A mathematical model of flavescence dorée epidemiology

A Mathematical Model of Flavescence Dorée Epidemiology

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Abstract

Flavescence dorée (FD) is a disease of grapevine transmitted by an insect vector, *Scaphoideus titanus* Ball. At present, no prophylaxis exists, so mandatory control procedures (e.g. removal of infected plants, and insecticidal sprays to avoid transmission) are in place in Italy and other European countries. We propose a model of the epidemiology of FD by taking into account the different aspects involved into the transmission process (acquisition of the disease, latency and expression of symptoms, recovery rate, removal and replacement of infected plants, insecticidal treatments, and the effect of hotbeds). The model was constructed as a system of first order nonlinear ODEs in four compartment variables. A bifurcation analysis shows that, in the absence of hotbeds, the state of healthy vineyard is stable, if removal and replacement of infected plants is implemented. In the presence of hotbeds, depending on the grapevine density, we find either a single family of equilibria in which the health of the vineyard gradually deteriorates for progressively more severe hotbeds, or multiple equilibria that give rise to sudden transitions from a nearly healthy vineyard to a highly deteriorated one when the severity of the hotbeds crosses a critical value. These results show the long-term risks in planting new vineyards in environmental situations where strong hotbeds of FD are present or may arise in the surroundings.

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1. Introduction

Flavescence dorée (hereafter FD) is a serious disease of grapevine, widespread in many European countries, caused by phytoplasmas belonging to 16SrV-C and 16SrV-D ribosomal groups (Malembic-Maher et al., 2009). Symptoms of FD include leaf yellowing or redness, lack of lignification of canes, lack of blossom. The infected plants generally stop producing grapes, and often die after a few years, although sometimes a full recovery is observed. Symptoms of FD are usually shown after a latency period of 1-3 years from infection; young plants are more likely to show symptoms just one year after infection (Osler et al., 2002; Morone et al., 2007). Other grapevine diseases caused by phytoplasmas, such as Bois noir, may show similar symptoms: discrimination is based on specific molecular analyses (Galetto et al., 2005).

FD is transmitted vine-to-vine by an insect vector, *Scaphoideus titanus* Ball (Hemiptera: Cicadellidae), native to North America and introduced into Europe in the late 1950s (Bonfils and Schvester, 1960; Chuche and Thiéry, 2014). *S. titanus* feeds and reproduces only on grapevine (*Vitis* spp.), has a single generation per year, and overwinters in the egg stage, laid under the bark of grapes (Vidano, 1964; Bagnoli and Gargani, 2011; Chuche and Thiéry, 2014). Eggs start to hatch during spring, and the insect over-goes through five nymphal instars before becoming adult during summer (Vidano, 1964; Chuche and Thiéry, 2014). Nymphs (mainly from the 3rd and later instars) acquire phytoplasmas when feeding on infected plants, and after a latency period lasting 4-5 weeks (meanwhile becoming adults) they are able to inoculate phytoplasmas to healthy plants (Bressan et al., 2005; Chuche and Thiéry, 2014). Once infective, insects retain vector capability through their lifetime; on the other hand, no transovarial transmission of 16 SrV phytoplasmas has been proved for *S. titanus* at present, therefore newly born insects have to feed on infected plants in order to acquire phytoplasmas (Alma et al., 1997; Chuche and Thiéry, 2014). Other insects are acknowledged to be occasional vectors (Filippin et al., 2009), however their role in the spread of Flavescence dorée to date is not considered to

be important.

Infected plants may be subject to recovery, with symptoms disappearing within a few years after the infection. Recovery rates depend on cultivar (Bellomo et al., 2007; Bosco and Marzachì, 2011; Pavan et al., 2012b) and age of plants, the youngest being the less able to recover (Schvester, 1970). Observed recovery rates are highly variable and range from 1% to 70% of the infected plants, but show a strong inverse dependence on the abundance of the vector insect, with the highest recovery rates observed in vineyards subject to aggressive insecticide treatments, and the lowest in vineyards subject to no treatments (Morone et al., 2007; Zorloni et al., 2008). This supports the notion that recovered plants are not immune from reinfection. However, recovered plants are not a source of phytoplasmas for insects (Galetto et al., 2014). There are some known instances in which plants develop some degree of resistance to further reinfection after recovering from phytoplasmosis (Osler et al., 2014). However, lacking any direct evidence of the development of a resistance to FD in grapes, we shall assume that recovered plants are just as susceptible as those that have never been infected.

In Italy FD is subject to mandatory control procedures, including sprays of insecticide against the vector and removal of the infected plants, which, however, may have higher cost than insecticide treatment. In many vine-growing areas abandoned vineyards and woods containing wild grapevine act as hotbeds of both phytoplasmas and *S. titanus* (Lessio et al., 2007; Pavan et al., 2012a; Lessio et al., 2014). Adults of *S. titanus* are able to move within and among vineyards. Outside a vineyard their density decays exponentially with the distance from the vineyard, and cannot be neglected in the first 300 m (Lessio et al., 2014).

In this paper we formulate a model of the time evolution of a FD infection in a single vineyard, by considering the different aspects involved into (or influencing the) transmission process. Given the knowledge of some parameters, the model may be used for forecasting the long-term outcome of an infection. More importantly, it highlights the key ecological factors involved in the infection process, and explains their mutual interactions and relative importance, thus

offering guidance for planning an adequate response. In particular it suggests that, with weak or no nearby hotbeds, removal and replacement of infected plants should be a sufficient response for maintaining the overall health of the vineyard, even without or with very mild insecticide treatments. It also suggest that high-density vineyards would be subject to sudden increases of the infected plants, and that in these cases insecticide treatments should help.

From a mathematical point of view, the model is a system of nonlinear, first-order, ordinary differential equations with four compartments that quantify the abundance of healthy full-grown (S), latent (L), infected (I), and young (G) plants.

The rest of the paper is structured as follows. In Section 2 we formulate the model and present the main mathematical results. In Section 3 we discuss the ecological significance of those results. Conclusions are given in Section 4. The Appendix 5 contains proofs and other mathematical details that, for brevity and clarity, were omitted in the main text.

2. The model

2.1. Formulation

Previous modeling efforts have focused on the short-term spread of the infection, on the economic viability of control strategies, or on the life-cycle of the *S. titanus* (Bressan et al., 2006; Morone et al., 2007; Pavan et al., 2012b; Maggi et al., 2013; Falzoi et al., 2014; Rigamonti et al., 2014). Although of undeniable interest, these models, some of which have a large number of free parameters, are not particularly suitable for understanding the long-term evolution of a vineyard subject to FD. Moreover, none of them explicitly takes into account the effect of hotbeds of infection. We have thus developed a simple model that describes the epidemiology of the infection on time scales longer than a year and up to a few decades.

Two facts lay the conceptual foundation of our model. The first is that the year-to-year population density of *S. titanus* in a vine growing area, in spite of the complicated life cycle, remains roughly constant, or at least of the

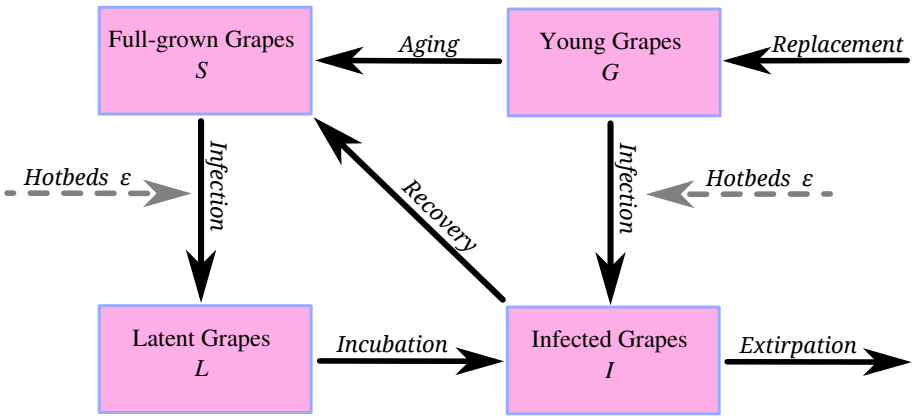

Figure 1: Graphical representation of the model. The grapevine population in a vineyard is split into four compartments, representing healthy, full-grown plants (S), healthy, young plants (G), latent plants (L) and infected plants (I). The solid black arrows represent the processes that increase (inward arrow) or decrease (outward arrow) the population of each compartment. The dashed gray arrows represent the effect of infection hotbeds close to the vineyard, triggering the infection fluxes in initially healthy vineyards.

same order of magnitude, if all known relevant factors (e.g. timing, number and effectiveness of insecticidal sprays, the presence of nearby hotbeds of infestation) are kept constant. (Lessio et al., 2011a,b; Maggi et al., 2013). The second is that there appears to be no transovarial transmission (Alma et al., 1997) of the phytoplasma: individuals of *S. titanus* become vectors of the phytoplasma by feeding on infected plants at the nymph stage, when the insect lacks the ability to move extensively from plant to plant and tend to cluster together on the same plant (Chuche and Thiéry, 2014). After the emergence, the much more mobile, flying adults spread across the vineyard. Those that have fed on infected plants at the nymph stage may then infect healthy plants. Therefore, at least as a first approximation, it seems safe to assume that the abundance of phytoplasma-carrying adults, at the scale of a vineyard, is fairly homogeneous and proportional to the number of infected plants. This upholds the modelling choice of omitting an explicit description of the population dynamics of *S. titanus*, which is simply considered as a coupling factor between the infected and the healthy plants. The processes that are explicitly modelled are shown in Figure 1.

