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Mathematical Analysis of Vector-Borne Diseases on Plants

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***Abstract*—Many models of vector borne infectious diseases have been constructed and analyzed mathematically. The host populations in such models are typically animals. This work deals with the specific case of a plant host population taking into consideration the particular properties of plants. The main epidemiological issues of transmission, persistency, thresholds, interventions, etc., are all considered in this setting and discussed on a representative set of two models - one epidemic and one endemic. The main properties of the models are formulated as theorems and illustrated via computer simulations. In particular, we provide some threshold parameters that summarize the dynamics of the system and help to choose appropriate and efficient control tools or strategies for crop protection. The full proofs of the theorems are omitted but their main ideas are discussed in some detail.**

***Keywords*-plant epidemiology, thresholds, endemic and epidemic models, mathematical analysis**

I. Introduction

For many diseases [1] and vector borne diseases, like Malaria [19], [15], Dengue [16] or Chikungunya [6] a lot of mathematical models have been developed and studied, using various mathematical theory [1]. Since 1911, and the Malaria model developed by Sir Ronald Ross [19], many progress have been made in mathematical epidemiology, with some great achievements. One of them being the famous Basic Reproduction Ratio [1], [4], $\mathcal{R}_0$, a threshold parameter aggregating important information related to the dynamic of the disease. In particular, for vector-borne disease, $\mathcal{R}_0$ gives precious indications on parameters that may be modified through vector control (insecticide, larvicide, biological control, etc.) in order to reduce the epidemiological risk.

Mathematical modeling applied to human and animal diseases has shown great results, and, in some sense, it seems natural to use the same kind of approach to study plant vector-borne disease. In this paper we consider mathematical models for vector-borne plant diseases constructing by using compartmental structuring of the involved populations. While these models share significant similarity with compartmental models of human and animal vector borne diseases like Dengue, Chikungunya, Malaria, they also need to account for some specific issues related to plants. The Mathematical Analysis is based on the tools and concepts developed already for compartmental population models, derived and/or computed here in the specific setting of plant diseases. Hence, all results (threshold quantities, short and long term predictions, damage estimates, risks) are particularly related to plant diseases. In order to keep this fairly general discussion also tangible, we focus the exposition on the construction and the mathematical analysis of two models - one of short term, e.g. annual, epidemic disease involving a noncirculative pathogen and one of a long term endemic disease caused by a circulative pathogen. Deriving and interpretation of threshold quantities, providing rigorous mathematical statements as well as computer simulations are essential part of this work. In order to make the paper as self-contained as possible we give in the next section a brief review of some basic facts and issues related to plant epidemiology. The endemic and the epidemic models mentioned above are discussed in Sections 3 and 4 respectively. In both sections the qualitative behavior of the solutions of the respective models is characterized in the threshold parameter known in epidemiology as reproduction number or reproduction ratio. The obtained threshold values provide for the design of control strategies to stop or to contain the disease. The theoretical results are supported by illustrative examples. Summary and some directions for future work are given in the conclusion.

II. About Plant Epidemiology

Like for humans and animals, plants have to deal with viruses (obligate parasites). However, plant viruses have difficulties to overcome some transmission restrictions due to plant immobility (no plant-to-plant contact) and the wall made of cellulose and pectin that surrounds all plant cells limiting the entry and exit of viruses. To circumvent these obstacles, plant viruses have developed different strategies to transfer efficiently from one host to another. Moreover, in the Plant kingdom, the transmission mechanisms are more complex.

Plant viruses, like some human or animal viruses need vectors. Among many vectors, Aphids are the most important: they transmit more than 50% of the plant viruses vectored by insects [17]. In England more than 200 aphid species have been reported to transmit virus. That is why a deep understanding of the vector and its behavior are

necessary to understand the spread of a disease and to apply efficient control tools.

Another very important difference between animal viruses and plant viruses lies in the transmission process. No mechanical or direct transmission is possible, an extrinsic incubation period is necessary such that an infected mosquito becomes infective, i.e. to be able to infect susceptible humans. For instance, it takes two days for the CHIKV virus to move from the salivary glands after oral infection [5] (for Dengue it takes almost ten days). During this extrinsic incubation period, mosquitoes are infected but not infectious.

For plant, virus transmission characteristics are different and depend on the interaction between the virus and the vector [7]. Mechanical and Biological transmissions are considered to be the main way for virus transmission by arthropod vectors. However theses terms are not always clear and do not represent efficiently the mechanism of insect transmission of plant-infecting viruses.

For plant, it is necessary to consider different type of transmission mechanisms, described by time events. Three main transmission modes have been defined:

- the nonpersistent mode, with viruses acquired within seconds, and not retained for more than a few hours by their vectors.
- the semi-persistent mode, with viruses acquired within minutes and retained for several hours.
- the persistent mode if they remain in the vector for the rest of its life. In that case the acquisition and inoculation times, as well as the latent periods are of days.

