where a delay term appears on the infected olive trees variable. We show that this model is well-posed and establish the existence of two equilibrium points: the disease-free equilibrium and the endemic equilibrium. An analysis of local stability is carried out, and for the disease-free equilibrium, a global stability analysis is performed using a Lyapunov-Krasovskii functional, using the basic reproduction number $\mathcal{R}_0$ depending on the delay τ . This number depends also in particular on the implantation rate Λ as well as on the mortality rates of healthy, infected and uprooted olive trees. Numerical simulations illustrate the theoretical results.

Keywords: hybrid mathematical model, delay differential equations, local and global stability, Lyapunov-Krasovskii functional.

1. Introduction

Infectious diseases represent a major threat to ecosystems either human, animal or plant, to public health and to the world's economy. The study of their spread has become a major preoccupation in a number of fields, notably the mathematical modeling, which proves its worth in understanding and predicting the evolution of diseases and suggesting effective control strategies to eradicate them. Mathematical modeling enables us to see the dynamics of epidemic spread through systems of ordinary differential equations, or partial differential equations, with crucial applications in phytopathology, i.e. plant diseases ([15]; [17]).

In plant epidemiology, diseases cause major losses for farmers and the global economy. Several factors, such as climate change and the use of chemical substances, as well as certain insect pests, contribute to the spread of infectious agents. One of the most emblematic example of destructive bacterium is *Xylella Fastidiosa*.

Xylella Fastidiosa is a Gram-negative bacterium that poses a considerable threat, as it can

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cause disease in the risk assessment zone, once established. In the presence of hosts and given the right environmental conditions, *Xylella Fastidiosa* is capable of infecting a variety of crops in Europe, including, for example, citrus, grapevine, pumpkin, olive and stone fruits such as almonds, peaches and plums. It can also affect a variety of ornamental plants such as oaks, sycamores and oleanders. It feeds on a wide variety of hosts, including many cultivated and wild plants ([5]; [11]).

The olive tree (*Olea europaea*), a symbol of peace and sustainability due to its exceptional longevity, an emblematic tree of the mediterranean basin, has been an essential part of the existence of mediterranean civilizations since immemorial time. It is the mainstay of many rural economies, the basis of age-old agri-food traditions. It has constantly satisfied various basic needs of daily life, thanks in particular to the oil derived from its fruit, which is one of the most appreciated oils for its nutritional, cosmetic and medicinal virtues. It is therefore considered an aromatic and medicinal tree, rich in natural compounds with proven benefits ([14]). In Algeria, the olive is the predominant fruit species in terms of planting, accounting for around two-thirds of the national olive orchard.([14]).

Xylella Fastidiosa has wreaked havoc in olive groves and is destroying this balance. The bacterium infects the plant's xylemic tissues, blocking the circulation of raw sap, causing leaf dryness, branch necrosis and ultimately tree death ([18]). Insect vectors, in particular the leafhopper *Philaenus spumarius*, ensure transmission between trees, making the epidemic difficult to contain. The ecological impact is also very significant, with the disappearance of trees, loss of biodiversity, weakening of soils and permanent modification of landscapes. Added to this is the disastrous economic impact for olive and olive oil producers, some of whom have lost their entire farms ([20]).

The spread of *Xylella Fastidiosa* in olive groves requires effective strategies to reduce its evolution. This is where mathematical modeling represents an essential tool. Thanks to mathematical models, we can describe the dynamics of the interaction between plants and the vectors that transmit this bacterium. These models, often inspired by the S-I, SIR or SEIR systems used in human epidemiology, are adapted to the plant world, taking into account the long incubation period, the role of vectors and the influence of climatic factors. ([4]).

In the case of this destructive bacterium, numerous mathematical studies have already explored the possibility of spatial and temporal control of the epidemic. These take into account vector-plant transmission dynamics, insect mobility, olive tree density and the effects of sanitary measures (felling, treatment, replanting). They are particularly useful for testing the impact of different strategies (see [7];[8]; [9], [19]; [21]).

The aim of this work is to propose an epidemiological model for olive trees under the spread of *Xylella Fastidiosa*, taking into account the biological peculiarities of the disease. It is well established that transmission of this bacterium is essentially via insect vectors, which play a crucial role in the dissemination of the pathogen between host plants. However, in this manuscript, we have chosen to explicitly neglect vector dynamics in a first approach, in order to simplify the model and better understand the epidemic mechanisms specific to the olive tree population. This assumption is based on the fact that the biological dynamics of insects vary over short timescales of a few weeks to months, while those of olive trees evolve over several years. Therefore the two populations live on very different timescales. By thinking in terms of long time steps, we can therefore consider that the aggregate effect of vectors can be implicitly integrated into the transmission rate between olive trees, without

explicitly modeling their population. This simplification will enable us to build a more accessible analytical model, without losing sight of the objective of a later extension to a model explicitly coupling hosts and vectors as already done in ([9]; [19]).
The paper is organized as follows: we begin by formulating an age-structured model of the dynamics of olive trees infected by *Xylella Fastidiosa* which looks like the one studied by Pierre Magal and Arnaud Ducrot [1]; [27]. Our approach consists in reducing this system in the next section to a system of delay differential equations. Then we study this system obtained from the model, showing in the third section that the problem is well posed. The existence of equilibrium points and their stability study is performed in the fourth, fifth and sixth sections. Finally, we present numerical simulations and conclusion in the last two sections.

