

Modeling epidemic and pest dynamics in plants

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Abstract

We present a reaction-diffusion model that describe ...

Key words: Population dynamics; reaction-diffusion model; infectious disease; plant disease; basic reproduction number; equilibrium; stability; next generation matrix; compound matrix.

Mathematics subject classifications (2000): XXXXX, 65N06, 76T20.

1 Introduction

1.1 Scope

Scientists worldwide are deeply committed to studying and analyzing infectious diseases and pests that affect a wide variety of living organisms, including animals, insects, plants, humans, and many others. This interest stems from the significant impact these diseases have on various aspects. On one hand, they have a negative impact on human health, causing severe illnesses and posing a risk to public health. Additionally, these diseases can have a significant environmental impact by altering biodiversity and the health of natural ecosystems. Specifically, pests or infectious diseases that affect the food chain pose an imminent risk to the food industry by disrupting agricultural and livestock production, which could trigger significant economic and social consequences.

The increase in the world population poses several additional challenges. On one hand, there is the need to meet the demand for food, including grains, fruits, vegetables, and meat. On the other hand, there is the need to conserve food diversity, the health of ecosystems, and the sustainability of the agricultural industry (production, processing, and marketing of agricultural products) in general. This industry is heavily affected by various infectious diseases or pests that currently arise, impacting grain crops, fruit tree plantations, and vegetable cultivation intended for human consumption. These events reduce food production and quality [2]. Furthermore, the ongoing process of climate change exacerbates this situation by altering climatic patterns and the necessary environmental conditions for food production. Among the tangible negative effects is the increase in both the frequency and severity of plant diseases or pests, generating social, health, and economic impacts.

From an environmental standpoint several countries constantly monitors and tries to prevent the entry and spread of pests and diseases that affect fruit crops, native forests and commercial plantations

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too, with the aim of protecting and improving the conditions of these plantations and, at the same time, reducing high-cost economic and environmental impacts that would be generated if these events were to occur inevitably. Given the introduction of new species or diseases affecting plants, animals, fruit tree plantations, and others, along with their environmental impact, it becomes crucial to forecast potential disease or pest outbreaks.

From the perspective of mathematical modeling, it is known that the study and analysis of mathematical models describing the spread of infectious diseases or pests in plantations provide an important tool and support in decision-making and resource management associated with large plantations, as they allow for simulations, design of control strategies, and evaluation of potential epidemic outbreaks in the agricultural environment in case these events occur.

1.2 Related work

Currently, national and international researchers are continuously seeking to apply mathematical modeling and the design of numerical methods to understand, simulate, and predict the behavior of infectious diseases in humans, animals, on forest or fruit plantations, as well as to understand other events (geophysical, industrial, retail problems) of high social, environmental, and economic impact.

Such models are mathematically expressed as a system of partial differential equations (PDEs) dependent on space and time in the following form;

$$\partial_t \mathbf{u} + \partial_{x_1} \mathbf{f}_1(\mathbf{x}, \mathbf{u}) + \dots + \partial_{x_d} \mathbf{f}_d(\mathbf{x}, \mathbf{u}) = \nabla \cdot (\mathbf{B}(\mathbf{u}) \nabla \mathbf{u}) + \mathbf{r}(\mathbf{u}), \quad \mathbf{x} \in \Omega \subset \mathbb{R}^d, \quad t > 0, \quad (1)$$

plus an appropriate initial and boundary condition. Here, t represents time, \mathbf{x} is the spatial variable, the matrices \mathbf{B} and $\mathbf{f}_1, \dots, \mathbf{f}_d$ are given, and the vector of unknowns is $\mathbf{u} = (u_1, \dots, u_N)^T$, which typically represents the density of a species. If $\mathbf{B} = 0$ and $\mathbf{r} = 0$, a system of conservation laws in d dimensions is obtained, given by

$$\partial_t \mathbf{u} + \partial_{x_1} \mathbf{f}_1(\mathbf{x}, \mathbf{u}) + \dots + \partial_{x_d} \mathbf{f}_d(\mathbf{x}, \mathbf{u}) = \mathbf{0}, \quad \mathbf{x} \in \Omega \subset \mathbb{R}^d, \quad t > 0. \quad (2)$$

The spatio-temporal spread of an infectious disease or pests on plantations can often be adequately described by reaction-diffusion systems of the form (1) in $d = 2$ dimensions, where convection is generally absent, i.e., $\mathbf{f}_i = \mathbf{0}$ for $i = 1, 2$, and the coordinates of the vector of unknowns \mathbf{u} represent the densities of different epidemiological states or compartments of a population (see, for example [6, 10, 14, 17]).

These descriptions are generally based on models of known ordinary differential equations (ODEs) [3, 11, 15, 16, 24] (*SIR*, *SEIR*, *SEIRD*, *SVEIR*, among others). The *SEIR* model distinguishes between compartments of susceptibles, exposed, infected, and recovered individuals, so in this case, the vector of unknowns is given by $\mathbf{u} = (S, E, I, R)^T$, and the corresponding “reaction” term $\mathbf{r}(\mathbf{u})$ represents transmission rates, birth and death rates, and the proportion between different compartments. The diffusion term $\nabla \cdot (\mathbf{B}(\mathbf{u}) \nabla \mathbf{u})$ expresses that the movement of individuals is determined by the local gradient $\nabla \mathbf{u}$ of all compartment densities and a gradual dispersal from regions of higher density to regions of lower density.