An additional classification has been considered by biologists: they distinguish viruses that remain "outside" the vectors, from those traversing the intestine via the body lumen to the salivary glands. The first type of viruses enter the category of noncirculative viruses, that have a more superficial and transient relationship with the vector: transmissible virus particles attach only to the exterior mouthpieces of the insect from which they are released into a new host. In the animal kingdom, this kind of transmission is also called mechanical transmission and is important in the epidemiology of many animal diseases (many Diptera species are responsible for mechanical transmission).

The second type of plant viruses are called circulative viruses that may be inoculated with the saliva into a new host plant. A circulative virus can be further classified as circulative propagative or circulative nonpropagative according as the virus replicates within the vector or not.

In general, noncirculative viruses are nonpersistent or semi-persistent, while circulative viruses are semi-persistent or persistent .

Altogether plant viruses strongly depend on vectors for their transmission and survival, indicating that modeling have to take into account the vectors population. Moreover, it seems natural to consider vector control as an efficient method to reduce the epidemiological risk.

Aphids are main vectors of plant viruses [2] because of their non-destructive feeding behavior. In fact, once landed on a plant, aphids first probe the prospective food source by short, only seconds lasting intracellular punctures in epidermis and mesophile cells that do not even kill the punctured cells.After these exploratory punctures and when they judge the plant as suited, the aphids insert their proboscis-like mouthpieces (stylets) into the phloem and feed from its sap for time spans that may exceed several hours. Depending on the tissues they infect, plant viruses can be acquired by aphids during either or only one of the two puncture phases. For example, Luteoviruses are only acquired from the vascular tissues, whereas Cauliflower mosaic virus is acquired from both tissues [18].

Thus, due to the diversity of disease transmissions, Plant diseases are very interesting from the Modelling point of view. Moreover, like for Human and Animal Epidemiology, Mathematical Modelling can be helpful to better understand the main dynamic of Plant Diseases and thus to improve control tools.

III. Epidemic model of vector-borne plant disease

Since the pioneering work of van der Plank in the sixties, several works have been done in the modelling of plant epidemiology to understand the spreading and/or the persistence of a disease for the aerial part of the plant or the root system. For a brief history and an overview see [8], [9], [10], [11], [20] among many others.

Here we consider a typical situation of annual plants in a cultivated field. The plants are all simultaneously planted and develop more or less in the same way. During the season no new plants are added to the population and typically very few are lost. Therefore, we have a situation of constant host population without vital dynamics. Let K be the total number of plants. In order to be more specific in the model construction we assume that the pathogen is non-persistent and non-circulative. We also assume that the virus does not kill the plant but still causes damage. One example is the Potato virus Y (PVY) mentioned earlier. In this case, the potato produced by infected plant are rendered unmarketable due to spots and deformations.

In principle, for the plant we consider the compartments of healthy plats (H_p), infected but not yet infective (L_p), infective (I_p) and removed/recovered (R_p). In the later category are plants that are no longer infective. Naturally we have $H_p + L_p + I_p + R_p = K$.

The principal method of transmission is by vectors feeding on the plants. In the case of PVY these are aphids. We assume that the vectors, as it is the case with aphids, cause little damage to the plant by itself. Since the transmission occurs in a non-persistent and non-circulative manner, the

vector loses its ability to infect plants after a few feedings on healthy plants (when it can actually infect them). Hence we have an interesting dynamics where the force of infection on plants is a "disinfecting" force on the vector. For non-circulative viruses the vector has only two states infective and susceptible. The respective compartments are denoted by I_v and S_v. We have $S_v + I_v = V$ - the total number of vectors. The vector density (number of vectors per plant) is $\rho = \frac{V}{K}$. The compartmental structure and flow chart are represented on Figure 1.

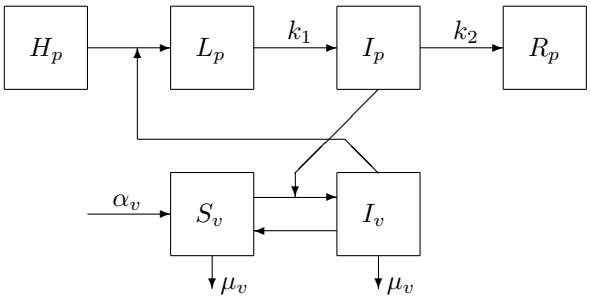

Figure 1. Compartmental structure and a population flow chart

The life span of the vector is typically small compared to the period of the development of the plan. Hence the vital dynamics of the vector population need to be considered. Usually the vector population grows as the crop develops. We assume some natural control over the population growth which induces a density dependent mortality rate. We also make the simplifying assumption that there is no immigration into the vector population. Emigration, if it exists, can be incorporated in the mortality rate. Hence, the vector population V is assumed to be governed by the following equation