A second choice is that of formulating a continuous-time model. We feel that

it is easier to describe the process of infection in a continuous-time setting, even if it has a strong seasonality. In addition, incubation, recovery and aging are processes that do not have an obvious discrete-time nature. On the other hand, replacement and extirpation are almost instantaneous events that occur once a year. The values of the model's variable, must thus be intended as representative of year-round averages. In Section 5 we propose a discrete-time version of the model, which takes into account the same processes of the continuous-time model, and has qualitatively the same equilibria and bifurcation diagram. We are thus confident that the conclusions deduced from the model do not depend on its continuous-time or discrete-time nature, but only on the physiological and ecological processes that are modelled.

Our model splits the grapevine population of a vineyard in four compartments (or stages), as shown in Figure 1. The variable S represents the density of healthy, full-grown plants (number of vines per unit area), and I represents the density of infected plants. The infection rate of the healthy plants is modeled by a term of the form

$$\text{Infection rate} = f(I)S \tag{1}$$

where f is an unknown function quantifying the efficiency of phytoplasma-carrying adults at infecting healthy plants. Obviously, f must be a growing function of the density of infected plants, with $f(0) = 0$. Laboratory experiments show that a non-negligible fraction of plants remains healthy, even after being exposed in insect-proof cages to a large number of infected individuals of *S. titanus* (Schvester et al., 1969; Mori et al., 2002). This suggests that many probes from infected adults are required for a plant to eventually contract FD. Therefore, a small number of infected plants in a vineyard cannot be very effective at spreading the disease, because the few phytoplasma-carrying insects originating from those plants would spread around and feed on many different healthy plants during their adult lifespan, and only rarely return on the same plant enough times to infect it, even in the presence of a moderate correlation between the movements of adults grown on the same plant. In mathematical terms, this

means that not only f, but also its derivative vanishes for $I \to 0$. Of course, if the density of infected plants is large, the probability of recurrent feeding on the same healthy plant of phytoplasma-carrying insects must be large as well. Thus, we argue that f should grow faster than linearly with I, at least at moderately low values of I. The simplest mathematical expression that captures these assumptions is

$$\text{Infection rate} = qSI^2 \tag{2}$$

where q is a constant whose value depends on the susceptibility to the infection of the particular cultivar which is being considered, on the local abundance of *S. titanus*, and on its acquisition efficiency, which is also cultivar-dependent, with the most susceptible cultivars being also the most efficient at transmitting the phytoplasmas to the insects (Bressan et al., 2005). The value of this constant is subject to large uncertainties. We estimate $q \approx 10^{-6}$ ha^2 plants^{-2} Y^{-1} , but reasonable values range from 10^{-7} to 10^{-5} ha^2 plants^{-2} Y^{-1} (see Appendix 5.1 for details). Because direct laboratory measurements of f are presently lacking, we have resisted the temptation of using more complicated functional forms. At the end of Section 3 we discuss the effect of choosing f proportional to I.

In the presence of hotbeds of infection nearby the modeled vineyard (such as infected wild grapes or an abandoned infected vineyard), the density of infected plants that enters in the infection rate terms should not be I, but rather $(I+\varepsilon)$, where the parameter ε quantifies the phytoplasma-carrying insects coming from the hotbeds, which appears to decay exponentially with the distance of the hotbed (Lessio et al., 2014).

Insecticidal sprays against *S. titanus* are applied generally twice a year, the first against nymphs and the second against adults (Bosco and Mori, 2013). However, the second treatment is generally made within the end of July with less persistent active ingredients, and is therefore largely ineffective against adults incoming from surrounding hotbeds (Bosco and Mori, 2013; Mori et al., 2013). Thus, the effect of the insecticides is that of decreasing the coupling between the healthy and the infected plants of the vineyard, but it cannot appreciably

Table 1: Value (or range of likely values) for the parameters appearing in the model (4).

Process	Parameter	Value	Reference
Farmer's intervention time	τ	$1\,\mathrm{Y}$	In Italy immediate eradication of infected plants is mandatory by law (DM 32442/2000). Similar measures are in place in France.
Vineyard's design density	D	2000 to $11000\,\mathrm{plants/ha}$	
Coupling infected-healthy	q	10^{-6} (10^{-7} to 10^{-5}) $\mathrm{ha^2 plants^{-2}\,Y^{-1}}$	
Recovery from infection	k_1	$0.4\,\mathrm{Y}^{-1}$	(Zorloni et al., 2008; Pavan et al., 2012b)
Latency before symptoms	k_2	$\frac{1}{3}\,\mathrm{Y}^{-1}$	(Osler et al., 2002; Morone et al., 2007)
Mortality of infected plants	k_3	τ^{-1} (managed vineyards); 0.15 to $0.05\,\mathrm{Y}^{-1}$ (unmanaged vin.)	By law (DM 32442/2000); (Pavan et al., 2012b)
Aging of new plants	k_4	$\frac{1}{5}\,\mathrm{Y}^{-1}$	(Pavan et al., 2012b)

decrease the coupling between the healthy plants and the external hotbeds. In the model we shall use the following expression for the infection rate:

$$\text{Infection rate} = qS\left(pI + \varepsilon\right)^2 \tag{3}$$

where the parameter p ranges between zero and one, with $p = 1$ corresponding to no insecticide treatments and $p = 0$ to a complete elimination of the insects, except for those coming from the hotbeds.

In time, some infected plants have a chance to recover from the disease, and to return symptom-free. Furthermore, they may be re-infected, thus recovered plants do not require a separate compartment. The process of recovery is modeled by a flux from the I to the S compartments quantified as $k_1 I$. Experimental data, taken in vineyards where insecticide treatments had brought to a negligible amount the presence of *S. titanus*, show that the constant k_1^{-1} ranges between 2 and 3 years for the popular *Barbera, Chardonnay, Merlot* and *Sauvignon* cultivars (Zorloni et al., 2008; Pavan et al., 2012b). For other cultivars these figures should be taken as representative of the order of magnitude, and not as accurate estimates of the recovery rate.

In full-grown plants, the symptoms of FD do not usually appear immediately after the inoculation. Inoculated individuals may remain in a latent, symptomless state for up to a few years. In our model the density of latent plants is quantified by the compartment L. The amount of latent plants that develops symptoms is quantified by the flux $k_2 L$ from the L to the I compartments. The time scale k_2^{-1} of the process is estimated to be approximately 3 years (Caudwell, 1990; Osler et al., 2002).

We assume that the farmer extirpates actively the infected plants, on a time scale $k_3^{-1} = \tau$. This causes a mortality of the infected plants quantified as $-k_3 I$. On the same time scale, the manager attempts to maintain a constant density D of plants in the vineyard, by planting healthy, young plants, whose density is quantified by the variable G. This process continues as long as the actual density of the vineyard (which is $S + L + I + G$) doesn't match the design density D. The constant τ quantifies the reaction time of the farmer. While τ can't be

smaller than one year (infected grapes are roughed at the end of summer, and nursery grapes are usually planted in the next spring in order to be productive in autumn) it may occasionally be larger, when economic constraints force a delay of the extirpation and replacement procedures. Young plants are subject to infection just in the same way as full-grown ones, with the only difference that they do not have a phase of latency, but develop the symptoms rapidly after having being infected (Osler et al., 1993). Thus, the process of infection produces a flux from G to I (rather than to L). The infection rate of young plants is quantified as $qG(I+\varepsilon)^2$, analogously to (2). In principle we could model a different susceptibility to the infection for the young and the full-grown plants by using different values of the constant q for the two compartments. However, lacking a direct empirical evidence of a clear disparity in susceptibility between young and full-grown plants, for simplicity, we prefer to use the same value of q for both. Young plants that do not become infected eventually turn into full-grown plants by aging. This process is modeled as a flux from the G to the S compartment quantified as $k_4 G$. For most cultivars the aging time is about $k_4^{-1} \approx 5$ years (Pavan et al., 2012b).

The model as described by the above considerations is embodied by the following system of first-order ordinary differential equations:

$$\begin{cases} S' = & -qS\left(pI+\varepsilon\right)^2 + k_1 I + k_4 G \\ L' = & \quad qS\left(pI+\varepsilon\right)^2 - k_2 L \\ I' = & \quad qG\left(pI+\varepsilon\right)^2 + k_2 L - k_1 I - k_3 I \\ G' = & -qG\left(pI+\varepsilon\right)^2 - k_4 G + \\ & \quad \tau^{-1}\left(D - S - L - I - G\right) \end{cases} \tag{4}$$

where the prime denotes differentiation with respect to time.

