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Modelling plant-aphid interactions explicitly considering the role of cultural practices: Peach (*Prunus persica*) - green aphid (*Myzus persicae*) as study case

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THÈSE DE DOCTORAT D'AVIGNON UNIVERSITÉ

École Doctorale 536
Agrosciences et sciences

Unité Plantes et Systèmes de culture Horticole (PSH)
INRAE - Institut National de Recherche pour
l'Agriculture, l'Alimentation et l'Environnement

Présentée par
Marta ZAFFARONI

Modélisation des interactions plant-puceron,
en considérant explicitement le rôle
des pratiques agricoles.

*Pêche (*Prunus persica*) - puceron vert*
*(*Myzus persicae*) comme cas d'étude.*

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Résumé

Les pucerons modifient le développement des plantes et peuvent transmettre des virus, représentant ainsi une menace majeure pour les cultures. Il est possible de réduire la pression exercée par les pucerons sur les plantes et d'améliorer la production agricole en facilitant certains processus écologiques en plus ou en remplacement de l'utilisation de pesticides. Les modèles mathématiques peuvent aider à prédire la direction et la force de ces processus écologiques et ils peuvent révéler l'impact des modes alternatifs de gestion des cultures. La thèse proposée vise à développer des modèles mathématiques basés sur les processus, couplant la physiologie des plantes et la démographie des pucerons, afin de favoriser l'intensification écologique et de réduire l'utilisation des pesticides. Les modèles tiennent compte *i)* des interactions entre la plante et le puceron, alors que la plupart des modèles de culture ne prennent en compte que l'effet du ravageur sur la plante et non l'inverse, ce qui nuit à la compréhension de la lutte antiparasitaire via les pratiques culturales; et *ii)* de l'effet des pratiques culturales et du résultat en termes de récolte, questions qui sont généralement absentes dans les modèles écologiques. Je couple tout d'abord un modèle mécaniste de croissance des plantes avec un modèle de population des pucerons, je le calibre pour un système pêche-

puceron vert et je l'utilise pour obtenir des informations sur les mécanismes qui sous-tendent la réponse des pucerons à la fertilisation et à l'irrigation. En outre, je développe un modèle épidémiologique qui tient compte explicitement de l'interférence entre deux pucerons vecteurs. J'applique ce modèle pour étudier l'effet de l'interférence inter-spécifique des pucerons dans la propagation des virus des plantes, en tenant compte de l'effet des pratiques agricoles.

Abstract

Aphids alter plant development and can transmit viruses, thus representing a major threat for crops. Aphid pressure on plant can be reduced and crop production can be enhanced by facilitating some ecological processes in addition, or in substitution, to the use of pesticides. Mathematical models can help in predicting the direction and strength of these ecological processes and they can reveal the impact of alternative ways of managing crops. The proposed thesis aims to develop process based mathematical models coupling plant physiology and aphid demography to drive ecological intensification and reduce the use of pesticides. The models consider *i)* interactions between plant and aphid, while most crop models only consider the effect of the pest on the plant and not vice versa hence impairing insights upon bottom-up pest control via cultural practices; and *ii)* the effect of cultural practices and the outcome in terms of harvest, issues that are usually absent in ecological models. Therefore, I firstly couple a mechanistic plant growth model with a pest population model, I calibrate it for a peach-green aphid system and I use it to get insights on the mechanisms behind the response of aphids to fertilization and irrigation. Furthermore, I develop an epidemiological model explicitly accounting for the interference between two aphid vectors. I apply the model

to explore the effect of inter-specific aphid interference in shaping the spread of plant viruses, considering the effect of agricultural practices.

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Introduction

Background

The insect family Aphididae comprises more than 4300 species, all of which are specialized to feed on plants phloem sap (Van Emden and Harrington, 2007). Aphids are mostly widespread in the temperate regions of the northern hemisphere, where almost every major crop is a host for at least one aphid species (Blackman et al., 2000). Aphids can damage crops in two main ways: by directly removing plant phloem, diverting resources allocated to plant growth, and indirectly by transmitting viruses, impairing plant development and production and, sometimes, causing plant death (Van Emden and Harrington, 2007; Zvereva et al., 2010; Ng and Perry, 2004). The more aphids present on a plant, the greater will be the direct damage (i.e. phloem feeding), while in terms of virus damage, aphid abundance is not as important as the ease with which they move from plant to plant (Van Emden and Harrington, 2007). In turn, plants infested by aphids can be induced to use carbon and nitrogen resource to defend themselves, to the detriment of growth (Will et al., 2013; Züst and Agrawal, 2016; Vyska et al., 2016). Induced plant defences can *i*) lower plant attractiveness to aphid visits (e.g.

through volatiles emission) (Zust and Agrawal, 2016), *ii*) reduce aphid accessibility to phloem (e.g. by phloem sealing) (Medina-Ortega and Walker, 2013; van Velzen and Etienne, 2015), and/or *iii*) decrease the rate at which ingested food is converted into progenies (e.g. by releasing toxic component in the phloem) (Zust and Agrawal, 2017).

Pesticides have been widely used in agriculture to control aphids since the middle of the twentieth century, particularly in more economically developed countries (Carvalho, 2006). Besides the well known environmental impacts due to the application of pesticides, they have been showed not to be an efficient solution for the long-term control of aphids. Regular and frequent pesticide applications can cause the emergence and spread of pesticide resistance in aphid populations (Hawkins et al., 2019). Moreover, pesticides may not be always effective in slowing the spread of aphid-borne viruses (Perring et al., 1999). Finding sustainable and lasting alternatives to control aphid outbreaks in agricultural field is thus necessary. In recent decades, agroecology has developed as discipline which aims to provide alternatives to the use of pesticide in agronomy to control pest. The rationale is that ecological concepts and principles can be applied to control pest populations while reducing the use of pesticides. For instance, the concept of ‘bottom-up’ control, according to which pest population dynamics are driven by quantity and quality of plant resources, is particularly highlighted by agroecologists. Indeed, a number of agricultural practices, affecting plant physiology and altering resources offered by plant to aphids, has the potential to control aphid abundance in the field (Coley and Bryant, 1985; Kytö et al., 1996).

Understanding the effects of fertilization and irrigation upon aphid abundance on the plant

Fertilization and irrigation are commonly used in agriculture to meet plants' nutrient and water needs, respectively (Gliessman, 2015a; Bommarco et al., 2013; Turner et al., 2019). In the last decades the potential of these agricultural practices in controlling aphid abundance on plants have been studied. Fertilization modifies nutrient balance in plants, enhancing plant tissue nutritional status, which may foster aphid growth, and influences the synthesis of defence compounds, which may reduce aphid phloem ingestion and fecundity (Awmack and Leather, 2002). Similarly, irrigation controls plant vigour, phloem nutritional quality and viscosity, possibly regulating aphid abundance (Girousse et al., 1996; Sevanto, 2014; Rousselin et al., 2018; Han et al., 2019). However, how plant nutrient and water status influence aphid population is not straightforward and empirical evidence is ambiguous, showing aphid performance increasing, decreasing or not changing with fertilization and irrigation (see e.g. Tamburini et al. (2018); Bethke et al. (1998); Tariq et al. (2012)). For example, there are some experimental evidence suggesting that practices such as fertilization and irrigation, supporting plant growth, can be associated with abundant aphid populations, since this habitat provide more resource (Rousselin et al., 2016; Tamburini et al., 2018). By contrast, other experimental evidence argues that aphid performs better on stressed plants (e.g. plants subjected to control irrigation deficit) that would not have resource to deploy defences and/or whose nutritional quality might be enhanced (Oswald and Brewer, 1997; Tariq et al., 2012). With such an unclear

picture of aphid response to fertilization and irrigation, efficiently applying the concept of bottom-up control to regulate aphid abundance on the plant is tricky. Understanding the mechanism behind the observed pattern is then fundamental to define an unified conceptual framework sufficiently flexible for the contrasting experimental evidence to find support. This can eventually help in designing appropriate management strategy to control aphid abundance on plant.

In addition, fertilization and irrigation are not lacking of environmental impacts (e.g. nitrogen leaching, greenhouse gasses production, reduction of water flow to waterbodies). Including those impacts when evaluating the effect of fertilization and irrigation on the plant-aphid system is thus important. Ultimately, this can contribute in designing management strategies which satisfy crop demand while also protecting the environment.

Understanding the effect of inter-specific aphid interference upon the spread of plant viruses

Direct and indirect (plant mediated) interference between insects have been showed to modify their behaviour and performance (Denno et al., 1995; Kaplan and Denno, 2007; Bird et al., 2019). This is particularly interesting in epidemiology, where inter-specific interference has the potential to shape the movement of vectors in the field and thus the spread of viruses (Crowder et al., 2019; Chisholm et al., 2019; Thaler et al., 2010). For instance, inter-specific interference can decrease the frequency of encounters between

vectors and plants and increase the vector propensity for leaving an area (Levins and Culver, 1971; Nee and May, 1992; Van Emden and Harrington, 2007; Long and Finke, 2015). Moreover, agricultural practices may alter insects inter-specific interference. For example, fertilization may increase vector abundance, possibly fostering inter-specific interference and pesticides may affect pest community structure, possibly resulting in species dominance shift favouring secondary pest outbreaks (Mohammed et al., 2019; Guedes et al., 2016, 2017). Exploring the effect of inter-specific interference, and how it is affected by agricultural practices, upon aphid movement is then important to understand and control the spread of plant aphid-borne viruses.

Mathematical models and their applications to the plant-aphid system

Mathematical model is an useful tool allowing to take an hypothetical mechanism and examine its consequences, making prediction and suggesting experiments that would verify or invalidate model's assumptions (Murray, 2002). In particular, process based model can be an helpful tool to analyze complex ecological system and processes such as those that determine plant-aphid interactions.

In agronomy a broad literature of process based models on plant physiology exists (Grossman and DeJong, 1994; Jones et al., 2003; Keating et al., 2003; Lescourret et al., 2011): they have been widely used to simulate crop yield under different environmental and management conditions (Miras-

Avalos et al., 2013; Li et al., 2015). They have also been used to evaluate losses due to pest outbreaks (Aggarwal et al., 2006; Chander et al., 2006; Willocquet et al., 2008; Dietze and Matthes, 2014). However, such models tends to neglect the dynamical interaction between the plant (or some of its component parts) and the pest, modelling the impact of a pest on the plant by varying one or more plant parameters, according to the pest disturbance level with no further interaction or feedback.

In ecology a broad literature of models on interactions (e.g. predation, consumption, competition etc.) between different species or organisms exists. They have been widely applied to study temporal and spatial dynamics in plant-pest (e.g. Levins and Schultz (1996); Bewick et al. (2016); Bevacqua et al. (2016)) and in plant-pathogen systems (e.g. (Cunniffe et al., 2016; Hyatt-Twynam et al., 2017; Laranjeira et al., 2020)). However such models tends to simplify the description of the plant, which in turn limits the possibility to consider key issue of agronomical research, such as the interest in final yield quality and quantity and the role played by agricultural practices.

In my thesis, I aim to fill this gap between agronomy and ecology by coupling models of plant growth with aphid population dynamics. This will allow me to explore some mechanisms behind the plant-aphid direct and indirect interactions, and the effect of some agricultural practices. I will try to develop the models using assumption based on widely accepted biological principles and, focusing on parsimony, to keep the models as simple as possible.

Objectives and structure of the thesis

In this thesis, I will develop and use two mechanistic models for achieving the following three objectives, each of them representing an aspect of the analysis of plant-aphid interaction presented in this introduction:

1. underline the mechanisms behind the contrasting observed responses of aphid performance to fertilization and irrigation;
2. explore optimal management scenarios considering both plant biomass production and environmental impact related to agronomic practices;
3. analyze the role of inter-specific aphid interference in shaping the transmission of plant viruses.

The thesis will be divided in three chapters, each of them addressing one of the objectives (see figure 1). The first chapter corresponds to a published article (Zaffaroni et al., 2020) and the other two chapters are written with the same structure as that of an article. In the first chapter, I will couple a mechanistic plant growth model with a pest population model and I will demonstrate its utility by applying it to the peach – green aphid system. In the second chapter, I will use the model presented in Ch.1 to evaluate the biomass production under different management scenarios and I will use multi-criteria decision analysis to identify optimal management scenarios considering both production and environmental objectives. In the third chapter, I will develop a single host - multi vector model and I will use it to explore the role of inter-specific aphid interference in shaping the spread of

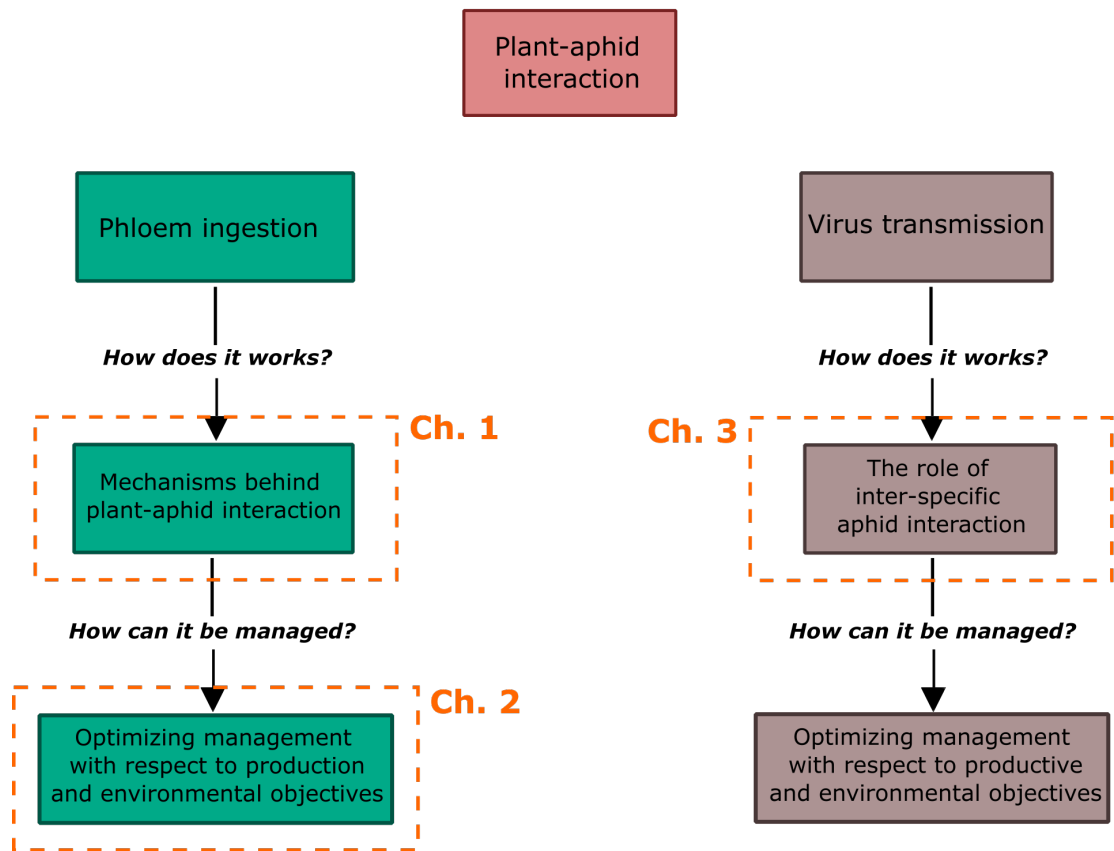


Figure 1: Thesis structure

plant viruses, considering the effect of agricultural practices (i.e. fertilization, pesticide and roguing).

Chapter 1

An ecophysiological model of plant-pest interactions: the role of nutrient and water availability

Résumé

Des études empiriques ont montré que des régimes particuliers d'irrigation/fertilisation peuvent réduire les populations de ravageurs dans les agro-écosystèmes. Cela semble promettre que le concept écologique de contrôle *bottom-up* peut être appliqué à la lutte contre les nuisibles. Cependant, un cadre conceptuel est nécessaire pour développer une base mécaniste pour les évidences empiriques. Ici, nous combinons un modèle mécaniste de croissance de la plante avec un modèle de population de ravageurs. Nous démontrons son utilité en

l'appliquant au système pèche - puceron vert. Les pucerons sont des herbivores qui se nourrissent du phloème de la plante, épuisent les ressources des plantes et transmettent (potentiellement) des maladies virales. Le modèle reproduit les propriétés du système observées dans les études de terrain et montre dans quelles conditions les hypothèses diamétralement opposées de *plant vigor* et *plant stress* trouvent un support. Nous montrons que l'effet de la fertilisation/irrigation sur la population de ravageurs ne peut pas être simplement réduit comme étant positif ou négatif. En fait, la magnitude et la direction de ces effets dépendent du niveau précis de fertilisation/irrigation et de la date d'observation. Nous montrons qu'une nouvelle synthèse de données expérimentales peut émerger en intégrant un modèle mécaniste de croissance de la plante, largement étudié en agronomie, dans un cadre de modélisation consommateurs-ressources, largement étudié en écologie. Le futur défi est d'utiliser ces connaissances pour éclairer la prise de décision pratique des agriculteurs et des producteurs.

Mots-clés: agro-écologie, modèle de population de pucerons, les défense des plantes induites, modèle de croissance de la plante, *plant stress* et *plant vigor hypotheses*, interactions plant-puceron

Note: in equation (2.1e) the term $\varphi_N \kappa \frac{C_S}{S} \frac{N_S}{S} S$ should be changed in $\varphi_N \kappa \frac{C_R}{R} \frac{N_R}{R} R$.

Research



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An ecophysiological model of plant–pest interactions: the role of nutrient and water availability

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Empirical studies have shown that particular irrigation/fertilization regimes can reduce pest populations in agroecosystems. This appears to promise that the ecological concept of bottom-up control can be applied to pest management. However, a conceptual framework is necessary to develop a mechanistic basis for empirical evidence. Here, we couple a mechanistic plant growth model with a pest population model. We demonstrate its utility by applying it to the peach–green aphid system. Aphids are herbivores which feed on the plant phloem, deplete plants' resources and (potentially) transmit viral diseases. The model reproduces system properties observed in field studies and shows under which conditions the diametrically opposed plant vigour and plant stress hypotheses find support. We show that the effect of fertilization/irrigation on the pest population cannot be simply reduced as positive or negative. In fact, the magnitude and direction of any effect depend on the precise level of fertilization/irrigation and on the date of observation. We show that a new synthesis of experimental data can emerge by embedding a mechanistic plant growth model, widely studied in agronomy, in a consumer–resource modelling framework, widely studied in ecology. The future challenge is to use this insight to inform practical decision making by farmers and growers.

1. Introduction

Chemicals have been widely used in agriculture to control pests since the middle of the twentieth century, particularly in more economically developed countries [1]. However, widespread application of agrochemicals carries an inherent environmental cost. There is also the significant challenge of declining efficacy owing to the emergence and spread of insecticide resistance in pest populations [2]. In recent decades, agroecology has developed as a discipline which aims to provide alternatives to the use of chemicals in agronomy to control pests. The rationale is that ecological concepts and principles can be applied to control pest populations while reducing the use of chemicals [3]. The concept of 'bottom-up' control, according to which population dynamics are driven by the quantity and quality of resources, is particularly highlighted by agroecologists. There are a number of agricultural practices that can affect plant physiology and that alter resources offered by plants to pests [4,5]. For example, fertilization modifies the nutrient balance in plants, enhancing plant tissue nutritional status, and influences the synthesis of defence compounds [6]. Similarly, irrigation controls plant vigour and phloem nutritional quality and viscosity, possibly regulating pest abundance [7–11].

Unfortunately, how pests might be affected by plant nutrient and irrigation status is far from obvious. Empirical evidence is ambiguous, potentially supporting diametrically opposed hypotheses. On the one hand, the plant vigour hypothesis (PVH) [12] argues that pest populations should increase most rapidly on vigorously growing plants (or organs), since these habitats provide more resources. In support of this hypothesis, there is some experimental

evidence suggesting that practices such as fertilization and irrigation, or favourable conditions for plant growth such as increased organic soil fertility, can be associated with abundant pest populations [13,14]. On the other hand, the plant stress hypothesis (PSH) [15] argues that pests perform better on stressed plants that would not have resources to deploy defences and/or whose nutritional quality might be enhanced. This has been determined experimentally to be the case for some aphid species feeding on plants subjected to controlled irrigation deficit [16,17].

In order to efficiently use the concepts of bottom-up control in agroecology, it is necessary to shed light on the mechanisms that are responsible for the observed patterns. We require a unified conceptual framework that is sufficiently flexible for both the PVH and PSH hypotheses to find support. Developing and validating such a framework requires integration of information from field experiments with mathematical modelling. Experimental data are clearly necessary to test the validity of theoretical hypotheses, but are often extremely costly and time consuming to obtain. Mathematical modelling, particularly mechanistic models, represent a useful tool to investigate which processes can be responsible for the observed patterns and to explore the consequences of different agricultural practices [18].

Here, we present an original, explicitly agroecological, model synthesizing elements of models as commonly used within the disciplines of agronomy and ecology. Agronomic models tend to empirically parametrize the detrimental effects of pests on plant biological rates (e.g. photosynthetic, growth, solute transport). However, such models invariably neglect the dynamical interaction between the plant (or some of its component parts) and the pest (e.g. [19–21]). That is, the impact of a pest on the plant is modelled by varying one or more plant parameters, according to the pest disturbance level with no further interaction or feedback. On the other hand, in ecology, there is a very broad literature of models on interactions (e.g. predation, consumption, competition etc.) between different species or organisms. These types of models have been widely used to study temporal and spatial dynamics in plant–pest (e.g. [22–24]) and particularly plant–pathogen systems (e.g. [25–27]). However, these types of model usually present a simplistic description of the plant (but see [28]), which in turn limits the possibility to consider the effects of agronomic practices. Some authors attempted to bridge the gap between agronomy and ecology by explicitly integrating pest dynamics in crop models [29,30]. However, and arguably, past works have over-emphasized realism and precision at the cost of parsimony, meaning that general principles cannot be revealed.

Here, focusing on parsimony, we couple a relatively simple plant growth model, which describes carbon and nitrogen assimilation and allocation to the shoot and root compartments of a plant, with a pest population model. With regard to the plant, we use the modelling framework proposed by Thornley in the early 1970s [31], and refined in the following decades [32–35], which represents a cornerstone in plant and crop modelling. With regard to the pest, we propose a novel population model that includes intraspecific competition in which pest birth and mortality rates depend on resource availability and quality. Moreover, we assume that the presence of the pest can induce the plant to produce defensive traits or compounds [36]. We demonstrate the utility of our model by applying it to the peach (*Prunus persica*)–green aphid (*Myzus*

persicae) system. Aphids are specialized herbivores which feed on the phloem of vascular plants. This depletes plants' resources, affecting growth and reproduction, as well as eventually impacting upon yield [37]. Moreover, aphids are the most common vector of plant viral diseases and so can often cause indirect damage far exceeding direct impacts via herbivory [38]. We use likelihood-based techniques to calibrate model parameters and select model assumptions against field data obtained under different conditions of irrigation and fertilization. The resulting model has the ability to reproduce different system properties observed in field studies, as well as showing under which conditions the PVH and PSH find more support. Our model also provides insights to conceive new targeted experiments to better understand this class of system and rethink the control of plant–aphid systems.