2. The mathematical model

To begin with, we construct a hybrid mathematical model structured in terms of age of infection.
Consider a population of olive trees, otherwise in good health, of size $N(t)$ at time t_0. The main variables in our description are,

- $S(t)$: The density of sensitive olive trees at the time t.
- $i(t,a)$: The density of infected olive trees over time t and age a.

The initial conditions of sensitive and infected olive trees are given by S_0 and $i_0(a)$ respectively.
The variable a denote the age of the disease (age of infection), *i.e.* the time that has elapsed since the infected olive trees became infected.
Moreover, we introduce,

- Λ: The density of new olive trees (implanted).
- μ_s: The mortality rate of healthy olive trees (felling or natural mortality).
- μ_i: The mortality rate of olive trees infected with the *Xylella Fastidiosa.*
- v_0: The rate of removal of infected olive trees, and α, the probability of infectivity that varies as the disease progresses in an infected olive tree.

Then, the model can be written as

$$\begin{cases} \dfrac{dS}{dt} = \Lambda - \mu_s S - S\int_0^A \alpha(a) i(t,a)da, & t \in (0,T), \\ \dfrac{\partial i(t,a)}{\partial t} + \dfrac{\partial i(t,a)}{\partial a} = -\mu_i(a) i(t,a) - v_0 i(t,a), & (t,a) \in (0,T)\times(0,A), \\ i(t,0) = S\int_0^A \alpha(a) i(t,a)da, & t \in (0,T), \\ i(0,a) = i_0(a), \quad a \in (0,A), \\ S(0) = S_0. \end{cases} \tag{1}$$

Note that $I = \int_0^A i(t,a)da$ is the total population of infected trees, and the initial condition i_0 is positive and in $(L^1 \cap L^\infty)(0,A)$.
This type of structuring, widely used in the modeling of chronic diseases, makes it possible to take into account the biological delay between initial infection and transmission capacity, or the onset of symptoms. However, this hybrid mathematical model can be simplified into a system while retaining the memory of the infectious process. We proceed by a reduction

to a delay model, in line with the approach formalized by ([1]).
Integrating the second equation of (1) with respect to age,we get:

$$\int_0^A [\frac{\partial i(t,a)}{\partial t} + \frac{\partial i(t,a)}{\partial a}]da = -\int_0^A (\mu_i(a) + v_0)i(t,a)da$$

so the following equation is obtained

$$\frac{dI}{dt} = -i(t,A) + i(t,0) - \int_0^A (\mu_i(a) + v_0)i(t,a)da$$

Substituting the boundary conditions, it follows that

$$\frac{dI}{dt} = -i(t,A) + S(t)\int_0^A \alpha(a)i(t,a)da - \int_0^A (\mu_i(a) + v_0)i(t,a)da \tag{2}$$

Using the characteristics of the PDE equation of system (1), by varying the variables t and a, we have

$$i(t,a) = i(t(s),a(s)) := U(s)$$

So,

$$\frac{dU}{ds} = \frac{\partial i}{\partial t}\frac{\partial t}{\partial s} + \frac{\partial i}{\partial a}\frac{\partial a}{\partial s} = -(\mu_i(a) + v_0)U(s)$$

With,

$$\frac{\partial t}{\partial s} = 1, \quad t(0) = t_0, \qquad \frac{\partial a}{\partial s} = 1, \quad a(0) = a_0$$
$$t(s) = s + t_0 \qquad\qquad a(s) = s + a_0$$

Then, $t(s) - a(s) = t_0 - a_0$. Also we have,

$$U(s) = U(0)e^{-\int_0^s (\mu_i(\tau+a_0)+v_0)d\tau} \tag{3}$$

where $U(0) = i(t_0,a_0)$. Along the characteristics, $i(t,a) = i(t_0,a_0)e^{-\int_0^s (\mu_i(\tau+a_0)+v_0)d\tau}$.
For $t_0 = 0$ and $a_0 > 0$, we have $t < a$, we have $t = s$ and $a = s + a_0$.We obtain

$$\begin{aligned} i(t,a) &= i(0,a-t)e^{-\int_0^t (\mu_i(\tau+a-t)+v_0)d\tau} \\ &= i_0(a-t)e^{-\int_0^t (\mu_i(\tau+a-t)+v_0)d\tau} \end{aligned} \tag{4}$$

And for $t_0 > 0$ and $a_0 = 0$, we have $t > a$, we have $a = s$ and $t = s + t_0$. We obtain

$$\begin{aligned} i(t,a) &= i(t-a,0)e^{-\int_0^a (\mu_i(\tau)+v_0)d\tau} \\ &= S(t-a)\int_0^A \alpha(\tau)i(t-a,\tau)d\tau e^{-\int_0^a (\mu_i(\tau+a-t)+v_0)d\tau} \end{aligned} \tag{5}$$