In Table 1 we summarize the estimates of the values (or value range) of the parameters that appear in equations (4), as deduced from evidence given in the accompanying references. From now on, we shall take $p = 1$, except than in Section 3.2, where we discuss the effect of insecticides.

The system (4) may be brought to non-dimensional form by using τ as the scale of time and $(q\tau)^{-1/2}$ as the scale of grapevine density. Defining the non-dimensional quantities

$$(\tilde{S}, \tilde{L}, \tilde{I}, \tilde{G}) = (q\tau)^{1/2}(S, L, I, G),$$

the system in non-dimensional form reads:

$$\begin{cases} \dot{\tilde{S}} = & -\tilde{S}\left(\tilde{I}+\epsilon\right)^2 + c_1\tilde{I} + c_4\tilde{G} \\ \dot{\tilde{L}} = & \tilde{S}\left(\tilde{I}+\epsilon\right)^2 - c_2\tilde{L} \\ \dot{\tilde{I}} = & \tilde{G}\left(\tilde{I}+\epsilon\right)^2 + c_2\tilde{L} - c_1\tilde{I} - c_3\tilde{I} \\ \dot{\tilde{G}} = & -\tilde{G}\left(\tilde{I}+\epsilon\right)^2 - c_4\tilde{G} + \\ & \mathcal{D} - \left(\tilde{S}+\tilde{L}+\tilde{I}+\tilde{G}\right) \end{cases} \tag{5}$$

where the dot denotes derivation with respect to the non-dimensional time, and the (positive) constants are $c_1 = k_1\tau$, $c_2 = k_2\tau$, $c_3 = k_3\tau$, $c_4 = k_4\tau$, $\mathcal{D} = (q\tau)^{1/2}D$, $\epsilon = (q\tau)^{1/2}\varepsilon$. For typographical clarity from now on we shall omit the tildes, and all quantities will be in non-dimensional form, unless otherwise specified.

2.2. Equilibria and their bifurcations

The model is meaningful for initial data such that $S, L, I, G \geq 0$ and $S + L + I + G \leq \mathcal{D}$. In this case the densities of the four compartments remain non-negative and bounded by the vineyard's design density $\mathcal{D}$ at all later times (see Appendix 5.2). If the initial condition is such that $S + L + I + G > \mathcal{D}$, then unacceptable solutions with negative values may develop. However, the only occurrence in which the density of the vineyard could be higher than the design density $\mathcal{D}$ is when a farmer decides to thin out the vineyard in order to attain a lower density. Modeling exceptional events of this sort is, of course, well beyond the aim of equations (5).

For $\epsilon = 0$ the system (5) has the obvious equilibrium

$$S = \mathcal{D}, \quad L = I = G = 0 \tag{6}$$

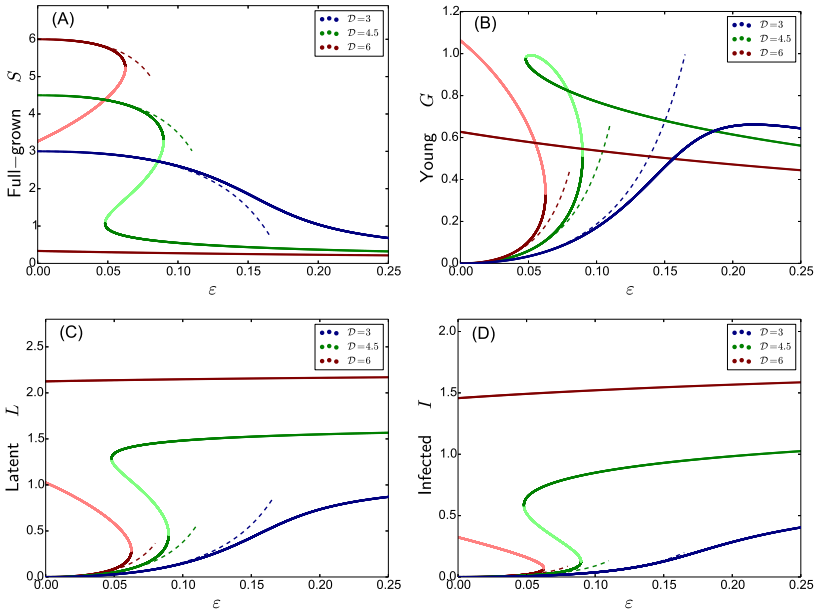

Figure 2: Equilibria of the vineyard model (5) as a function of the hotbed strength ϵ, showing the three different possibilities that may occur for different vineyard densities $\mathcal{D}$. Panels (A), (B), (C), (D) represent, respectively, the density of healty, full-grown plants (the S compartment); healthy, young plants (the G compartment); latent plants (the L compartment); infected plants (the I compartment). Dark/light color shades represent, respectively, stable and unstable equilibria. The dashed lines are the approximate expressions (15) of the equilibria close to the state (6) of healthy vineyard.

corresponding to an uninfected vineyard. As the parameter ϵ varies, an analytical and numerical investigation (see Kuznetsov, 1995, Chapter 10, and appendix 5.3 for details) finds only three possible outcomes, regardless of the actual values of the constants $c_1, \ldots, c_4$. For low densities $\mathcal{D}$ there exists only one fixed point, which is stable, and corresponds to a situation in which the vineyard's health progressively decreases as the strength ϵ of the hotbeds increases. For higher non-dimensional densities there are multiple equilibria in an interval of values of ϵ. In particular, there are two stable equilibria, corresponding to a nearly-healthy and to a severely deteriorated vineyard, and there is an intermediate unstable equilibrium. For even higher values of $\mathcal{D}$ multiple equilibria occur even in the absence of hotbeds. An example for $c_1 = 0.4$, $c_2 = 1/3$, $c_3 = 1$, $c_4 = 1/5$ is shown in Figure 2. In this example the transition to multiple equilibria occurs for $\mathcal{D} \approx 3.9$.

The bifurcation diagram shows that very weak (or very far) hotbeds have almost no effect, and the corresponding equilibrium has just a minimal amount of latent, infected and young plants. This notion is made precise by means of a perturbative analysis: if $\epsilon \ll 1$, then there exists an equilibrium which differs only by $O(\epsilon^2)$ from the healthy vineyard. Explicit, approximate expressions of this equilibrium are given by eq. (15) in Appendix 5.4.

When there are multiple equilibria, the equilibrium corresponding to a nearly-healthy vineyard may suddenly disappear if the strength of the hotbeds is increased beyond a critical value. This occurs at the fold that joins the branch of unstable equilibria to the branch of nearly-healthy equilibria. An approximate expression, accurate for large $\mathcal{D}$, for both the strength of the hotbeds and the density of infected plants at the fold is given by:

$$\epsilon_{fold} \approx I_{fold} \approx \frac{c_1 + c_3}{4\mathcal{D}}. \tag{7}$$

Figure 3 shows a comparison between the critical values determined numerically and the approximation (7). Appendix 5.5 gives further details. Note that the values S_{fold}, L_{fold}, G_{fold} of, respectively, the healthy, latent and young plant densities at the fold can be computed by using (7) in (13).