2. Material and methods

2.1. Model outline and assumptions

The model, which describes the temporal variation, during a growing season, of plant dry mass (partitioned into shoots and roots, in turn composed of structural mass, carbon and nitrogen substrates), its induced defensive level and the aphid population dwelling on the plant, is schematically represented in figure 1. According to Thornley and Johnson's seminal works [18,31–33], carbon is assimilated from the atmosphere via photosynthesis and stored in shoots, as shoot carbon substrate (C_S), or transported and then stored in roots as root carbon substrate (C_R). Similarly, nitrogen is assimilated from the soil, stored in roots as root nitrogen substrate (N_R), or transported and then stored in shoots as shoot nitrogen substrate (N_S). Carbon and nitrogen substrates are used, in a fixed ratio, to constitute structural shoot (S) and root (R) dry mass. With respect to the original model of Thornley, we added the assumption that the constitution of new plant mass is regulated by changes in the photo-period [39]. Such an assumption permits us to model the fact that perennial plants suspend growth, in favour of reserve constitution, before entering winter dormancy [40]. The assimilation of substrate (C_S or N_R) per unit of plant organ (S or R) decreases with organ mass because of shoot self-shading and root competition for nitrogen and it is inhibited by substrate concentration in the organ [33].

We coupled the plant model of carbon and nitrogen assimilation and partitioning with an aphid population model by assuming that aphids, which penetrate growing shoots of the host plant with a stylet and feed on the phloem [41], intercept a fraction of the substrates (C_S and N_S) directed towards the shoot structural mass compartment (S) to support their growth [42]. We assume that aphids act in a scramble competition context [43] and therefore any aphid ingests its maximum daily amount of food when the *per capita* available resource is sufficient, but that otherwise the resource is evenly shared among all the individuals: all other things being equal, the larger the aphid population gets, the lower the *per capita* ingested resource. The aphid birth rate depends on the *per capita* ingested food [44]: it is maximum when aphids have access to their maximum daily amount of food and decreases when aphids evenly share the limited resource. Whenever the aphid birth rate becomes lower than the mortality rate the aphid population declines. We assume that crowding can induce aphids to leave the plant [45].

We assumed that the infested plant can be induced to use carbon and nitrogen substrates to defend itself, to the detriment of growth [41,46,47]. This can result in the production of chemical and/or morphological and physiological changes that can reduce aphid accessibility to the resource (e.g. by phloem sealing) [48,49] and/or decrease the rate at which ingested food is

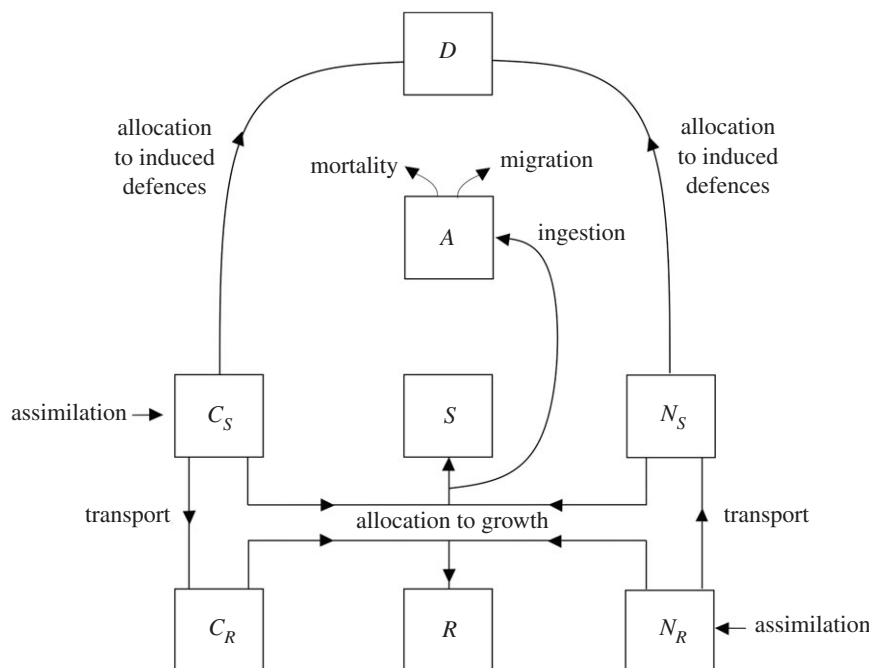


Figure 1. Schematic representation of the plant–aphid model where the plant is constituted by shoot (S) and root (R) structural dry mass and carbon (C) and nitrogen (N) substrates in shoots ($i = S$) and roots ($i = R$). The aphid population (A) intercepts a fraction of substrates allocated to constitute shoot structural mass and the plant diverts shoot substrates (carbon and nitrogen) to produce defensive compounds (D). More details are given in the main text.

converted into progeny, e.g. by releasing toxic components in the sieve that can even repel or kill the aphid [36]. We assumed that the production of induced defence compounds increases with the abundance of aphids [41].

2.2. Model equations

In quantitative terms, we describe the temporal variation of the plant–aphid system with the following system of ordinary differential equations.

$$\begin{aligned} \dot{C}_S &= \sigma_C S \left[\left(1 + \frac{S}{\nu}\right) \left(1 + \frac{C_S}{S_{\text{C}}}\right) \right]^{-1} - \varphi_C \kappa \frac{C_S N_S}{S} S - \left(\frac{C_S}{S} - \frac{C_R}{R}\right) (SR)^{\eta} \cdot (S^{\eta} + R^{\eta})^{-1} - \alpha \frac{C_S}{S} A, & (2.1a) \\ \dot{N}_S &= \left(\frac{N_R}{R} - \frac{N_S}{S}\right) (SR)^{\eta} \cdot (S^{\eta} + R^{\eta})^{-1} - \varphi_N \kappa \frac{C_S N_S}{S} S - \alpha \frac{N_S}{S} A, & (2.1b) \\ \dot{S} &= \begin{cases} \Phi \kappa \frac{C_S N_S}{S} S \left(1 - \frac{\theta A}{\Phi \kappa \frac{C_S N_S}{S} S}\right) & \text{if } \theta \cdot A \leq \Phi \kappa \frac{C_S N_S}{S} S \left(1 - \beta_1 \frac{D^{\delta_1}}{\pi_1^{\delta_1} + D^{\delta_1}}\right) \\ \Phi \kappa \frac{C_S N_S}{S} S \left(\beta_1 \frac{D^{\delta_1}}{\pi_1^{\delta_1} + D^{\delta_1}}\right) & \text{otherwise,} \end{cases} & (2.1c) \\ \dot{C}_R &= \left(\frac{C_S}{S} - \frac{C_R}{R}\right) (SR)^{\eta} \cdot (S^{\eta} + R^{\eta})^{-1} - \varphi_C \kappa \frac{C_R N_R}{R} R, & (2.1d) \\ \dot{N}_R &= \sigma_N R \left[\left(1 + \frac{R}{\nu}\right) \left(1 + \frac{N_R}{R_{\text{N}}}\right) \right]^{-1} - \varphi_N \kappa \frac{C_S N_S}{S} S - \left(\frac{N_R}{R} - \frac{N_S}{S}\right) (SR)^{\eta} (S^{\eta} + R^{\eta})^{-1}, & (2.1e) \\ \dot{R} &= \Phi \kappa \frac{C_R N_R}{R} R, & (2.1f) \\ \dot{D} &= (\varepsilon_C \alpha \frac{C_S}{S} + \varepsilon_N \alpha \frac{N_S}{S}) A, & (2.1g) \\ \dot{A} &= \begin{cases} \left(\xi \theta \left(1 - \beta_2 \frac{D^{\delta_2}}{\pi_2^{\delta_2} + D^{\delta_2}}\right) - \mu - \omega \frac{A}{S}\right) A & \text{if } \theta \cdot A \leq \Phi \kappa \frac{C_S N_S}{S} S \left(1 - \beta_1 \frac{D^{\delta_1}}{\pi_1^{\delta_1} + D^{\delta_1}}\right) \\ \left(\xi \Phi \kappa \frac{C_S N_S}{S} S \left(1 - \beta_1 \frac{D^{\delta_1}}{\pi_1^{\delta_1} + D^{\delta_1}}\right) \frac{1}{A} \left(1 - \beta_2 \frac{D^{\delta_2}}{\pi_2^{\delta_2} + D^{\delta_2}}\right) - \mu - \omega \frac{A}{S}\right) A & \text{otherwise.} \end{cases} & (2.1h) \end{aligned}$$

In our model C_S , N_S , S , C_R , N_R and R are expressed in grams (g); D is expressed in an arbitrary defence unit (DU) and A in individuals (ind.); and t represents the number of days (d) that have passed since 1 January of the year of the considered growing season. In equation (2.1a), $\sigma_C S \left[\left(1 + \frac{S}{\nu}\right) \left(1 + \frac{C_S}{S_{\text{C}}}\right) \right]^{-1}$ is the carbon substrate assimilated in shoots; $\varphi_C \kappa (C_S/S)(N_S/S)S$ is the shoot carbon substrate allocated to shoot growth or reserves; $(C_S/S - C_R/R)(SR)^{\eta} \cdot (S^{\eta} + R^{\eta})^{-1}$ is the shoot carbon substrate transported towards roots; and $\alpha (C_S/S)A$ is the shoot carbon substrate diverted to defences, in a unit of time. In equation (2.1b), $(N_R/R - N_S/S)(SR)^{\eta} \cdot (S^{\eta} + R^{\eta})^{-1}$ is the nitrogen substrate transported from roots towards shoots; $\varphi_N \kappa (C_S/S)(N_S/S)S$ is the shoot nitrogen substrate allocated to shoot growth or reserves; and $\alpha (N_S/S)A$ is

the shoot nitrogen substrate diverted to defences, with each of these quantities being measured as rates per unit of time. In equation (2.1c), the time-dependent parameter $\Phi = \lambda^{\eta} / (\lambda^{\eta} + t^{\eta})$ determines the suspension of plant growth-driven changes in the photo-period. The term $\Phi \kappa (C_S/S)(N_S/S)S$ is the increase in structural shoot dry mass in the absence of any phloem withdrawal by the aphids, with κ being the maximum rate of utilization of the substrates. The term $\theta A / (\Phi \kappa (C_S/S)(N_S/S)S)$ represents the fraction of substrates diverted from the allocation to plant growth, because of ingestion by aphids, when aphid *per capita* intake is limited by aphid maximum daily food intake, θ , and not by resource availability. The term $\beta_1 ((D/S)^{\delta_1} / (\pi_1^{\delta_1} + (D/S)^{\delta_1}))$ indicates the fraction of phloem that is protected by plant defences and

that is therefore inaccessible to aphids. When aphid *per capita* intake is limited by the resource availability, aphids ingest all the phloem they can access and the *per capita* intake is reduced. The dynamics of the variables in the root compartments (C_R, N_R, R) follow similar rules to those for assimilation of substrates, transport and allocation to root growth and we assumed that they are not directly affected by the presence of aphids. In equation (2.1*h*), we assume that the aphid birth rate is proportional to the *per capita* food intake (θ or $\Phi\kappa(C_S/S)(N_S/S)S(1 - \beta_1((D/S)^{\delta_1}/(\pi_1^{\delta_1} + (D/S)^{\delta_1})))1/A$) and that it can decrease as a result of a possible action of the defences. In other words, we assume that plant defences can determine an extra mortality rate, per unit of ingested food, modelled as $\beta_2((D/S)^{\delta_2}/(\pi_2^{\delta_2} + (D/S)^{\delta_2}))$. We modelled both the fraction of the phloem that can be protected and the phloem 'toxicity' as an increasing function of the concentration of defences, D/S . The shape of this function is given by the value of parameter δ_i . Namely, if $\delta_i > 1$ it is convex for $D/S < \pi_i$ and concave for $D/S > \pi_i$; if $0 < \delta_i < 1$, it is strictly concave. The parameter ω is the strength of possible density-dependent mechanisms inducing aphid migration. Details of the model variables and parameters are reported in table 1.

2.3. Model calibration

We apply the model to a system composed of 44 peach plants infested by green aphid subjected to four different treatments obtained by combining two levels of fertilization and irrigation. The shoot growth and the aphid infestation level were measured weekly on each plant: details of the experiment and of the observations are reported in Rousselin *et al.* [9] and in the electronic supplementary material. According to available data, we set the initial conditions of the system at the first observation date (i.e. 29 April, which was the 119th day of the year 2013) (see electronic supplementary material). We set the value of model parameters according to information available from peer-reviewed literature or experimental data whenever possible (table 1; electronic supplementary material). On the other hand, no information was available to *a priori* derive reliable estimates for parameters σ_N (net N assimilation rate) and κ (maximum rate of utilization of the substrates), which depend on environmental conditions that possibly varied in the different treatments: parameter q , affecting substrate transport within the plant and depending on the plant architecture [33], and six parameters relevant to the production of defences (α) and their effect ($\pi_1, \delta_1, \beta_2, \pi_2, \delta_2$). We estimated these unknown parameters by minimizing a cost function expressed as the sum of two negative log-likelihood functions, computed with respect to observations of shoot dry mass and aphid abundance (see the electronic supplementary material for details). We assessed the empirical distributions of calibrated parameters by making use of the moving block bootstrap [54]. In particular, we reconstructed bootstrapped time series for each of the observed variables and we fitted the values of the unknown parameters. We repeated this process 1000 times and we generated the 99% confidence intervals (CI) for each parameter via the percentile method [55].

2.4. Model selection

To account for possible different mechanisms regarding aphid ecology, the plant response to aphid infestation and its consequences, we contrasted the 'full' model reported in equation (2.1) with a set of nested models lacking some processes (figure 2). Namely, the full model (M10) assumes that aphid crowding promotes aphid migration and that the plant produces defences that make a fraction of resources inaccessible to aphids and kill, or repel, aphids if ingested. Three models nested in M10 assume a crowding effect on aphid migration and the induced production of defences. Yet, they can differ regarding the effect

of defences: killing/repulsion effect (M9), reduction of phloem accessibility (M8) or no effect (M7). There is also a simpler model that neglects the production of defences (M6). We also considered five analogous models ignoring the effect of aphid crowding, $\omega = 0$ (M1, M2, M3, M4, M5).

We tested if the effect of irrigation and fertilization can be represented in the models through a variation in some parameters κ and σ_N . The rationale is that the rate of utilization of the substrates (parameter κ) and the nitrogen assimilation rate (parameter σ_N) are expected to decrease in water [8,56] and nutrient [35,57] stress conditions, respectively. We then contrasted each of the 10 models assuming that (i) κ and σ_N , respectively, vary with irrigation and fertilization treatments; (ii) κ varies with irrigation and σ_N does not vary with fertilization; (iii) κ does not vary with irrigation and σ_N varies with fertilization; (iv) neither σ_N nor κ vary with fertilization and irrigation. Therefore, we calibrated two values for nitrogen assimilation rate per unit of root (σ_N^+ , σ_N^-) in cases (i) and (iii) and a unique value (σ_N^\pm) in cases (ii) and (iv). Analogously, we calibrated two values for the allocation of substrates to plant growth (κ^+ and κ^-) in cases (i) and (ii) and a unique value (κ^\pm) in cases (iii) and (iv).

Overall, we compared 40 different models (figure 2), obtained by incorporating five hypotheses on plant defences, two on density dependence of aphid migration and four on the effect of irrigation and fertilization, with one another. We selected the best model, that is, the one providing the best compromise between the goodness of fit to observed data and parsimony, through a model selection procedure based upon the Akaike information criterion [58]. For each model, we computed a value of $AIC = 2C + 2n_p$, where C is the minimum of the log-likelihood-based cost function estimated for the model and n_p is the number of calibrated parameters. Then, we ranked the models according to their AIC values and we computed the AIC differences (ΔAIC_i) between the AIC value of the i th model and the minimum AIC among all considered models (table 2). Models with $\Delta AIC_i < 2$ can be considered as equivalent [59,60] and, among equivalent models, we selected the simplest one (i.e. the one with fewest estimated parameters) as the best.

2.5. Sensitivity analysis

To assess the robustness of the model outputs to uncertainty affecting model parameters, we performed a sensitivity analysis of the model to (small) perturbations of the default parameter values reported in table 1. According to Thornley & Johnson [18], we computed the sensitivity of the variable Y (where for Y we considered the maximum value of S , of A and of their ratio A/S over the growing season) to small variations of parameter p_i as

$$\psi(Y, p_i) = \frac{\partial Y}{\partial p_i} \frac{p_i}{Y} \simeq \frac{\delta Y}{Y} \frac{p_i}{\delta p_i}. \quad (2.2)$$

In practice, after having changed the value of each parameter by +5% [18], we computed the value of ψ ; if $\psi(Y, p_i) > 1$ we concluded that the parameter has a more-than-linear effect on the variable.

2.6. The role played by fertilization and irrigation

After having ascertained that parameters κ and σ_N are likely to vary with irrigation and fertilization treatments, respectively, we used the selected model to simulate the temporal dynamics of the system for different values of these parameters. This allowed us to perform an *in silico* experiment to explore whether or not the model was able to reproduce the observed empirical patterns that claimed support for the plant vigour or the plant stress hypotheses. The *in silico* experiment is intended to test if the aphid density is affected by the fertilization (or irrigation) treatment. We considered five levels for the fertilization treatment (i.e. σ_N equal to 0.0012, 0.0024, 0.012, 0.06 and

Table 1. Model variables and parameters. For those parameters calibrated in the present work the calibrated 99% confidence intervals are reported in brackets. Cal, calibrated in the present work; Fix, fixed in the present work; see electronic supplementary material.

variable		dim.	description		
S		g	shoot structural dry mass		
R		g	root structural dry mass		
C_S		g	shoot carbon substrate		
C_R		g	root carbon substrate		
N_S		g	shoot nitrogen substrate		
N_R		g	root nitrogen substrate		
D		DU	plant-induced defences		
A		ind.	aphid population		
parameter	value	dim.	description		source
C and N assimilation					
σ_C	0.1	d^{-1}	assimilation rate of C		[33]
σ_N	6.70–17.08 10^{-3} [1.76–19.54] [4.56–43.04] 10^{-3} ^a	d^{-1}	assimilation rate of N		Cal
ν	1000	g	shoot (root) mass halving substrate assimilation due to self-shading (competition)		[33]
κ_C	0.1	/	semi-saturation C concentration		[33]
κ_N	0.01	/	semi-saturation N concentration		[33]
C and N substrate allocation to plant growth					
φ_C	0.50	/	unit of substrate C per unit of structural dry mass		[33]
φ_N	$2.50 \cdot 10^{-2}$	/	unit of substrate N per unit of structural dry mass		[33]
κ	142–223 [56–601] [48–876] ^b	d^{-1}	maximum rate of substrate utilization		Cal
η	73	/	switch-off function of plant growth: steepness		Fix
λ	169	d	switch-off function of plant growth: date of equal partitioning between growth and reserves		Fix
transport					
q	0.86 [0.84–0.89]	/	plant architecture scaling parameter		Cal
defences development					
α	0.02 [0.01–0.03]	$g \cdot d^{-1} \cdot ind^{-1}$	allocation of substrates to defences per unit of aphid		Cal
ε_C	$5 \cdot 10^{-2}$	$DU \cdot g^{-1}$	conversion efficiency of C substrate in defences		[50]
ε_N	1	$DU \cdot g^{-1}$	conversion efficiency of N substrate in defences		[50]
aphid					
θ	$1.12 \cdot 10^{-3}$	$g \cdot d^{-1} \cdot ind^{-1}$	maximum food intake per aphid		[51]
ξ	171	$ind \cdot g^{-1}$	maximum conversion efficiency of ingested food into descendants		[52]
μ	0.04	d^{-1}	aphid natural mortality rate		[53]
π_1	$8.52 \cdot 10^{-3}$ [3.70–23.12] 10^{-3}	$DU \cdot g^{-1}$	switch-on function of defences protected phloem fraction: concentration of defences at which the defence effect is half-saturated		Cal
δ_1	0.65 [0.55–0.73]	/	switch-on function of defences protected phloem fraction: steepness		Cal
π_2	0.05 [0.03–0.08]	$DU \cdot g^{-1}$	switch-on function of defences induced mortality/repulsion rate: concentration of defences at which the defence effect is half-saturated		Cal
δ_2	118 [27–251]	/	switch-on function of defences induced mortality/repulsion rate: steepness		Cal
β_1	1	/	switch-on function of defences protected phloem fraction: asymptotic value		Fix
β_2	28 [13–76]	/	switch-on function of defences induced mortality/repulsion rate: asymptotic value		Cal

^aValues refer to different fertilization treatments. ^bValues refer to different irrigation treatments.

0.12 d^{-1}) and five levels for the irrigation treatment (i.e. κ equal to 18, 36, 182, 910 and 1820 d^{-1}) corresponding to very low–low–average–high–very high levels of fertilization (or

irrigation). We varied the level of one treatment while keeping the other fixed at its average value, thus obtaining nine different combinations of factorial levels. Since in real factorial

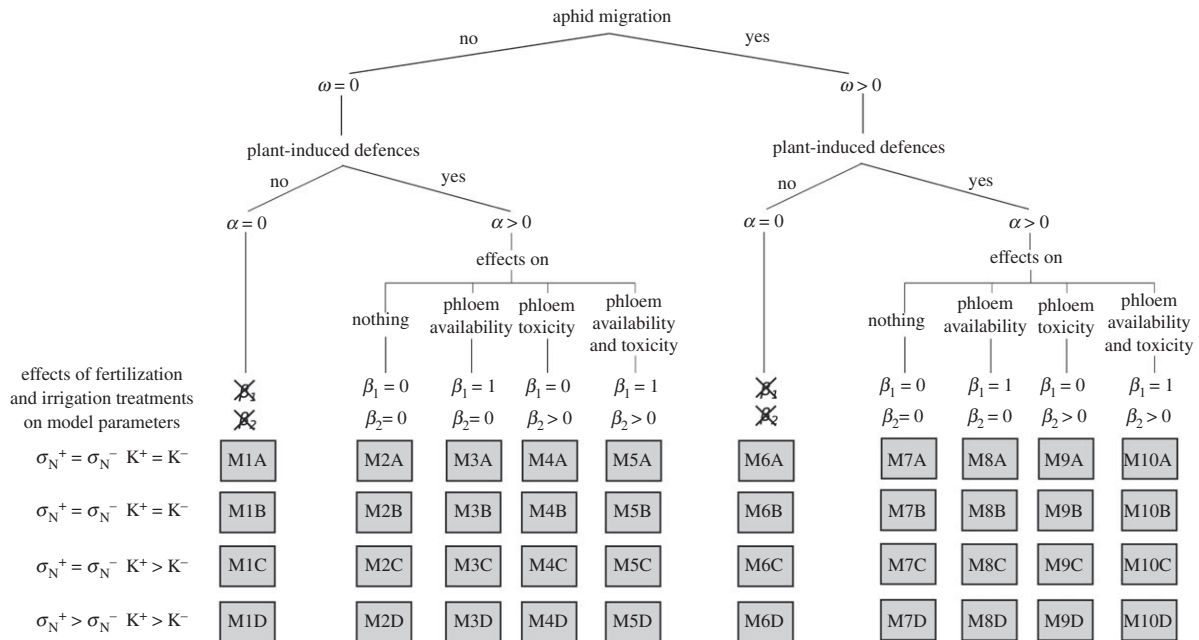


Figure 2. Schematic representation of the mechanisms considered in the different models M_i ($i \in [1, 10]$) nested in equation (2.1): (i) density-dependent aphid migration (ω), (ii) plant-induced defences development (α) and (iii) effect of induced defences on phloem availability to aphids (β_1) and on phloem toxicity (β_2). When the model parameter is set to zero, the relevant mechanism is ignored. Each model can be based on different hypotheses about the variation in the nitrogen assimilation rate σ_N (equal (A,C) or different (B,D) across fertilization treatments) and the substrate utilization rate k (equal (A,B) or different (C,D) across irrigation treatments).