$$\frac{dV}{dt} = \alpha_v V - (\mu_1 + \mu_2 V)V \tag{1}$$

where α_v is the birth rate and the mortality rate $\mu_v = \mu_1 + \mu_2 V$ comprises a density independent mortality rate μ_1 (that is the natural lifespan is $\frac{1}{\mu_1}$ time units, and a density dependent mortality rate $\mu_2 V$. The transfer rates k_1 and k_2 relate to the average waiting period for the respective compartment, that is the average length of the latent period of an infected plant is $\frac{1}{k_1}$ and then the plant is infective for a mean time $\frac{1}{k_2}$ days.

The essential part of the model is the mathematical representation of the forces of infection.

Force of infection from plant to vector: The force is the probability that a susceptible vector is infected in a unit of time. It is the product of the number of plants ϕ a vector visits per unit time, the probability a of transmission if the plant is infective and the probability that a plant is infective. Thus it equals $\phi a \frac{I_p}{K}$. Then the number of newly infected vectors per unit time is $\phi a S_v \frac{I_p}{K}$.

Force of infection from vector to plant: This force is the probability that a susceptible plant is infected in a unit time. It is the product of the average number of vectors visiting a plant per unit time, that is $\frac{\phi V}{K}$, the probability b of transmission if the vector is infective and the probability that a vector is infective. Thus it equals $b\frac{\phi V}{K}\frac{I_v}{V} = \phi b \frac{I_v}{K}$. Then the number of newly infected plants per unit time is $\phi b I_v \frac{H_p}{K}$.

"Disinfecting" force on the vectors: Since the virus does not multiply on the vector, after sometime it disappears. Then we have a transfer rate of δI_v from infective to susceptible, where $\frac{1}{\delta}$ is the average time of existence of the virus on the vector. However, we also need to consider that during the time $\frac{1}{\delta}$ the vector can be infected again. Considering the respective force of infection under the assumption that the number of infected plants remain constant this transfer rate is reduced by the factor of $e^{-\frac{\phi a I_p}{\delta K}}$. Let us note that when δ is large this factor is close to one and can be neglected. When δ is small then this factor makes the transfer rate even smaller and then can be assumed zero.

Using the forces of infection so outlined, the flow chart on Figure 1 is represented by the following system of differential equation

$$\begin{cases} \frac{dH_p}{dt} = -\phi b I_v \frac{H_p}{K}, \\ \frac{dL_p}{dt} = \phi b I_v \frac{H_p}{K} - k_1 L_p, \\ \frac{dI_p}{dt} = k_1 L_p - k_2 I_p, \\ \frac{dR_p}{dt} = k_2 I_p. \end{cases} \tag{2}$$

$$\begin{cases} \frac{dS_v}{dt} = \alpha_v V - (\mu_1 + \mu_2 V)S_v - \phi a S_v \frac{I_p}{K} + \delta I_v e^{-\frac{\phi a I_p}{\delta K}}, \\ \frac{dI_v}{dt} = \phi a S_v \frac{I_p}{K} - \delta I_v e^{-\frac{\phi a I_p}{\delta K}} - (\mu_1 + \mu_2 V)I_v \end{cases} \tag{3}$$

When studying its qualitative behavior it is more convenient to write the model in terms of the fractions of the populations in the respective compartments rather then actual numbers. The substitution $h_p = \frac{H_p}{K}$, $l_p = \frac{L_p}{K}$, $i_p = \frac{I_p}{K}$, $r_p = \frac{R_p}{K}$, $s_v = \frac{S_v}{V}$, $i_v = \frac{I_v}{V}$ leads to the following system

$$\frac{dh_p}{dt} = -\phi b i_v h_p \rho, \tag{4}$$

$$\frac{dl_p}{dt} = \phi b i_v h_p \rho - k_1 l_p, \tag{5}$$

$$\frac{di_p}{dt} = k_1 l_p - k_2 i_p, \tag{6}$$

$$\frac{dr_p}{dt} = k_2 i_p, \tag{7}$$

$$\frac{ds_v}{dt} = \alpha_v i_v - \phi a s_v i_p + \delta i_v e^{-\frac{\phi a i_p}{\delta}}, \tag{8}$$

$$\frac{di_v}{dt} = \phi a s_v i_p - \delta i_v e^{-\frac{\phi a i_p}{\delta}} - \alpha_v i_v, \tag{9}$$

$$\frac{d\rho}{dt} = \beta\rho(\hat{\rho} - \rho) \tag{10}$$

where equation (10) for the vector density, $\rho = \frac{V}{K}$, is obtained from (1) by denoting $\beta = \mu_2 K$ and $\hat{\rho} = \frac{\alpha_v - \mu_1}{\mu_2 K}$.