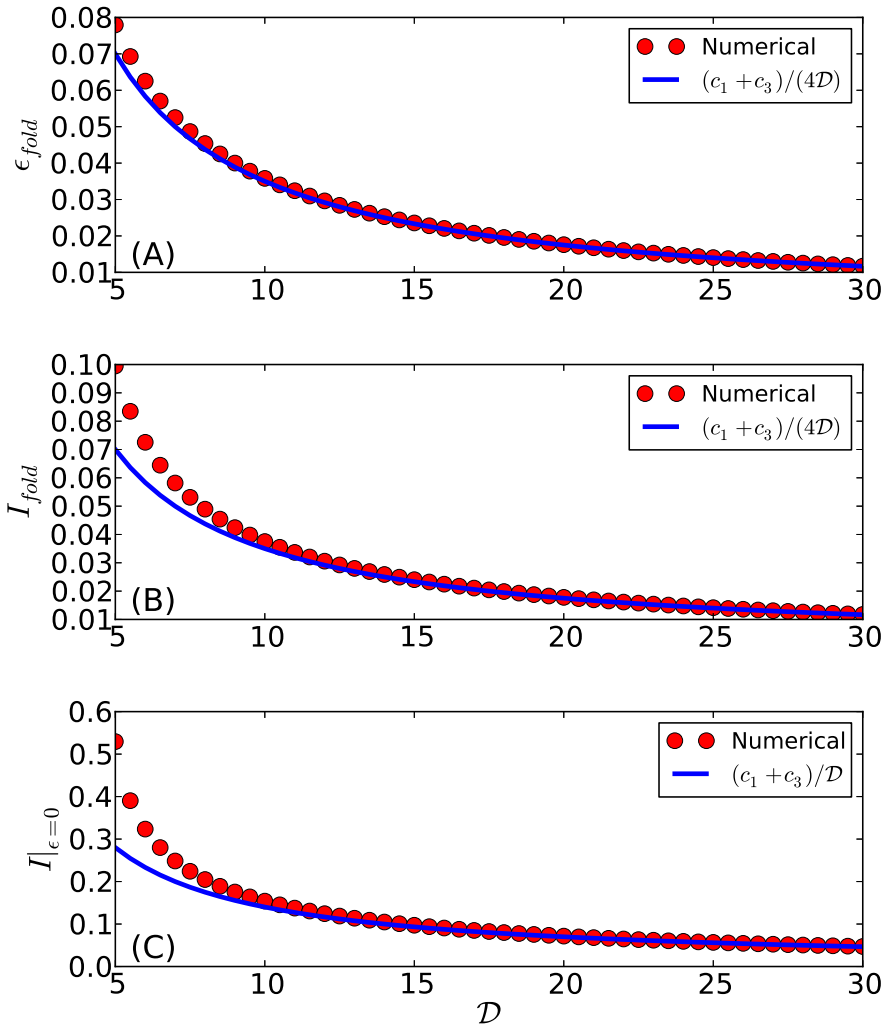

Figure 3: Panels (A) and (B): values of ϵ and of I at the fold bifurcation between the healthy vineyard branch and the unstable branch of equilibria for several values of $\mathcal{D}$. Panel (C): value of I of the unstable equilibrium at $\epsilon = 0$. The red dots are values computed numerically, the blue lines are the approximations discussed in the Appendix 5.5.

2.3. The case of an abandoned vineyard

Sometimes, for economic reasons, vineyards are left unmanaged. In the absence of insecticide treatments and of active replacement of infected plants, unmanaged vineyards may become hotbeds of infection. A similar role is played by wild grapevines living in woodlands and shrublands. Equations (5) may be used to model these cases, simply by omitting the G compartment of the young plants. The equations then read

$$\begin{cases} \dot{S} = & -SI^2 + c_1 I \\ \dot{L} = & SI^2 - c_2 L \\ \dot{I} = & -(c_1 + c_3)\, I + c_2 L \end{cases} \tag{8}$$

The values of the constants c_1 and c_2 may be taken the same as before, with the convention that the time unit used for defining the non-dimensional quantities is $\tau = 1$ year. The mortality rate of the infected plants c_3 is, instead, much smaller, because plants showing symptoms are not eradicated once a year by a farmer, but rather die as a consequence of the infection. The mortality time scale depends on the cultivar. Some (e.g. *Merlot*) appear to be very robust, and others (e.g. *Perera*) die more easily (Pavan et al., 2012b). An intermediate, order-of-magnitude estimate is about 10 years (Table 1). In this very simplified approach we have omitted to introduce terms modeling the reproduction and the natural mortality of healthy grapevines. Owing to the long lifespan of grapevine plants, these processes occurs on time scales which are much longer than those involving the spread of FD, and should therefore be negligible in the present context. For simplicity, we have also omitted any coupling term with other nearby hotbeds: we assume that the abandoned vineyard is already infected, and we are interested in the time evolution of the most virulent phase of the infection, during which the abundance of infected individuals of *S. titanus* is determined by the local density of infected plants, and any inflow from external sources becomes negligible.

Starting from an initially small (but not negligible) amount of infect plants, typical solutions of the system of equations (8) look like that shown in Figure 4.

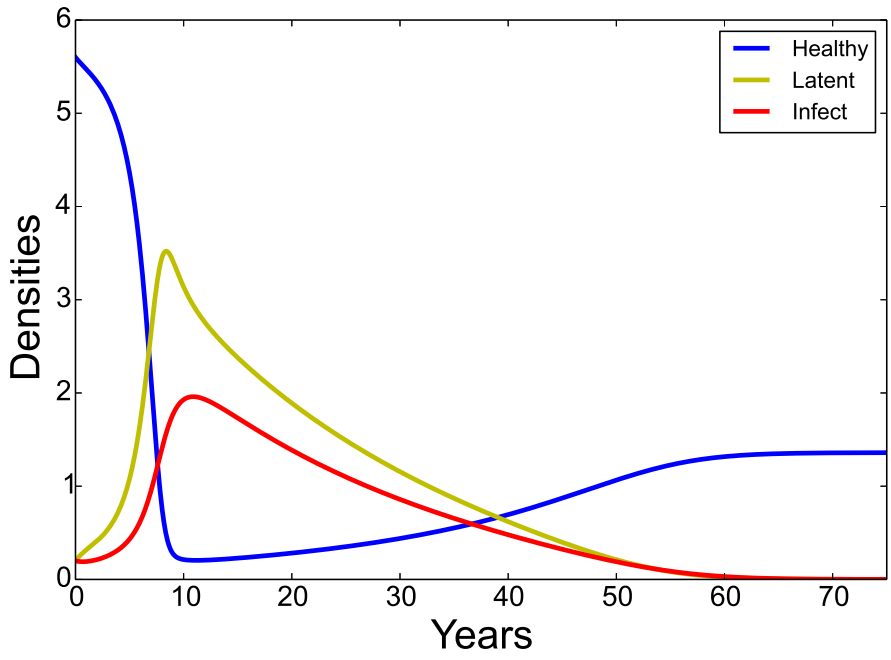

Figure 4: Numerical solution of equation (8) with the initial conditions $S(0) = 5.6$, $L = 0.2$, $I = 0.2$. The non-dimensional constants c_1, c_2, c_3 are computed from the parameter values given in Table (1).

In the first few years there is a sharp drop in the density of the healthy plants, and a corresponding sudden rise of the latent and infect plants. A peak occurs in about ten years, after which the healthy plants slowly increase in number, while latent and infect decrease. In the absence of external perturbations, and, in particular, assuming no hotbeds, this gradual recovery would continue for several decades, and finally reach a new healthy state, at a density much lower than that of the initial condition. Changes in the numerical values of the constants c_1, c_2, c_3, within ecologically reasonable intervals, do not change this general qualitative behavior.

During the initial, rapid phase, as the number of infected plants increases, so does the number of plants that recover and become healthy again. Those that do not recover die, decreasing the overall grape density. The epidemic peaks when the recovered plants become a substantial fraction of the healthy plants. This is in qualitative agreement with the results of the experiments of Morone et al. (Morone et al., 2007). After the peak, the continuing recovery of a fraction of the many infected plants causes the slow increase in the number of healthy plants. The sole recovery process would not avoid the death of all the grapes, because recovered grapes can be reinfected. When mortality reduces enough the density of infected grapes, then the arguments that lead to the expression (2) suggest that further transmission of FD would be hampered. This justifies the ultimate disappearance of the infected plants and the survival of a few healthy ones. In Appendix 5.6 we offer a mathematical justification of these statements.

Although equations ((8)) should represent fairly well the initial evolution of an hotbed of infection, we do not expect the very long-term behavior of the solutions to be realistic: on time scales of decades the land of an abandoned vineyard would be re-allocated, and an appropriate modeling of wild grapes should take into account other factors (e.g. reproduction, competition with other species, etc.). However, we feel that the long-term healing shown by the model ((8)) should provide a good illustration of the properties of the interaction term 2) and, in particular, that self-propagation of FD is particularly ineffective when the density of infected plants is very low.

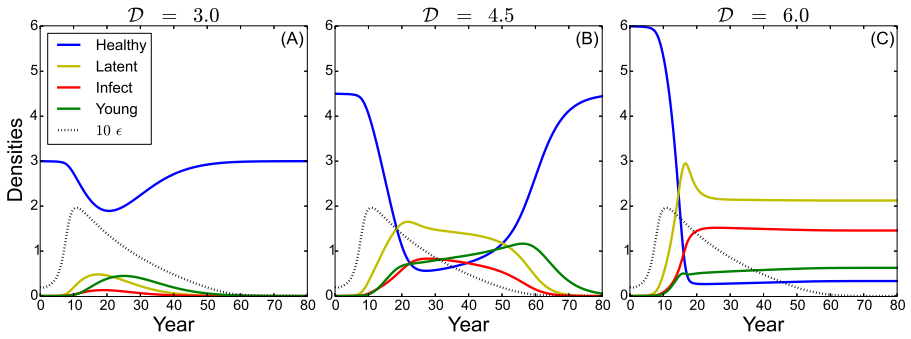

Figure 5: Time evolution of of a vineyard with design density $\mathcal{D} = 3$ (A), $\mathcal{D} = 4.5$ (B), and $\mathcal{D} = 6$ (C) exposed to hotbeds with a time-varying strength ϵ. Note that, for clarity, the value of ϵ (represented by the dotted black line) is multiplied by ten.

3. Discussion

3.1. Practical implications of the structure of the bifurcation diagram

The bifurcation diagram of Figure 2 shows that the two most important parameters determining the epidemiology of a vineyard subject to FD are the strength of the external hotbeds, and the non-dimensional density of the grapes. Changes in the other parameters do not affect the general structure of the diagram, although they quantitatively change some details (e.g. the position of the fold bifurcations, see eq. (7) and appendix 5.4).