Table 2. Comparison among candidate models for the plant–aphid system. For each model, we give its identifier ID (see text and figure 2 for details); its complexity assessed by the number of calibrated parameters n_p ; its Akaike score AIC; its ΔAIC_i computed as the difference between its AIC and the lowest obtained from all the models, i.e. $AIC = 6519.0$.

ID	n_p	AIC	ΔAIC_i	ID	n_p	AIC	ΔAIC_i
M10D	12	6519.0	0.0	M8A	7	6751.6	232.6
M5D	11	6520.8	1.8	M2D	6	6756.2	237.2
M8D	9	6570.8	51.8	M4D	9	6762.2	243.2
M10B	11	6576.1	57.1	M3C	7	6773.4	254.4
M5B	10	6590.5	71.5	M2B	5	6775.4	256.4
M3D	8	6624.5	105.5	M4B	8	6781.3	262.3
M7D	7	6628.4	109.4	M4C	8	6785.0	266.0
M6D	6	6632.1	113.1	M3A	6	6786.7	267.7
M9D	10	6634.2	115.2	M6C	5	6794.0	275.0
M3B	7	6641.5	122.5	M7C	6	6795.5	276.5
M8B	8	6641.9	122.9	M6A	4	6798.5	279.5
M7B	6	6646.4	127.4	M7A	5	6800.5	281.5
M9B	9	6651.6	132.6	M2C	5	6865.2	346.2
M6B	5	6696.0	177.0	M2A	4	6871.9	352.9
M9C	9	6708.6	189.6	M4A	7	6877.0	358.0
M10C	11	6712.6	193.6	M9A	8	6878.7	359.7
M8C	8	6721.6	202.6	M1B	4	7216.0	697.0
M5C	10	6727.9	208.9	M1D	5	7228.4	709.4
M10A	10	6742.9	223.9	M1A	3	7241.7	722.7
M5A	9	6746.8	227.8	M1C	4	7262.4	743.4

experiments the number of replicates (i.e. different plant individuals) is limited, we chose to simulate 10 replicates for each combination of factors, which corresponds to a realistic experiment with 90 plants being monitored. We simulated 10

possible trajectories of the system variables, for the same combination of factors, by running the model with 10 different parameter sets drawn from the empirical distribution obtained in the calibration process.

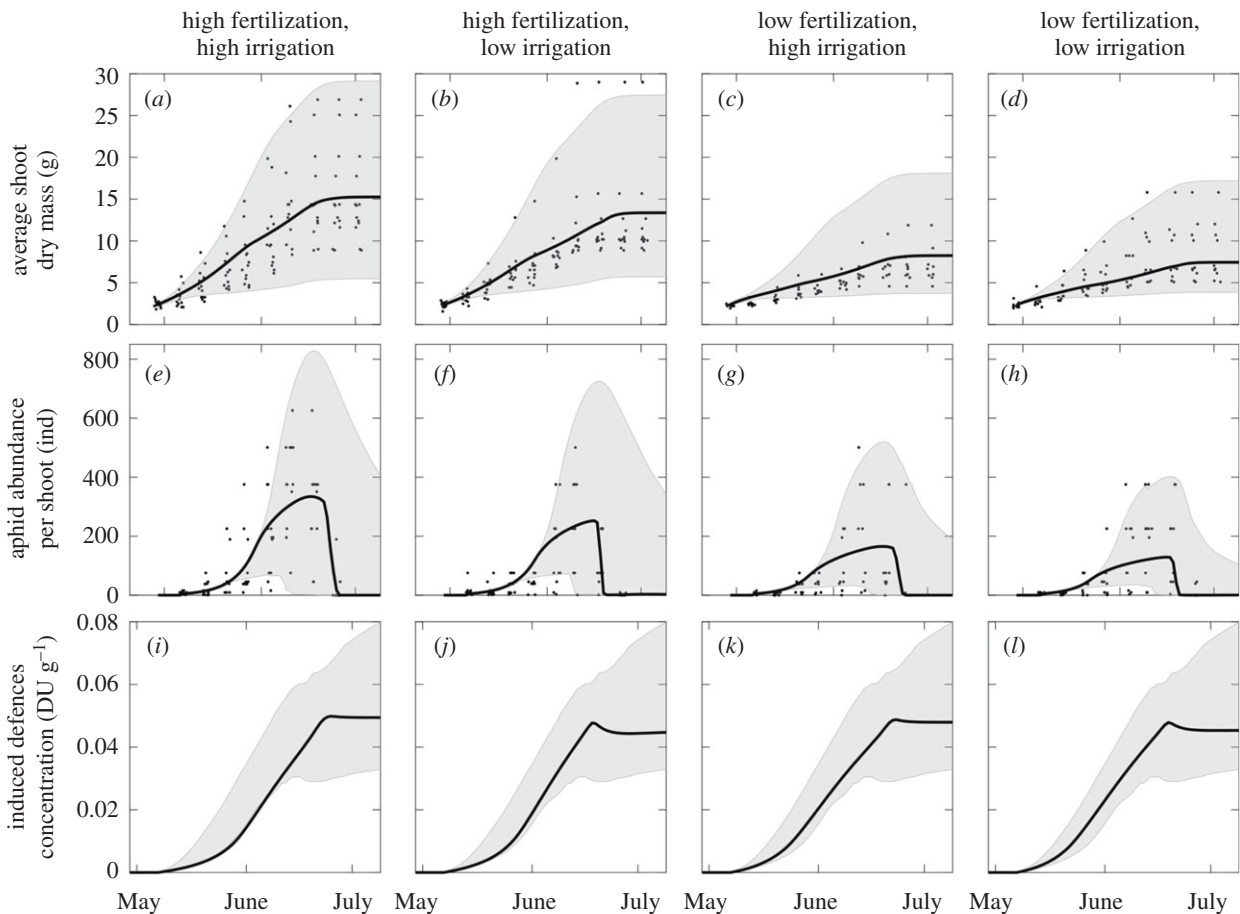


Figure 3. Observed (black points) and predicted (black lines) values of average shoot dry mass (*a–d*), average aphid abundance per shoot (*e–h*) and induced defences concentration (*i–l*) under different fertilization and irrigation treatments: high fertilization and irrigation (*a,e,i*), high fertilization and low irrigation (*b,f,j*), low fertilization and high irrigation (*c,g,k*), low fertilization and irrigation (*d,h,l*). Grey shaded areas indicate the predicted 99% confidence bands.

3. Results

3.1. Model calibration and selection

The best model ('the model', hereafter) assumes that (i) aphid migration due to crowding can be neglected; (ii) aphid presence induces the plant to divert resources from growth to defence; (iii) defences reduce phloem accessibility to aphids and, at higher concentrations, make the phloem sufficiently toxic to kill or repel aphids (electronic supplementary material, figure S1); (iv) the rates of nitrogen assimilation and substrate utilization differ for different levels of fertilization and irrigation, respectively.

The model fitted all four datasets, reproducing the main observed temporal patterns and differences between treatments (figure 3). Shoot growth is enhanced in high fertilization treatments while the water treatment considered here plays only a relatively minor role. The time course for shoot mass is linear and was followed by a stop towards the end of June. This is consistent with a potential exponential course, in the first part of the season [61], which has been prevented by the presence of the aphids. On the other hand, the stop in shoot growth at the end of June is induced by changes in the day-length. Note that parameter $\phi(t) = 0.5$ for $t = \lambda = 169$, corresponding to 18 June. Aphid population growth is initially sigmoidal, followed by a decay towards the end of June when the plant growth is halted (figure 3) and the concentration of defences attains the critical value of $\pi_2 = 0.03–0.08$, which makes ingestion from the phloem detrimental rather than beneficial to aphids. The initial

phase of aphid growth is enhanced in high fertilization treatments characterized by more vigorous plants.

The model gives biologically plausible parameter estimates (table 1) and the estimated variability in parameters permits most of the variability observed in the data to be captured. The calibrated values of σ_N , κ and q are consistent with previously published values (i.e. $\sigma_N = 0.02 \text{ d}^{-1}$, $\kappa = 200 \text{ d}^{-1}$ and $q = 0.67 - 1$ in [33]). The estimated values of parameters $\delta_1 < 1$ and $\delta_2 > 1$ suggest that the fraction of phloem that is protected from aphid withdrawal quickly increases for low concentrations of defences, whereas the phloem toxicity is switched on when the concentration of defences exceeds a threshold value (electronic supplementary material, figure S1). On the other hand, the model parameters relevant to the production of defences and their effect on aphids have no equivalent in the literature for a direct comparison.

3.2. Sensitivity analysis

Ranked values of the sensitivity, ψ , of shoot production and maximum aphid abundance and density to small changes in the parameter values are reported in electronic supplementary material, table S1. Negative values of ψ indicate a negative correlation between a change in a parameter value and the corresponding variable of interest. As expected, increasing the parameter λ results in an increase in shoot production, as it determines an increase in the growing season of 8.45 d, being $0.05\lambda = 8.45$, and consequently more resources to sustain a bigger aphid population, maintaining similar aphid densities.

Similarly, an increase in q results in an increase both in shoot production and in the peak of aphid abundance and density, as it determines a more efficient transport of substrates C and N between roots and shoots. This translates into bigger plants able to sustain higher peaks of aphid population densities.

With the exception of q and λ , our sensitivity analysis indicates that none of the model parameters has important (e.g. $\psi > 1$) consequences, indicating that the model is robust. However, our sensitivity analysis nevertheless provides some interesting insights. For instance, it shows that an increase in all those parameters positively related to the plant growth (σ_c , σ_n , ι_c , ι_n , k , ν) determine an increase in the maximum aphid abundance and, to a lesser extent, in maximum aphid density. If the aphids were more efficient in converting food into progeny (higher ξ), aphid density would increase but the overall population abundance would diminish as the resource would be over-exploited. An increase in the parameter α , determining a higher rate of resources devoted to defences, would have almost no effect on the shoot production but it would decrease aphid abundance and density. Yet, the plant could take advantage of a lower aphid abundance, since aphids are important vectors of viral diseases [41].

3.3. The role played by fertilization and irrigation

Shoot growth follows a sigmoidal pattern and it increases with fertilization and irrigation (figure 4*a,b*). The concentration of carbon substrates in shoots varies between 3% and 23% during the growing season with peaks at its beginning, when plant growth is limited by the nitrogen supply, and at its end, when plant growth halts in response to day length decreases, but carbon assimilation continues. Carbon concentration is enhanced in stressful conditions (very low to low fertilization/irrigation treatments) that limit plant growth rather than carbon assimilation (figure 4*c,d*). The concentration of nitrogen substrates varies between 0.1% and 1.4% during the growing season (figure 4*e,f*). It decreases in the first weeks of growth, but, in the case of very high/high fertilization or very low irrigation, it increases until the second week of May. In fact, for high fertilization treatments, nitrogen is not initially consumed by plant growth, which is limited by carbon supply, and, for low watering, nitrogen concentration increases as plant growth is impaired while N assimilation is not. The peak concentration of defences is delayed in time for higher fertilization and irrigation (figure 4*g,h*). When the plant is well watered, the time of the peak aphid population density is delayed by one week. This is due to the fact that defences need more time to reach significant concentrations in bigger plants (figure 4*i,j*). The positive effect of fertilization and irrigation upon aphid abundance becomes evident at the end of May. In the first part of the season, aphid density is enhanced by a low/average value of fertilization (or irrigation) while later in the season aphid density is higher in a well-fertilized (irrigated) plant (figure 4*k,l*).

The results of our virtual experiment show that one can draw very different conclusions depending on the considered fertilization/irrigation levels and the date of observations. For instance, one could infer that fertilization (i) enhances aphid population by observing aphid density in the mid- to late part of the season for very low to average values of fertilization (figure 5*c–e*); (ii) decreases it, by observing aphid density in the early to mid-season for average to very high values of fertilization (figure 5*a–c*); (iii) has no effect, by observing aphid density

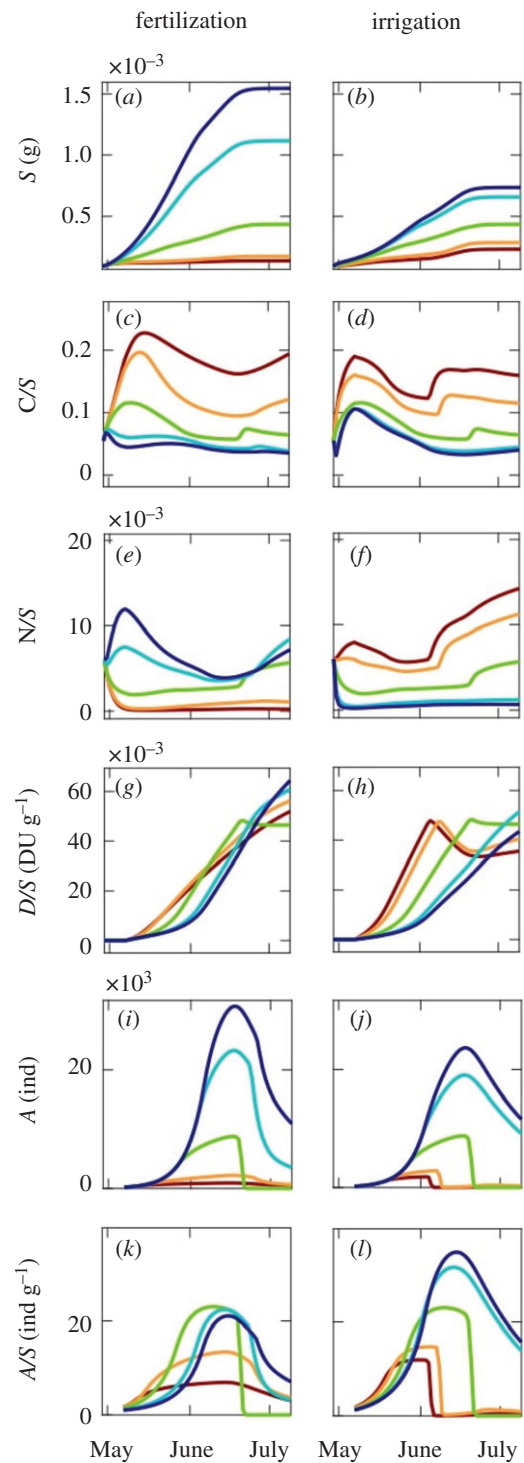


Figure 4. Simulated effect of fertilization (left column) and irrigation (right column) on the plant–aphid system: average shoot dry mass S (*a,b*), carbon C/S (*c,d*) and nitrogen N/S (*e,f*) substrate concentration in shoots, defences concentration in shoot D/S (*g,h*), aphid abundance A (*i,j*) and density A/S (*k,l*). Lines—colour identifies fertilization (or irrigation) level: very low (red), low (orange), average (green), high (light blue) and very high (blue).

early and late in the season, for high to very high values of fertilization (figure 5*a–e*). Similarly, different conclusions can be drawn regarding the effect of irrigation: positive (figure 5*f*), negative (figure 5*b*) or null (figure 5*d*, from average to very high values of irrigation). The explicit consideration of inter-individual variability in growth trajectories shows that patterns emerging from a limited (i.e. 10) number of replicates per treatment become less clear at the end of the growing season (see longer boxes in figure 5*e,f*). We purposefully avoided performing statistical tests on our results because

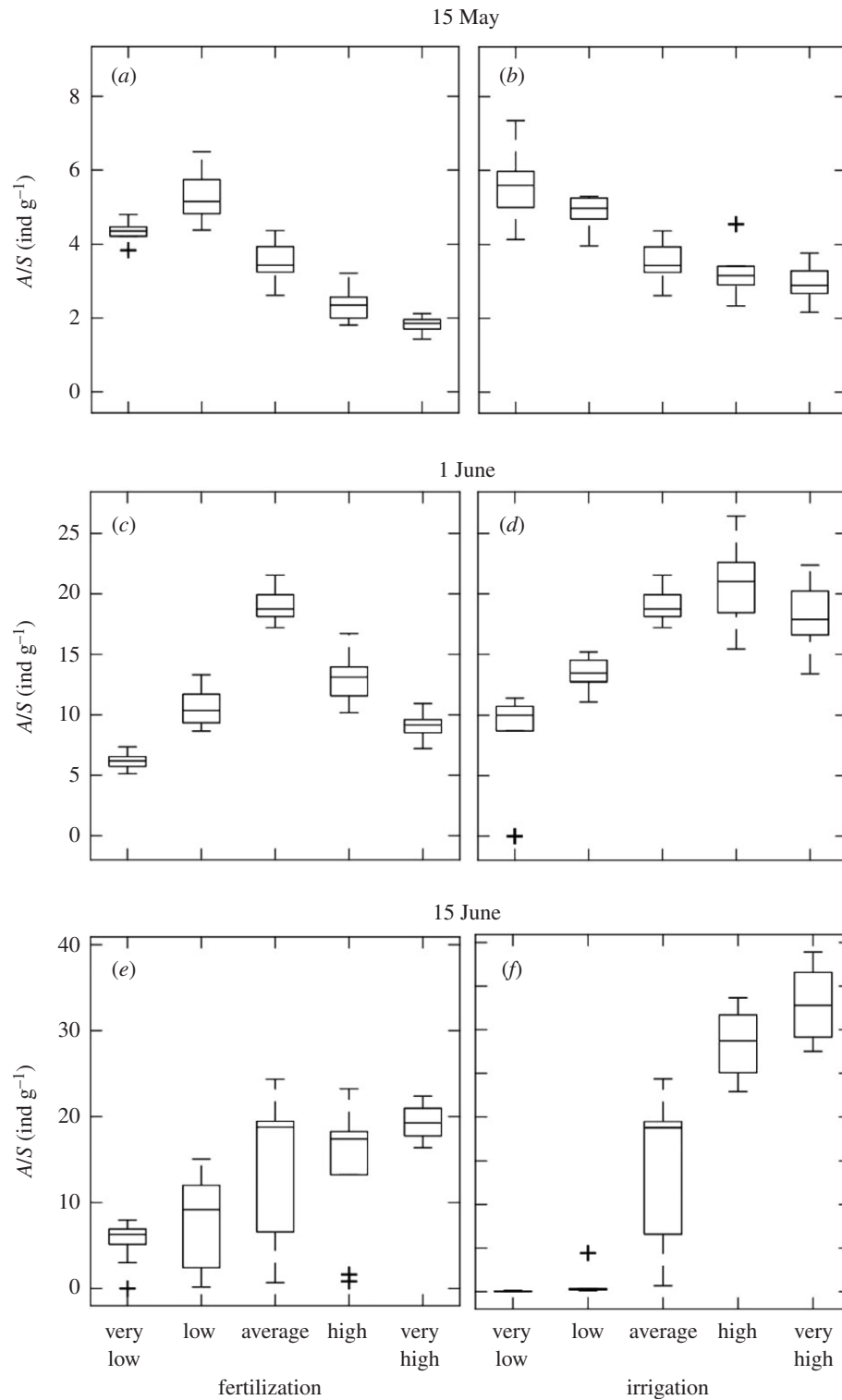


Figure 5. Simulated effect of fertilization (*a,c,e*) and irrigation (*b,d,f*) on aphid density on 15 May, 1 June and 15 June. Boxes represent the first and third quartiles (25% and 75%) with a line inside indicating the median of 10 simulated replicates of each treatment. The whiskers extend $\pm 1.5 \times$ the interquartile range (75th percentile—25th percentile) from the third and first quartiles. Values outside the whiskers are considered outliers and plotted individually using the ‘+’ symbol.

the number of replicates, which can be easily varied in a virtual experiment, would have increased the statistical power to detect changes in aphid density (see [62] for a similar exercise).

4. Discussion

In this work, we showed that embedding a mechanistic plant growth model, widely studied in agronomy, in a consumer–resource modelling framework, widely studied in ecology, might be a promising approach for agroecology. We demonstrated the ability of such a novel approach in understanding

the consequences of irrigation and fertilization treatments in a plant–aphid system. Yet, the proposed model has the ambition of being physiologically rigorous and general enough to be applied to different plant–pest systems and to incorporate the description of other agronomic practices.

4.1. The selected model and model calibration and selection

A recent review [41] suggested that infested plants can put in place phloem-sealing mechanisms to interfere with aphids’

access to plant resources and produce a number of secondary metabolites (e.g. cardenolides, glucosinolates and benzoxazinoids), which, if ingested, impair aphid viability [41]. Our study suggests that both defensive mechanisms are at play in the peach–green aphid system. According to our calibration, impairing phloem accessibility is the most effective at low concentrations of defences, while ‘intoxicating’ aphids is the most effective at higher concentrations. This is in accordance with works on the *Arabidopsis-Myzus persicae* system, for which reductions in aphid fecundity, up to 100%, have been reported in response to high concentrations of some plant defensive compounds [63,64]. The model application to a real study case subjected to different irrigation×fertilization treatments indicates that parameters relevant to plant nitrogen assimilation (σ_N) and plant utilization of substrates (κ), originally proposed within a theoretical framework [31], can be linked to agronomic practices and then manipulated by the grower. However, in order to effectively use the proposed model to define effective agronomic recommendations, further studies on the response of the model parameters to effective practices are clearly required.