Which brings us back to,

$$i(t,a) = \begin{cases} i_0(a-t)\exp(-\int_0^t (\mu_i(a-t+\tau)+v_0)d\tau), & t \le a \\ S(t-a)\int_0^A \alpha(\tau)i(t-a,\tau)d\tau \exp(-\int_0^a (\mu_i(s)+v_0)ds, & t > a \end{cases} \tag{6}$$

Let assume that ([1]),

$$\mu_i(a) = \mu_i > 0, \ \forall a \ge 0, \qquad \alpha(a) = 1_{[\tau,A]}(a), \ \tau > 0, \qquad i(t,A) = 0, \ \forall t \ge 0.$$

Then, the system (1) can be written as a delay model as follows (see [1]),

$$\begin{cases} \dfrac{dS}{dt} = \Lambda - \mu_s S(t) - S(t)I(t-\tau)e^{-(\mu_i+v_0)\tau}, \\ \dfrac{dI}{dt} = S(t)I(t-\tau)e^{-(\mu_i+v_0)\tau} - (\mu_i+v_0)I(t), \\ S(0) = S_0, \quad I(0) = \int_0^A i_0(a)da \\ S(t) = \varphi_1(t), \quad I(t) = \varphi_2(t), \qquad ,t \in [-\tau, 0] \end{cases} \tag{7}$$

Assume that the initial condition i_0 of the age-structured system (1) is continuous on $[0, \tau]$. Then the initial condition of (7), (φ_1, φ_2), that are in $C([-\tau, 0], \mathbb{R}^2_+)$ can be taken as (see[26])

$$\varphi_1(t) = S_0 \quad \text{and} \quad \varphi_2(t) = i_0(-t)e^{-(\mu_i+v_0)t}, \quad t \in [-\tau, 0] \tag{8}$$

This model corresponds to a S-I type system with delay. It comprises two compartments,

- $S(t)$: density of susceptibles healthy olive trees at time t.
- $I(t)$: density of infected (non-curable) olive trees at time t.

The parameters are those from model (1) with,

- τ: time between infection and a tree becoming infectious (or symptomatic).
- $e^{-(\mu_i+v_0)\tau}$ probability that a tree infected τ time units ago is still alive and infectious.

The delay formulation of the infection, results from the integration of the infected density into the hybrid model, using the method of characteristics. Thus, the term $S(t)I(t-\tau)e^{-(\mu_i+v_0)\tau}$ expresses the current flow of infection induced by individuals infected τ time ago and who have survived mortality and culling until present time.

3. Existence, positivity and uniqueness of solutions

In this section we will prove the existence and uniqueness of the solution of the model (7) and see if it is positive.

We note that the right-hand side of each equation of the system (7) is a function of class C^1. Morover, I is continuous for all $t \geq -\tau$ if and only if the initial condition $\varphi_2(t)$ satifies the compatibility condition, $\varphi_2(0) = i_0(0) = S_0\varphi_2(-\tau)e^{-(\mu_i+v_0)\tau}$, so according to the existence and uniqueness theorem (see [22]), for all initial condition (φ_1, φ_2), the solution to system exists and is unique.

Moreover for $S(t_0) = 0$, we have from the first equation in (7), $S'(t_0) = \Lambda > 0$, and therefore $S(t)$ is a strictly increasing function, so if $S_0 > 0$, we get $S(t) > 0$ for all $t > 0$.

And for $I(t_0) = 0$, as $S(t_0) > 0$, from the second equation we obtain $I'(t_0) > 0$, and so if $I_0 > 0$, we get $I(t) > 0$ for all $t > 0$.

Introduce

$$N(t) = S(t) + I(t) \tag{9}$$

When we calculate the time derivative of N,we get

$$\begin{aligned} N'(t) &= S' + I' \\ &= \Lambda - \mu_s S - (\mu_i + v_0)I \\ &\leq \Lambda - \sigma N(t) \\ N(t) &\leq (N(0) - \frac{\Lambda}{\sigma})e^{-\sigma t} + \frac{\Lambda}{\sigma}. \end{aligned} \tag{10}$$

where $\sigma = \min_{t\in[0,T]} \{\mu_s, (\mu_i + v_0)\}$.

It follows that,

- If $N(0) \leq \dfrac{\Lambda}{\sigma}$, then $N(t) \leq \dfrac{\Lambda}{\sigma} \quad \forall t \geq 0$.
- If $N(0) \geq \dfrac{\Lambda}{\sigma}$, then $N(t) \leq N(0) \quad \forall t \geq 0$.

So $N(t) \leq \max(N(0), \dfrac{\Lambda}{\sigma})$. From there, we conclude that $N(t)$ is a bounded function, consequently S and I are a bounded functions. This leads us to the following theorem

Theorem 3.1.
The system (7) has a unique positive global solution $(S(t), I(t))$.