Note that the system can be simplified in the following ways: (i) equation (7) can be decoupled; (ii) using $s_v + i_v = 1$ equation (8) can be eliminated; (iii) equation (10) can be solved independently. Then we have

$$\begin{aligned}
\frac{dh_p}{dt} &= -\phi b i_v h_p \rho(t), && (11)\\
\frac{dl_p}{dt} &= \phi b i_v h_p \rho(t) - k_1 l_p, && (12)\\
\frac{di_p}{dt} &= k_1 l_p - k_2 i_p, && (13)\\
\frac{di_v}{dt} &= \phi a (1 - i_v) i_p - \delta i_v e^{-\frac{\phi a i_p}{\delta}} - \alpha_v i_v. && (14)
\end{aligned}$$

Here the function ρ is obtained as a solution of (10) in an explicit form. However, the relevant properties are that $\rho(t)$ is nonnegative and that $\rho(t) \to \hat{\rho}$ as $t \to \infty$. These two properties are used in the next two theorems.

Theorem 1: For any continuous nonnegative function ρ the system of differential equations (11)–(14) defines a (positive) dynamical system on the compact domain

$$\Omega = \left\{ x = (h_p, l_p, i_p, i_v)^T \in \mathbb{R}^4 \;\middle|\; \begin{array}{l} x \geq 0, \\ h_p + l_p + i_p \leq 1, \\ i_v \leq 1 \end{array} \right\} \quad (15)$$

By equating the right-hand side of (11)–(14) to zero we obtain that the equilibria of the system comprise the set

$$\mathcal{P} = \{x = (h_p, 0, 0, 0) : 0 \leq h_p \leq 1\} \subset \Omega.$$

The stability of equilibria of epidemiological models is studied by the basic reproduction ratio of the pathogen. It is typically determined by using a next generation matrix (NGM), [21]. For our model we proceed as follows. We write the equation for the vector $y = (l_p, i_p, i_v)^T$ of infected compartments in the form

$$\begin{aligned}
\frac{dy}{dt} &= F(x) - G(x) \\
&= \begin{pmatrix} \phi b i_v h_p \rho \\ k_1 l_p \\ \phi a i_p \end{pmatrix} - \begin{pmatrix} k_1 l_p \\ k_2 i_p \\ \phi a i_v i_p + \delta i_v e^{-\frac{\phi a i_p}{\delta}} + \alpha i_v \end{pmatrix}
\end{aligned}$$

Then for any $x \in \mathcal{P}$ we have

$$\begin{aligned}
NGM(x) &= \left(\frac{dF}{dy} \right) \left(\frac{dG}{dy} \right)^{-1} \Bigg|_{y=0} \\
&= \begin{pmatrix} 0 & 0 & \phi b \rho h_p \\ k_1 & 0 & 0 \\ 0 & \phi a & 0 \end{pmatrix} \begin{pmatrix} k_1 & 0 & 0 \\ 0 & k_2 & 0 \\ 0 & 0 & \delta + \alpha_v \end{pmatrix}^{-1} \\
&= \begin{pmatrix} 0 & 0 & \frac{\phi b \rho h_p}{\delta + \alpha_v} \\ 1 & 0 & 0 \\ 0 & \frac{\phi a}{k_2} & 0 \end{pmatrix}.
\end{aligned}$$

The reproduction ratio at x is defined as the spectral radius of $NGM(x)$. Hence we have

$$\mathcal{R}(x) = \sqrt[3]{\frac{\phi^2 a b \rho h_p}{k_2(\alpha_v + \delta)}}.$$

The basic reproduction ratio $\mathcal{R}_0$ is the largest value of $\mathcal{R}(x)$ and is obtained when all plants are healthy, that is $h_p = 1$. Thus the "standard" basic reproduction Number $\mathcal{R}_0$ is related to the equilibrium $(1, 0, 0, 0)$, that is

$$\mathcal{R}_0 = \sqrt[3]{\frac{\phi^2 a b \rho}{k_2(\alpha_v + \delta)}}.$$

The stability of an equilibrium x is related to the condition $\mathcal{R}(x) \leq 1$. Since $\rho(t) \to \hat{\rho}$ as $t \to \infty$ the latter inequality is required for $\rho = \hat{\rho}$. Then this inequality can equivalently be written as

$$h_p \leq h_p^* = \frac{k_2(\alpha_v + \delta)}{\phi^2 a b \hat{\rho}} \quad (16)$$

Using the threshold value h_p^* of h_p the long term behavior of the solutions of the model (11)–(14) is characterized as follows.