One of the main features of the peach–green aphid system is that, at the beginning of summer, aphid populations dwelling on peach trees drop. This occurs because aphids die, or abandon their primary host, or give birth to winged newborns that migrate to secondary herbaceous hosts [65]. However, the underlying mechanisms triggering these processes are far from being clear. Our findings suggest that the reduction in resource availability, due to the investment in defensive traits and to photo-period-driven interruption of shoot growth, along with the reduction of the phloem nutritional value, due to the accumulation of defensive compounds possibly toxic to the aphid, are the mechanisms most likely to be responsible for the observed patterns. In principle, the crash in aphid population could be due to other factors such as the arrival of predators attracted by high aphid density [66] or the possible reduction of the phloem nutritional value due to plant ageing [6]. However, if the aphid population drop were driven by density-dependent mechanisms, one would probably expect to observe fluctuations in the aphid population rather than a constant decline [67]. Moreover, in previous modelling works, it has been shown that observed population trends in different aphid species could be reproduced by considering a *per capita* death rate positively related to the aphid cumulative population size [68–70]. Such a relationship coherently emerges as a property of our model if the pest presence induces the plant to produce defences that accumulate, and not if the phloem nutritional value declines throughout the season, independently from aphid presence.

Performing experiments to find correct numerical values for parameters of biological models is virtually impossible because many parameters cannot be directly measured. For this reason, we were forced to numerically calibrate nine parameters via our likelihood-based model fitting procedure. However, biologically plausible parameter estimates and good fitting do not guarantee that parameter estimates are correct, because of possible correlations among the parameters [71] and model identifiability problems that can arise from an imbalance between model complexity and available data [72]. The proposed modelling framework would therefore benefit enormously from experimental works dedicated to the measurement, or at least a sound assessment, of some model parameters. Despite the importance of the parameter

q in Thornley’s models, we found no studies on its assessment. Similarly, although it is well known that a plant can divert resources from growth to defence [73], we found no quantitative relationships relevant to the cost of making defences (parameter α in our model) in terms of growth loss, nor between the presence of defences and pest performances. Our exercise provides a preliminary assessment of these parameters that needs to be confirmed or confuted by dedicated field and/or laboratory works.

4.2. The role played by fertilization and irrigation

Variations in plant growth, and in the concentrations of C and N substrates in plant tissues, for different levels of fertilization and/or irrigation are well acknowledged [56,74] and they have already been shown to be emerging properties of the original model for plant growth used in this work [31]. Our pest-plant model maintains these properties (figure 4a–f) and allows further insights regarding the variations observed in an aphid population. The aphid population response to fertilization and irrigation has been explored in a number of empirical works not providing a straightforward picture. Some authors observed no effect of fertilization in the wheat–Russian wheat aphid system [75], or negative effects of irrigation in the apple–rosy apple aphid and in the cotton–cotton aphid systems, respectively [76,77]. Other authors observed the highest aphid abundance at an average level of fertilization, and no effect of irrigation, in the chrysanthemum–cotton aphid system [78]. The intrinsic rate of oat aphid population increase in three grass species was observed to be favoured by irrigation in [79]. On the other hand, aphid population was observed to be maximal for moderate water stress in the cabbage–green aphid and cabbage–cabbage aphid systems [17], and in one out of three genotypes tested for the poplar–woolly poplar aphid system [80]. Our model, parametrized for the peach–green aphid system, shows that all this apparently contrasting empirical evidence can emerge from the same biological principles governing plant–pest dynamics and that both plant vigour and plant stress hypotheses can find support when observing a plant–pest system evolving in time and subject to different levels of changes in the environmental conditions. The aphid population dynamics reproduced by our model (figure 5) indicate that the effect of fertilization and irrigation on the pest population cannot be simply reduced to positive or negative. In fact, its sign and strength depends on the considered levels of fertilization/irrigation and on the date of observation along the growing season. The contribution of our work is to show how a new synthesis of the experimental data can emerge by using mechanistic modelling. The challenge for our future work is to show how this insight—as well as the model developed here—can be used to inform practical decision making by farmers and growers.

Data accessibility. Datasets for this research are included in Rousselin *et al.* [9] and are reported in the electronic supplementary material.

Authors’ contributions. D.B. conceived the study. D.B. and M.Z. designed and implemented the study. All three authors discussed the results and their implications throughout the study, and wrote the paper.

Competing interests. We declare we have no competing interests.

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Supplementary Information

- **A.1** Available data
- **A.2** Model initialization and off-line calibration
- **A.3** Model calibration

Chapter 2

Maximizing plant production and minimizing environmental impact: comparing agricultural management scenarios with multi criteria decision analysis

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Résumé

Satisfaire la demande de produits agricoles tout en protégeant l'environnement contre les effets négatifs de l'expansion de l'agriculture est un défi majeur auquel la gestion des cultures est confrontée. Nous avons appliqué un modèle de simulation plante-ravageur et une analyse décisionnelle multicritères pour considérer simultanément deux objectifs: (1) maximiser la production de biomasse végétale et (2) minimiser l'impact environnemental lié à la fertilisation, à l'irrigation et aux pesticides. Nous avons utilisé un modèle plante-ravageur pour simuler la production de biomasse végétale pour 27 scénarios de gestion, qui couvrent une gamme de niveaux de fertilisation, d'irrigation et de pesticide. Nous avons calculé l'impact environnemental lié à chaque scénario de gestion au moyen d'une somme pondérée des coûts attribués aux niveaux de fertilisation, d'irrigation et de pesticide. En analysant le front de Pareto, nous avons identifié les scénarios optimaux par rapport aux objectifs considérés. Ces scénarios se caractérisent principalement par une fertilisation maximale et l'absence de pesticide. Nous avons évalué la multifonctionnalité des scénarios optimaux au travers du coefficient de Gini: le scénario assurant le mieux l'égalité entre les deux objectifs était caractérisé par une fertilisation élevée, une irrigation intermédiaire et l'absence de pesticide. Même si nos résultats doivent être considérés comme qualitatifs et non immédiatement transférables à des pratiques agronomiques, notre cadre analytique a été un outil utile pour comprendre les compromis entre deux objectifs contrastés et la manière dont ils peuvent être conciliés simultanément.

Mots-clés: production de biomasse végétale; impact environnemental; analyse décisionnelle multicritère; gestion agricole multi-objectifs; modélisation

Abstract

Satisfying the demand for agricultural products while also protecting the environment from negative impacts of agriculture expansion is a major challenge that crop management is facing. We applied a plant-pest simulation model and multi-criteria decision analysis to simultaneously consider two objectives: (1) maximizing plant biomass production and (2) minimizing environmental impact related to fertilization, irrigation and pesticide deployment. We used a plant-pest model to simulate plant biomass production under 27 management scenarios, that span a range of fertilization, irrigation and pesticide levels. We computed the environmental impact linked to each management scenario by means of a weighted sum of the costs assigned to fertilization, irrigation and pesticide levels. By analysing the Pareto front we identified the optimal scenarios respect to the considered objectives. These scenarios were mostly characterized by the highest fertilization and no pesticide. We evaluated the multi-functionality of the optimal scenarios by mean of the Gini coefficient: the scenario better assuring the equality between the two objectives was characterized by high fertilization, intermediate irrigation and no pesticide. Even if our results should be considered as qualitative and not immediately transferable to agronomic practices, our analytical framework was a useful tool in understanding trade-offs among two contrasting objectives and how they can be simultaneously accommodated.

Keywords: plant biomass production; environmental impact; multi-criteria decision analysis; multi-objective agricultural management; modelling

Introduction

The world population continuously increases and, according to estimates, it would be 1.5 times the current population by 2100 (Li et al., 2019; Deknock et al., 2019). To ensure global food security, an increase in crop yield is essential (Li et al., 2019). Industrial forms of modern agriculture aim to remove limitations to plant productivity mainly by increasing *i*) chemical fertilizers application, to meet plants' nutrient needs (Gliessman, 2015a; Bommarco et al., 2013); *ii*) irrigation, to supply for increasing crop water demand due to warming temperature (Turner et al., 2019); *iii*) pesticide use, to control pests, which reduce crop productivity by feeding and transmitting viruses (Deknock et al., 2019; Oerke, 2006).

The way how agricultural crops are managed can significantly affect the environment and biodiversity (Bommarco et al., 2013; Seppelt et al., 2013; Demestihis et al., 2017). Mineral fertilization fosters nitrogen leaching, which declines the quality of drained water, resulting in a series of environmental impacts that include surface water eutrophication, groundwater pollution, and soil degradation (Demestihis et al., 2017; Li et al., 2019). Furthermore, common fertilizer tends to volatilize into the air in the form of N_2O , a greenhouse gas with global warming potential (GWP) 298 times that of the reference gas, CO_2 (Xiao et al., 2019). The agricultural sector is the largest

consumer of water: agriculture uses 69% of all freshwater withdrawals (UNESCO, 2020). Exploitation of both surface and ground water resources to sustain crops irrigation can reduce water flow to rivers, lakes and wetlands, and cause sustained drawdown in aquifer head levels (Calzadilla et al., 2010; Turner et al., 2019). The uneven distribution of water (and population) among world regions has made water supply critical for a growing number of countries: this trend is likely to be exacerbated by climate change (IPCC, 2014). The accumulation of pesticide residues in the environment is a threat for human and environmental health, accounting for the contamination of drinking water supplies and food sources and the reduction of biodiversity (Deknock et al., 2019). In addition, pesticide may impair bee colonies, consequently decreasing pollination, an essential service for crop production, the value of which has been estimated at €153 billion (Gallai et al., 2009).

Satisfying crop demand while guaranteeing other ecosystem services represents one of the greatest challenges facing crop management. Yet, the presence of several interacting components and biophysical processes makes the relationships between agricultural practices, crop production and environmental impact not straightforward (Demestihis et al., 2017). Models may help in attaining an understanding of agroecological systems and processes (Grimm, 1994). Moreover, they can be used to analyse the effects of a wide range of management scenarios, supplementing field experiments for identifying best management strategies (Malik and Dechmi, 2020). For example, Malik and Dechmi (2020) use a crop growth model to simulate the effect of different nitrogen management practices on maize, wheat, barley, sunflower and alfalfa fields, considering crop production and N losses. Demestihis et al.

(2018) use a crop model to explore how agricultural management practices, such as planting density, irrigation and fertilization, affect the production of ecosystem services by altering soil fertility, greenhouse gas emission, water quality and fruit production in apple orchards.

Production and environmental objectives are often contrasting. One approach for evaluating management alternatives facing conflicting objectives is multi-criteria decision analysis (MCDA). MCDA is a collection of formal methods to structure and formalize the comparison of multiple and/or conflicting objectives to support the decision making (Belton and Stewart, 2002). MCDA has been widely and successfully applied to agriculture (Craheix et al., 2016; De Luca et al., 2017; Scharfy et al., 2017; Chukalla et al., 2018; Balezentis et al., 2020), forest (Kangas and Kangas, 2005; Diaz-Balteiro and Romero, 2008; Schwenk et al., 2012), and to city and tourism (Ferretti et al., 2014; Michailidou et al., 2016; Pesce et al., 2018; Langemeyer et al., 2020) management.

In the current study, we use Zaffaroni et al. (2020) model, which couples plant growth and aphid dynamics and is calibrated for the system peach *Prunus persica* - green aphid *Mizus persicae*, to compute plant biomass production under different management scenarios (i.e. combination of different levels of fertilization, irrigation and pesticide). We compute the environmental impact linked to each management scenario by mean of a weighted sum of the cost arbitrarily assigned to fertilization, irrigation and pesticide level. We apply MCDA to support the overall goal of managing agricultural crop with respect to two contrasting objectives: to maximize plant biomass production and to minimize environmental impact. We explore the optimal

alternatives respect to the two objectives through the analysis of the Pareto-front. In addition, we evaluate the multi-functionality of the Pareto-optimal alternatives by means of the Gini coefficient, which accounts for inequalities in objectives values.

Materials and methods

The model

The plant-aphid model is described in details elsewhere (see Zaffaroni et al. (2020)). We provide here only an overview of its main features, schematically represented in figure 2.1. The model describes the temporal variation during a growing season of *i*) dry biomass of total shoots and roots (S and R) of an average plant, *ii*) carbon and nitrogen substrates in shoots and roots (C_i , N_i $i = S, R$), *iii*) plant induced defensive level (D), and *iv*) aphid population feeding plant phloem and impairing plant growth (A). Plant is induced by aphids presence to use carbon and nitrogen substrates to defend itself (Will et al., 2013; Züst and Agrawal, 2016; Vyska et al., 2016). This results in the production of chemical and morphological/physiological changes that reduces aphid accessibility to the phloem (e.g. by phloem sealing) (Medina-Ortega and Walker, 2013; van Velzen and Etienne, 2015) and decreases the rate at which ingested food is converted into progeny (e.g. by releasing toxic components in the sieve that can even repel or kill the aphids) (Züst and Agrawal, 2017). The rationale behind model assumptions is discussed in detail in Zaffaroni et al. (2020). We describe the dynamics of the plant-aphid

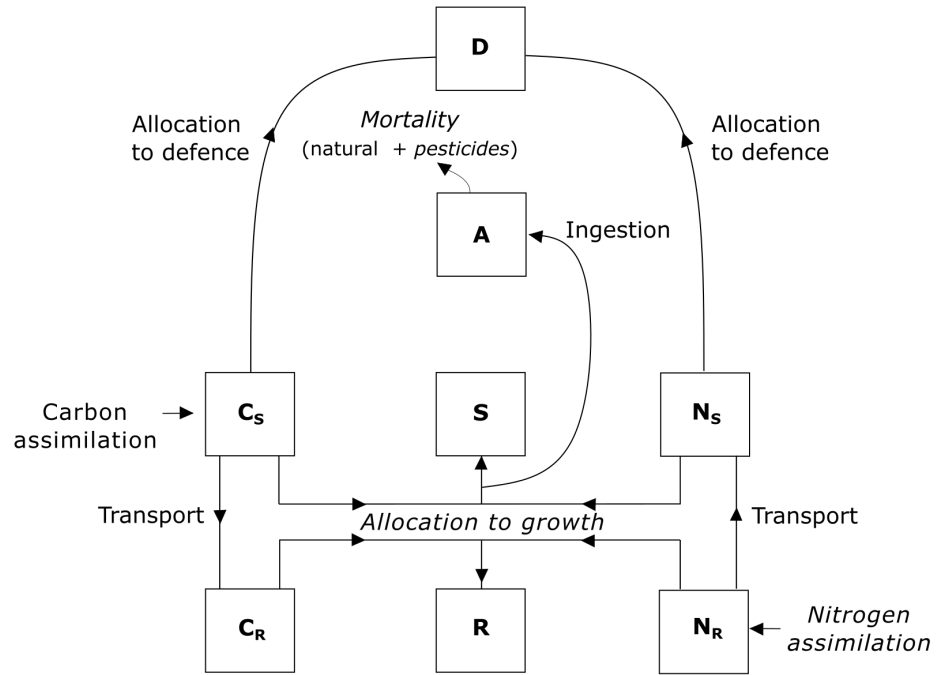


Figure 2.1: Schematic representation of the plant-aphid model where the plant is constituted by shoot (S) and root (R) structural dry mass, carbon (C_i) and nitrogen (N_i) substrates in shoots ($i = S$) and roots ($i = R$). The aphid population (A) intercepts a fraction of substrates allocated to constitute shoot structural mass and the plant diverts shoot substrates (carbon and nitrogen) to produce defences compounds (D). Fertilization increases nitrogen assimilation, irrigation increases plant allocation to growth, and pesticides increase aphid mortality. More details are given in the main text.

system with the following system of ordinary differential equations, the list of the processes (in capital Greek letters in the model) along with the list of parameters can be found in *Appendix B* :

$$\left\{ \begin{array}{l} \dot{C}_S = \Theta_S - \kappa\Gamma_S - T_C - \Delta_C \quad (2.1a) \\ \dot{N}_S = T_N - \kappa\Lambda_S - \Delta_N \quad (2.1b) \\ \dot{S} = \kappa\Omega_S - \Psi \quad (2.1c) \\ \dot{C}_R = T_C - \kappa\Gamma_R \quad (2.1d) \\ \dot{N}_R = \sigma_N\Theta_R - \kappa\Lambda_R - T_N \quad (2.1e) \\ \dot{R} = \kappa\Omega_R \quad (2.1f) \\ \dot{D} = E_C + E_N \quad (2.1g) \\ \dot{A} = (\Xi - \mu_p)A \quad (2.1h) \end{array} \right.$$

In equation (2.1a), Θ_S is the carbon assimilated by shoots from the atmosphere via photosynthesis and stored in shoots. The term $\kappa\Gamma_S$ is the carbon substrate allocated to shoot growth or reserves. The parameter κ is the rate of substrate utilization for shoot biomass growth, which we assume to decrease in water stress condition (Muller et al., 2011; Sevanto, 2014). The term T_C is the carbon substrate transported from shoots to roots, which depends on carbon concentration difference between shoots and roots divided by a resistance, and Δ_C is the carbon substrate allocated to produce induced defences, which is proportional to aphid abundance. In equation (2.1b), T_N is the nitrogen substrate transported from roots to shoots, which, similar to carbon transport, depends on nitrogen concentration difference between roots and shoots divided by a resistance. The term $\kappa\Lambda_S$ is the nitrogen

substrate allocated to shoot growth or reserves, and Δ_N is the nitrogen substrate allocated to produce induced defences, which is proportional to aphid abundance. In equation (2.1c), $\kappa\Omega_s$ is the increase in structural shoot dry mass in the absence of any phloem withdrawal by the aphids: it depends on the rate of substrates utilization for shoots growth and it accounts for the suspension of plant growth driven changes in the photo-period. The terms Ψ represents the amount of phloem ingested by aphid population on plant, which decreases with plant defences. The dynamics of carbon and nitrogen substrate in roots (C_R and N_R) and of roots dry mass (R) follow similar rules for nitrogen substrate assimilation, transport and allocation to root growth and we assumed that they are not directly affected by the presence of aphids. In equation (2.1e), the term $\sigma_N\Theta_R$ is the nitrogen assimilated by roots from the soil and stored in roots: the parameter σ_N is the nitrogen assimilation rate, which we assume to decrease in nutrient stress condition (Connor et al., 2011). In equation (2.1g), the terms E_C and E_N are the amounts of, respectively, carbon-base and nitrogen-base induced defences. In equation (2.1h), Ξ is the aphid intrinsic growth rate, accounting for birth rate, which increase with the ingested phloem and decrease with plant defences, and the natural mortality rate. The term μ_p is the pesticide induced aphid mortality rate.

We simulate the effect of variation in fertilization treatments through a variation of nitrogen assimilation rate variation (σ_N), which directly affects the amount of nitrogen assimilate by roots. We simulate the effect of variation in irrigation treatment through a variation of the rate of substrates utilization for plant growth, which directly affects the allocation of carbon and nitrogen substrates to shoot and root growth. The rationale behind these assumptions

is discussed in detail in Zaffaroni et al. (2020). In the present work, we add the term for aphid pesticide induced mortality (μ_p) which we assume to increase with pesticide.

Management scenarios

We consider 27 management scenarios obtained by combining three different levels (i.e. low, intermediate, high) of the three considered decision variables: fertilization, irrigation and pesticide (table 2.1). Note that when we refer to a low pesticide level we refer to no pesticide application. For each scenario we simulate the trajectory of shoot dry mass $S(t)$: we assume that the shoot biomass at the end of the vegetative season (S^*) is a good proxy of overall plant growth and relevant crop.

Identification of objectives

We evaluate the performance of each management scenario with respect to two objectives: to maximize the plant biomass production and to minimize the environmental impact (EI). Since maximizing plant biomass production is equivalent to minimizing plant biomass loss (BL), for the sake of simplicity, we will consider the latter as objective in our analysis. We compute the plant biomass loss as the fraction of shoot biomass lost respect to the maximum potential value ($BL_i = \frac{\max[S_1^*; S_2^*; \dots; S_{27}^*] - S_i^*}{\max[S_1^*; S_2^*; \dots; S_{27}^*]}$). This implies that BL is equal 0 when the shoot production is maximum and is equal to 1 when the shoot production is null. For each management scenario we assign a cost (c_k) to the three *decision variables* (i.e. fertilization (n), irrigation (h), pesticide (p)) as

follows: *i*) $c_k = 0$ when fertilization (irrigation/pesticide) is low; *ii*) $c_k = 0.5$ when fertilization (irrigation/pesticide) is intermediate; *iii*) $c_k = 1$ when fertilization (irrigation/pesticide) is high. We combine the decision variables' costs to establish the environmental impact of the scenarios by mean of a weighted sum ($EI_i = \sum_{k=\{n,h,p\}} \omega_{k,i} c_{k,i}$). Assuming that the environmental impacts due to fertilization, irrigation and pesticides has the same importance for the decision makers, we consider equal weights for each decision variable (*i.e.* $\omega_n = \omega_h = \omega_p = 0.33$). As for BL, EI values range between 0 and 1.

Multi criteria decision analysis

MCDA is a multi-step process comprising a family of methods to structure and formalize the comparison of management scenarios to support the decision-making (Pesce et al., 2018; Zanchi and Brady, 2019). We firstly explore the possible trade-off between the two objectives (minimizing BL and EI) through the identification of the Pareto front. The Pareto front specifies the groups of Pareto-optimal scenarios, *i.e.* the management scenarios for which it is not possible to modify the decision variables (*i.e.* fertilization, irrigation and/or pesticide) to improve the performance respect to one objective (*i.e.* decreasing BL) without worsening at the same time the performance respect to the other objective (*i.e.* increasing EI) (Kennedy et al., 2008). Since the weights assigned to the decision variables influences the value of the objective EI and consequently the Pareto-optimal scenarios, we considered other weights combination to mimic different decision makers' concerns relate to fertilization, irrigation and pesticide environmental impacts. Thus,

we assign a cost $c_k = 0.9$ to the decision variable whose environmental impact concerns the decision makers the most, and a cost $c_k = 0.05$ to the other two decision variables and we analyze the variation in the scenarios composing the Pareto front.

For each Pareto-optimal scenario we compute the Gini coefficient, which describes the inequality in the objectives values and indicate the multi-functionality of a scenario. Although Gini coefficient has originally been employed in economic analysis as a valid index to measure the income inequality of inhabitants, a growing number of references have demonstrated that it can be used in ecological application as well (Accatino et al., 2018; Wang et al., 2019; Li et al., 2017). We computed the Gini coefficient for each scenario i as Dorfman (1979):

$$G_i = \frac{\sum_{j=1}^n \sum_{k=1}^n |x_{j,i} - x_{k,i}|}{2n \sum_{j=1}^n x_{j,i}} \quad (2.2)$$

where n ($= 2$) is the number of objectives and x_j and x_k are the objective values for the considered scenario. A Gini coefficient of zero expresses perfect equality between the objectives, increasing the value of the Gini coefficient the inequality between the objectives increases and the multi-functionality of a scenario decreases.