4. Existence of equilibrium points

In this section, we analyze the existence of equilibrium points of (7), denoted by (S^*, I^*).
For $\dfrac{dS}{dt} = 0$ and $\dfrac{dI}{dt} = 0$, the system (7) gives

$$\begin{cases} \Lambda - \mu_s S^* - S^* I^* e^{-(\mu_i + v_0)\tau} = 0 \\ S^* I^* e^{-(\mu_i + v_0)\tau} - (\mu_i + v_0) I^* = 0 \end{cases} \tag{11}$$

If $I^* = 0$, we obtain the disease-free equilibrium $E_0 = \left(\dfrac{\Lambda}{\mu_s}, 0\right)$.
If $I^* > 0$, we obtain, from the second equation, $S^* = (\mu_i + v_0) e^{(\mu_i + v_0)\tau}$. By injecting it into the first equation, we find

$$I^* = \frac{\Lambda - \mu_s (\mu_i + v_0) e^{(\mu_i + v_0)\tau}}{(\mu_i + v_0)}.$$

So we have the endemic equilibrium

$$E_1 = \left((\mu_i + v_0) e^{(\mu_i + v_0)\tau}, \frac{\Lambda - \mu_s S^*}{\mu_i + v_0} \right)$$

.
Note that the endemic equilibrium exist if and only if, $I^* > 0$ which means if and only if $\Lambda > \mu_s (\mu_i + v_0) e^{(\mu_i + v_0)\tau}$.
Define,

$$\mathcal{R}_0(\tau) := \frac{\Lambda e^{-(\mu_i + v_0)\tau}}{\mu_s (\mu_i + v_0)} \tag{12}$$

$\mathcal{R}_0(\tau)$ can be seen as the basic reproduction rate, which is the number of new olive trees infected by an average infectious olive tree during its infectivity period, in a population made up entirely of healthy olive trees.

5. Local stability of equilibrium points

In this section, we study the local stability of equilibrium points based on the linearization. Let (S^*, I^*) be an equilibrium point of the system (7).
Introduce

$$U_1(t) = S(t) - S^*, \quad U_2(t) = I(t) - I^* \tag{13}$$

We obtain the following system

$$\begin{cases} \dfrac{dU_1}{dt} = \Lambda - \mu_s(U_1(t)+S^*) - (U_1(t)+S^*)(I^* + U_2(t-\tau)e^{-(\mu_i+v_0)\tau} \\ \dfrac{dU_2}{dt} = (U_1(t)+S^*)(I^*+U_2(t-\tau))e^{-(\mu_i+v_0)\tau} - (\mu_i+v_0)(I^*+U_2(t)) \end{cases} \tag{14}$$

Then we get,

$$\begin{cases} \dfrac{dU_1}{dt} = -(\mu_s + I^* e^{-(\mu_i+v_0)\tau})U_1(t) - S^* U_2(t-\tau)e^{-(\mu_i+v_0)\tau} \\ \dfrac{dU_2}{dt} = I^* U_1(t) e^{-(\mu_i+v_0)\tau} + (S^* e^{-(\mu_i+v_0)\tau} - (\mu_i+v_0))U_2(t-\tau) \end{cases} \tag{15}$$

Define

$$J(\lambda) = \begin{pmatrix} \lambda + \mu_s + I^* e^{-(\mu_i+v_0)\tau} & S^* e^{-(\mu_i+v_0)\tau} e^{-\lambda\tau} \\ -e^{-(\mu_i+v_0)\tau} I^* & \lambda + (\mu_i+v_0) - S^* e^{-(\mu_i+v_0)\tau} e^{-\lambda\tau} \end{pmatrix}.$$

The characteristic equation corresponding to the stationary state (S^*, I^*) is given by $det(J(\lambda)) = 0$, that gives

$$(\lambda+\mu_s+I^*e^{-(\mu_i+v_0)\tau})(\lambda+(\mu_i+v_0)-S^*e^{-(\mu_i+v_0)\tau}e^{-\lambda\tau}) + S^*I^*e^{-2(\mu_i+v_0)\tau}e^{-\lambda\tau} = 0. \tag{16}$$

5.1. **Local stability of disease-free equilibrium.** For $E_0 = (\dfrac{\Lambda}{\mu_s}, 0)$, the caracteristic equation (16) becomes,

$$(\lambda+\mu_s)(\lambda+(\mu_i+v_0) - \frac{\Lambda}{\mu_s}e^{-(\mu_i+v_0)\tau}e^{-\lambda\tau}) = 0 \tag{17}$$

then we have, $\lambda_1 = -\mu_s < 0$ and λ_2 is a solution of the equation

$$\lambda = (\mu_i+v_0)(\mathcal{R}_0(\tau)e^{-\lambda\tau} - 1)$$

Theorem 5.1.

- *If* $\mathcal{R}_0(\tau) < 1 \ \forall\ \tau \geq 0$ *the disease-free equilibrium is locally asymptotically stable for all* $\tau \geq 0$.
- *If* $\mathcal{R}_0(\tau) > 1$ *the disease-free equilibrium is unstable for those* $\tau \geq 0$.