Theorem 2: If the function $\rho(t)$ is nonnegative and such that $\rho(t) \to \hat{\rho}$ as $t \to \infty$, the set

$$\mathcal{P}_s = \{x = (h_p, 0, 0, 0) : 0 \leq h_p \leq \min\{1, h_p^*\}\} \subset \mathcal{P}$$

consists of all stable equilibria of the dynamical system defined via (11)–(14). The equilibria in $\mathcal{P}_u = \mathcal{P} \setminus \mathcal{P}_s$ are unstable. The set $\mathcal{P}_s$ is a stable invariant set with basin of attraction $\Omega \setminus \mathcal{P}_u$. More precisely, every trajectory initiated in $\Omega \setminus \mathcal{P}_u$ converges to a point in $\mathcal{P}_s$.

Some trajectories representing the progression of a disease ($l_p + i_p$ versus h_p) in the case when $h_p^* < 1$ are presented in Figure 2 and Figure 3. The values of the parameters are $\phi = \hat{\rho} = 1$, $a = b = 0.2$, $k_1 = 0.2$, $k_2 = 0.1$, $\alpha_v = 0.05$, $\beta = 0.01$, $\delta = 0.2$. One has to note that while variations in the values of the parameters may change the shape of the curves, they influence the asymptotic behavior only jointly through the value of h_p^*. Further, the shape of the curves is affected by the size of the initial infection and wether it is brought in by the plants, e.g. infected seeds, see Figure 2, or via the vectors, see Figure 3. Nevertheless, in all cases eventually any trajectory approaches a limit in the interval $[0, h_p^*]$. Moreover, one can observe on Figure 2 and Figure 3 that if the disease is introduced in a fully susceptible population, that is $r_p = 0$, the trajectories of all solutions are above the separatrix via the disease free equilibrium (1,0,0,0). In this case the limits of all solutions are in a much smaller interval. One may further note that the lower the size of the initial infection the larger the fraction of the remaining healthy plants. The explicit formula (16) for the threshold h_p^* can be used to derive strategies for control of the disease. For example, one can protect the plants to reduce ϕ or reduce the equilibrium viral density $\hat{\rho}$.