Results

We report the values of the two objectives evaluated for different management scenarios in table 2.1 and in figure 2.2. Biomass loss varies between 0 and

0.96, while the environmental impact varies between 0 and 1. The Pareto front highlights that as biomass loss decreases, ecological impact increases and vice versa. That clearly shows a trade-off between the considered objectives. The Pareto front is composed by 7 out of 27 management scenarios (hereafter the Pareto-optimal scenarios). Among these: the 72% is characterized by an high level of fertilization (scenarios 19, 22, 23, 26, 27), the 14% by, respectively, a low and intermediate levels of fertilization (scenarios 1 and 10, respectively); the 42.6% is characterized by a low irrigation (scenarios 1, 10, 19), the 28.7% by, respectively, an intermediate and high level of irrigation (scenarios 22, 23 and 26, 27 respectively); the 57% is characterized by a low pesticide level (scenarios 1, 10, 19, 22), the 29% by an intermediate level of pesticide (scenarios 23 and 26) and the 14% by an high level of pesticide (scenario 27).

The weights assigned to the decision variables influence the EI and consequently which scenarios are scored as optimal. Yet the response of the Pareto-optimal scenarios to the weights assigned to the decision variables scores (ω_k) showed that the majority of Pareto-optimal scenarios are characterized by high fertilization (figure B.1 A-G-J in *Appendix B*) and low pesticide (figure B.1 C-F-I-L in *Appendix B*), independently by the weights combination, except when fertilization causes the highest environmental impact (figure B.1 D in *Appendix B*): in that case, the majority of Pareto-optimal scenario are characterized by intermediate fertilization.

The baseline scenario, which is characterized by an intermediate value for fertilization, irrigation and pesticides application (scenario 14 in figure 2.2), is not an optimal scenario. Starting from the baseline scenario, we identify

four scenarios groups: *i*) win-win scenarios, where both the objectives are improved (or do not change) respect to the baseline scenario; *ii*) win-lose scenarios, where BL decreases (or does not change), but EI increases; *iii*) lose-win scenarios, where EI decreases (or does not change), but BL increases; *iv*) lose-lose scenarios, where both the objectives are worsened respect to the baseline scenario. Win-win scenarios (i.e. scenarios 16, 19, 20, 22) are the most interesting and, in our case, they are all characterized by an higher fertilization level respect to the baseline scenarios.

The values of Gini coefficient for the Pareto-optimal scenarios are reported between parenthesis in figure 2.2. The Gini coefficient ranges between 0.02 to 0.50, representing large differences of multi-functionality between the optimal scenarios. Scenario 22 is the one which better assures the equality between the environmental and the production objectives. It is characterized by high fertilization, intermediate irrigation and low pesticide. Scenarios 1 and 27 presents the highest inequality between the objectives: in scenario 1 the environmental objective dominates, while in scenario 27 the production objective dominates.

Discussion

In this work, we show that a mechanistic model reproducing plant-aphid interaction can be used to simulate the impact of agricultural practices on a plant-aphid system. The strengths of our model are, arguably, that it is relatively simple and transparent, providing clear connections between assumed mechanisms and predicted response. Transparency is important, so

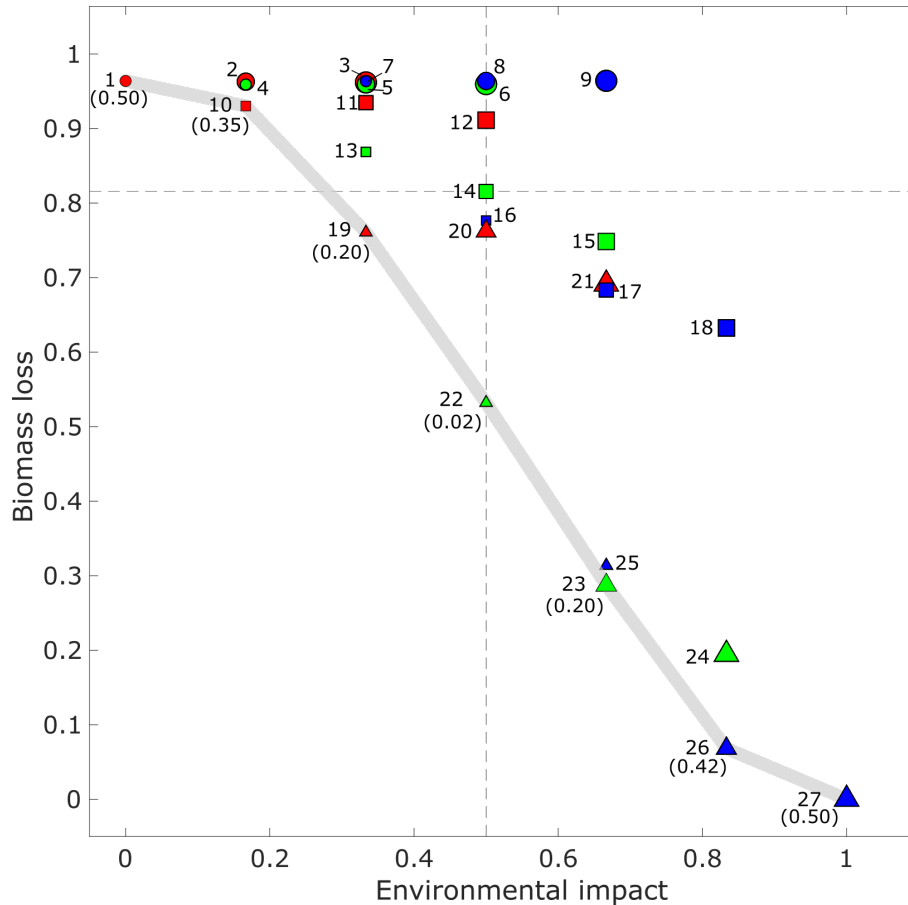


Figure 2.2: Biomass loss and environmental impact for different management scenarios characterized by different values of fertilization (σ_N) (circle = low, square = intermediate, triangle = high); irrigation (κ) (red = low, green = intermediate, blue = high); and pesticides (μ_p) (small = low, intermediate = intermediate, big = high). The grey line represents the Pareto front. Scenario 14 is the baseline scenario, considering an intermediate level of fertilization, irrigation and pesticides applications. Dashed lines identify four group scenarios: win-win (bottom-left), win-lose (bottom-right), lose-lose (top-right), lose-win (top-left). The Gini coefficient for the optimal scenarios is indicated between parentheses.

Table 2.1: Summary of the scenarios characterized by different levels (low = L, intermediate = I, high = H) of fertilization (L: $\sigma_N = 0.0012 \text{ d}^{-1}$, I: $\sigma_N = 0.012 \text{ d}^{-1}$, H: $\sigma_N = 0.12 \text{ d}^{-1}$), irrigation (L: $\kappa = 18 \text{ d}^{-1}$, I: $\kappa = 182 \text{ d}^{-1}$, H: $\kappa = 1820 \text{ d}^{-1}$) and pesticides (L: $\mu_p = 0 \text{ d}^{-1}$, I: $\mu_p = 0.0625 \text{ d}^{-1}$, H: $\mu_p = 0.125 \text{ d}^{-1}$), of the estimated biomass loss (BL) and environmental impact (EI).

Scenario	Fertilization	Irrigation	Pesticide	BL	EC
1	L	L	L	0.964	0.000
2	L	L	I	0.963	0.167
3	L	L	H	0.962	0.333
4	L	I	L	0.959	0.167
5	L	I	I	0.959	0.333
6	L	I	H	0.960	0.500
7	L	H	L	0.964	0.333
8	L	H	I	0.964	0.500
9	L	H	H	0.964	0.667
10	I	L	L	0.930	0.167
11	I	L	I	0.935	0.333
12	I	L	H	0.911	0.500
13	I	I	L	0.869	0.333
14	I	I	I	0.815	0.500
15	I	I	H	0.748	0.667
16	I	H	L	0.777	0.500
17	I	H	I	0.683	0.667
18	I	H	H	0.632	0.833
19	H	L	L	0.761	0.333
20	H	L	I	0.776	0.500
21	H	L	H	0.691	0.667
22	H	I	L	0.532	0.500
23	H	I	I	0.287	0.667
24	H	I	H	0.194	0.833
25	H	H	L	0.314	0.667
26	H	H	I	0.068	0.833
27	H	H	H	0.000	1.000

that complex and sometimes unexpected interactions between agricultural practices, plant growth and aphid population dynamics can be disentangled. For example, our model assumes that fertilization has a double role: it fosters

both plant growth (Robertson and Vitousek, 2009) and the production of induced defences, which eventually reduce aphid pressure on the plant (Zust and Agrawal, 2016). This mechanism likely explains why the scenarios with the highest plant biomass production all consider an high fertilization level. Moreover, our model assumes that both nitrogen and carbon are essential to plant to produce new biomass. This likely explains why, increasing 10 times nitrogen assimilation rate, without varying carbon assimilation rate, does not result in a proportionate increase in the production of new plant biomass: for example by increasing scenario 18 fertilization level we obtained an increase in biomass production of 2.7 times. The weakness of our model, however, is that the obtained results should be interpreted as qualitative pattern that cannot immediately be transferred to agronomic practices. In fact, to be applicable to agriculture, one should define the functional relationships which link model parameters to effective practices. Models exists that linked soil nitrate content to nitrogen uptake by crop (see e.g. Cárdenas-Navarro et al. (1999); Devienne-Barret et al. (2000)): further works should investigate how this type of model can be adapted to be used in mechanistic plant models to reproduce different fertilization treatments.

We show that embedded plant growth simulation modelling with MCDA has the potential to identify optimal management scenarios and evaluate trade-offs between production and environmental objectives. The methodology we use to compute the environmental impact associated to agricultural practices is suitable for general plant growth model as ours, which does not explicitly reproduce the effect of agronomic practices on the environment. Yet, our methodological framework could be expanded and adapted for more

complex models which explicitly simulate the effect of agricultural practices on the environment: for example STICS model (Brisson et al., 1998) allows to compute water and nitrogen leaching associated to a management scenario.

Our work show a trade-off between plant biomass loss and environmental impact of the considered management scenarios. Such trade-off is a common finding in both empirical (Jiang et al., 2013; Raudsepp-Hearne et al., 2010; Tilman et al., 2002) and simulation (Goldstein et al., 2012; Nelson et al., 2009; Kirchner et al., 2015) works. Pareto-optimal scenarios are mostly characterized by high fertilization level. As highlight before, this is due to the fact that in the model fertilization sustains both plant growth and the production of induced defences. The environmental impacts linked to fertilization are mainly due to the fact that crops take up only about 30%-50% of nitrogen fertilization applied leaving most of the remainder available for loss by volatilization and leaching (Tilman et al., 2002; Malik and Dechmi, 2020). Agricultural practices that allow to increase nitrogen use efficiency, i.e. the fraction of applied nitrogen that is absorbed and used by the plant (σ_N in the model), represents a promising solution to increase plant biomass production and reduce the environmental impact of fertilization (Tilman et al., 2002; Malik and Dechmi, 2020). For example, fertilize applications during periods of greatest crop demand, in close proximity of plant roots, and in smaller and more frequent doses have the potential to improve nitrogen use efficiency, reducing fertilizer losses while maintaining or improving yields and quality (Tilman et al., 2002; Cui et al., 2008; Xiao et al., 2019).

The majority of Pareto optimal scenarios is characterized by no pesticide addition. This is likely due to the fact that aphid's feeding effect on plant

biomass production is modest respect to those of other pest (e.g. defoliators) (Van Emden and Harrington, 2007). Yet, optimal scenarios considering no pesticide are characterized by medium to high plant biomass loss, thus producers may be reluctant to implement this prescription fearing for economical losses. Nevertheless, as the willingness to pay of the consumers might increase for products issued from sustainable agronomic practices that minimize the use of pesticide, differences in economical return per *ha* between the organic alternative and conventional farming may be small (Pimentel and Burgess, 2005). In the event that producers are more oriented towards scenarios minimizing biomass loss, pesticide addition is necessary. In this case, agricultural practices such as biological control have the potential to limit pest outbreaks acting on aphids mortality (μ_p) through predation or parasitism (Murdoch et al., 1985), and to eliminate the environmental impact due to pesticide.

Regular deficit irrigation (RDI) is a key water-saving technology in agriculture, which has been widely applied in field crops (e.g. wheat, maize, cotton, rice, soybean, sunflower) and woody plant species (e.g. grapevine, nectarine, olive trees, apple) with the aim of reducing water application without decreasing the yield (see Chai et al. (2016) and citations therein). This kind of practice allows to increase plant biomass production and reduce the environmental impact due to irrigation. Moreover RDI has the potential to control pest pressure (see e.g. Costello (2008); Mercier et al. (2008); Rouselin et al. (2016); Bevacqua et al. (2019)) which, if accompanied by a lower pesticide application, may further increase plant biomass production and reduce environmental impact. Yet, timing and the extent to which RDI is applied plays a critical role in determining yield production and pest control

(Chai et al., 2016).

While the methodology we present is flexible enough to apply in many contexts, we should point out several considerations in interpreting the results of this analysis. First, we use a plant-pest model general enough to be applied to different plant-pest system. We calibrate it for young peach tree without fruits to compute the plant biomass loss objective (see Zaffaroni et al. (2020)). The assumption we make to use shoot production as a proxy for relevant crop is generally true for annual plants, while for perennial plants is true if we consider the average production in the whole lifespan of plants, because they requiring a number of growth cycles before fruit is produced. Another consideration involves the assumption of independent effects of fertilization and irrigation on plant growth: by contrast, in drought stress condition N uptake by root may be restricted (McDonald and Davies, 1996). A water model (e.g. that presented in Thornley (1996)), describing the water movement from the soil to the shoot can be incorporate to our plant-pest model to explicitly take into account the interaction between nitrogen uptake and soil water content. Yet, despite the simplifying assumptions outlined above, our work offers new insights for sustainable crop management, demonstrating the utility of analytical approaches that combine plant simulation modelling with MCDA. Furthermore, the framework we propose can be readily modified to consider different plant-pest system, to include others agricultural practices and to incorporate alternative plant models.

Supplementary Information

- **B.1** Model processes and parameters

Chapter 3

The role of vectors interference in a shared host-multi vector system

Authors

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Résumé

Les pucerons sont le principal vecteur des virus à transmission non persistante (TNP) des plantes. La plupart des études existantes se sont concentrées sur les pucerons migrants en tant que vecteurs des virus TNP, car ils peuvent sonder de nombreuses plantes avant de quitter une zone, et éventuellement acquérir et/ou inoculer des particules virales. Cependant, les pucerons colonisateurs jouent un rôle car ils peuvent *i)* transmettre les virus TNP et *ii)* interférer avec la présence des pucerons migrants sur le terrain, à la fois directement et indirectement (par l'intermédiaire des plantes). Au travers d'un modèle épidémiologique, nous montrons que l'interférence interspécifiques, en réduisant le nombre de plantes visitées et la durée de la permanence des pucerons migrants sur le terrain, conditionne la transmission des virus TNP. Nos résultats montrent que la fertilisation peut diminuer la propagation des virus TNP dans les champs, car l'augmentation de la population de pucerons colonisateurs augmente l'interférence envers les pucerons migrants. De plus, nous montrons que les pesticides, en réduisant la population de pucerons colonisateurs, peuvent affaiblir l'interférence envers les pucerons migrants, favorisant ainsi la propagation des virus TNP. Nos résultats suggèrent qu'une meilleure compréhension de la manière dont les vecteurs interagissent les uns avec les autres peut améliorer notre compréhension de l'écologie des maladies, favorisant ainsi le développement de stratégies de gestion des maladies.

Mots-clés: agent pathogène transmis par les pucerons, virus à transmission non persistante, modèle épidémiologique, interférence inter-spécifique

Abstract

Aphids are the principal vector of plants non persistent transmitted (NPT) viruses. Most existing studies focused on migrant aphids as NPT viruses vectors because they can probe many plants before leaving an area, possibly acquiring and/or inoculating virus particles. However, colonizer aphids play a role as they can *i*) transmit NPT viruses and *ii*) interfere with migrant aphids' presence in the field, both directly and indirectly (plant-mediated). By mean of an epidemiological model, we show that inter-specific interference, reducing the number of plants visited and the duration of the permanence of migrant aphids in the field, shapes the transmission of NPT viruses. Our results show that fertilization may decrease the spread of NPT viruses in the field, because increasing colonizer aphid population increases plant-mediated interference towards migrant aphids. We show that pesticide, reducing colonizer aphid population, may weaken the interference towards migrant aphids, favoring the spread of NPT viruses. Our results suggest that a better understanding of how vectors interact each other may enhance our understanding of disease ecology, supporting the development of disease management strategies.

Keywords: aphid-borne pathogen; non persistent transmitted virus; epidemiological model; inter-specific interference

Introduction

Aphid species are responsible for the transmission of nearly 30% of known plant virus species (Brault et al., 2010). Aphids spread the majority of viruses through nonpersistent transmission (NPT), a form of vectoring in which virus particles (virions) attach loosely to the insect's stylets (Brault et al., 2010). Virions are rapidly acquired from infected plants, retained by their vectors for less than a few hours and inoculated to healthy plants during plant sampling probes, which is a mechanism aphids use to recognize the plant they landed on (Nault, 1997; Andret-Link and Fuchs, 2005; Ng and Falk, 2006). NPT viruses are responsible for severe damage to cereal, vegetable, fruit, and floral crops (Andret-Link and Fuchs, 2005). For instance, the *Plum pox virus* (PPV), which is spread worldwide by more than 20 aphid species, is responsible for sharka, the most devastating disease of stone fruit trees (Cambra et al., 2006; Rimbaud et al., 2015a). Another example is the *Potato virus Y* (PVY), which is spread by more than 50 aphid species, threatens the production of a range of solanaceous crops, including potato, tomato, tobacco, and pepper (Radcliffe and Ragsdale, 2002; MacKenzie et al., 2013).

The epidemiology of NPT viruses is closely related to the behaviour of aphid vectors, in particular to *i*) aphids' ability to acquire and inoculate the virus during sampling probes and *ii*) their propensity for moving among plants (Ng and Perry, 2004). According to their patterns of mobility and preferred host species, aphid species can be classified as: "colonizers", which under favourable conditions, spend most of their life on the same host plant, or "migrants", which land and probe numerous plant individuals and species

during their life (Van Emden and Harrington, 2007; Fereres and Moreno, 2009). Most studies on the epidemiology of NPT viruses focus on migrant aphids because they probe many plants without colonizing them, possibly acquiring and/or inoculating virions over large spatial areas (Powell et al., 2006). Moreover, pesticides are inefficient to control viral transmission by migrant vectors, as these aphids spend only few minutes on treated plants (Perring et al., 1999). However, colonizer aphids can also efficiently transmit NPT viruses when they are induced to change their host, for example in response to crowding or to change in plant nutrient contents (Müller et al., 2001). For instance, the green peach aphid *Myzus persicae*, despite colonizing and reproducing on peach *Prunus persica* and potato *Solanum tuberosum* plants (Van Emden and Harrington, 2007; Robert et al., 2000), was the most efficient vector at laboratory conditions of PPV and PVY (Rimbaud et al., 2015b,a; Robert et al., 2000).

In addition, the presence of colonizer aphids may affect migrant aphids' movement between plants (Kaplan and Denno, 2007; Bird et al., 2019; Chisholm et al., 2019). For example, Mehrparvar et al. (2014) studied the influence of aphid presence on a plant on the choice of other aphid species to visit that plant. Their experiment was conducted with three species of aphids specialized on tansy (*Tanacetum vulgare* L.). They showed that *Macrosiphoniella tanacetaria* rarely chooses a plant occupied by *Metopeurum fuscoviride* and, in turn, *M. fuscoviride* rarely chooses a plant occupied by *Uroleucon tanaceti*. Different kinds of interferences might characterize interactions between colonizer and migrant aphid species. Colonizer aphids can *i)* directly interfere through the production of pheromones that can have a repelling effect to-

wards other aphid species (Van Emden and Harrington, 2007); *ii*) indirectly induce the host plant to produce volatile compounds as a defensive mechanism, which may lower plant attractiveness to other aphid species (Zust and Agrawal, 2016). Therefore, such interference mechanisms are likely to reduce the number of plants visited by migrant aphids in a given area and increase their propensity for leaving the area (Levins and Culver, 1971; Nee and May, 1992), possibly interfering with the spread of NPT viruses by migrant aphids.

As far as we know, the role of colonizer aphids in the spread of NPT viruses has never been explored. Understanding NPT viruses spread is complex because experimentation is costly and difficult: symptoms may be difficult to detect and experimental trials in the vicinity of susceptible commercial crops may be restricted (Cunniffe et al., 2014; Picard et al., 2017). Mathematical models are thus particularly useful to provide complementary insights on virus spread at a scale matching that of typical epidemics (Picard et al., 2017), and to design and test management strategies, while circumventing the difficulties associated with experiments (Fabre et al., 2012; Cunniffe et al., 2014; Rimbaud et al., 2015b; Cunniffe et al., 2015b; Hilker et al., 2017). Numerous models have been developed to study the role of vector population dynamics and vector-host-pathogens interactions on the spread of NPT viruses (e.g. Sisterson and Stenger (2016); Shaw et al. (2017); Jeger et al. (2018); Allen et al. (2019); Donnelly et al. (2019); Crowder et al. (2019)). Shaw et al. (2017) developed a conceptual model to assess the contributions of vector life history (e.g. growth rates, fecundity, and longevity) and behavior (e.g. vector preferences for settling and feeding) to pathogen spread. Crowder et al. (2019) developed a model where the vector vital traits (e.g.

fecundity, mortality, host encounter rates) were varied to explore the effect of species interactions (e.g. predation, competition and mutualism between a vector and a non vector species) on vector fitness and behavior and on the spread of plant pathogens. However, in these studies, the authors consider only a single vector species, which limits the possibility to assess the effects of interference between two or more vector species.

In the present work, we develop a general epidemiological model which describes the temporal variation of the number of susceptible and infected plants, and of the number of non viruliferous and viruliferous colonizer and migrant aphids. We apply the model to explore the role of inter-specific interference upon colonizer and migrant aphid behaviour and the resulting effects on the invasion, persistence and control of NPT viruses. We use the model to analyze the effects of common agricultural practices, such as fertilization, pesticide application and roguing, upon the spread of NPT viruses. We apply the model to a general pathosystem composed by a NPT virus vectored by a colonizer and a migrant aphid species. Examples of such a pathosystem are: *Plum pox virus*, transmitted to peach trees by the colonizer *Myzus persicae* and the migrant *Aphis spiraecola* (Rimbaud et al., 2015b); *Zucchini yellow mosaic virus*, transmitted to cucurbit crops by the colonizer *Aphis gossypii* and the migrant *Aphis craccivora* (Lecoq and Desbiez, 2012); *Potato virus Y*, transmitted to potato plants by the colonizer *Myzus persicae* and the migrant *Sitobion avenae* (Robert et al., 2000; Fox et al., 2017).

Methods

Model outlines and assumption

The model, which describes the temporal variation of the number of plants, colonizer aphids and migrant aphids, is schematically represented in figure 3.1.