In order to give an example of how the structure of the bifurcation diagram shapes the time evolution of a FD epidemics in a vineyard exposed to a nearby hotbed, we have coupled the model (5) for a managed vineyard, with the model (8) for an abandoned vineyard, playing the role of a hotbed in which the infection builds-up, peaks and then slowly wanes as the plants die off. In the equations (5) we have set $\epsilon = c_o I_a$, where I_a is the density of infected plants that occurs in the hotbed, according to the equations (8). With $c_o = 0.1$, keeping the parameters of the managed vineyard as in Figure 2, and those of the hotbed as in Figure 4, we obtain the numerical solutions shown in Figure 5.

In the first few years, while the number of infected plants builds up in the hotbed, the amount of infected and latent plants in the managed vineyard is minimal. The system remains in the state of nearly-healthy vineyard, while

the parameter ϵ moves rightward in the diagrams of Figure 2. After this initial phase, the subsequent time evolution is determined by the non-dimensional density of the vineyard. At low densities (Figure 5A) the system never undergoes a bifurcation: the health of the vineyard deteriorates while the intensity of the hotbed peaks and then wanes, and, after a lag determined by the latency, the managed vineyard returns to a healthy state. About two-thirds of the plants remain healthy and productive (the exact amount may change with the parameters $c_{1,\ldots,4}$). The remaining third is composed mostly by young and latent plants. The extirpation and replacement procedures are able to maintain the presence of infected plants at very low levels.

At intermediate and high densities (Figure 5B,C) after the initial build-up phase, the system crosses the fold bifurcation that marks the end of the nearly-healthy state. A further gradual deterioration of the vineyard cannot occur. Instead, the system moves toward the only other stable state, corresponding to a severely infected vineyard, dominated by latent and infected plants. In this state extirpation and replacement simply fuels the infection, as the young plants are unlikely to remain healthy for long.

From an abstract point of view, the situation at intermediate densities is different than at higher densities. In the former case when the intensity of the hotbed decreases enough, the system crosses the second fold bifurcation present in the green curves of Figure 2 and (slowly) returns to the healthy state in a classical hysteresis cycle. At higher densities the second bifurcation is not present, and the system remains stuck in the severely degraded state even when the influence of the hotbed disappears. In practice, for most of the reasonable values of the parameters, the hysteresis cycle occurs on time scales that are too long for being of any practical value: the useful life-span of a vineyard is about thirty years. Thus, the difference between intermediate and high densities might be irrelevant in practice.

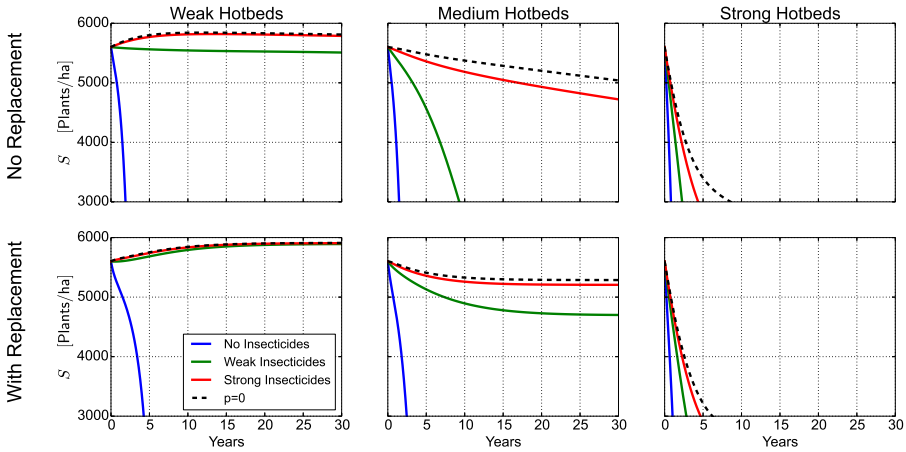

Figure 6: Density of healthy plants as a function of time in a vineyard with a cultivar highly susceptible to FD infection ($q = 3 \cdot 10^{-6}$). The upper panels shows the case in which the infected plants are not replaced with young ones; in the lower panels the replacement occurs on a yearly basis. The left, middle and right panels corresponds to hotbeds having an effective density of infected plants of, respectively, 25, 75, 225 plants/ha. The blue, green, red and dashed black curves correspond, respectively, to $p = 1$, $1/4$, $1/16$, 0.

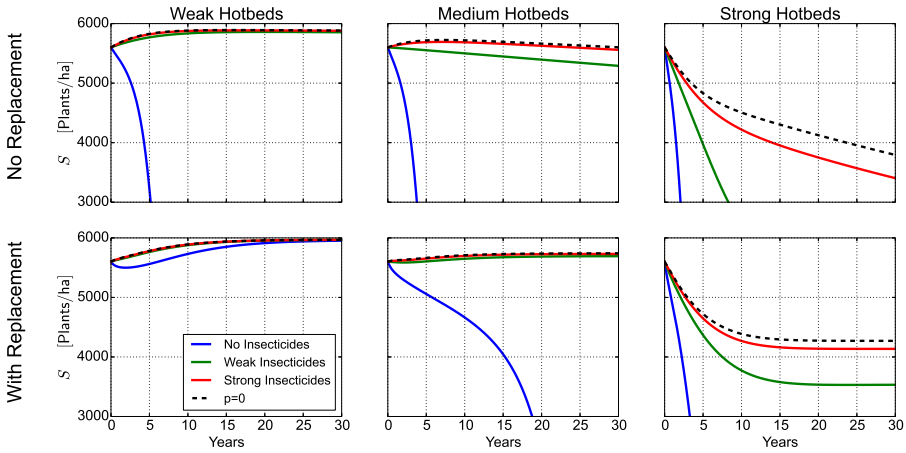

Figure 7: As Figure 6, but with a moderately susceptible cultivar ($q = 10^{-6}$).

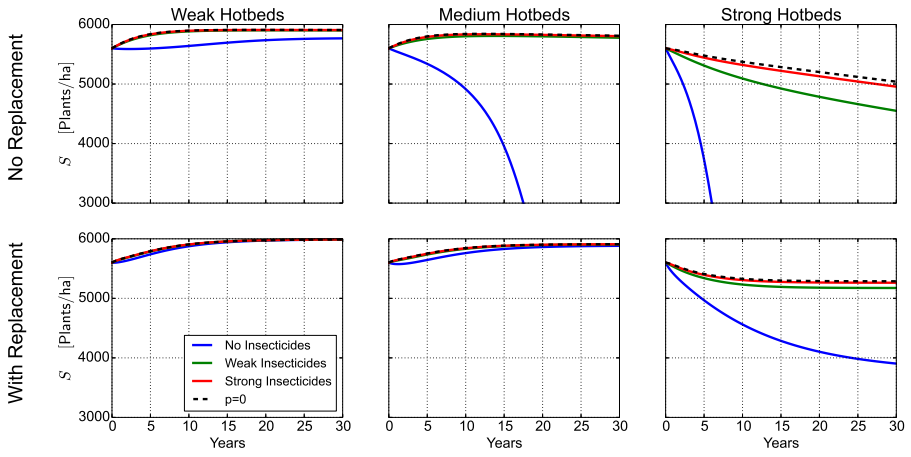

Figure 8: As Figure 6, but with a robust cultivar ($q = 0.\overline{3} \cdot 10^{-6}$).

3.2. The effect of the insecticides

Insecticide treatments reduce the coupling between the infected and the healthy plants of a vineyard. In the absence of hotbeds, this translates to a decrease of the value of the parameter q which appears in the infection rate (2). In non-dimensional units, this is equivalent to a reduction of the vineyard design density, since we have $\mathcal{D} = (q\tau)^{1/2} D$. Even if the insecticide does not hamper the coupling between the healthy plants and the hotbeds, this suggests (as we have verified numerically) that insecticide treatments tend to remove the fold bifurcations, and produce curves of equilibria qualitatively similar to those that occur without treatments at low densities, such as the blue curve in Figure (2).

Because the process of extirpation and replacement of infected plants is expensive, farmers are sometimes tempted to omit it (or perform it in a less timely fashion) and attempt to manage a FD infection with insecticides alone. Therefore it is interesting to assess the model results with insecticides, and both with and without replacement. We thus simulate three vineyards (Figures 6, 7, 8) planted with cultivars that are, respectively, highly susceptible ($q = 3 \cdot 10^{-6}$), moderately susceptible ($q = 10^{-6}$), and robust ($q = 0.\overline{3} \cdot 10^{-6}$) to FD infection. All three vineyards have a design density $D = 6000$ plants/ha, and initially contain 200 plants/ha of both infected and latent plants, and no

young plants. We show the time evolution of the density of healthy plants without replacement (upper three panels) and with replacement (lower three panels). The left, central and right panels, named "Weak Hotbeds", "Medium Hotbeds", and "Strong Hotbeds" correspond to an equivalent hotbed density of $\varepsilon = 25, 75, 225$ plants/ha. Each panel has four curves, corresponding to $p = 1$ (no insecticide), $p = 1/4$ (weak insecticide treatment), $p = 1/16$ (strong insecticide treatment) and $p = 0$ (the ideal case in which the insecticide kills all the vectors present in the vineyard at the moment of treatment). All simulations continue for thirty years, which is the typical lifespan of a vineyard.

