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Modeling plant virus propagation with delays

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ABSTRACT

Plants play a vital role in the everyday life of all organisms on earth. Sometimes, however, plants become infected with a virus. This can have a devastating effect on the ecosystem that depends on it. An insect–vector can cause the transmission of the virus from plant to plant. In this paper, a system of ordinary differential equations was first used to model the interaction between the insects and the plants. We found the equilibria of the model, and we analyzed the stability using the Reproductive number, derived by the next generation matrix. Afterwards, we introduced a delay to the model and a system of delay differential equations (DDE) was obtained. Analysis of the DDEs dynamics was performed including equilibria, stability, and bifurcations. Then, numerical solutions of the ODE and the DDE were compared and conclusions are given.

1. Introduction

Plants are essential to not only man's existence, but to every species on Earth. Sometimes, plants become infected with a disease. There are many different ways that a plant may contract a disease. One of which is bacterial. For example, *Magnaporthe oryzae* is a bacteria that causes rice blast which can cause rice production to decrease up to 90% [1]. Also, a plant may become infected with a fungal disease. One such fungus is *Botrytis cinerea*, and it destroys the fruits the plant produces [2]. In this paper, we will be interested in modeling the interaction between plants, a plant virus, and the insect–vector that transfers the virus from one plant to another.

In order to work with the viruses, we must first understand how they replicate. For a virus to replicate, it must invade a healthy cell and use the cell's DNA or RNA to reproduce. The infected cell bursts and several copies of the virus exit. The new virus particles infect other cells. The viruses continue this process until there are no more healthy cells to invade. Different virus processes have been widely studied. See for example [3–5], and [6].

Plant viruses cause many diseases some of which that affect many plants all over the world. For instance, the *Citrus tristeza* virus once wiped out millions of trees in Brazil [7]. But in order for an infected plant to infect another, a virus from an infected plant must come in contact with a healthy plant. This may happen in several different ways. For example, a field worker might contact the juices of an infected plant and contact a healthy plant. Or maybe the interaction is more organic in that an infected plant might have its juices fall directly onto a plant underneath it. Another, and the most common, way for the transference is by an insect–vector. Insect–vectors transmit more than 70% of all known plant viruses [7]. Many vectors that transport these viruses include aphids, whiteflies, leafhoppers, etc.

There are many ways that plant viruses interact with the vectors, but in this paper we are concerned with circulative, persistent transmission. This transmission works in the following way. The vectors consume sap from an infected host through their stylets. The viruses in the sap enter the salivary glands, circulate within the vector, and then cause infection.

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This process can take a few hours or up to a day depending on the insect–vector interaction. The vector will hold the infection for the rest of its life. When the infected vector contacts a healthy plant, some virus particles leave the vector and invade the plant [8–10]. Once the virus has circulated and propagated throughout the plant, the plant may use defense mechanisms to combat the virus. On example is by antiviral RNA silencing, a process by which slicing or translation repression of viruses occurs [11].

Many physical and biological processes (gestation, maturation, reproduction, infection) take time to complete. In the case of a viral infection, it takes time for a virus to invade a cell, reproduce, and spread in order throughout the host. This process time is a delay time. Processes with delay times can be modeled using delay differential equations (DDE). DDEs have been used to model time delays of many biological processes. For example the authors in this Ref. [12], used delays to model the delay time between infection of a $CD4^+$ T-cell and the introduction of the HIV virus to the host. The authors notice that the introduction of the delay gave rise to oscillations in their solutions. Aside from introducing oscillations to the model, delay times can change the dynamics of the model in many other ways. For example, delays may change the solutions, cause discontinuities in the derivative, affect uniqueness, or change the stability. Despite the complications and numerical difficulties, results are more realistic from the biological and physical points of view. The effects of the delay times are highly coupled with the parameters of the model. See for example [13,14].

Although there are many models that describe the interaction between vectors and humans, there are not as many that describe the relationship between plants and vectors. In this paper, we construct a model assuming that the virus gets transmitted by plant and insect contact, and is modeled using Holing type II [15], since insects can only bite a limited number of plants. In [8] a similar model is presented that also consider that infection can be transmitted from plant to plant. We do not consider such transmission because there is empirical evidence it is not common [9]. In Section 2, we will further develop the assumptions of the model and introduce the system of ordinary differential equations. Then equilibria and stability will be analyzed with the basic reproduction number, using the next generation matrix approach. In Section 3, we will introduce a delay to the model. We perform a stability analysis in this section as well. In Section 4, we compare the numerical solutions to the systems of ODEs and DDEs. Also, some numerical bifurcation analysis will be presented. Finally in Section 5, some conclusions are given.

2. Model assumptions and system of ODEs

In this model we do not consider a specific virus, plant, or insect–vector, but we make the following assumptions. These are similar assumptions made in [8]. We assume three populations of plants: Susceptible, S, healthy but subject to be infected by the virus, Infective, I, already infected by the virus, and Recovered, R. Each of these variables describes their respective population at time, t. The total number of plants will be denoted by the fixed positive constant K, $K = S + I + R$. It is reasonable to assume K is fixed, because when a plant dies by the virus or natural death in farms, it is replaced with a new healthy plant. The new plant shares the same characteristics of the plant it replaced, before it was infected.

For the insect–vectors, there will be two populations: Susceptible, X, and Infective, Y. Each of which describes the populations at time, t, as well. The total number of insects will be denoted by $N = X + Y$. Also, the rate at which the insects enter the system, by birth or immigration, is constant. There is no vertical transmission of the virus, and vectors cannot transmit the virus to another vector. In addition, vectors do not get killed by the virus nor do they defend against it. The vector will keep the virus for its lifespan and does not recover. The infective insects do not get sick from the virus, they are just carriers.

As far as the interaction between the insects and the plants, an infected insect–vector can only infect a susceptible plant. The only way for the vector to become infected is through coming in contact with an infected plant. The interaction between vector and plant is of predator–prey Holling type 2 [15].

The following table lists the parameters of the model.

Parameter	Description	Value
K	Total plant host population	50–1000
N	Total insect–vector population	50–100
β	Infection rate of plants due to vectors	0.01–0.02
β_1	Infection rate of vectors due to plants	0.01–0.02
α	Saturation constant of plants due to vectors	0.01
α_1	Saturation constant of vectors due to plants	0.02
μ	Natural death rate of plants	0–0.1
m	Natural death rate of vectors	0–0.5
γ	Recovery rate of plants	0–0.25
Λ	Replenishing rate of vectors (birth and/or immigration)	5
d	Death rate of infected plants due to the disease	0.1

Fig. 1 is a flow diagram for the interactions.

In the flow diagram, the solid lines represent an individual moving from one class to the next. Whereas the dashed line indicates contact between the two classes.