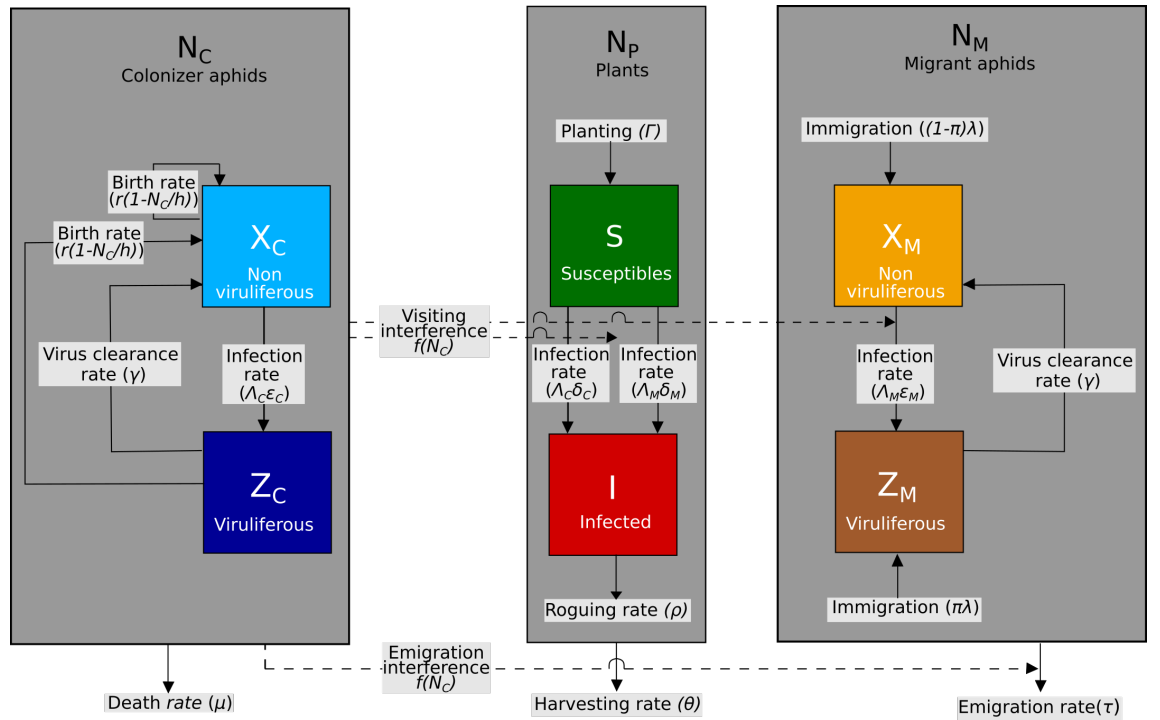


Figure 3.1: Schematic representation of the single host-multi vectors model, where the host plant is partitioned into susceptible (S) and infected (I) individuals. Aphids are partitioned into non viruliferous (X_i) and viruliferous (Z_i), and are classified as colonizer ($i = C$) or migrant ($i = M$). Dashed lines represent the effects of interference exerted by colonizer towards migrant aphids. Details on the involved processes are given in the main text.

The plant population (ind. ha^{-1}) is structured into two compartments: susceptible (S) and infected (I). Aphid population (ind. plant^{-1}) is struc-

tured into colonizers (C) and migrants (M), which in turn, are structured into non viruliferous (X_C and X_M) and viruliferous (Z_C and Z_M). The total number of plants per hectare is N_P ($N_P = S + I$), the average number of colonizer aphids per plant is N_C ($N_C = X_C + Z_C$) and the average number of migrant aphids visiting a plant per unit time is N_M ($N_M = X_M + Z_M$). A susceptible plant can be infected if it enters in contact with viruliferous colonizer (Z_C) or migrant (Z_M) aphids. The probability per unit time that a viruliferous aphid infects a susceptible plant depends on *i*) the number of plants visited per unit time (Λ_i , $i = C, M$, $\Lambda_M > \Lambda_C$ due to the different aphid moving behaviour), *ii*) the probability of inoculating the virus to the plant per visit (δ_i , $i = C, M$), and *iii*) the probability that the visited plant is susceptible ($\frac{S}{N_P}$). Therefore the number of infected plants per unit time exerted by viruriferous aphids at the plant population scale is $\sum_{i=C,M} \Lambda_i \delta_i \frac{S}{N_P} Z_i N_P$. We assumed that infected plants are removed with roguing rate (ρ) and that both susceptible and infected plant are harvested at a rate θ . Furthermore, Γ new susceptible plants are planted per unit time.

A non viruliferous aphid can become viruliferous if it enters in contact with an infected plant. The probability per unit time that a non viruliferous aphid acquires the virus from an infected plant depends on *i*) the number of plants visited per unit time (Λ_i , $i = C, M$), *ii*) the probability of acquiring the virus per visit (ϵ_i , $i = C, M$) and *iii*) the probability that the visited plant is infected ($\frac{I}{N_P}$). Viruliferous aphids lose viruliferousness at a rate γ . We assume that the population of colonizer aphids varies following a logistic function with a density dependent growth rate $r(1 - \frac{N_C}{h})$, where r is the intrinsic growth rate and h is the plant hosting capacity, and a constant

aphid mortality rate μ (Donnelly et al., 2019; Crowder et al., 2019). We assume that an average of λ migrant aphids per plant immigrate into the system (i.e. 1 ha patch) per unit time and a fraction π of immigrant aphids can be viruliferous. Such a parameter depends on the disease prevalence in the surrounding area. Migrant aphids emigrate from the system at a rate τ , which is the inverse of their average sojourn time in the system in absence of colonizer aphids. We assume that such an average sojourn time can be reduced in the presence of colonizer aphids.

Model equations

The model outlined above can be described by the following set of differential equations:

$$\left\{ \begin{array}{l} \dot{S} = \Gamma - (\Lambda_C \delta_C Z_C N_P + \Lambda_M \delta_M f(N_C) Z_M N_P) \left(\frac{S}{N_P}\right) - \theta S \\ \dot{I} = (\Lambda_C \delta_C Z_C N_P + \Lambda_M \delta_M f(N_C) Z_M N_P) \left(\frac{S}{N_P}\right) - (\rho + \theta) I \\ \dot{X}_C = r N_C \left(1 - \frac{N_C}{h}\right) - \mu X_C - \Lambda_C \epsilon_C \frac{I}{N_P} X_C + \gamma Z_C \\ \dot{Z}_C = \Lambda_C \epsilon_C \frac{I}{N_P} X_C - (\gamma + \mu) Z_C \\ \dot{X}_M = (1 - \pi) \lambda - \Lambda_M \epsilon_M f(N_C) \frac{I}{N_P} X_M + \gamma Z_M - \tau g(N_C) X_M \\ \dot{Z}_M = \pi \lambda + \Lambda_M \epsilon_M f(N_C) \frac{I}{N_P} X_M - \gamma Z_M - \tau g(N_C) Z_M \end{array} \right. \quad (3.1)$$

Where the dot represents the derivative with respect to time t , and parameters are as in Table 3.1. Functions $f(N_C)$ and $g(N_C)$ represent, respectively, the effects of “visiting interference” and of “emigration interference” exerted by colonizer aphids towards migrant aphids, details on their functional forms

are given in the following section.

The set of differential equations can be used to represent a plant virus epidemic under very general circumstances. To gain insight on disease transmission, we assume that: *i*) the total number of plants (N_P) is constant, which implies that the number Γ of new susceptible plant per unit time is given by

$$\Gamma = \theta S + (\rho + \theta)I \quad (3.2)$$

where θ is plant harvesting rate and ρ is infected plant roguing rate;

ii) the average number of migrant aphids (M) per plant in absence of colonizer aphids is given by the average number λ of migrant aphids immigrating into the system per plant per unit time, multiplied by the average sojourn time ($\frac{1}{\tau}$) of a migrant in the system in absence of colonizer aphids

$$M = \frac{\lambda}{\tau} \quad (3.3)$$

Modelling interference between colonizer and migrant aphids

We assume that interference exerted by coloniser towards migrant aphids can independently induce them to: *i*) visit less plants per unit time; and/or *ii*) reduce the average resident time in the system. To account for plants becoming less attractive to migrant aphids when coloniser aphids are present, we introduced the “visiting interference” via a functional form $f(N_C; \alpha_1, V_1, \beta_1, h)$ controlling the proportionate decrease in the rate at which migrant aphids

visit plants, with

$$f(N_C) = \frac{1}{1 + \left(V_1 \frac{N_C}{h^{\beta_1}}\right)^{\alpha_1}} \quad (3.4)$$

This is a generalisation of the competition function proposed by Bellows (1981), extending in continuous time the model of Smith and Slatkin (1972). It is sufficiently flexible to account for a range of possible types of interference (figure 3.2). The “curvature” parameter α_1 allows for responses in which the strength of competition accelerates ($\alpha_1 > 1$) or decelerates ($0 < \alpha_1 \leq 1$) with coloniser density. The “strength” parameter V_1 controls the magnitude of interference, and so the density of colonisers that is required to appreciably affect the behaviour of migrant aphids. The number of colonizer N_C is scaled by h^{β_1} , where h is the aphids hosting capacity and the parameter $\beta_1 \in [0, 1]$ allows to represent different interference mechanisms. When $\beta_1 = 1$ the function $f(N_C)$ represents direct interference (e.g. competition for space, (Van Emden and Harrington, 2007)) and its value decreases with the density of colonizer on the plant ($\frac{N_C}{h}$): this implies that, at the same aphid abundance (N_C), the exerted interference is weaker on a bigger plant (with higher h). When $\beta_1 = 0$, the function $f(N_C)$ represents indirect interference (e.g. release of plant volatiles, (Zust and Agrawal, 2016)) and its value decreases with the absolute number of colonizer aphids. Values of $0 < \beta_1 < 1$ correspond to forms of interference intermediate between these two extremes.

To assure that the value of parameter V_1 in equation (3.4) is biologically relevant when the scaling parameter β_1 changes, we assumed that there is a “reference” value of aphids hosting capacity ($h = h_R$) for which - all other things being equal (i.e. the values of α_1 and N_C) - the value of the inter-

ference function is the same independently from the underline interference mechanism (i.e. the value of β_1). This implies that the strength parameter V_1 can be rewritten as conditioned on the value of β_1 (i.e. $V_1 = \frac{\nu_1}{h_R^{1-\beta_1}}$, further details in *Appendix C*) and the interference function can be reformulated as:

$$f(N_C) = \frac{1}{1 + \left(\frac{\nu_1}{h_R^{1-\beta_1}} \frac{N_C}{h^{\beta_1}} \right)^{\alpha_1}} \quad (3.5)$$

The functional form selected for $f(N_C)$ ensures that there is no interference when there are no coloniser aphids (i.e. $f(0) = 1$ for all $\alpha_1, \nu_1, \beta_1, h$ and h_R), meaning that migrant aphids visit plants at the underlying rate Λ_M .

Similarly, we introduced the ‘‘emigration interference’’ $g(N_C, \alpha_2, \nu_2, \beta_2, h, h_R)$, which is the proportionate increase in the rate at which migrant aphids emigrate out of the system when the population of coloniser aphids is N_C , and where:

$$g(N_C) = 1 + \left(\frac{\nu_2}{h_R^{1-\beta_2}} \frac{N_C}{h^{\beta_2}} \right)^{\alpha_2} \quad (3.6)$$

The emigration interference function is parameterised via curvature, strength and scaling parameters (α_2, ν_2 and β_2), which have identical interpretations to those controlling the visiting interference. The functional form selected for $g(N_C)$ ensures that there is no interference when there are no coloniser aphids (i.e. $g(0) = 1$ for all $\alpha_2, \nu_2, \beta_2, h$ and h_R), meaning that migrant aphids leave the system at the underpinning aphid emigration rate τ . In the most general form of our model, the analogous parameters for the two functions are entirely independent (e.g. $\alpha_1 \neq \alpha_2$), which provides sufficient flexibility to allow inter-specific interference to affect visiting and/or emigration independently.

However in all numerical work in this paper we assumed that $\alpha_1 = \alpha_2 = \alpha$, $\nu_1 = \nu_2 = \nu$ and $\beta_1 = \beta_2 = \beta$, and so, in turn, that proportionate effects of the density of colonizer aphids on visiting and emigration rates were similar.

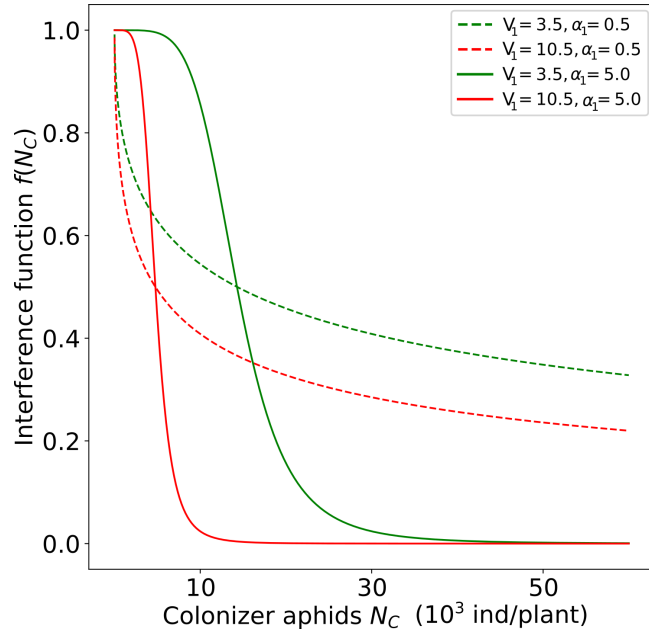


Figure 3.2: Proportionate decrease of the number of plants visited by a migrant aphid in the unit of time as a function of the abundance of colonizer aphids per plant, for two values of parameter V_1 and α_1 , in a plant displaying a reference aphid hosting capacity (i.e. $h = h_R$).

Table 3.1: Model state variables and parameters.

Sources: (1) Jeger et al. (1998); (2) Jeger et al. (2004); (3) Jeger et al. (2008); (4) Holt et al. (1997); (5) Madden et al. (2000); (6) Rimbaud et al. (2019); (7) Roussel et al. (2016); (8) Jevremović et al. (2016); (9) Arbitrarily fixed in present work

Variable	Description	Dimensions	Values	Source
S	Susceptible plants	ind. ha ⁻¹		(9)
I	Infected plants	ind. ha ⁻¹		(9)
X_C	Non viruliferous colonizer aphids per plant	ind. plant ⁻¹		(9)
Z_C	Viruliferous colonizer aphids per plant	ind. plant ⁻¹		(9)
X_M	Non viruliferous migrant aphids per plant	ind. plant ⁻¹		(9)
Z_M	Viruliferous migrant aphids per plant	ind. plant ⁻¹		(9)
Parameter	Description	Dimensions	Values	Source
Γ	Number of new susceptible plants planted	ind. day ⁻¹	$(\rho + \theta)I$	(9)
Λ_C	Number of plant visited by a colonizer aphid	ind. day ⁻¹	0.05	(9)
Λ_M	Number of plant visited by a migrant aphid	ind. day ⁻¹	8.5	(9)
δ_C	Virus inoculation rate from colonizer aphid	ind. ⁻¹	0.04	(1)
δ_M	Virus inoculation rate from migrant aphid	ind. ⁻¹	0.04	(1)
ϵ_C	Virus acquisition rate by colonizer aphid	ind. ⁻¹	0.02	(1)
ϵ_M	Virus acquisition rate by migrant aphid	ind. ⁻¹	0.02	(1)
α_1	Visiting interference curvature	dimensionless	1.00	(9)
ν_1	Visiting interference strength (for direct interference)	dimensionless	3.5-10.5	(9)
β_1	Visiting interference scaling	dimensionless	[0,1]	(9)
α_2	Emigration interference curvature	dimensionless	1.00	(9)
ν_2	Emigration interference strength (for direct interference)	dimensionless	3.5-10.5	(9)
β_2	Emigration interference scaling	dimensionless	[0,1]	(9)
ρ	Infected plant roguing rate	day ⁻¹	0.02	(2,3)
θ	Plant harvesting rate	day ⁻¹	0.003	(4)
r	Intrinsic rate of growth of colonizer aphids	day ⁻¹	0.21	(4)
h	Aphids hosting capacity	ind. plant ⁻¹	50,000	(7)
h_R	Reference aphids hosting capacity	ind. plant ⁻¹	50,000	(9)
μ	Mortality rate of colonizer aphids	day ⁻¹	0.08	(9)
γ	Virus clearance rate in aphid vectors	day ⁻¹	4	(1)
λ	Average number of migrant aphids per plant	ind. plant ⁻¹ day ⁻¹	τM	(9)
π	Fraction of viruliferous immigrant aphids	dimensionless	0	(9)
τ	Migrant aphids emigration rate in absence of colonizer aphids	day ⁻¹	0.7	(2,5)
N_P	Total number of plants	ind. ha ⁻¹	720	(6)
M	Average number of migrant aphids per plant in absence of colonizer aphids	ind. plant ⁻¹	500	(8)

Agricultural practices and disease control

The effect of a number of agricultural practices commonly used to *i)* increase plant growth, *ii)* control aphid population and *iii)* mitigate the effect of plant diseases can be taken into account by modifying the values of some model parameters. Practices such as fertilization, commonly used to foster crop growth, also increase the abundance of colonizer aphids dwelling on the fertilized plants (Huberty and Denno, 2006; Tamburini et al., 2018) and in our model this translates into an increase of the parameter h (while parameter h_R is not varied). Pesticides are commonly used in crops to reduce the number of coloniser aphids and in our model this translates into an increase colonizer aphid mortality μ , while the dynamics of migrant aphids is not affected (Perring et al., 1999). In case of spread of plant diseases, frequently producers try to identify as soon as possible the infected plants and replace them with new healthy plants (Sisterson and Stenger, 2013). Such a practice is modelled by the roguing rate ρ , which is defined as the inverse of the average time an infected plant remains in the system before being identified and removed ($\rho = \frac{2}{\Delta}$, where Δ is the average surveillance time) (Cunniffe et al., 2014).

Methods of analysis

We first determine the reproduction number of the pathogen, R_0 , and the system equilibria. Then, we explore the responses of R_0 and of the density of infected plant at the equilibrium, \bar{I} , to variations of the aforementioned agricultural practices (modeled through parameters h , μ and ρ) under four com-

binations of interference type scenarios (indirect ($\beta = 0$) vs. direct ($\beta = 1$)) and of interference strength scenarios (weak ($\nu = 3.5$) vs. strong ($\nu = 10.5$)). In particular, we vary one parameter at a time and we explore the effectiveness of the respectively agricultural practices in controlling and eradicating the disease. Finally, we analyse the effectiveness of couples of agricultural practices in controlling and eradicating the disease by exploring the response of R_0 to simultaneous variation of two parameters at a time. In our analysis, we use biologically plausible parameter values and ranges to reflect a broad range of single host - multi vector system rather than restricting the analysis to a specific system.

Results

Basic reproduction number: definition and response to agricultural practices

The basic reproduction number of the disease is computed assuming that only non viruliferous migrant aphids immigrate into the system ($\pi = 0$) and is expressed as:

$$R_0 = \frac{1}{\rho + \theta} \left(\frac{\Lambda_C^2 \delta_C \epsilon_C \bar{N}_C}{\gamma + \mu} + \frac{\Lambda_M^2 \delta_M \epsilon_M f(\bar{N}_C)^2 \bar{N}_M}{\gamma + \tau g(\bar{N}_J null C)} \right) \quad (3.7)$$

where \bar{N}_C and \bar{N}_M are, respectively, the size of the colonizer and migrant aphid populations at the infection-free steady state. The term $(\rho + \theta)$ is the inverse of the mean time that an infected plant stays in the system before being removed, $(\gamma + \mu)$ is the inverse of the mean time that a colonizer

aphid carries the virus before losing the ability to transmit it or dying, and $(\gamma + \tau g(\bar{N}_C))$ is the inverse of the mean time that a migrant aphid carries the virus before losing the ability to transmit it or emigrating from the system. The terms $\Lambda_C \delta_C$ and $\Lambda_M \delta_M f(\bar{N}_C)$ are the rates at which a susceptible plant is infected by a colonizer or a migrant aphid, respectively. The terms $\Lambda_C \epsilon_C$ and $\Lambda_M \epsilon_M f(\bar{N}_C)$ are the rates at which a colonizer or a migrant aphid acquires the virus from an infected plant, respectively. The basic reproduction number can be written as the sum of two components: one considering virus transmission by colonizer aphids $\left(R_0^C = \frac{\Lambda_C^2 \delta_C \epsilon_C \bar{N}_C}{(\rho + \theta)(\gamma + \mu)}\right)$ and one considering virus transmission by migrant aphids $\left(R_0^M = \frac{\Lambda_M^2 \delta_M \epsilon_M f(\bar{N}_C)^2 \bar{N}_M}{(\rho + \theta)(\gamma + \tau g(\bar{N}_C))}\right)$, which is typical for plant disease models with multiple routes of transmission (Jeger et al., 2009; Cunniffe and Gilligan, 2010; Cunniffe et al., 2012; Hilker et al., 2017).

When $\mu < r$, the size of colonizer and migrant aphid populations at the infection-free steady state is $\bar{N}_C = h(1 - \frac{\mu}{r}) > 0$ and $\bar{N}_M = \frac{M}{g(\bar{N}_C)}$, respectively (see Appendix C for further details). The value of the visiting interference function is given by

$$f(\bar{N}_C = h(1 - \mu/r)) = \frac{1}{1 + \left(\frac{\nu}{h_R^{1-\beta}} \frac{h(1 - \mu/r)}{h^\beta}\right)^\alpha} \quad (3.8)$$

It follows that when $\beta = 1$ (e.g. interference for space), the value of $f(\bar{N}_C)$ does not depend on the aphid hosting capacity h of the plant, while when $\beta = 0$ (e.g. emission of plant defensive volatiles) the value of $f(\bar{N}_C)$ decreases with h . Moreover, when h is not varied, we set the value of h to its reference

value ($h = h_R$), consequently, the value of $f(\bar{N}_C)$ is not affected by the value of β . Similar consideration can be done for the emigration interference $g(\bar{N}_C)$.