Proof.

To begin with, let $\tau = 0$

We get $\lambda_2 = (\mu_i+v_0)(\mathcal{R}_0(0) - 1)$, therefore we have the following result

- If $\mathcal{R}_0(0) < 1$, then $\lambda_{1,2} < 0$, so the disease-free equilibrium is locally asymptotically stable.
- If $\mathcal{R}_0(0) > 1$, then $\lambda_2 > 0$ so, the disease-free equilibrium is unstable.

If $\tau > 0$ we have,

$$\lambda + (\mu_i+v_0) - (\mu_i+v_0)\mathcal{R}_0(\tau)e^{-\lambda\tau} = 0 \tag{18}$$

If we assume that $\lambda = \dfrac{z}{\tau}$, $p = -\tau(\mu_i+v_0)$ and $q = \tau(\mu_i+v_0)\mathcal{R}_0(\tau)$, this equation can be express as follows ([10]),

$$z - p - qe^{-z} = 0 \tag{19}$$

This is an equation identical to that of Cooke and Grossman ([10]), which shows that the necessary and sufficient conditions for all roots to have a negative real part are :

(1) $p < 1$, fulfilled for all $\tau > 0$ since $-(\mu_i+v_0) < 0$.

(2) $q + p < 0$. Since we have, $q = \tau(\mu_i + v_0)\mathcal{R}_0(\tau) < \tau(\mu_i + v_0) = -p$, it is satisfied for all $\tau > 0$ if and only if $\mathcal{R}_0(\tau) < 1 \; \forall \; \tau \geq 0$.

If $q > -p$, which means $\mathcal{R}_0(\tau) > 1$, there exists λ with positive real part.

To conclude, if $\mathcal{R}_0(\tau) < 1$ for every positive τ, the characteristic equation has no complex solution with $\Re(\lambda) > 0$. If $\mathcal{R}_0(\tau) > 1$, the characteristic equation has at least one complex solution with $\Re(\lambda) > 0$. □

Remark If

$$\frac{\Lambda}{\mu_s(\mu_i + v_0)} < 1$$

then $\mathcal{R}_0(\tau) < 1$ for every positive τ.

5.2. Local stability of the endemic equilibrium. For

$$E_1 = \left((\mu_i + v_0)e^{(\mu_i+v_0)\tau}, \frac{\Lambda - \mu_s S^*}{\mu_i + v_0} \right),$$

$S^* = (\mu_i + v_0)e^{(\mu_i+v_0)\tau}$, recall the characteristic equation (16), where we have,

$$(\lambda + \mu_s + I^* e^{-(\mu_i+v_0)\tau})(\lambda + (\mu_i + v_0) - S^* e^{-(\mu_i+v_0)\tau} e^{-\lambda\tau}) + S^* I^* e^{-2(\mu_i+v_0)\tau} e^{-\lambda\tau} = 0. \quad (20)$$

We can write (20) as follow,

$$(\lambda + \mu_s\mathcal{R}_0(\tau))(\lambda + (\mu_i + v_0)(1 - e^{-\lambda\tau})) + \mu_s(\mu_i + v_0)(\mathcal{R}_0(\tau) - 1)e^{-\lambda\tau} = 0. \quad (21)$$

Theorem 5.2.

If $\mathcal{R}_0(\tau) > 1$ for $\tau \in [0, \tau_0]$, then the endemic equilibrium is locally asymptotically stable.

Proof.

First of all, let us assume that $\tau = 0$. From (21) , the following second-degree equation is obtained

$$\lambda^2 + \mu_s\mathcal{R}_0(0)\lambda + \mu_s(\mu_i + v_0)(\mathcal{R}_0(0) - 1) = 0 \quad (22)$$

Since $\mu_s\mathcal{R}_0(0) > 0$ and $\mu_s(\mu_i + v_0)(\mathcal{R}_0(0) - 1) > 0$, it follows that all roots λ of (22) have $\Re(\lambda) < 0$.

And so, for $\tau = 0$ the endemic equilibrium E_1 is locally asymptotically stable.

If $\tau \in (0, \tau_0]$, the equation (21), can be expressed as follows ([2], [6])

$$P(\lambda) + Q(\lambda)e^{-\lambda\tau} = 0, \quad (23)$$

where

$$P(\lambda) = \lambda^2 + (\mu_s\mathcal{R}_0(\tau) + (\mu_i + v_0))\lambda + \mu_s\mathcal{R}_0(\tau)(\mu_i + v_0),$$
$$Q(\lambda) = -(\mu_i + v_0)(\lambda + \mu_s),$$

Since there is stability for $\tau = 0$, it can be lost if there exists $\lambda = iy$ ($y \in \mathbb{R}$) a root of ((23)). In this situation it follows that :

$$|P(iy)| = |Q(iy)|,$$

Introducing $P(iy) = P_{\Re}(y) + iP_{Im}(y)$ and $Q(iy) = Q_{\Re}(y) + iQ_{Im}(y)$, we obtain :

$$P(iy) = -y^2 + iy(\mu_s\mathcal{R}_0(\tau) + (\mu_i + v_0)) + \mu_s\mathcal{R}_0(\tau)(\mu_i + v_0),$$
$$Q(iy) = -(\mu_i + v_0)(iy + \mu_s) = -(\mu_i + v_0)\mu_s - i(\mu_i + v_0)y.$$