Less general models than (1), with reaction-diffusion and diffusion terms similar to the ones to be obtained, analyzed, and numerically solved in this paper, can be found in [8, 18, 19, 1, 5], some with spatial structure in [20, 23, 25, 7, 21, 13, 4], representing an important starting point for this study.

The study and analysis of epidemiological models aim to provide supportive information in decision-making and resource management associated with plantations, as they allow for simulations, design of control strategies, and evaluation of potential epidemic outbreaks in the agricultural environment. In this context, compartmental epidemiological models (system of ordinary differential equations)

have the important property that the basic reproduction number R_0 can be calculated and can be established for each disease [22], representing a threshold parameter that indicates if $R_0 > 1$, the disease will spread, and if $R_0 < 1$, it will die out. From an epidemiological standpoint, R_0 denotes the number of secondary cases produced in a completely susceptible population by the typical infected individual. Such a property is not naturally inherited when spatial variables are included in the modeling. In [9], the authors study the effects of diffusion and advection in an *SIS* epidemiological model (susceptible-infected-susceptible), provide a definition of the basic reproduction number R_0 , and study the existence of the endemic equilibrium when $R_0 > 1$. However, the definition is much more complicated than what is achieved with models lacking a spatial component. In [12], an *SIR* model with free boundary with reaction-diffusion is presented, and the authors provide sufficient conditions for the extinction or spread of the disease. Their main result shows that the disease will not spread throughout the area if the basic reproduction number $R_0 < 1$ or if the initial radius of infected individuals h_0 is sufficiently small, even for $R_0 > 1$.

1.3 Outline of the paper

2 Mathematical model

In this section, we present a compartmental mathematical model structured into two main groups of compartments that represent the disease dynamics in plants and insect vectors. The first group refers to the compartments S , I , and R , which correspond to susceptible, infected, and recovered plants, respectively. This group captures the vegetation's state in relation to the infection. The second group includes the compartments X and Y , representing susceptible and infected insect vectors, respectively. These vectors are responsible for transmitting the disease to plants, and their infection dynamics are key to understanding the disease's spread. This model allows us to examine how plant and vector populations interact over time, providing a foundation for predicting and controlling the progression of the infection.

Some assumptions are necessary to understand the model formulation. The assumptions considered in this model are as follows:

- The total population of insect vectors is divided into two categories, X and Y , which represent the densities of susceptible and infectious vectors at time t , respectively.
- For the host plant population, it is divided into three categories: S , I , and R , representing the numbers of susceptible, infectious, and recovered plants at time t , respectively.
- The total number of plants $K = S + I + R$ is a positive constant. This assumption considers that the number of plants in an area is fixed, allowing for the maintenance of the total number by adding a new plant whenever one dies.
- We assume that newly added plants are susceptible, with the birth rate of susceptible plants defined as $f(S, I) = \mu K + dI$.
- Susceptible plants can be infected not only by infectious insect vectors but also through contact with infected plants.
- A susceptible vector can only become infected through contact with an infected host plant, and once infected, it carries the pathogen for life. Additionally, vertical transmission is not considered.

- The replacement rate of insect vectors is a positive constant, and all newly born vectors are susceptible.
- The model includes a nonlinear incidence rate of the disease to better capture the transmission dynamics.

Under these assumptions, the general model in terms of partial differential equations is given by:

$$\begin{aligned}
\dot{S} - d_1 \Delta S &= f(S, I) - \mu S - \left(\frac{\beta_p Y}{1 + \alpha_p Y} + \frac{\beta_S I}{1 + \alpha_S I} \right) S, \\
\dot{I} - d_2 \Delta I &= \left(\frac{\beta_p Y}{1 + \alpha_p Y} + \frac{\beta_S I}{1 + \alpha_S I} \right) S - (d + \mu + \gamma) I, \\
\dot{R} - d_3 \Delta R &= \gamma I - \mu R, \\
\dot{X} - d_4 \Delta X &= \Lambda - \frac{\beta_1 I X}{1 + \alpha_1 I} - \alpha_4 c X P - m X, \\
\dot{Y} - d_5 \Delta Y &= \frac{\beta_1 I X}{1 + \alpha_1 I} - \alpha_4 c Y P - m Y, \\
\dot{P} - d_6 \Delta P &= \alpha_4 c (X P + Y P) - \delta P, \\
\partial_\eta S &= \partial_\eta I = \partial_\eta R = \partial_\eta X = \partial_\eta Y = \partial_\eta P = 0, \quad x \in \partial\Omega, t > 0,
\end{aligned} \tag{3}$$

with $f(S, I) = \mu K + dI$ and the initial condition given by: $S(0, x) = S^0(x)$, $I(0, x) = I^0(x)$, $R(0, x) = R^0(x)$, $X(0, x) = X^0(x)$, $Y(0, x) = Y^0(x)$ and $P(0, x) = P^0(x)$.