When the interference between colonizer and migrant aphids is indirect ($\beta = 0$), the response of the value of R_0 to changes in h is non-monotonic (figure 3.3A) and depends upon the balance between the effects of parameter changes on the R_0 's components. Progressively increasing h , initially reduces R_0 because the interference exerted by colonizer towards migrant aphids increases, reducing their contribution in spreading the disease (R_0^M decreases). Yet, if fertilization increases the population of colonizer aphids above the population abundance needed to control the spread of the virus by migrant aphids (i.e. to reduce R_0^M below 1), the surplus of colonizer aphids does not contribute to inter-specific interference, but only contributes to increase the spread of NPT virus (R_0^C , and consequently R_0 increase) (3.3A). By contrast, when the interference between colonizer and migrant aphids is direct ($\beta = 1$), the response of the value of R_0 to changes in colonizer aphid hosting capacity (h) is monotonic (figure 3.3B): increasing h increases the spread of the NPT virus (R_0 increases). Indeed, increasing fertilization increases the abundance of colonizer aphids, thus their contribution in spreading of the virus (R_0^C increase). Yet the contribution of migrant aphids in spreading the virus is constant (R_0^M constant). This is due to the fact that increasing fertilization increases both plant size and colonizer aphid hosting capacity, keeping constant the colonizer aphid density on the plant, and, consequently, the interference exerted towards migrant aphids ($f(\bar{N}_C)$). The greater the increase of colonizer aphid mortality (μ), the greater the reduction of the col-

onizer aphid population size: on the one hand, this reduces the spread of the virus by colonizer aphids (R_0^C decreases), on the other hand this reduces the interference exerted by colonizer aphids, favouring the spread of the virus by migrant aphids (R_0^M increases). In the case of weak interference, the increase of μ decreases more the interference towards the migrant aphids than the spread of the virus by colonizer aphids, resulting in monotonically increases R_0 (figure 3.3C). In the case of strong interference, small increase of colonizer aphid mortality ($0.04 < \mu < 0.06$) reduces more the spread of virus by colonizers than the interference exerted toward migrant aphids, resulting in decreasing R_0 (figure 3.3C.1). Instead, for $\mu > 0.06$ the reduction of the interference is higher than the reduction of the spread of the virus by colonizer aphids, resulting in increasing the spread by migrant aphids and R_0 (figure 3.3C.1). To investigate if these patterns are general or due to the selected parameters, we generated 100,000 random parameters sets and, for each of them, we explored the response of R_0 to variation of μ (see *Appendix C* for further details). In the case of weak interference, R_0 increases monotonically with μ in 44% of the cases, while in 56% of the case the response is non monotonic. In the case of strong competition, R_0 increases monotonically with μ in 0.01% of the cases, while in 0.99% of the case the response is non monotonic. The response of the value of R_0 to changes in the surveillance interval ($2/\rho$) is predictably monotonic (figure 3.3D): the more effort is put into this control measure (i. e. the smaller the surveillance interval gets), the greater the effect in reducing the spread of virus by both colonizer (R_0^C decreases) and migrant aphids (R_0^M decreases). As long as $h = h_R$ when μ and ρ are varied, the value of the interference functions, and consequently

the value of R_0 , does not depend on β (figure 3.3C-D).

If $\mu \geq r$ colonizer aphids go extinct, $\bar{N}_C = 0$, consequently $f(\bar{N}_C) = 1$ and $g(\bar{N}_C) = 1$. The value of R_0 is then defined only by the contribution of migrant aphids in the spread of the virus ($R_0 = R_0^M$).

Table 3.2: Response of R_0 and its components R_0^C and R_0^M to parameter changes. The symbol \downarrow indicates that the parameter change in question leads to a decrease in R_0 (or R_0^C , or R_0^M), \uparrow indicates that it leads to an increase and $\downarrow \uparrow$ indicates that it leads first to a decrease then to an increase depending upon the magnitude of the change.

	Colonizer aphids present $\mu < r$				Colonizer aphids absent $\mu \geq r$		
	Increasing h		Increasing μ	Increasing $2/\rho$	Increasing h	Increasing μ	Increasing $2/\rho$
	Indirect interf. $\beta = 0$	Direct interf. $\beta = 1$					
R_0^C	\uparrow	\uparrow	\downarrow	\uparrow	0	0	0
R_0^M	\downarrow	constant	\uparrow	\uparrow	constant	constant	\uparrow
R_0	$\downarrow \uparrow$	\uparrow	\uparrow or $\downarrow \uparrow^a$	\uparrow	constant	constant	\uparrow

^aDepending on parameters values, see main text for details

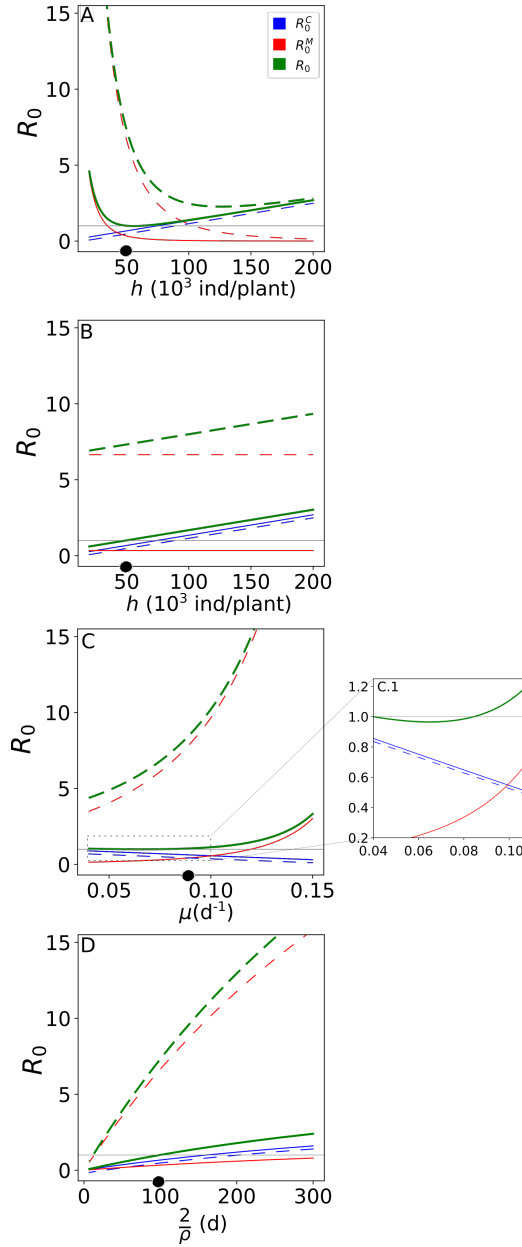


Figure 3.3: Response of the basic reproduction number R_0 and its components R_0^C and R_0^M to changes in aphids hosting capacity (h) under indirect ($\beta = 0$, A) and direct ($\beta = 1$, B) interference scenarios, (C) colonizer aphids mortality (μ), with a close-up in C.1, (D) average surveillance interval ($2/\rho$). Dashed lines identify weak interference scenario ($\nu = 3.5$) and continuous lines strong interference scenario ($\nu = 10.5$) (note: the value of R_0^C is insensitive to the value of ν , so dashed and continuous blue lines overlap). Black dot represents the default value of the varying parameter. The other model parameters are set to default values (Table 3.1).

Equilibrium analysis

The behaviour of the model at equilibrium is summarized in Table 3.3. When $\mu \geq r$, colonizer aphids are not able to survive ($\bar{X}_C = \bar{Z}_C = 0$) and the disease is spread exclusively by migrant aphids. When $\mu < r$ both colonizer and migrant aphids are present in the system and may spread the disease. When all the incoming migrant aphids are non viruliferous (i. e. $\pi = 0$), the disease is able to persist only if $R_0 = R_0^C + R_0^M$ is higher than 1. When a fraction of incoming migrant aphids is viruliferous ($\pi > 0$), the disease is always able to persist, because there will always be an influx of some new viruliferous aphids into the system, and infections of plants will result not just from infected plants in the system (through the activity of aphids present in the system), but also from viruliferous individuals originating outside the system. Note that the patterns presented in Table 3.3 can be explained analytically, with the mathematical details derived in *Appendix C*.

Inter-specific interference and disease control

The responses of R_0 and infected plants density at the equilibrium \bar{I} to variations of the strength of visiting and emigration interferences are shown in figure 3.4. Disease eradication is possible also when only one interference mechanism is active, however, when visiting and emigration interferences are combined, the effect in controlling the disease is stronger than when only one source of interference is at play.

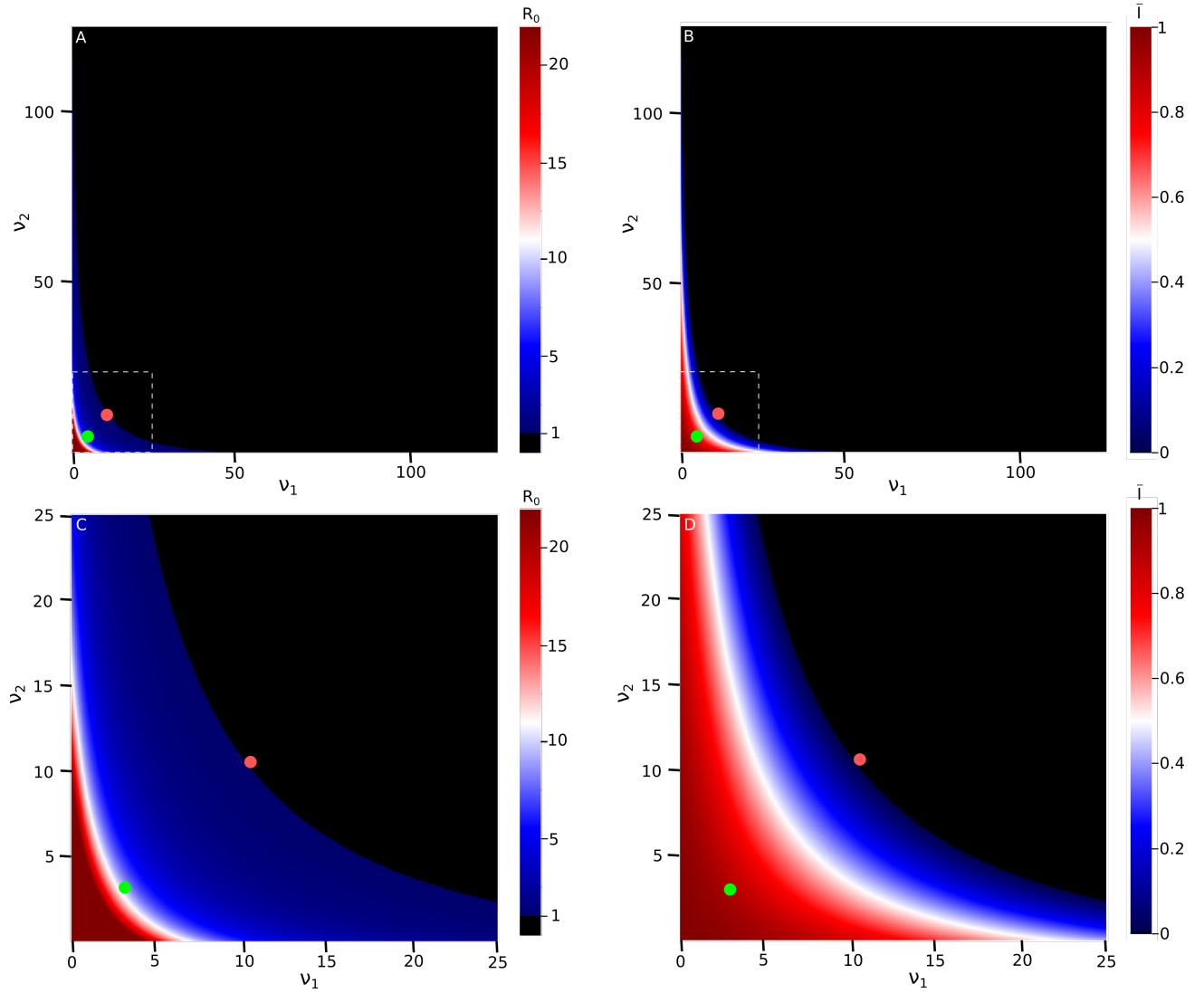


Figure 3.4: Response of R_0 (A-C) and the density of infected plants at the equilibrium (B-D) to changes in visiting interference strength (ν_1) and emigration interference strength (ν_2). C-D are close ups of the highlighted areas in A-B, respectively. Black areas identify values of $R_0 < 1$ (A-C) or infected host density equal to 0 (B-D), corresponding to disease eradication. Green and red dots represent the default value of the varying parameter in weak and strong interference scenarios, respectively. Other model parameters are set to default values (Table 3.1).

Table 3.3: Summary of equilibrium behaviour.

Viruliferous aphids enter the system ($\pi > 0$)	Colonizer aphids are present ($\mu < r$)	Invasion condition	$(\bar{S}, \bar{I}, \bar{X}_C, \bar{Z}_C, \bar{X}_M, \bar{Z}_M)$	Explanation
\times	\times	$R_0^M < 1$	$(+, 0, 0, 0, +, 0)$	Colonizer aphids go extinct, the disease is spread by the migrant aphids but it does not persist in the system.
\times	\times	$R_0^M > 1$	$(+, +, 0, 0, +, +)$	Colonizer aphids go extinct, the disease is spread by the migrant aphids.
\times	\checkmark	$R_0 < 1$	$(+, 0, +, 0, +, 0)$	Colonizer and migrant aphids spread the disease, but it does not persist in the system.
\times	\checkmark	$R_0 > 1$	$(+, +, +, +, +, +)$	Colonizer and migrant aphids spread the disease.
\checkmark	\times	Not relevant	$(+, +, 0, 0, +, +)$	Colonizer aphids go extinct, migrant aphids spread the disease. Migrant aphids can bear the disease from outside the system.
\checkmark	\checkmark	Not relevant	$(+, +, +, +, +, +)$	Colonizer and migrant aphids spread the disease. Migrant aphids can bear the disease from outside the system.

Agricultural practices and disease control

The response of the density of infected plants at equilibrium (\bar{I}) to variations in control parameters is shown in figure 3.5. Note that we assume that the fraction of infected migrant aphids entering the system is equal to 0 (i.e. $\pi = 0$). In the indirect interference scenario ($\beta = 0$), the density of infected

plants at equilibrium exhibits a non-monotonic response to aphids hosting capacity both in the weak and strong interference scenario (figure 3.5A). The eradication of the disease is possible only for the strong interference scenario, for value of h between 48,000-66,000 ind. plant⁻¹. In the direct interference scenarios ($\beta = 1$), the density of infected plants at equilibrium increases with aphids hosting capacity (figure 3.5B). The eradication of the disease is possible only for the strong interference scenario, for value of h between 20,000 and 51,000 ind. plant⁻¹. Since $h = h_R$ when μ and ρ are varied, the effects of visiting and emigrating interference upon \bar{I} are the same regardless the interference mechanism (i.e. the value of β) (figure 3.5C-D). The density of infected plants at equilibrium increases monotonically with colonizer aphid mortality both in the weak and in the strong interference scenario (figure 3.5C). Disease eradication is possible only in the strong interference scenario for values of μ between 0.040 and 0.085 day⁻¹. Note that the non monotonic response highlighted in figure 3.3C.1 for $0.04 < \mu < 0.08$ does not emerge here, because $R_0 < 1$ in this μ values range, thus $\bar{I} = 0$. The density of infected plants at equilibrium increases with the surveillance interval (i.e the average amount of time that a plant remains in the system before roguing) and the eradication of the disease is possible for values of $2/\rho$ lower than 12 days in the weak interference scenario, and lower than 100 days in the strong interference scenario (figure 3.5D). We analyze the response of the patterns shown in figure 3.5 to variation in the value of π (see *Appendix C* figure C.3): the qualitative responses of the density of infected plants to agricultural practices are generally maintained for higher fractions of viruliferous immigrant aphids, although the response of \bar{I} to variation of parameters h , μ , ρ is less

and less evident with increasing value of π . The density of infected plants increases with the value of π .

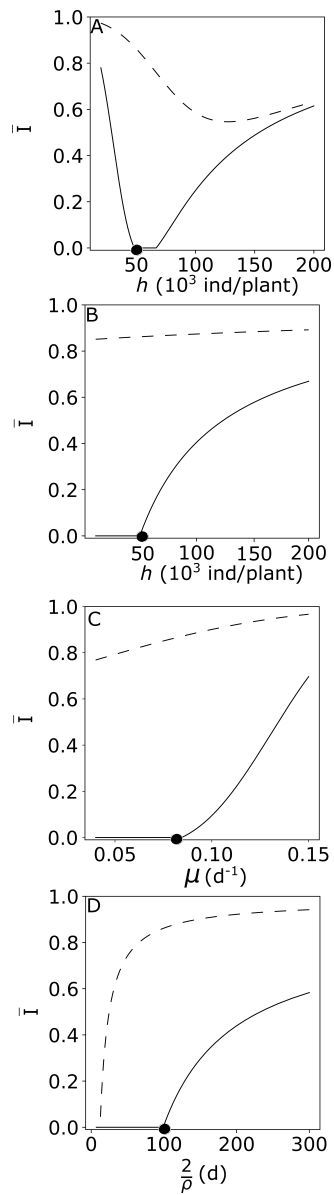


Figure 3.5: Response of the density of infected plants at the equilibrium to changes in (A-B) aphids hosting capacity (h) under indirect ($\beta = 1$, A) and direct ($\beta = 0$, B) interference scenarios; (C) colonizer aphids mortality (μ); (D) average surveillance interval ($2/\rho$). Dashed lines identify weak interference scenario ($\nu = 3.5$) and continuous lines strong interference scenario ($\nu = 10.5$). Black dot represents the default value of the varying parameter. The other model parameters are set to default values (Table 3.1).

The response of R_0 to the simultaneous variation of two control paramete-

ters for different interference scenarios is reported in figure 3.6. Also in this case, we assume that migrant aphids entering the system are non viruliferous (i.e. $\pi = 0$). It is always possible to eradicate the disease for some combination of two of the considered parameters, except for (h, μ) combinations in the case of weak interference (figure 3.6A-B), and the region of parameter combinations that lead to disease eradication is bigger in the strong than in the weak interference scenarios. Eradication is possible without the addition of pesticides ($\mu = 0.04 \text{ day}^{-1}$, i.e. colonizer aphid natural mortality) for values of h between 20,000 and 51,000 ind. plant⁻¹ in the direct strong interference scenarios (figure 3.6D) and for value of h between 39,000 and 51,000 ind. plant⁻¹ in the indirect strong interference scenario (figure 3.6C). Disease eradication is not possible for values of colonizer aphid mortality (μ) higher than 0.11 day^{-1} , independently from the value of h , in the strong direct interference scenarios (figure 3.6D), while it is possible in the strong indirect interference scenario, where the value of μ that leads to disease eradication, increases with the value of h (figure 3.6C). Reducing surveillance interval ($2/\rho$) increases the range of h and μ values that lead to disease eradication. For example, in the strong direct interference scenario, if $2/\rho$ is lower than 30 days, disease eradication is possible for all the considered h values (figure 3.6H) and for all the considered μ values (figure 3.6L). Since $h = h_R$ when μ and ρ are varied, the response of R_0 to combinations of $(2/\rho, \mu)$ does not depend on the interference mechanism (i.e. the value of β), but only on the interference strength (i.e. the value of ν): that is why figure 3.6I is equal to figure 3.6K and figure 3.6J is equal to figure 3.6L.

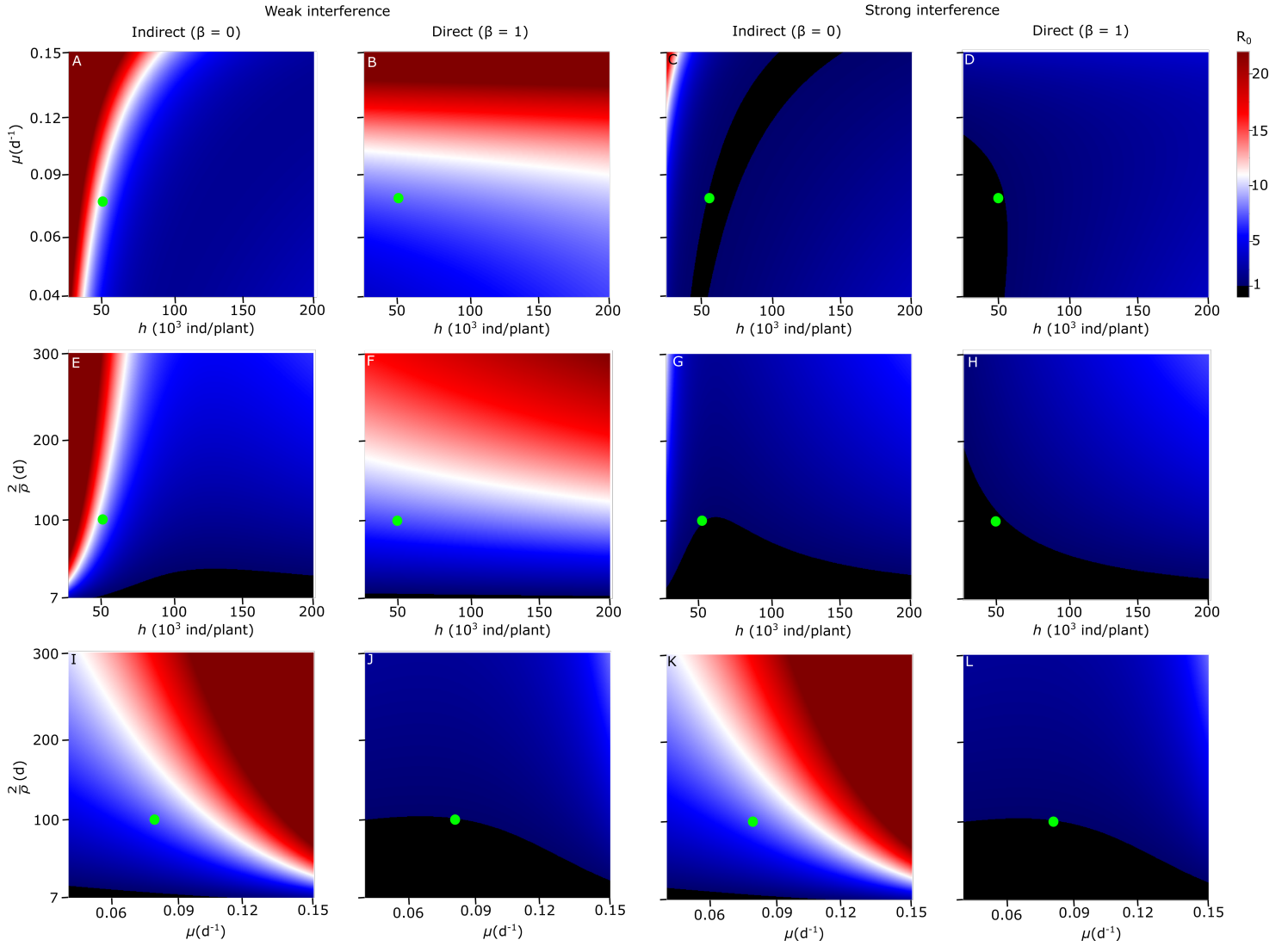


Figure 3.6: Response of R_0 to changes in: aphids hosting capacity (h) and colonizer aphids mortality (μ) (A-B-C-D); aphids hosting capacity (h) and surveillance interval ($2/\rho$) (E-F-G-H); colonizer aphids mortality (μ) and surveillance interval ($2/\rho$) (I-J-K-L), under different interference scenarios (weak $\nu = 3.5$ and strong $\nu = 10.5$). Black areas identify values of $R_0 < 1$, corresponding to disease eradication. Green dot represents the default values of the varying parameters. Other model parameters are set to default values (Table 3.1).