The real and imaginary parts are :

$$P_{\Re}(y) = -y^2 + \mu_s\mathcal{R}_0(\tau)(\mu_i + v_0), \qquad P_{Im}(y) = y(\mu_s\mathcal{R}_0(\tau) + (\mu_i + v_0)),$$
$$Q_{\Re}(y) = -(\mu_i + v_0)\mu_s, \qquad Q_{Im}(y) = -(\mu_i + v_0)y.$$

Let the function

$$\begin{aligned} F(y) := & \quad |P(iy)|^2 - |Q(iy)|^2 \\ = & \quad (-y^2 + \mu_s \mathcal{R}_0(\tau)(\mu_i + v_0))^2 + y^2(\mu_s \mathcal{R}_0(\tau) + (\mu_i + v_0))^2 \\ & -(\mu_i + v_0)^2 \mu_s^2 - (\mu_i + v_0)^2 y^2 = 0. \end{aligned} \tag{24}$$

Consequently, the solutions of $|P(iy)|^2 = |Q(iy)|^2$, are the roots of ((24)).
The equation (24), by substituting $y^2 = Z$, can be written as follows,

$$Z^2 + \mu_s^2 \mathcal{R}_0^2(\tau) Z + \mu_s^2 (\mu_i + v_0)^2 (\mathcal{R}_0^2(\tau) - 1) = 0 \tag{25}$$

We have $\mu_s^2 \mathcal{R}_0^2(\tau) > 0$, and $\mu_s^2(\mu_i + v_0)^2(\mathcal{R}_0^2(\tau) - 1) > 0$, since $\mathcal{R}_0(\tau) > 1 \, \forall \, \tau \in (0, \tau_0]$. So, all roots of (25) verify $Z < 0$. Consequently, the function F has no real solution y, which means that the quation (23) has no pure imaginary solution, i.e. the roots λ don't cross the imaginary axis([2]).
In conclusion if $\mathcal{R}_0(\tau) > 1$, for all $\tau \in [0, \tau_0]$ the characteristic equation (21) have complex solutions λ with $\Re(\lambda) < 0$. □

6. Global stability of the disease-free equilibrium

In this section we analyze the global stability of the disease-free equilibrium point $(S^0, I^0) = \left(\frac{\Lambda}{\mu_s}, 0\right)$ by introducing a Lyapunov-Krasovskii ([13]) functional.

Theorem 6.1.
1.If $\mathcal{R}_0(\tau) < 1$, for every $\tau \in [0, \tau_0]$, then the disease-free equilibrium (S^0, I^0) is globally asymptotically stable in the invariant set $\{0 \leq N \leq \frac{\Lambda}{\mu_s}\}$.
2. If $R_0(\tau) > 1$, the disease-free equilibrium is unstable.
Proof.
Let

$$V(t) = (S(t) - S^0 - S^0 \ln(\frac{S(t)}{S^0})) + I(t) + (\mu_i + v_0) \int_{t-\tau}^{t} I(s) ds. \tag{26}$$

The latter is a function of class C^1, moreover $V(t) \geq 0$ and $V(t) = 0$ if and only if $I = 0$ and $S = S^0$.
Let's calculate the time derivative of V along the system (7).Using the fact that $\Lambda = \mu_s S^0$,

$$\begin{aligned} V'(t) & = (S'(t) - S^0 \frac{\frac{S'(t)}{S^0}}{\frac{S(t)}{S^0}}) + I'(t) + (\mu_i + v_0)(I(t) - I(t-\tau)) \\ & = S(t)I(t-\tau)e^{-(\mu_i + v_0)\tau} - (\mu_i + v_0)I(t) + (1 - \frac{S^0}{S})(\Lambda - \mu_s S(t)) \\ & -S(t)I(t-\tau)e^{-(\mu_i + v_0)\tau}) + (\mu_i + v_0)(I(t) - I(t-\tau)) \\ & = S(t)I(t-\tau)e^{-(\mu_i + v_0)\tau} + (1 - \frac{S^0}{S})(\mu_s S^0 - \mu_s S(t) \\ & -S(t)I(t-\tau)e^{-(\mu_i + v_0)\tau}) - (\mu_i + v_0)I(t-\tau) \\ & = S(t)I(t-\tau)e^{-(\mu_i + v_0)\tau} - (\frac{S^0}{S} - 1)(\mu_s(S^0 - S(t))) \\ & -(1 - \frac{S^0}{S})(S(t)I(t-\tau)e^{-(\mu_i + v_0)\tau}) - (\mu_i + v_0)I(t-\tau) \end{aligned} \tag{27}$$