The simulations show that insecticide treatments with no replacement lead to satisfactory results in the case of weak hotbeds. As the strength of the hotbeds increases, the sensitivity of the cultivars becomes an important factor, and only for the robust one an infection of FD appears to be manageable with insecticides alone. Conversely, if replacements of infected plants is performed, even moderately effective insecticide treatments are able to control the infection, except for strong hotbeds and highly susceptible cultivars. In this case the vineyard appears to be not salvageable.

Our results suggest that extremely aggressive insecticide treatments are essentially useless: the simulations corresponding to $p = 0$ always give results very similar to those with $p = 1/16$, and, often, even to those with $p = 1/4$. In the presence of strong hotbeds, this is understandable by observing that once the insecticides have reduced the amount of insect vectors originating within the vineyard below that of those coming from external sources, then any further use of insecticides should yield no appreciable differences. For weaker hotbeds, and in the presence of replacement, the results shows that even a moderate decoupling between healthy and infected plants is sufficient to move away the fold bifurcation, and maintain the vineyard in a nearly-healthy state.

Finally, we observe that, for sufficiently weak hotbeds and sufficiently robust cultivars, the replacement procedure alone appears to be sufficient to control the infection (although, in practice, the feasibility of this strategy may be hampered by the difficulty and inherent costs of early detection and replacement of all

symptomatic plants in a vineyard). In the extreme case of weak hotbeds and robust cultivar (upper left panel of Figure 8) the vineyard self-recovers, with no insecticides and no replacement. This is because the density of the infected plants is too low to further spread the disease, as discussed in Section 2.3.

3.3. The functional form of the infection rate

What is known about the physiology and ecology of *S. titanus* strongly suggests that the infection rate should depend quadratically on the density of infected plants (see eq. 2), at least for small and moderate densities of the infected plants. However, this hypothesis has never been tested directly. It is therefore interesting to investigate which would be the properties of the model if the infection rate depended linearly on I, substituting (2) with the following expression

$$\text{Infection rate} = \hat{q}SI \tag{9}$$

where $\hat{q}$ is expressed as $\text{ha}\,\text{plants}^{-1}\text{Y}^{-1}$ and the scale of grapevine density is $(\hat{q}\tau)^{-1}$.

As a function of the strength of the hotbeds, the resulting equations admit two families of equilibria, of which one is stable and the other is unstable. They are, respectively, the analogous of the states of nearly healthy vineyard and of severely deteriorated vineyard of the model (5). These families are not subject to bifurcations for changing strength of the hotbeds. Their stability depends on the following simple criterion: the nearly healthy equilibrium is stable if

$$\mathcal{D} < c_1 + c_3. \tag{10}$$

If the inequality is reversed, then the the stable equilibrium is that of severely deteriorated vineyard.

At low densities (or for robust cultivars) both the model with the linear and that with the quadratic dependence uphold the same qualitative scenario: a gradual degradation of the vineyard for progressively stronger hotbeds. At higher densities (or for more susceptible cultivars) there is a stark difference

between the two models: that using (9) predicts that the vineyard would precipitate into a severely deteriorated state no matter how small are the hotbeds. In principle, even a single infected insect would be sufficient to disrupt the healthy state. In contrast, using (2) we obtain a model where the healthy state is insensitive to truly small perturbations, and requires the presence of hotbeds (non necessarily in large amount, if the vineyard is densely planted or has a highly susceptible cultivar) for triggering the fall to the severely deteriorated state.

Unfortunately, there is not a large body of studies attempting to assess the long-term importance of hotbeds for the epidemiology of FD. The few that focus on the role of the hotbeds (Lessio et al., 2007; Pavan et al., 2012a; Lessio et al., 2014) always agree that external sources of S. titanus play an important role, although no one has ever looked for threshold effects, such as those predicted by the model (4) in the presence of a fold bifurcation.

There are in the literature reports of very rapid deterioration of FD infections. For example in Serbia, several vineyards of cultivar "Plovdina", very susceptible to FD, developed up to 100% symptomatic grapes in three years starting from less than 5% of infected plants (Kuzmanović et al., 2008). The observations are consistent both with the infection rate (9) (the healthy state would be unstable and the vineyard would precipitate to the deteriorate state at the first occurrence of infected insects) and with the infection rate (2) (a small and unnoticed buildup of hotbeds, after some time, destabilizes the healthy vineyards and precipitates them into the deteriorated state). Taking into account that occurrences of FD had already been reported in the region (and motivated the three-years long survey), we tentatively assume the second case as more fitting.

4. Conclusions

We have developed a model for the time evolution of a FD epidemics in a vineyard. The presence of the vector of the disease (the leafhopper *Scaphoideus titanus* Ball) is not explicitly modeled, but is parameterized as an interaction

term between the infected and the healthy grapevine plants. The presence of infection hotbeds near the vineyard appears as a parameter in this interaction term. In addition to infection, the model takes also into account incubation, recovery and aging processes, and management actions operated by the farmer, namely extirpation and replacement of infected plants and insecticide treatments.

The model shows that, in the presence of abundant populations of *S. titanus*, or, equivalently, for vineyards with moderate and high plant density or cultivars susceptible to FD, two stable equilibria are possible. One of these corresponds to a situation with just a few infected plants, where the infection is kept under control by the extirpation and replacement process. The other equilibrium corresponds to a vineyard dominated by infected plants, where extirpation and replacement is ineffective. When the strength of the hotbeds crosses a critical threshold, only the latter equilibrium survives, and the former disappears. Therefore, vineyards infected with FD may undergo an irreversible transition from a near-healthy state to a severely compromised one.

If the initial stages of the infection go unnoticed, or if the hotbeds are too strong, then extirpation and replacement alone is insufficient to maintain a nearly-healthy vineyard state. In these cases our results show that insecticide treatments are determinant for recovering the infected vineyard. Although there are cases in which FD infections have been solved just by insecticide treatments (e.g. for the Prosecco cultivar in the Conegliano-Valdobbiadene grape-growing area (Osler et al., 2002)) our model shows that the best results would be obtained by combining both insecticides and replacement of infected plants. This, too, was observed in the Soave area (Sancassani et al., 1997). Furthermore, it shows that when replacement is performed timely and accurately, insecticide treatments of mild intensity should yield results just as satisfactory as very intense treatments.

The model predicts two important and novel features.

One is the role of the vineyard density. The non-dimensional density in the model is defined as a combination of the physical density of the vineyard

(number of plants per unit of surface) and of a parameter that quantifies the ease of transmission of FD from infected to healthy plants. The latter, in turn, depends on the cultivar and on the abundance of insect vectors, and can be lowered by insecticides. Thus, lowering the physical density of the vineyard would be equivalent, according to the model, to using a less susceptible cultivar, or to having a lower level of insect vectors.

The second is the role of hotbeds. Their intensity is the most important control parameter determining the health of a vineyard. In particular, the model shows the possibility of threshold effects: as long as hotbeds remain below a threshold, FD infections would be easily manageable. When they cross the threshold, a rapid deterioration of the vineyard health should be expected, if the intensity of insecticide treatments is kept constant. Planting new vineyards in areas with the presence of strong hotbeds is therefore not suggested, especially in the case of highly susceptible cultivars.

Because both features were somewhat unanticipated, there are no available data to directly confirm or disprove them. However, the model's results are able to reproduce the observed phenomenology. The scenarios of Figures 6, 7, 8 reflect the variety of outcomes of FD epidemics observed in the field. For example, in the presence of strong hotbeds, losses of about 20% within ten years, even if insecticide treatments are performed, agree with the authors' personal observations of vineyards with moderately susceptible cultivars in many wine growing areas of Piedmont (see the right panels of Figure 7), but much more rapid, catastrophic deterioration have been observed (e.g. in Serbia, see Section 3.3) for very susceptible cultivars, just as suggested by the model.