We will begin the analysis of this model by considering the simplified case where $P(t) = 0$ for all $t \geq 0$ and $d_i = 0$ for $i = 1, \dots, 6$.

$$\begin{aligned}
\dot{S} &= f(S, I) - \mu S - \left(\frac{\beta_p Y}{1 + \alpha_p Y} + \frac{\beta_S I}{1 + \alpha_S I} \right) S, \\
\dot{I} &= \left(\frac{\beta_p Y}{1 + \alpha_p Y} + \frac{\beta_S I}{1 + \alpha_S I} \right) S - (d + \mu + \gamma) I, \\
\dot{R} &= \gamma I - \mu R, \\
\dot{X} &= \Lambda - \frac{\beta_1 I X}{1 + \alpha_1 I} - m X, \\
\dot{Y} &= \frac{\beta_1 I X}{1 + \alpha_1 I} - m Y.
\end{aligned} \tag{4}$$

The descriptions of the model parameters are provided in Table 2.

2.1 Analysis of model behavior

In this section, we conduct a detailed analysis of the models, focusing on the positivity and boundedness of the variables, as well as the existence and uniqueness of solutions. This analysis aims to ensure that the model's variables remain within realistic ranges, verify that solutions do not exhibit unbounded growth, and confirm the mathematical well-posedness of the model. Such insights are essential for validating the model's robustness and ensuring its applicability to real-world scenarios.

We begin by noting that an equation for the total population of vector insects can be derived by summing equations (4.4) and (4.5), yielding the following differential equation:

$$\dot{N} - mN = \Lambda, \tag{5}$$

Parameter	Description	Default value
S	number of the susceptible plant hosts	-
I	number of the infected plant hosts	-
R	number of the recovered plant hosts	-
K	sum of the total plant hosts	$50 - 1,000$
X	density of the susceptible insect vectors	-
Y	density of the infected insect vectors	-
N	sum of the total insect vectors density	$50 - 100$
β_1	infection ratio between infected hosts and susceptible vectors	$0.01 - 0.02$
β_p	biting rate of an infected vector on the susceptible host plants	$0.01 - 0.02$
β_s	infection incidence between infected and susceptible hosts	$0.01 - 0.02$
α_1	determines the level at which the force of infection saturates	0.1
α_p	determines the level at which the force of infection saturates	0.2
α_s	determines the level at which the force of infection saturates	0.2
γ	the conversion rate of infected hosts to recovered hosts	$0 - 0.25$
μ	natural death rate of plant hosts	$0 - 0.1$
Λ	birth or immigration of insect vectors	5
m	natural death rate of insect vectors	$0 - 0.5$
d	disease-induced mortality of infected hosts	0.1

Table 1: Parámetros del modelo y sus valores predeterminados

with $N = X + Y$. De 5, se obtiene que $N \rightarrow \frac{\Lambda}{m}$ cuando $t \rightarrow \infty$. Noting that $S + I + R = K$ and considering the previous observation, we conclude that for the analysis it is only necessary to study the dynamics of the following reduced subsystem:

$$\begin{aligned}
\dot{S} &= \mu K + dI - \mu S - \left(\frac{\beta_p Y}{1 + \alpha_p Y} + \frac{\beta_s I}{1 + \alpha_s I} \right) S, \\
\dot{I} &= \left(\frac{\beta_p Y}{1 + \alpha_p Y} + \frac{\beta_s I}{1 + \alpha_s I} \right) S - (d + \mu + \gamma)I, \\
\dot{R} &= \gamma I - \mu R, \\
\dot{Y} &= \frac{\beta_1 I}{1 + \alpha_1 I} \left(\frac{\Lambda}{m} - Y \right) - mY.
\end{aligned} \tag{6}$$

The reaction-diffusion version of this system in one spatial dimension is given by

$$\begin{aligned}
\dot{S} - d_1 \frac{\partial^2 S}{\partial x^2} &= \mu K + dI - \mu S - \left(\frac{\beta_p Y}{1 + \alpha_p Y} + \frac{\beta_s I}{1 + \alpha_s I} \right) S, \\
\dot{I} - d_2 \frac{\partial^2 I}{\partial x^2} &= \left(\frac{\beta_p Y}{1 + \alpha_p Y} + \frac{\beta_s I}{1 + \alpha_s I} \right) S - (d + \mu + \gamma)I, \\
\dot{R} - d_3 \frac{\partial^2 R}{\partial x^2} &= \gamma I - \mu R, \\
\dot{Y} - d_4 \frac{\partial^2 Y}{\partial x^2} &= \frac{\beta_1 I}{1 + \alpha_1 I} \left(\frac{\Lambda}{m} - Y \right) - mY, \\
\partial_\eta S &= \partial_\eta I = \partial_\eta R = \partial_\eta Y = 0, \quad x \in \partial\Omega, t > 0.
\end{aligned} \tag{7}$$

plus the initial condition $S(0, x) = S^0(x)$, $I(0, x) = I^0(x)$, $R(0, x) = R^0(x)$ and $Y(0, x) = Y^0(x)$ for both systems.