then,

$$\begin{aligned} V'(t) &= -\mu_s\frac{(S^0-S)^2}{S} + [1-(1-\frac{S^0}{S})](S(t)I(t-\tau)e^{-(\mu_i+v_0)\tau}) \\ &-(\mu_i+v_0)I(t-\tau) \\ &= -\mu_s\frac{(S^0-S)^2}{S} + \frac{S^0}{S}S(t)I(t-\tau)e^{-(\mu_i+v_0)\tau} - (\mu_i+v_0)I(t-\tau) \\ &= -\mu_s\frac{(S^0-S)^2}{S} + (\mu_i+v_0)\frac{S^0e^{-(\mu_i+v_0)\tau}}{(\mu_i+v_0)}I(t-\tau) - (\mu_i+v_0)I(t-\tau) \\ &= -\mu_s\frac{(S^0-S)^2}{S} + (\mu_i+v_0)\mathcal{R}_0(\tau)I(t-\tau)) - (\mu_i+v_0)I(t-\tau) \\ &= -\mu_s\frac{(S^0-S)^2}{S} - (\mu_i+v_0)I(t-\tau)(1-\mathcal{R}_0(\tau)) \end{aligned} \tag{28}$$

And so $V' \leq 0$ if $\mathcal{R}_0(\tau) \leq 1$. Even more $V' = 0$ if and only if $S = S^0 = \dfrac{\Lambda}{\mu_s}$, and $\mathcal{R}_0 = 1$ or $I = 0 = I^0$. But if we put $I = 0$ in the first equation of (7), we get $S(t) = \dfrac{\Lambda}{\mu_s} = S^0$. Consequently, the set $\omega = \{(\dfrac{\Lambda}{\mu_s}, 0)\}$ is the largest compact invariant set of $\Omega = \{(S^0, I^0) \in \mathbb{R}^2_+ | V' = 0\}$. And then, according to Barbashin-Krasovskii-LaSalle invariance principle ([12],[13],[23], [24]), ([25], Theorem 5.3.1), we get the result. □

7. Numerical simulations

In this part, we will illustrate our theoretical results using some numerical simulations. The main parameters used in the calculations are grouped together in the table below (1).

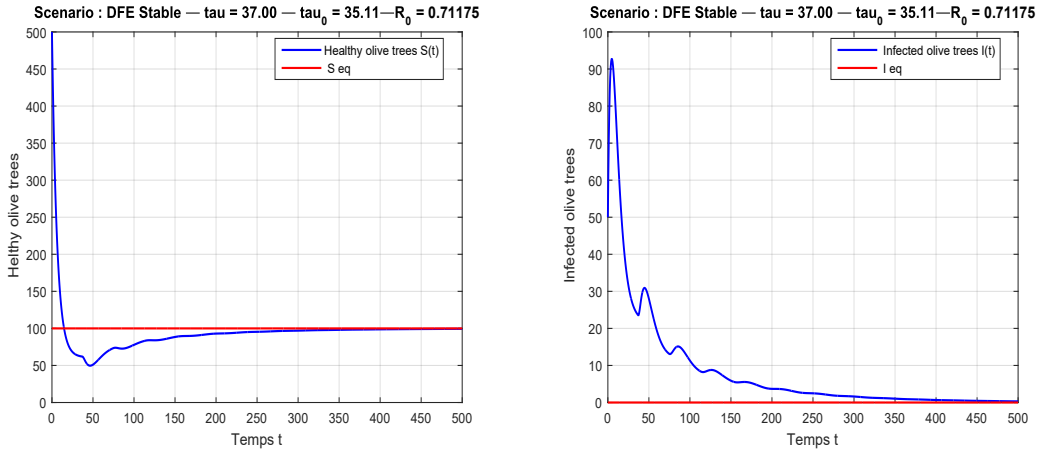
Parameters	Symbol	Value	Reference
The density of new olive trees	Λ	10 year^{-1}	This study
The mortality rate of healthy olive trees	μ_s	0.1 year^{-1}	[21]
The mortality rate of olive trees infected	μ_i	0.09 year^{-1}	[21]
The rate of removal of infected olive trees	v_0	0.09 year^{-1}	[21]
The initiale density of sensitive olive trees	S_0	500	This study
The initial density of infected olive trees	I_0	50	This study

TABLE 1. Parameters used in numerical simulations

We are therefore looking for the value of τ_0 for which the basic reproduction number $\mathcal{R}_0(\tau)$ exceeds 1. This value marks the critical threshold at which an epidemic can emerge.
We now proceed to its calculation, recall that,