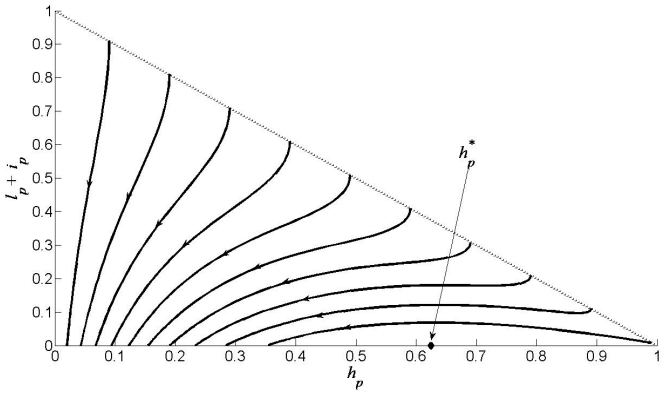

Figure 2. Infection introduced in plant

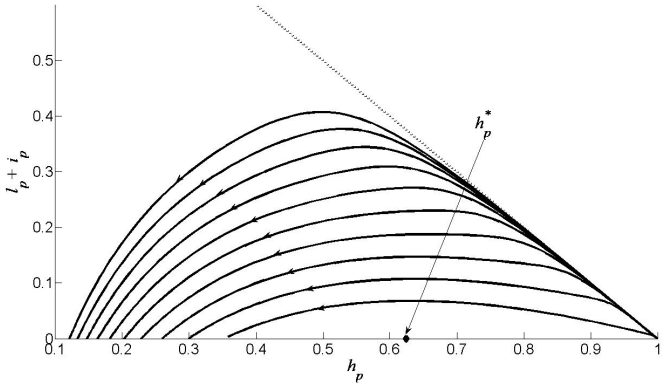

Figure 3. Infection introduced in vector

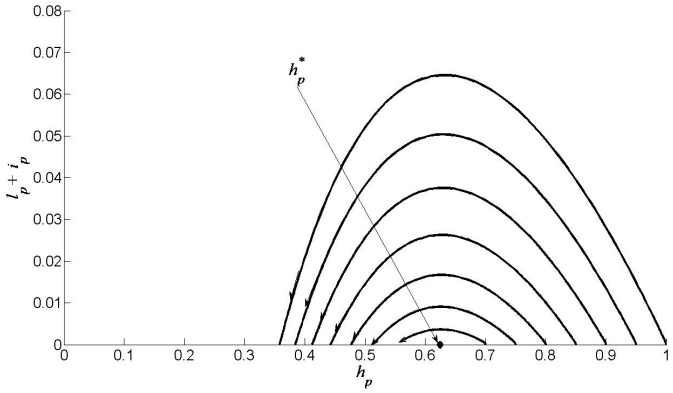

Figure 4. "Diluted" crop population

The model further suggests that one can reduce the effect or even prevent an epidemic by using "dilution" of the population of plants. Indeed, by definition, compartment R_p consists of plants that can not transmit the disease. In principle, these need to be good hosts for the vectors but need not be the same plant species. Suppose that there is a plant species which a good host for the vector but it is not susceptible to the pathogen and suppose that this species is planted on the same field randomly mixed with the original plants. Then these non-susceptible plants are accounted for in the R_p compartment and we have $h_p(0) + l_p(0) + i_p(0) < 1$. Typical trajectories for varying fraction of the "diluting" plant are presented on Figure 4. One can easily observe that in such situation the damage to the crops is much smaller. For example, if the diluting plant is 30%, then eventually the healthy plants are around 56%. So the loss is $\frac{70-56}{70} = 20\%$ of the originally planted crops. Moreover, if the "diluting" plant is a fraction larger than $1 - h_p^*$ and the source of infection is small, the infection quickly dies out and the damage is minimal, e.g. about equal to the originally infected plants if the plants are the source. Finally, let us note that this damage estimate is based on the assumption that the epidemic runs its course during the lifetime of the plant. Very often this is not the case and by taking into account the time dynamics of the system suitable intervention can be designed, e.g. delaying the introduction of the infection so that it cannot spread widely before the crops are harvested. However, this issue is very species specific and cannot be properly addressed in a general presentation as this one.

IV. Endemic model of vector-borne plant disease

In the model (2)–(3) or (4)–(9), considered in the preceding section, the infection eventually dies out due to the lack of sufficient number of susceptible plants. In the case when there is a constant supply of new susceptible plants the disease can establish itself in a long term endemic equilibrium. The recruits in the class healthy (susceptible) plants come either from the vital dynamics (new plants) or as a transfer from recovered plants (plants that after recovery can be reinfected). Here we consider a model which involves vital dynamics of the plant population and the pathogen is circulative, constructed similarly to [12]. The basic compartmental structure and flow chart are given on Figure 5, where the rate of births (new plants) and deaths in the plant population are assumed equal so that $H_p + L_p + I_p + R_p = K$ is constant.

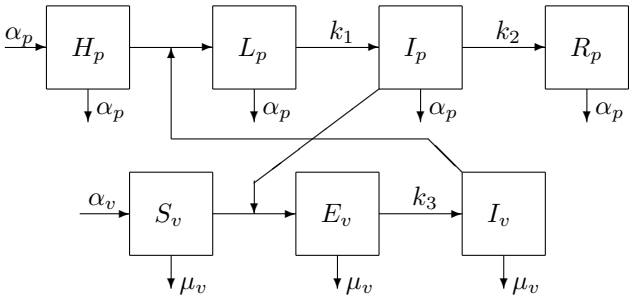

Figure 5. Compartmental structure and a population flow chart

Since the pathogen is assumed circulative, typically it requires time to develop within the vector. During that time the vectors are not yet infective. These vectors comprise the compartment E_v (exposed vectors). The transfer rate from

E_v to I_v is denoted by k_3 and corresponds to an average time of $\frac{1}{k_3}$ from inoculation to becoming infective. In a similar way to the epidemic model one can derive a system of equations for the fractions $h_p = \frac{H_p}{K}$, $l_p = \frac{L_p}{K}$, $i_p = \frac{I_p}{K}$, $r_p = \frac{R_p}{K}$, $s_v = \frac{S_v}{V}$, $e_v = \frac{E_v}{V}$, $i_v = \frac{I_v}{V}$ of the respective populations in each compartment. With the equation about r_p being decoupled and ρ being given by (10), we obtained the system in the following form

$$\begin{aligned}
\frac{dh_p}{dt} &= \alpha_p(1-h_p) - \phi b i_v h_p \rho(t), && (17)\\
\frac{dl_p}{dt} &= \phi b i_v h_p \rho(t) - (\alpha_p + k_1) l_p, && (18)\\
\frac{di_p}{dt} &= k_1 l_p - (\alpha_p + k_2) i_p, && (19)\\
\frac{ds_v}{dt} &= \alpha_v(1-s_v) - \phi a s_v i_p, && (20)\\
\frac{de_v}{dt} &= \phi a s_v i_p - (\alpha_v + k_3) e_v, && (21)\\
\frac{di_v}{dt} &= k_3 e_v - \alpha_v i_v, && (22)
\end{aligned}$$

Note that using $s_v + e_v + i_v = 1$ we can eliminate the equation for s_v. However, this creates technical difficulties for the mathematical analysis and it is not implemented here. We have the following theorem.