Theorem 1 *For non-negative initial conditions, the solutions $(S(t), I(t), R(t), Y(t))$, $t > 0$, of the model (6) are non-negative.*

Proof 1 From system (6), we have that:

$$\left. \frac{dS}{dt} \right|_{S=0} = \mu(I + R) + dI \geq 0, \quad (8)$$

$$\left. \frac{dI}{dt} \right|_{I=0} = \frac{\beta_p Y S}{1 + \alpha_p Y} \geq 0, \quad (9)$$

$$\left. \frac{dR}{dt} \right|_{R=0} = \gamma I \geq 0, \quad (10)$$

$$\left. \frac{dY}{dt} \right|_{Y=0} = \frac{\beta_1 I}{1 + \alpha_1 I} \frac{\Lambda}{m} \geq 0, \quad (11)$$

when S, I, R, Y are non-negative. This implies that the solutions remain non-negative for non-negative initial states.

Theorem 2 The model described in (6) has bounded solutions for any set of non-negative initial conditions.

Proof 2 The total population of host plants is represented by the following expression

$$\dot{S} + \dot{I} + \dot{R} = 0, \quad (12)$$

then $S + I + R = \text{cte}$. On the other hand, for the vector species

$$\dot{X} + \dot{Y} = \Lambda - m(X + Y), \quad (13)$$

then $X + Y \leq \frac{\Lambda}{m}$. Therefore, all variables are bounded.

To analyze the existence and uniqueness of the solution to system (6) in the region $[t_0, T] \times \Omega$, where $\Omega = \{(S, I, R, Y) \in \mathbb{R}^4 : \max\{|S|, |I|, |R|, |Y|\} \leq \lambda\}$ and $T < +\infty$, we apply the Picard-Lindelöf theorem. To do this, we demonstrate that the right-hand side of model (6) satisfies the Lipschitz condition. For this purpose, we consider the vectors $X_1 = (S, I, R, Y)$ and $\bar{X}_1 = (\bar{S}, \bar{I}, \bar{R}, \bar{Y})$.

Theorem 3 For each initial condition $X_{t_0} = (S^0, I^0, R^0, Y^0) \in \Delta$, there exists a unique solution $X(t) \in \Delta$ to the system (6), which is defined for all $t \geq t_0$.

Proof 3 loading...

2.2 The basic reproduction number R_0

The basic reproduction number, R_0 , is intuitively defined as “the average number of secondary infection cases generated by a primary case in a fully susceptible host population”. Mathematically, it is given by the dominant eigenvalue of the next-generation operator. Thus, R_0 is a biologically meaningful quantity that characterizes the reproductive fitness of the pathogen. In deterministic models, it determines the invasion threshold: if $R_0 > 1$, the disease will spread within the population; otherwise, if $R_0 < 1$, the pathogen will eventually die out. Therefore, R_0 can be used to estimate the critical proportion of the host population that needs to be immunized (i.e., vaccinated or otherwise protected) in order to eradicate the disease. Furthermore, R_0 often provides an estimate for the disease’s eventual equilibrium level in the population [?].

Theorem 4 *The basic reproductive number R_0 is given by*

$$R_0 = \frac{\beta_s K}{\omega} + \frac{\beta_1 \beta_p \Lambda K}{m^2 \omega},$$

with $\omega = d + \mu + \gamma$.

Proof 4 *Using the next-generation matrix method, where the rate of new infections is determined by the matrix F , and the transfer rates into and out of the infected state class are represented by the matrix V , respectively.*

$$F = \begin{pmatrix} \frac{\partial F_I}{\partial I} & \frac{\partial F_I}{\partial y} \\ \frac{\partial F_y}{\partial I} & \frac{\partial F_y}{\partial y} \end{pmatrix} = \begin{pmatrix} \beta_s K & \beta_p K \\ 0 & 0 \end{pmatrix}$$

y

$$V = \begin{pmatrix} \frac{\partial V_I}{\partial I} & \frac{\partial V_I}{\partial y} \\ \frac{\partial V_y}{\partial I} & \frac{\partial V_y}{\partial y} \end{pmatrix} = \begin{pmatrix} d + \mu + \gamma & 0 \\ -\frac{\beta_1 \Lambda}{m} & m \end{pmatrix}.$$

Letting $\omega = d + \mu + \gamma$, the next-generation matrix is

$$FV^{-1} = \begin{pmatrix} \frac{\beta_s K}{\omega} + \frac{\beta_1 \beta_p \Lambda K}{m^2 \omega} & \frac{\beta_p K}{m} \\ 0 & 0 \end{pmatrix},$$

obtaining the basic reproduction number

$$R_0 = \frac{\beta_s K}{\omega} + \frac{\beta_1 \beta_p \Lambda K}{m^2 \omega}.$$

3 Numerical simulations

To solve the system of partial differential equations (7) we use the finite difference method in one spatial dimension, we will discretize both time and space in N_x points with step Δx and N_t steps with step Δt . Then we get that

$$\begin{aligned} x_j &= j\Delta x \text{ for } j = 0, 1, \dots, N_x \\ t^n &= n\Delta t \text{ for } n = 0, 1, \dots, N_t. \end{aligned} \tag{14}$$