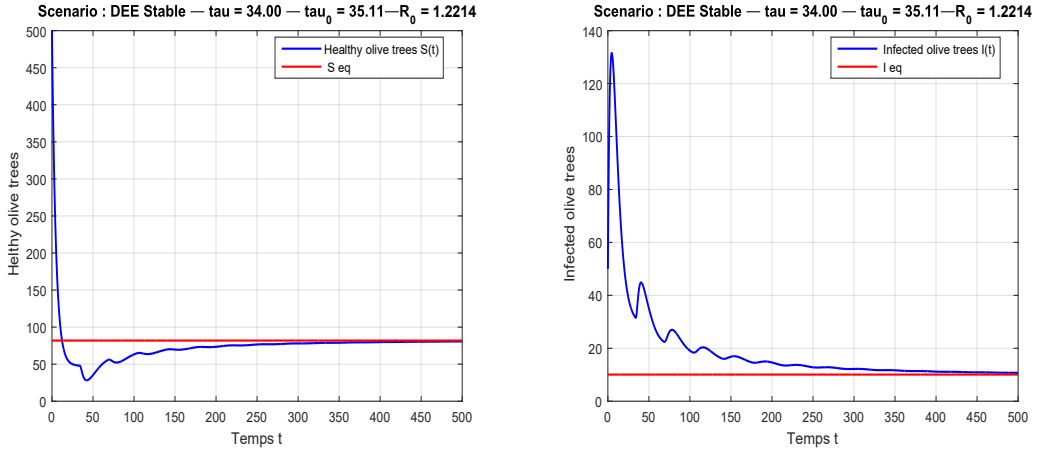
$$\begin{aligned} \mathcal{R}_0(\tau_0) := \frac{\Lambda e^{-(\mu_i+v_0)\tau_0}}{\mu_s(\mu_i+v_0)} &> 1 \\ e^{-(\mu_i+v_0)\tau_0} &> \frac{\mu_s(\mu_i+v_0)}{\Lambda} \\ -(\mu_i+v_0)\tau_0 &> \ln\left(\frac{\mu_s(\mu_i+v_0)}{\Lambda}\right) \\ \tau_0 &< -\frac{1}{\mu_i+v_0}\ln\left(\frac{\mu_s(\mu_i+v_0)}{\Lambda}\right) \end{aligned}$$

Using the parameters in the table (1), we find $\tau_0 < 35.11$, and also, we present numerical simulations illustrating the evolution of the system (7) and the stability of the associated equilibrium point.



(a) (b)

Figure 1. Populations of olive trees, healthy in (a) and infected in (b). Case $\mathcal{R}_0(\tau) = 0.71175 < 1$, $\tau = 37$. We observe that the curves tend to the disease-free equilibrium point $S^0 = 100$ and $I^0 = 0$, shown in red, which agrees with Theorem **6**.1.



(a) (b)

Figure 2. Populations of olive trees, healthy in (a) and infected in (b). Case $\mathcal{R}_0(\tau) = 1.2214 > 1$, $\tau = 34$. We observe that the curves tend to the endemic equilibrium point $S^* = 82$ and $I^* = 10$ shown in red, which agrees with Theorem **5**.2.

8. Conclusion

In this work, an age-structured model (1) that describes the dynamics of olive trees in the face of infection by *Xylella Fastidiosa*, is reduced to a delayed model (7), which captures the essential epidemic mechanisms while taking into account the separation of time scales between the rapid dynamics of insect vectors and the slower evolution of olive trees. This simplification enabled us to focus on the internal dynamics of the trees population, with the transmission rate implicitly reflecting the average impact of vectors over time.
Our mathematical study is based on the well-posed problem in the first place, and the search for equilibria and the demonstration of stability, which give us an idea on the behavior of system's solutions.
The conditions on local stability of equilibria involve the parameters and the net reproduction rate $\mathcal{R}_0(\tau)$ of the model (7) (see Th **5**.1, Th **5**.2).
If the net reproduction rate $\mathcal{R}_0(\tau) < 1$, the disease-free equilibrium $(S^0, 0)$ is locally asymptotically stable, indicating the eventual disappearance of the infection. In the case $\mathcal{R}_0(\tau) > 1$, the disease-free equilibrium change it's stability nature and becomes unstable.
The endemic equilibrium point exists in the case $\mathcal{R}_0(\tau) > 1 \; \forall \, \tau \in [0, \tau_0]$ and is stable for all delays $\tau \in [0, \tau_0]$ which means that the epidemic is persisting in olive groves.
We then moved on to global stability of the disease-free equilibrium $(S^0, 0)$. By using a suitable Lyapunov-Krasovskii functional , we proved that the solutions of the system tend towards the equilibrium point.
If the net reproduction rate $\mathcal{R}_0(\tau) < 1$, the disease-free equilibrium $(S^0, 0)$ is globally asymptotically stable, and unstable if $\mathcal{R}_0(\tau) > 1$ (see Th **6**.1).
Finally, we supplemented this theoretical knowledge with numerical simulations that illustrate the dynamics of *Xylella Fastidiosa* invasion and eradication, and confirm the consistency of the model's predictions, (see Fig 1, Fig2).
This work was carried out while deliberately neglecting the disease's insect vectors. This simplification enables us to focus on the dynamics specific to infected olive trees, without the complexity associated with vector biology. Despite this omission, the model developed effectively captures the temporal structure of interactions between healthy and infected trees. It shows how a delay model can accurately describe the evolution of an epidemic in a plant system.
This analytical framework remains clear, rigorous and, above all, extensible. It forms a solid foundation on which we can later explicitly integrate insect dynamics, seasonal factors or targeted control measures.

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