Theorem 3: For any continuous nonnegative function ρ the system of differential equations (17)–(22) defines a (positive) dynamical system on the compact domain

$$\Omega = \left\{ x = (h_p, l_p, i_p, s_v, e_v, i_v)^T \in \mathbb{R}^6 \;\middle|\; \begin{array}{l} x \geq 0, \\ h_p + l_p + i_p \leq 1, \\ s_v + e_v + i_v = 1 \end{array} \right\} \quad (23)$$

In the analysis of the asymptotic behavior of the solutions we need to consider that $\rho(t) \to \hat{\rho}$ as $t \to \infty$. It is easy to show that the long term behavior of the solutions of the system (17)–(22) for any initial value of $\rho(0)$ is the same as the solution of this system for $\rho = \hat{\rho}$ which we assume in the following discussion.

Due to the involvement of the vital dynamics of the plants the model has only one disease free equilibrium $DFE = (1, 0, 0, 1, 0, 0)^T$. As usual, the stability of DFE is characterized in terms of the basic reproduction ratio. To obtain the next generation matrix we write the equation for the vector $y = (l_p, i_p, e_v, i_v)^T$ of infected compartments in the form

$$\begin{aligned}
\frac{dy}{dt} &= F(x) - G(x)\\
&= \begin{pmatrix} \phi b i_v h_p \hat{\rho} \\ k_1 l_p \\ \phi a i_p s_v \\ k_3 e_v \end{pmatrix} - \begin{pmatrix} (\alpha_p + k_1) l_p \\ (\alpha_p + k_2) i_p \\ (\alpha_v + k_3) e_v \\ \alpha_v i_v \end{pmatrix}
\end{aligned}$$

Then

$$NGM = \left(\frac{dF}{dy}\right)\left(\frac{dG}{dy}\right)^{-1}\Bigg|_{x=DFE} =$$

$$\begin{pmatrix} 0 & 0 & 0 & \phi b \hat{\rho} \\ k_1 & 0 & 0 & 0 \\ 0 & \phi a & 0 & 0 \\ 0 & 0 & k_3 & 0 \end{pmatrix} \begin{pmatrix} \alpha_p{+}k_1 & 0 & 0 & 0 \\ 0 & \alpha_p{+}k_2 & 0 & 0 \\ 0 & 0 & \alpha_v{+}k_3 & 0 \\ 0 & 0 & 0 & \alpha_v \end{pmatrix}^{-1}$$

$$= \begin{pmatrix} 0 & 0 & 0 & \frac{\phi b \hat{\rho}}{\alpha_v} \\ \frac{k_1}{\alpha_p + k_1} & 0 & 0 & 0 \\ 0 & \frac{\phi a}{\alpha_p + k_2} & 0 & 0 \\ 0 & 0 & \frac{k_3}{\alpha_v + k_3} & 0 \end{pmatrix}$$

Then $\mathcal{R}_0$ calculated as the spectral radius of NGM is

$$\mathcal{R}_0 = \sqrt[4]{\frac{\phi^2 a b k_1 k_3 \hat{\rho}}{(\alpha_p + k_1)(\alpha_p + k_2)(\alpha_v + k_3)\alpha_v}}\,.$$

Theorem 4: Given $\rho(t) \to \hat{\rho}$ as $t \to \infty$, DFE is globally asymptotically stable equilibrium on Ω if and only if $\mathcal{R}_0 \leq 1$. Moreover, if $\mathcal{R}_0 > 1$ DFE is unstable and repelling.

As a consequence of Theorem 3 and Theorem 4 we obtain that if $\mathcal{R}_0 > 1$ the number of infected plants is bounded away from zero as $t \to +\infty$. This means that the pathogen persists in the plant population indefinitely, that is, the disease is endemic. The precise behavior of the solutions of the dynamical system is difficult to prove mathematically. By equating the right hand side of (17)–(22) to zero in addition to DFE we obtain a unique endemic equilibrium (EE). The simulations presented on Figure 6 indicate that this equilibrium is stable and attractive with basin of attraction $\Omega \setminus \{x \in \Omega : l_p = i_p = e_v = i_v = 0\}$. As mentioned before the parameters of the model influence the qualitative behavior of the solutions only jointly via $\mathcal{R}_0$. In the simulations the set of parameter values is the same as in the Epidemic Model augmented with $\alpha_p = 0.002$, $k_3 = 0.2$. For these values we have $\mathcal{R}_0 = 1.579$. It is assumed that the pathogen is first introduced in the plant population.