We denote the approximations of the state functions $S(x, t)$, $I(x, t)$, $R(x, t)$, and $Y(x, t)$ at the point (x_j, t^n) as S_j^n , I_j^n , R_j^n , and Y_j^n , respectively. For the temporal derivative discretization of \dot{S} , \dot{I} , \dot{R} , and \dot{Y} , we use a forward time approximation to get an explicit method:

$$\frac{\partial S}{\partial t} \approx \frac{S_j^{n+1} - S_j^n}{\Delta t}, \frac{\partial I}{\partial t} \approx \frac{I_j^{n+1} - I_j^n}{\Delta t}, \frac{\partial R}{\partial t} \approx \frac{R_j^{n+1} - R_j^n}{\Delta t}, \frac{\partial Y}{\partial t} \approx \frac{Y_j^{n+1} - Y_j^n}{\Delta t}. \tag{15}$$

For the second order spatial derivatives $\partial^2 S/\partial x^2$, $\partial^2 I/\partial x^2$, $\partial^2 R/\partial x^2$, and $\partial^2 Y/\partial x^2$, we use a centered difference approximation,

$$\begin{aligned}\frac{\partial^2 S}{\partial x^2} &\approx \frac{S_{j+1}^n - 2S_j^n + S_{j-1}^n}{(\Delta x)^2}, \\ \frac{\partial^2 I}{\partial x^2} &\approx \frac{I_{j+1}^n - 2I_j^n + I_{j-1}^n}{(\Delta x)^2}, \\ \frac{\partial^2 R}{\partial x^2} &\approx \frac{R_{j+1}^n - 2R_j^n + R_{j-1}^n}{(\Delta x)^2}, \\ \frac{\partial^2 Y}{\partial x^2} &\approx \frac{Y_{j+1}^n - 2Y_j^n + Y_{j-1}^n}{(\Delta x)^2},\end{aligned}\tag{16}$$

respectively. Finally, substituting the temporal and spatial derivatives approximations (15) and (16) into the system of equations (7), we get the following discrete version

$$\begin{aligned}\frac{S_j^{n+1} - S_j^n}{\Delta t} - d_1 \frac{S_{j+1}^n - 2S_j^n + S_{j-1}^n}{(\Delta x)^2} &= \mu K + dI_j^n - \mu S_j^n - \left(\frac{\beta_p Y_j^n}{1 + \alpha_p Y_j^n} + \frac{\beta_s I_j^n}{1 + \alpha_s I_j^n} \right) S_j^n \\ \frac{I_j^{n+1} - I_j^n}{\Delta t} - d_2 \frac{I_{j+1}^n - 2I_j^n + I_{j-1}^n}{(\Delta x)^2} &= \left(\frac{\beta_p Y_j^n}{1 + \alpha_p Y_j^n} + \frac{\beta_s I_j^n}{1 + \alpha_s I_j^n} \right) S_j^n - (d + \mu + \gamma) I_j^n \\ \frac{R_j^{n+1} - R_j^n}{\Delta t} - d_3 \frac{R_{j+1}^n - 2R_j^n + R_{j-1}^n}{(\Delta x)^2} &= \gamma I_j^n - \mu R_j^n \\ \frac{Y_j^{n+1} - Y_j^n}{\Delta t} - d_4 \frac{Y_{j+1}^n - 2Y_j^n + Y_{j-1}^n}{(\Delta x)^2} &= \frac{\beta_1 I_j^n}{1 + \alpha_1 I_j^n} \left(\frac{\Lambda}{m} - Y_j^n \right) - m Y_j^n\end{aligned}\tag{17}$$

plus the following initial and boudary conditions S_j^0 , I_j^0 , R_j^0 , and Y_j^0 at $t = 0$ and

$$\partial_\eta S = \partial_\eta I = \partial_\eta R = \partial_\eta Y = 0, \quad x \in \partial\Omega, \quad t > 0$$

These Neumann boundary conditions are implemented by setting the spatial derivatives at the boundaries to zero, i.e.,

$$\begin{aligned}S_{-1}^n &= S_1^n, \quad S_{N_x+1}^n = S_{N_x-1}^n, \quad I_{-1}^n = I_1^n, \quad I_{N_x+1}^n = I_{N_x-1}^n, \\ R_{-1}^n &= R_1^n, \quad R_{N_x+1}^n = R_{N_x-1}^n \quad \text{and} \quad Y_{-1}^n = Y_1^n, \quad Y_{N_x+1}^n = Y_{N_x-1}^n.\end{aligned}$$

This finite difference scheme is explicit in time, which may lead to instability for certain values of Δt and Δx . To enhance stability, we could employ an implicit or semi-implicit method, or ensure that Δt and Δx satisfy the Courant-Friedrichs-Lewy (CFL) stability condition. Specifically, the time step Δt should meet the condition

$$\Delta t \leq \frac{(\Delta x)^2}{2 \max(d_1, d_2, d_3, d_4)},$$

to help maintain stability in the explicit finite difference scheme throughout the simulation.