Unfortunately, a quantitative assessment of the presence of hotbeds is not routinely carried out. Most of the available data in the literature do not quote this important parameter, or do so just in vague terms. Thus, a quantitative comparison between the available data and the model outputs, if the strength of the hotbeds is not precisely constrained by the observations, is more an exercise in data fitting (as in Figure 9) than a validation of the model. However, there are already mounting concerns suggesting that hotbeds are more than

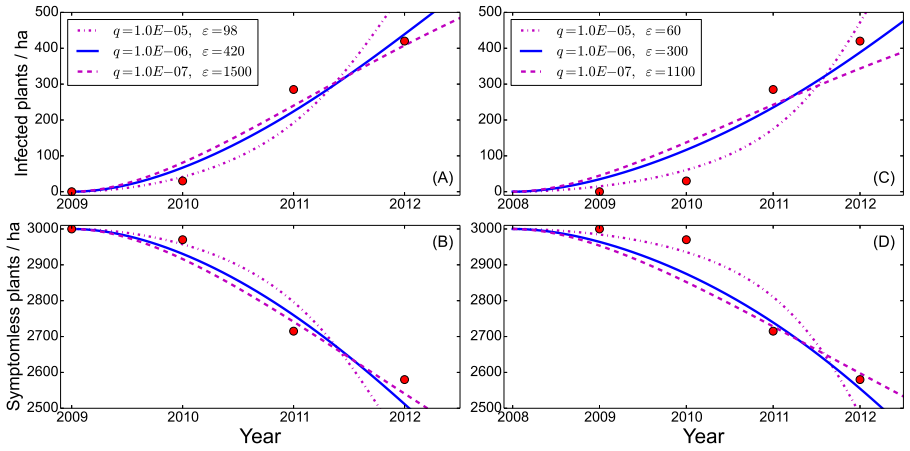

Figure 9: Comparison between observed data (dots) and the model (lines) for different values of the parameter q. In panels (A), (C) the dots represent the observed density of infected plants in an experimental vineyard, the lines are the I compartment. In panels (B), (D) the dots represent the observed density of symptomless plants, the lines are the sum of the S and L compartments. In panels (A), (B) the numerical solution starts in 2009, and uses the q $\left(\mathrm{ha}^2\mathrm{plants}^{-2}\mathrm{Y}^{-1}\right)$ and ε $\left(\mathrm{plants\,ha}^{-1}\right)$ values given in the inset of panel (A). In panels (C), (D) the solution starts in 2008 and the q, ε values are given in the inset of panel (C).

just a transmission pathway of the infection, but rather that their presence and strength is a very important factor in shaping the time evolution of a FD epidemics. This motivated the recent start of accurate mapping campaigns of cultivated, wild and reverted to the wild grapes in selected wine-growing areas of Piedmont, financed by the regional administration. Therefore, it appears that in the near future it will be possible to quantitatively validate the model and, hopefully, use it as a practical tool in the management of FD infections.

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5. Appendix

5.1. An estimate of the value of q

As part of an experiment conducted in the province of Cuneo (Italy), a small vineyard of 0.475 ha was monitored from 2009 to 2012. The vineyard had an initial density $D = 3000$ plants/ha and no infected plants. Flavescence Dorée was already well established in the surrounding territory. However we lack quantitative data allowing for the estimation of the appropriate value of ε. No insecticide treatments, nor extirpation of infected plants was performed. Every year, the number of infected and simptomless plants was assessed (the red dots in Figure 9 (A), (C) and (B),(D), respectively). The assessment would not distinguish between healthy and latent plants, both classified as symptomless.

In order to determine a reasonable range of values of q, we apply the model (4) by setting $G = 0$ and dropping the last equation (which models the replacement of infected plants with young ones). Figure (9) shows a comparison between the model results and the observed data, for several choices of the parameters q and ε. The other coefficients are those of Table 1. The density of symptomless plants is compared with the sum of the S and L compartments. The initial condition is $S = D$, $L = I = 0$. In Figures 9 (A), (B) the numerical solution starts in 2009, the last year without infected plants. In Figures 9 (C), (D) the solution starts in 2008. This allows for the hypothesis that for one year all the inoculated plants remained in the latent state, or with symptoms as weak as to evade detection. In the first case, $q \approx 10^{-6}\,\mathrm{ha^2 plants^{-2}\,Y^{-1}}$ gives a reasonable fit of the data, while in the second a value as high as $q \approx 10^{-5}\,\mathrm{ha^2 plants^{-2}\,Y^{-1}}$ yields a more convincing fit. Values as low as $q \approx 10^{-7}\,\mathrm{ha^2 plants^{-2}\,Y^{-1}}$ also give an acceptable fit, if the initial condition refers to 2009, but should probably be ruled out, because they require unrealistically high values of ε.

5.2. Boundedness and non-negativity of the solutions

For non-negative initial conditions such that $S+L+I+G \leq \mathcal{D}$ the solutions of the model equations (5) remain non-negative and bounded by $\mathcal{D}$ at all later

times. In fact, by adding together the four equations in (5), and defining the total vineyard density $x = S + L + I + G$, we obtain

$$\dot{x} = \mathcal{D} - x - c_3 I. \tag{11}$$

Considering I as a known function of time, we have that the solution of (11) is

$$x(t) = \mathcal{D} + \left(x(0) - \mathcal{D} - c_3 \int_0^t e^s I(s)\, \mathrm{d}s \right) e^{-t}. \tag{12}$$

This shows that, if the initial vineyard density is $x(0) \leq \mathcal{D}$, then, as long as I remains non-negative, it will be $x(t) \leq \mathcal{D}$. If at any time t we have $S, L, I, G \geq 0$ and $x(t) \leq \mathcal{D}$, then from (5) we deduce $S = 0 \Rightarrow \dot{S} \geq 0$, $L = 0 \Rightarrow \dot{L} \geq 0$, $I = 0 \Rightarrow \dot{I} \geq 0$, and $G = 0 \Rightarrow \dot{G} \geq 0$. Therefore, none of the four compartments can become negative. Thus we have that $0 \leq x(t) \leq \mathcal{D}$ at all times, which implies $0 \leq S, L, I, G \leq \mathcal{D}$.

5.3. Determination of the equilibria of the model

Imposing the right-hand side of (5) to be zero, for $I + \epsilon \neq 0$, the other equilibria of the model may be expressed as solutions of the following system of non-linear algebraic equations:

$$\begin{cases} S = \dfrac{c_1 I}{(I+\epsilon)^2} + \dfrac{c_3 c_4 I}{(I+\epsilon)^2 \left(c_4 + (I+\epsilon)^2 \right)} \\ L = \dfrac{c_1}{c_2} I + \dfrac{c_3 c_4 I}{c_2 \left(c_4 + (I+\epsilon)^2 \right)} \\ G = \dfrac{c_3 I}{c_4 + (I+\epsilon)^2} \\ I = c_3^{-1} \left(\mathcal{D} - (S + L + I + G) \right) \end{cases} . \tag{13}$$

By substituting the first three expressions of (13) in the fourth, and then multiplying by $c_2 (I+\epsilon)^2 \left((I+\epsilon)^2 + c_4 \right)$ we obtain that the equilibrium densities of infected plants are the non-negative roots of the fifth-order polynomial

$\mathcal{P}(I) = \sum_{n=0}^{5} q_n I^n$, whose coefficients are

$$\begin{aligned}
q_5 &= \quad c_2\,(c_3+1) + c_1 \\
q_4 &= -c_2\mathcal{D} + 4q_5\epsilon \\
q_3 &= \quad c_2\,(c_3+1)\,c_4 + (c_1+c_3)\,(c_2+c_4) + 4q_4\epsilon - 10q_5\epsilon^2 \\
q_2 &= -c_2c_4\mathcal{D} + 2q_3\epsilon - 2q_4\epsilon^2 \\
q_1 &= \quad c_2\,(c_3+c_1)\,c_4 + 2q_2\epsilon - 3q_3\epsilon^2 + 4q_4\epsilon^3 - 5q_5\epsilon^4 \\
q_0 &= -c_2c_4\mathcal{D}\epsilon^2 - c_2\mathcal{D}\epsilon^4
\end{aligned} \tag{14}$$

Recalling that $c_1, \ldots, c_4 > 0$ and $\mathcal{D} > 0$, from the last equation in (4) it follows that there are no equilibria with $I \geq \mathcal{D}$ and $S, L, G \geq 0$. Because from (13) it follows that to any non-negative root of $\mathcal{P}$ corresponds an equilibrium with non-negative values for all the four compartments, then we deduce that $\mathcal{P}$ cannot have real roots larger than $\mathcal{D}$.

Note that the coefficients $q_5, \ldots, q_0$ are polynomials in ϵ. We observe that, for any given $\mathcal{D}$, there are sufficiently small values of ϵ so that the coefficients of the odd powers are positive and those of the even powers are negative. Then, from Descartes' rule of signs, it follows that $\mathcal{P}$ has no negative roots. Hence, being an odd-degree polynomial, it must have at least one non-negative real root. In the special case $\epsilon = 0$ then $q_0 = 0$, and a real root is $I = 0$, which yields the equilibrium (6). We also observe that for any positive value of ϵ as small as to make $q_3, q_1 > 0$, $2q_3 > q_4\epsilon$, there exist sufficiently small values of $\mathcal{D}$ such that $q_4, q_2 > 0$. Then, from Descartes' rule of signs, it follows that $\mathcal{P}$ has one, and only one positive root.

An extensive numerical exploration for reasonable values of the parameters has never yielded more than three positive real roots for $\mathcal{P}$. Neither we found numerical evidence of limit cycles or deterministic chaos. We therefore are confident that the bifurcation diagrams shown in Figure 2 determine all the qualitative dynamics of the model equations (5).