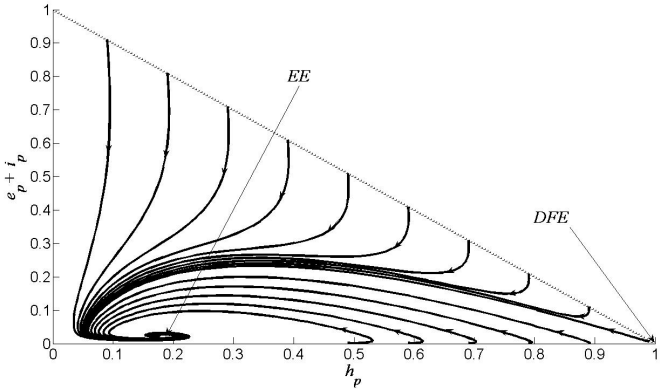

Figure 6. Endemic Equilibrium

One obvious way of reducing $\mathcal{R}_0$ is by reducing the vector density. For example, reducing $\hat{\rho}$ by a factor of ten yields $\mathcal{R}_0 = 0.888 < 1$. Then DFE is stable and globally attractive as illustrated on Figure 7.

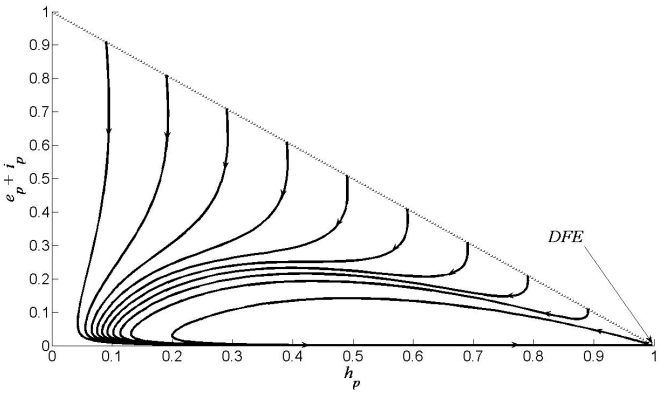

Figure 7. DFE stable and globally attractive

V. On the proofs of the theorems

The sets denoted as Ω in the epidemic and in the endemic model, see (15) and (23), give the biologically feasible region for the values of the involved variable. Therefore, proving Theorem 1 and Theorem 3 is an important step in showing the relevance of the models. In both cases the statement can be proved by showing that the vector field defined by the right hand side of the model is directed inwards at the boundary of Ω. For invariance of the equality in (23) we add the equations (20)–(22) to show that if $s_v + e_v + i_v = 1$ then $\frac{d(s_v+e_v+i_v)}{dt} = 0$.

The stability properties of equilibria are typically addressed by using the eigenvalues of the Jacobian of the right hand side of the system. This is the approach used in both Theorem 2 and Theorem 4 regarding the stability of the equilibria. We note that for the epidemic model the equilibria in the set $\mathcal{P}_s$ are stable but not attractive, that is they are not asymptotically stable. Nevertheless, the set $\mathcal{P}_s$, which is also stable invariant set of the system, has a basin of attraction containing the whole Ω except for $\mathcal{P}_u$. This result is obtained by using that h_p is decreasing and bounded. Hence it has a limit $\overline{h}_p$ as $t \to +\infty$. Using the LaSalle Invariance Principle it is enough to consider the largest invariant set satisfying $h_p = \overline{h}_p$. Then similar argument gives that l_p converges to a limit and so on. Further, if a solution of the system has a limit as $t \to +\infty$ then this limit is an equilibrium. The fact that this equilibrium is in $\mathcal{P}_s$ is easily seen.

Let us note that proving global attractiveness or a basin of attraction of an equilibrium is often a complicated issue since there is no general method for establishing such properties. In every case one needs to utilize in some way the particular structure of the system of equations as in the method for Theorem 2 mentioned above. A method for proving global asymptotic stability of the disease free equilibrium using the specific structure of compartmental epidemiological models is outlined in [14] and [3]. This method is applied in the proof of Theorem 4. The proof of a similar theorem specifying the basin of attraction of the Endemic Equilibrium as depicted in Figure 6 is still an outstanding issue.

VI. Conclusion

Plant diseases, similarly to animal and human diseases, are often transmitted by a vector. Mathematical modelling of such diseases can contribute to better understanding of their transmission dynamics, threshold quantities and intervention opportunities, thus making a contribution to better control strategies. These models are quite different in nature and between themselves cover most of the important epidemiological issues of vector borne diseases on plants. The explicit form of the thresholds characterizing the properties of the models are also indicators of possible control measures. Naturally, these measures in any specific case are related to the type of the virus, where one needs to consider the available controls, the cost of intervention, damage on the crop by the pathogen, damage on the crop by the intervention, etc.

The models discussed here can be adapted better to any particular pathogen-host-vector situation by removing some of the simplifying assumptions and by respectively enlarging the set of parameters, e.g. by considering vector migration, vectors feeding on other plants (that may or may not susceptible), climatic factors and others. In the future we intend to consider also plant growth in order to take into account plant-insect interactions. While this topic is interesting in itself it also relates to the impact of the pathogen on crop yields which depends on the time the disease appear during plant growth. Further, in our future research we plant to study specific cases, where our main interest is the identification of sustainable and "green" control strategies.

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