3.1 Example 1: Model simulation without diffusion

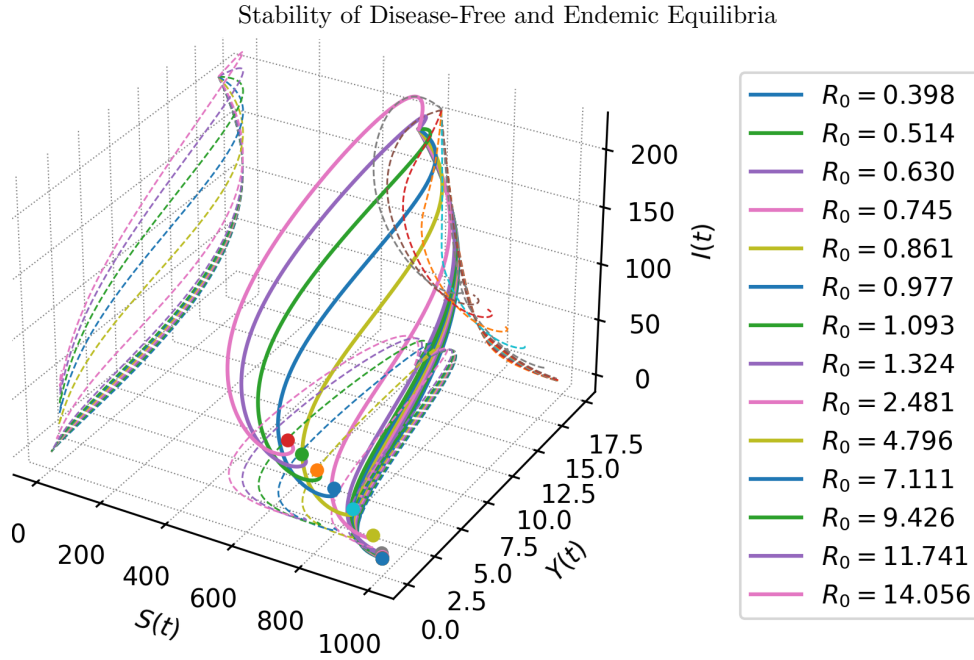


Figure 1: Stability of Disease-Free and endemic equilibria for de model (6).

3.1.1 Stability of the Endemic Equilibrium

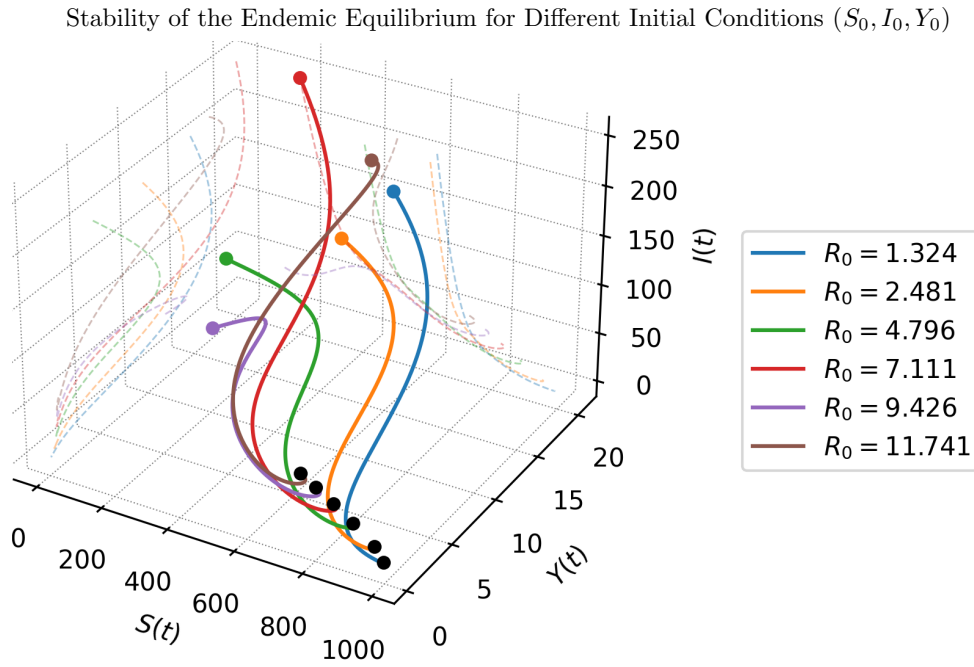


Figure 2: Stability of the endemic equilibrium for different initial conditions for de model (6).