5.4. *Approximate explicit expressions for the equilibria near the state of healthy vineyard*

For $\epsilon \ll 1$, explicit, approximate expressions for the equilibria of the model (5) may be sought perturbatively, assuming an expansion of the form

$$\begin{aligned} S(\epsilon) &= \mathcal{D} + \epsilon S_1 + \epsilon^2 S_2 + \epsilon^3 S_3 + \cdots \\ L(\epsilon) &= \epsilon L_1 + \epsilon^2 L_2 + \epsilon^3 L_3 + \cdots \\ I(\epsilon) &= \epsilon I_1 + \epsilon^2 I_2 + \epsilon^3 I_3 + \cdots \\ G(\epsilon) &= \epsilon G_1 + \epsilon^2 G_2 + \epsilon^3 G_3 + \cdots \end{aligned}$$

which represents a small correction upon the healthy vineyard equilibrium. The perturbative analysis reveals that $S_1 = L_1 = I_1 = G_1 = 0$. That is, weak hotbeds at first perturbative order have no effect on a healthy vineyard. The second and higher orders are non-zero, and the information that they carry is best conveyed by using Padé approximants. The (2,1) Padé approximation of the equilibrium computed with the perturbative expansion up to the third order is the following

$$\begin{aligned} S(\epsilon) &= \mathcal{D} - \frac{\left(c_2 c_3 + c_4\left(c_1 + c_2 + c_3\left(c_2 + 1\right)\right)\right)}{c_2 c_4\left(c_1 + c_3 - 2\epsilon\mathcal{D}\right)}\epsilon^2 \\ L(\epsilon) &= \frac{\left(c_1 + c_3\right)\mathcal{D}}{c_2\left(c_1 + c_3 - 2\epsilon\mathcal{D}\right)}\epsilon^2 \\ I(\epsilon) &= \frac{\mathcal{D}}{\left(c_1 + c_3 - 2\epsilon\mathcal{D}\right)}\epsilon^2 \\ G(\epsilon) &= \frac{c_3\mathcal{D}}{c_4\left(c_1 + c_3 - 2\epsilon\mathcal{D}\right)}\epsilon^2 \end{aligned} \tag{15}$$

5.5. *Approximate position of the fold bifurcation*

If the vineyard's desired density $\mathcal{D}$ is sufficiently high, for $\epsilon = 0$ there are three equilibria: the healthy vineyard stable node (6) (with no infected plants), a saddle (with an intermediate number of infected plants), and another stable node (with a high number of infected plants). As the parameter ϵ grows, the branch of stable nodes which passes through (6) and the branch of saddles move close to each other, and meet in a fold (also known as saddle-node) bifurcation at ϵ_{fold} (e.g. Figure 2(D) for $\mathcal{D} = 6$). The value of ϵ_{fold} and of the corresponding

equilibrium value of infected plants I_{fold} may be approximated with explicit expressions, as shown in Figure 3.

First we observe that for positive ϵ and sufficiently large $\mathcal{D}$ the polynomial $\mathcal{P}$ has one real positive root of size $O(\mathcal{D})$. The other roots, as $\mathcal{D} \to \infty$, tend to the solutions of

$$I^4 + c_4 I^2 + c_4 \epsilon^2 + \epsilon^4 = 0$$

(where we have used the expressions (14) divided by $\mathcal{D}$). But this polynomial does not have real solutions. Therefore we conclude that for fixed $\epsilon > 0$ and asymptotically large $\mathcal{D}$, the polynomial $\mathcal{P}$ has only one real root, which is positive.

For $\epsilon = 0$ the polynomial $\mathcal{P}$ has the root $I = 0$. The other equilibria are given by the solutions of

$$\frac{q_5}{\mathcal{D}} I^4 - c_2 I^3 + \frac{q_3}{\mathcal{D}} I^2 - c_2 c_4 I + \frac{c_2 c_4 (c_1 + c_3)}{\mathcal{D}} = 0 \tag{16}$$

where q_5, q_3, q_1 are given by (14) with $\epsilon = 0$. For $\mathcal{D} \to \infty$ one of the solutions of (16) approaches zero. Therefore it may be approximated by neglecting the terms of order higher than the first, yielding

$$I|_{\epsilon=0} \approx \frac{c_1 + c_3}{\mathcal{D}}. \tag{17}$$

For $0 \leq \epsilon \leq \epsilon_{fold}$, a smooth family of equilibria connects the equilibrium corresponding to (17) to the healthy vineyard equilibrium (6), changing stability at ϵ_{fold}. But for $\mathcal{D} \to \infty$ it must be $\epsilon_{fold} \to 0$ because for large $\mathcal{D}$ and positive ϵ, $\mathcal{P}$ has only one real solution. Thus, if the family of equilibria is a smooth curve, asymptotically for large $\mathcal{D}$, it must be $0 < I_{fold} < I|_{\epsilon=0}$ and $\epsilon_{fold} \propto I_{fold}$. We have verified numerically for a large number of values of c_1, c_3 and $\mathcal{D}$, that

$$I_{fold} = \epsilon_{fold} = \frac{I|_{\epsilon=0}}{4}$$

is a very good approximation for the position of the fold bifurcation, except for the values of $\mathcal{D}$ so low that for $\epsilon = 0$, $\mathcal{P}$ has only the real root $I = 0$ (e.g. the case $\mathcal{D} = 4.5$ in Figure 2(D)).

5.6. Time evolution of the abandoned vineyard model

Equations (8) admit the infinite set of equilibria $\mathcal{H} = \{S = S_o,\, L = 0,\, I = 0\}$, where S_o is an arbitrary positive constant. This is also the set of states without infection. By linearizing the equations around the equilibria we find that $\mathcal{H}$ is a normally hyperbolic manifold having two negative eigenvalues (namely, $\lambda_1 = -c_1 - c_3$ and $\lambda_2 = -c2$). It is also the center manifold of each equilibrium (Kuznetsov, 1995, Chapter 5). Therefore, initial conditions involving a very small number of infected and latent plants tend to fall back to an infection-free state in $\mathcal{H}$ without experiencing an appreciable growth of infected plants.

If the initial density of infected plants in the initial conditions is not very small, a more complicated dynamics occurs, as illustrated in Figure (4). From (8) we have

$$\frac{d}{dt}\left(L + I\right) = SI^2 - \left(c_1 + c_3\right) I,$$

thus, if initially it is $SI > c_1 + c_3$, then the density of latent and infected plants will continue to grow as long as the latter inequality is satisfied. This produces, in the span of a few years, a dramatic decrease of the density of healthy plants mirrored by a corresponding rise of infected and latent plants. This rise peaks when the density of healthy plants has dropped so much that $SI < c_1 + c_3$.

5.7. A discrete-time version of the model

As we have discussed in sec. 2.1, the processes depicted in Figure 1 could also be modelled by means of an iterated map. In non-dimensional form, the year-to-year change of the vine density in the four compartments is given by

$$\begin{cases} S_{n+1} = \left(S_n + b_4 G_n\right) e^{-\left(I_n+\epsilon\right)^2} + b_1 I_n \\ L_{n+1} = S_n \left(1 - e^{-\left(I_n+\epsilon\right)^2}\right) + \left(1 - b_2\right) L_n \\ I_{n+1} = G_n \left(1 - e^{-\left(I_n+\epsilon\right)^2}\right) + (1 - b_1 - b_3) I_n + b_2 L_n \\ G_{n+1} = G_n e^{-\left(I_n+\epsilon\right)^2} \left(1 - b_4\right) + \mathcal{D} - S_n - L_n - I_n - G_n \end{cases} . \qquad (18)$$

Here $\mathcal{D}$ and ϵ have the same meaning as in the continuous-time version. In the time interval from year n to year $n + 1$: b_1 is the fraction of infected plants that recover, b_2 is the fraction of latent plants that become infected, b_3 is the

fraction of infected plants that die, $b_4 e^{-(I_n+\epsilon)^2}$ is the fraction of young plants that become healthy adults. Obviously $0 < b_{1,\cdots,4} < 1$. In well-managed vineyards the mortality of infected plants is determined by the yearly extirpation process which would tend to eliminate all the plants that appear infected, thus it should be $b_1 + b_3 \approx 1$.

The infection process is modeled according to the principles discussed in sec 2.1. In particular, if the density of infected plants and of hotbeds is small, both the number of healthy plants that becomes latent and the number of young plants that becomes infected is proportional to $(I_n + \epsilon)^2$. The exponential functions insure that all the variables remain non-negative even at high densities of infected plants. Other functional forms could have been used, with no qualitative changes in the results.

It can be shown that the model (18) has a bifurcation diagram with the same structure as that of its continuous-time counterpart (5) shown in Figure 2. In particular, for low vineyard densities, as the strength of the hotbeds increases, there exists only a single equilibrium, which is stable. At higher densities there is an interval of hotbed strengths in which there are two stable and an unstable equilibrium, with fold bifurcations at the extremes of this interval. At even higher densities the interval of multiple equilibria includes $\epsilon = 0$.

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