3.1.2 Stability of the disease-free equilibrium

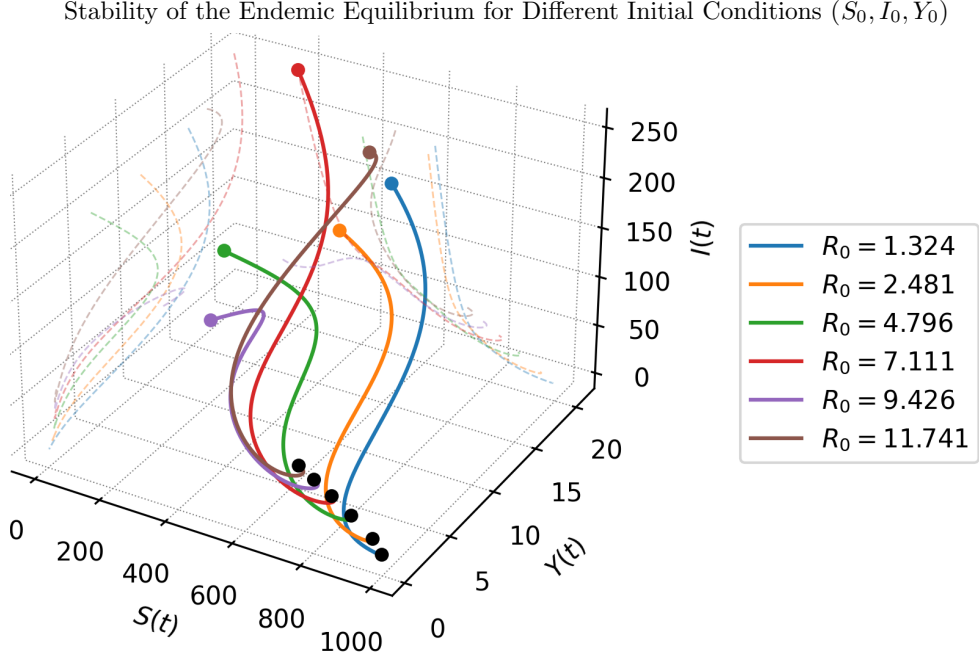


Figure 3: Stability of the disease-free equilibrium for different initial conditions for the model (6).

3.2 Example 2: Simulation of the reaction-diffusion Model

A numerical simulation of the reaction-diffusion model (7) is presented below, using the following initial conditions for each variable

$$\begin{aligned}
 S(x, 0) &= 0.5 + 0.1 \sin\left(\frac{\pi x}{L}\right), \\
 I(x, 0) &= 0.2 + 0.05 \sin\left(\frac{2\pi x}{L}\right), \\
 R(x, 0) &= K - S(x, 0) - I(x, 0), \\
 Y(x, 0) &= 0.3 + 0.2 \sin\left(\frac{2\pi x}{L}\right).
 \end{aligned}$$

Neumann boundary conditions have been applied for this simulation. The numerical results were obtained using the explicit numerical method introduced at the beginning of this section. The following figures display the densities of susceptible, infected, and recovered plants, as well as the density of insect vectors, at each point x in the spatial domain and over the time interval $t \in [0, 20s]$.

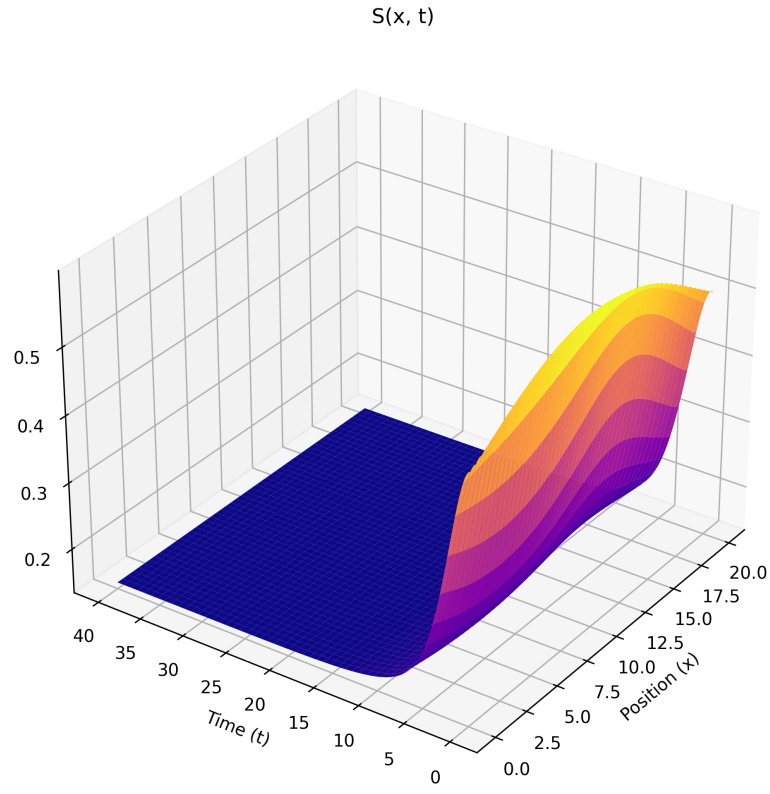


Figure 4: Density of the susceptible plant hosts $S(x, t)$ from (7).

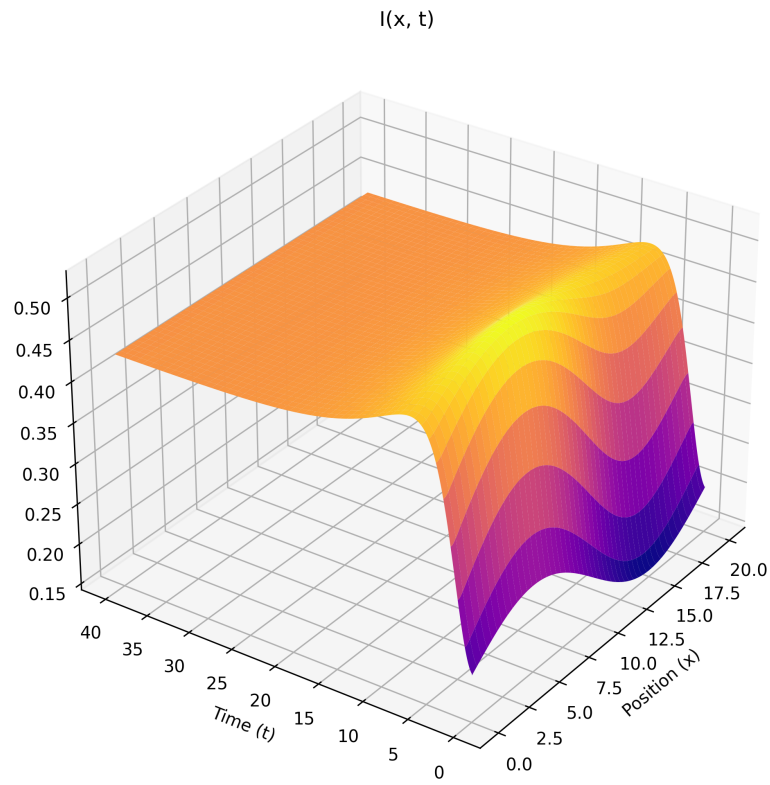


Figure 5: Density of the infected plant hosts $I(x, t)$ from (7).

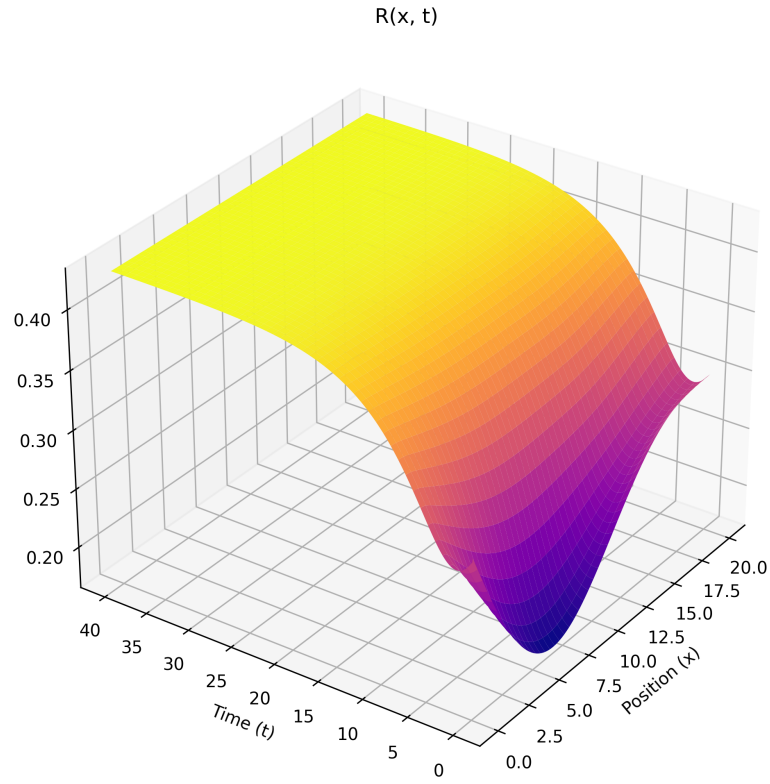


Figure 6: Density of the recovered plant hosts $R(x, t)$ from (7).

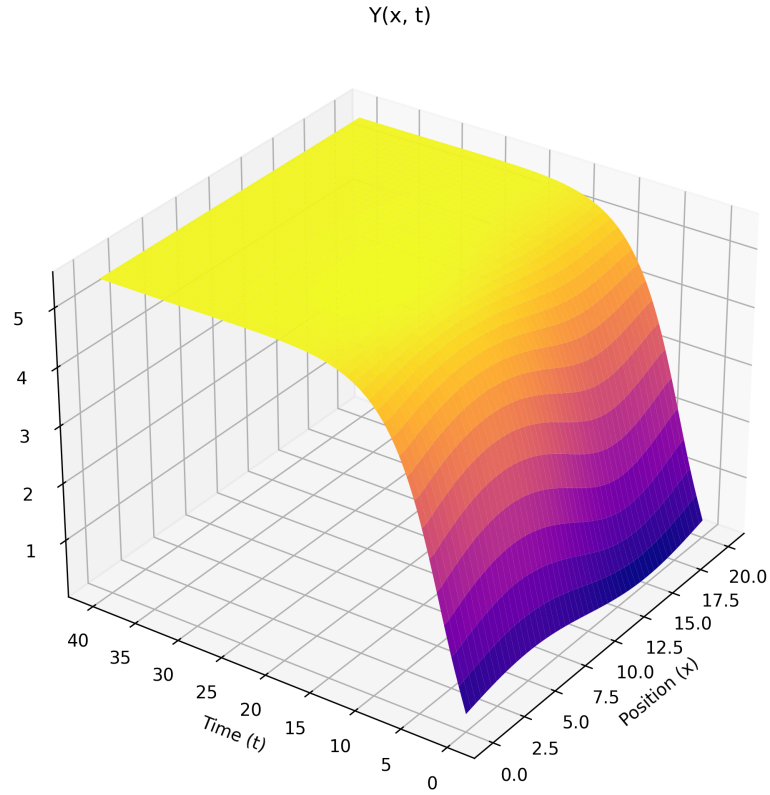


Figure 7: Density of the infected insect vectors $Y(x, t)$ from (7).

4 Conclusions

loading...

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