Discussion

Although the established evidence from meta-analytical evaluations (Bird et al., 2019; Kaplan and Denno, 2007; Denno et al., 1995) suggests that both direct and indirect (i.e. plant mediated) interferences between herbivorous insects shape their behaviour and performance, these interference mechanisms have been ignored in epidemiological analyses despite a few exceptions (see e.g. Crowder et al. (2019); Chisholm et al. (2019); Thaler et al. (2010)). Yet, these works only consider interaction between vector and non-vector insects. In the present work, we modeled the interaction between two aphid vectors by mean of two interference functions, which account for the reduction of the number of plants visited per unit time and the average resident time in the system of migrant aphids. The interference functions allow to represent both direct and indirect interferences, through the parameter β . By means of mathematical and numerical analyses of our model, we have demonstrated that the considered interference mechanisms towards migrant aphids can have profound effects on the invasion, persistence and control of plant NPT viruses. No matter the underlying mechanism is direct or indirect, the interference affecting the number of visited plants (visiting interference $f(N_C)$ in our model) appears squared because it affects both the acquisition and the inoculation of the virus. This explains why we found stronger effect of this interference mechanism in reducing the spread of the virus. In theoretical work, ecological interactions that limit vector movement have already been showed to be more effective in controlling the spread of NPT viruses than those targeting vector abundance (Crowder et al., 2019). Our theoret-

ical results confirm this finding: visiting interference reduces the number of plants visited per day by a migrant aphid, which reduces both the probability of acquired and inoculate the virus and diminishes the incidence of the disease.

Moreover, our result suggest that largely used agricultural practices, such as fertilization, pesticide application and roguing of diseased plants, can have unexpected results upon the spread of NPT viruses. Fertilization is largely used in agriculture to meet plants' nutrient needs and increase plant growth and production (Gliessman, 2015b; Bommarco et al., 2013). Furthermore fertilization can impact disease development, possibly affecting plant physiology, pathogens and/or vector population dynamics (Dordas, 2008; Miller et al., 2015). Yet its effect on virus spread is poorly understood (Dordas, 2008). On the one hand, most of the studies reviewed by Dordas (2008), showed that application of fertilizers decreases the incidence of disease in crop plants. This is due to the fact that nitrogen is involved in the resistance mechanisms of the host plant, i.e. its ability to limit the penetration, development and reproduction of the invading pathogen (Dordas, 2008). On the other hand, fertilization can affect vector population dynamics via changes in plant nutrition: vector populations are often N limited, and N addition can increase vector abundance. For example, Miller et al. (2015) observed that fertilization increased wheat curl mite, vector of the *Wheat streak mosaic virus*, growth rate on winter wheat. Liang et al. (2019) observed that fertilization increased the pressure of the bird cherry-oat aphid (*Rhopalosiphum padi L.*), vector of *Barley yellow dwarf virus*, on winter wheat. Vector population increase can subsequently result in increasing virus transmission

and spread (Thomas and Hein, 2003; Borer et al., 2009). Our results show that increase in colonizer vector due to increase in fertilization may, for low level of fertilization, decrease the spread of the disease due to the increase of indirect interference between colonizer and migrant aphids. For medium to high value of fertilization, however, increasing fertilization increases the population of colonizer aphids resulting in increasing the incidence of the disease.

Pesticide application is the most common aphid control method, but it is well known that its ability to prevent the spread of NPT viruses by migrant aphids is limited because viruses inoculation occurs rapidly and before a pesticide can take effect on the migrant vector (Perring et al., 1999). Furthermore pesticide may affect local pest community structure as differential susceptibility to pesticide may result in species dominance shift favoring secondary pest outbreaks (Mohammed et al., 2019; Guedes et al., 2016, 2017; Zhao et al., 2017). In their experiments with bird cherry-oat aphid (*Rhopalosiphum padi* L.) and the English grain aphid (*Sitobion avenae* F.), Mohammed et al. (2019) showed that pesticide exposure led to a shift in the outcome of interspecific competition between the two aphid species, compromising the dominance of *R. padi* in pesticide-free plants, while favoring the prevalence of the *S. avenae* under pesticides exposure. Similarly, Zhao et al. (2017), in their experiments with two thrip species, observed that *Thrips tabaci*, which is competitively superior to *Frankliniella occidentalis* on cabbage foliage in the absence of pesticide, were displaced by *F. occidentalis*, which became the predominant species when insecticides were applied. Our results show that small pesticide application has the potential to reduce the

spread of NPT viruses. Yet, large pesticide application, reducing the interference exerted by colonizer towards migrant aphids, is counter productive in reducing NPT viruses, because it favours the prevalence of migrant aphids, increasing the spread of the virus by the more mobile vector.

Roguing infected plants has often been implemented to control the spread of plant pathogens (Dallot et al., 2004; Sisterson et al., 2008; Sisterson and Stenger, 2013; Rimbaud et al., 2015a). The success of roguing in slowing disease spread depends on how rapidly infected plants are identified and removed (Dallot et al., 2004; Cunniffe et al., 2015a). Yet, there are various logistical issues associated with identification and removal of infected plants in large-scale agriculture. Firstly, the identification of disease plants may be hampered by a lack of appropriate and/or cost-effective diagnostic tests or personnel trained to recognize symptoms. Further, growers can be reluctant to remove diseased plants as soon as symptoms are identified, since infected plants may continue to produce a marketable yield (Sisterson and Stenger, 2013). Particularly, some perennial crops may take years to become fully productive (Muraro et al., 1996; Edwin and Masters, 2005), thus a mature infected plant may be more productive over the remaining lifetime of the crop than an uninfected immature replant (Sisterson et al., 2008). Finally, the degree of coordination among farmers concerning the decision of roguing is likely to affect the success in slowing disease spread (Sisterson and Stenger, 2013; Laranjeira et al., 2020). Our results unsurprisingly suggest that the incidence of the disease decreases with the effort put in roguing. Yet, they would benefit from further economic evaluations, given the cost of roguing and replanting operations.

Despite our efforts to provide a realistic representation of the complex epidemiological and ecological components of an agroecosystem, we had to introduce a number of simplifying assumptions. We have assumed the colonizer aphid mortality rate to be constant. In reality, the effects of chemical control on pest mortality do not remain constant, but vary with the repeated application and subsequent decay of pesticides concentration. Focusing on parameter parsimony, according to several authors (see e.g. Crowder et al. (2019); Donnelly et al. (2019); Allen et al. (2019); Sisterson and Stenger (2016); Madden et al. (2000)), we considered a time constant mortality rate. We have assumed that NPT viruses do not manipulate host-vector behavior in order to enhance their own transmission, for example by making infected plant more attractive to aphids but inhibit aphid settling on infected plants (Mauck et al., 2012). This may not always hold, for example, it was shown that squash plants (*Cucurbita pepo*) infected with *Cucumber mosaic virus* firstly emit a blend of volatile organic compounds that attracts aphids (Mauck et al., 2010; Pickett and Khan, 2016), and secondly produce anti-feedant compound, which deter aphids from prolonged feeding (Mauck et al., 2010). Yet, a non negligible proportion of the experiments analyzed by Mauck et al. (2012) performed with NPT viruses, including several potyviruses (i.e. PPV), do not follow this “attract and deter” trend (Rimbaud et al., 2015a). Finally, it is possible that plants put in place other types of defensive mechanisms which may impair colonizer aphid fecundity (Will et al., 2013; Zust and Agrawal, 2016) and which can be fostered by fertilization (Zaffaroni et al., 2020). Although all these mechanisms could be included in our model, we have chosen to avoid the proliferation of parameters which

would have been associated with more complex models, possibly hiding the underlying message of this work. Yet, despite the simplifying assumptions outlined above, we are confident that our analysis highlights the importance of understanding interference mechanisms between vectors and the occasionally counter-intuitive effect of common agronomic practices to build effective strategies for the control and eradication of aphid vectored diseases

The model we developed is general enough to be applicable to a large number of pathosystems. Indeed, it is common that both colonizer and migrant aphids are involved in the spread of NPT viruses, with the former being often more efficient in transmitting the virus and the second being more mobile in the field (Robert et al., 2000; Lecoq and Desbiez, 2012; Rimbaud et al., 2015b; Clafin et al., 2017). Noting that further experimental works are clearly required to confirm our findings, our work suggests that the impacts of inter-specific interference should be incorporated more broadly into the planning of disease management strategies to regulate the invasion and persistence of NPT viruses.

Supplementary Information

- **C.1** Scaling of the interference strength parameter V_i ($i = 1,2$)
- **C.2** Response of R_0 to μ under different parametrizations
- **C.3** Non dimensionalization
- **C.4** Basic reproduction number
- **C.5** Epidemic equilibrium

Conclusion and perspectives

The aim of my thesis was to develop models which explicitly considered the interactions between plant and aphids and use them to explore the potential of common agricultural practices to regulate aphid direct and indirect damages to plant.

I developed two models: the first one (hereafter the plant-aphid model) coupled plant growth and aphid population dynamics, accounting for the direct damage of aphids on plant (i.e. phloem feeding) and the development of induced plant defences. The second one (hereafter the single-host, multi-vector model) simulated the spread of non persistent transmitted (NPT) viruses to a plant host by colonizer and migrant aphid species, accounting for the interference exerted by colonizer towards migrant aphids. Both the models explicitly represented direct or indirect interaction between plant and aphids: they were thus suitable to explore the effects of agronomic practices on the plant and their feedback on aphid dynamics. The two models I developed have the ambition of being general enough to be applied to different plant-pest systems and to incorporate the description of other agronomic practices. In addition the single-host, multi-vector model, with proper adjustments, can be adapted to simulate the spread of semi-persistent and

persistent viruses (see e.g. Madden et al. (2000); Crowder et al. (2019)).

I demonstrated that common agricultural practices (i.e. fertilization, irrigation and pesticide) have the potential to control aphid direct and indirect damages to plant. However, the effect of agricultural practices on aphid population is not monotonic (i.e. it cannot be simply reduced as positive or negative), but it may depend on the considered level of treatment and on the date of observation along the growing season. Furthermore, some agricultural practices may reduce the direct but increase the indirect damage of aphids to plants and vice-versa. Indeed, agricultural practices that affect aphid abundance on the plant are more likely to influence the direct damage, while agricultural practices that affect the movement of aphids between plant are more likely to influence the indirect damage (Van Emden and Harrington, 2007). For example, my results suggested that reducing fertilization may lower aphid population, and consequently their phloem uptakes, because of a reduction in plant nutrient content. However a reduction in aphid population decreases the interference exerted towards migrant aphid species, possibly favouring the spread of plant viruses. Similarly, the application of pesticide directly reduces phloem feeding by aphids, reducing the direct damage, but it may reduce the interference towards migrant aphid species, leaving the plant more expose to migrant aphid species visits and thus to viruses infection. I believe that the effect of agricultural practices on aphid population dynamics should be further investigated by considering at the same time aphid direct and indirect damages, for example by coupling the plant-aphid and the single-host, multi-vector models.

Furthermore, since the key interest of farmers is the final yield quality

and quantity, it is important to evaluate the effect of agricultural practices not only on aphid population dynamics, but on the crop production, which indeed is influenced by aphid population dynamics. In my work, I applied the plant-aphid model to explore optimal management alternatives respect to two contrasting objectives: to maximize plant biomass production and to minimize environmental impact. My work shown that the majority of optimal scenarios are characterized by high fertilization, since we assumed it fosters plant growth and favours the production of nitrogen-based induced defences, which control aphids outbreaks. It would be interesting to use the single-host, multi-vector model, adjusted to evaluate the production of host plants, to analyse the optimal management alternatives respect the two over mentioned objectives considering the role of aphid as vector of plant viruses.

The results I presented in my thesis are based on the assumptions I made to develop the plant-aphid and the single-host, multi-vector models. The proposed plant-aphid model would enormously benefit from experimental works dedicated to analyse the quantitative relationship relevant to *i)* the plant cost of making defences in term of growth loss, and *ii)* the effect of plant defences on aphid performance. Similarly, experimental works are needed to confirm the results obtained with the single-host, multi-vector model, in particular the effectiveness of inter-specific aphid interference in shaping the spread of plant viruses. Hence, the results I presented in my thesis should be interpreted as qualitative pattern that can't immediately be transferred to agronomic practices. Indeed, quantitative evaluation of the relationship between treatment (fertilization, irrigation and aphid mortality) level and model's associated parameter (σ_N, κ, μ_p) are needed. Yet, despite the simpli-

ifying assumptions I brought out throughout this manuscript, I am confident that my analysis highlighted the importance of understanding the effects of common agricultural practices in regulating both the direct and indirect damage of aphids to plant. The challenge for the future works would be to show how the insights highlighted in my thesis - as well as the models developed here - can be used to inform practical decision making by farmers and growers.

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Appendix A

Supplementary Information - Chapter 1

A.1 Available data

Data come from 44 peach *Prunus persica* trees cultivated at the Institut National Recherche Agronomique (INRA) station of Avignon (southern France, 43°60' N, 4°49' E). The plants were grafted in February 2011 and the experiment took place in spring 2013, from the end of April (corresponding to approximately 30 days after bloom) to the beginning of July (corresponding to the end of vegetative growth). Plants were subjected to four different treatments obtained by combining two levels of fertilization (N^+ and N^-) and two levels of irrigation (H^+ and H^-). On May 2nd, two growing shoots per plant were selected and infested with five wingless adult aphid females each. The shoot growth and the aphid infestation level was measured weekly on each plant. Shoot growth was measured by counting the number of leaves

per shoot. Aphid infestation was measured by assigning to each shoot an infestation class: C0 (no aphids), C1 (1-5 ind.), C2 (6-25 ind.), C3 (26-125 ind.), C4 (126-625 ind.) and C5 (> 625 ind.). Available data are reported in tables A.2 and A.3. Details on the experiment are reported in the original paper Rousselin et al. (2016).

We computed dry shoot $S(t)$ mass as a function of the counted shoot leaves $n_S(t)$ via the allometric relationships $S(t) = 0.26 \cdot n_S(t)$ where S is expressed in grams Génard et al. (2000); Grossman and DeJong (1994). We computed aphid abundance $A(t)$ from the measured infestation class by setting it equal to 3, 16, 76, 376 and 625, respectively for classes from C1 to C5. We scaled up all the measures at the shoot level to the whole plant level by multiplying them by the number of growing shoots per plant.

A.2 Model initialization and off-line calibration

In order to run the model (i.e. integrate the system of ODE's) one must define the initial time t_0 and the corresponding values of the model state variables. We set t_0 to April 29th (i.e. 119th day of year DOY) and $S(t_0) = 19.7$ g and $A(t_0 + 3) = 41.5$ ind., per plant, according to field observations. Following Thornley Thornley (1998), we assumed that at the beginning of the vegetative season, root:shoot ratio equals equals 0.3 and that the concentration of substrates in shoots and roots is 5.5% for C and 0.6% for N. We then set $R(t_0) = 5.9$ g, $C_S(t_0) = 1.09$ g, $N_S(t_0) = 0.12$ g, $C_R(t_0) = 0.33$ g,

$N_R(t_0) = 0.04$ g. We set $D(t_0) = 0$ by assuming that induced defences are not present before infestation.

We used available data to assess the time at which the plant stopped growing. For each pairs of consecutive observation dates we evaluated via a t-test the probability P that the means of the shoot dry masses were equal. If the plant has significantly grown in the considered period, the P level will be ≤ 0.01 . We assumed that the first date of the pair for which $P > 0.01$ (i.e. $t=169$) corresponds to the parameter λ of the switch off function $\Phi = \frac{\lambda^\eta}{\lambda^\eta + t^\eta}$. We assumed that at the first date t^* of the pair for which $P > 0.05$ (i.e. $t^*=176$) the value of Φ drops to a critical value $\Phi^* = 0.05$. After some algebra, one obtains $\eta = \frac{\ln(\frac{1}{\Phi^*}-1)}{\ln(t^*)-\ln(\lambda)} = 73$.

A.3 Model calibration

We estimated model unknown parameters by minimizing a cost function C expressed as the sum of two negative log-likelihood functions $C = -(\ell_y + \ell_x)$ with:

$$\ell_i = -N_i \log(\sqrt{2\pi\sigma_i^2}) - \frac{1}{2\sigma_i^2} \sum_P \sum_t (i_{P,t} - \hat{i}_{P,t})^2$$

where $i_{P,t}$ are the values of the average shoot dry mass ($i = y$) and the average aphid abundance per shoot ($i = x$) observed on the plant P at time t , total samples size equal to N_i and $\hat{i}_{P,t}$ are the corresponding values simulated by the model.

We assumed that the errors between each observation and the corresponding value estimated by the model follow a Gaussian distribution with mean

0 and unknown variance σ_i^2 . To derive the log-likelihood functions, we assumed that the error structure is additive. We found the set of parameters that minimized the cost function C using the Matlab command "fminsearch" (Nelder-Mead algorithm).

Figure

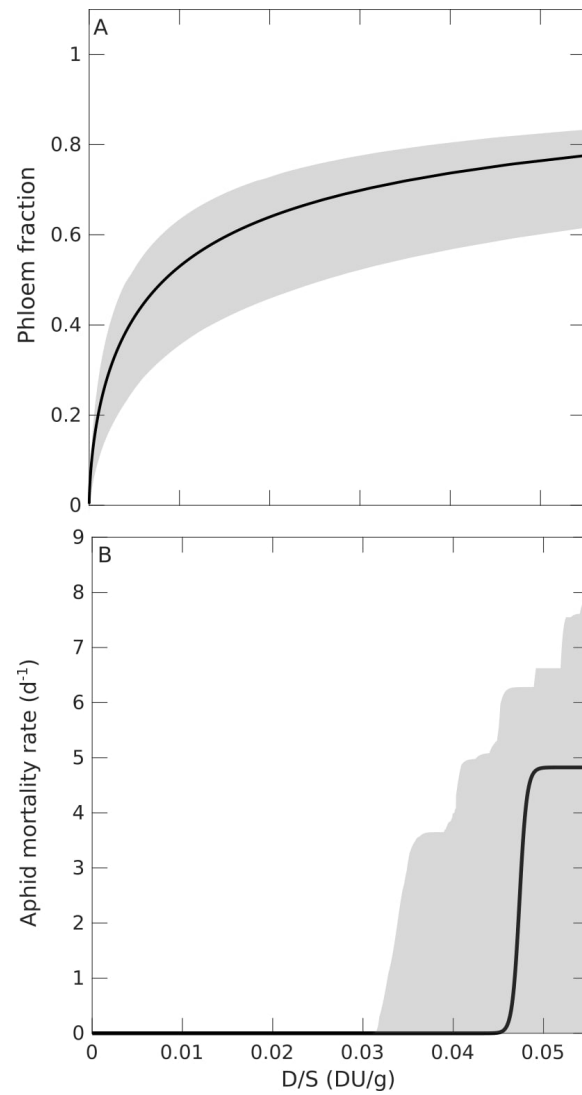


Figure A.1: Effect of the concentration of plant defences on (A) Fraction of phloem that is unavailable to aphids and (B) aphid mortality induced per mg of ingested phloem. Grey curves indicate 99% confidence bands due to the uncertainty in the estimate of parameters π_1 and δ_1 (A), and π_2 , δ_2 and β_2 (B).

Tables

Table A.1: Numerical approximation of the sensitivity, ψ (see equation (2.2) in Chapter 1), of the maximum value of shoot mass (S), aphid abundance (A) and density (A/S) to small changes in the parameters.

	$\psi(S)$	$\psi(A)$	$\psi(A/S)$
δ_2	0.00	0.00	0.00
δ_1	0.01	-0.35	-0.27
β_2	0.02	0.00	0.00
μ	0.02	-0.31	-0.32
α	0.02	-0.65	-0.43
π_1	-0.05	0.07	0.26
η	-0.06	-0.01	0.01
ϵ_N	0.15	-0.10	-0.10
ν	0.17	0.10	0.05
ξ	-0.18	-0.17	0.72
κ	0.22	0.46	0.23
ϵ_C	0.25	-0.12	-0.16
π_2	-0.25	0.00	0.00
θ	-0.25	-0.68	0.19
ι_C	0.26	0.21	0.06
ι_N	0.29	0.27	0.04
φ_C	-0.53	-0.71	-0.15
σ_N	0.63	0.64	0.06
σ_C	0.65	0.63	0.16
φ_N	-0.66	-0.76	0.03
q	1.56	1.79	0.33
λ	5.87	1.31	0.04

Table A.2: Number of leaves observed on two growing shoots (id_sh: a, b) on 44 peach trees subjected to different fertilization and irrigation treatments (tr: N+H+, N-H+, N+H-, N-H-). The observation dates are reported DOY 2013.

id_tree	id_sh	tr	DOY										
			119	126	133	141	147	155	161	169	176	183	
1	a	N+H+	11	16	25	28	37	49	55	55	55	55	
2	a	N+H+	10	14	23	31	35	47	61	61	61	61	
3	a	N+H+	7	8	10	15	17	23	30	30	30	30	
4	a	N+H+	8	9	14	20	23	28	33	33	33	33	
5	a	N+H+	8	8	22	32	33	37	48	51	51	51	
6	a	N+H+	9	10	13	18	18	25	32	36	36	36	
7	a	N+H+	11	20	26	32	36	46	63	63	63	63	
8	a	N+H+	8	8	13	17	18	32	45	53	53	53	
9	a	N+H+	12	22	30	42	55	88	126	129	129	129	
10	a	N+H+	10	12	19	25	29	40	57	57	57	57	
11	a	N+H+	10	10	12	18	20	26	34	37	37	37	

Table A.2 (continued)

id_tree	id_sh	tr	DOY										
			119	126	133	141	147	155	161	169	176	183	
12	a	N+H+	9	10	11	20	24	30	67	76	76	76	
13	a	N+H+	9	12	13	23	28	45	56	56	56	56	
1	b	N+H+	14	28	41	62	76	103	131	137	137	137	
2	b	N+H+	10	13	20	27	29	45	49	49	49	49	
3	b	N+H+	7	8	11	18	19	27	39	39	39	39	
4	b	N+H+	10	18	33	45	50	60	77	77	77	77	
5	b	N+H+	7	8	9	13	17	25	39	42	42	42	
6	b	N+H+	9	9	12	14	16	22	32	32	32	32	
7	b	N+H+	10	12	14	18	21	27	35	35	35	35	
8	b	N+H+	9	9	12	18	19	41	56	56	56	56	
9	b	N+H+	11	16	26	39	44	56	74	77	77	77	
10	b	N+H+	9	11	14	23	25	32	38	38	38	38	
11	b	N+H+	11	11	13	21	22	28	39	48	52	52	

Table A.2 (continued)

id_tree	id_sh	tr	DOY										
			119	126	133	141	147	155	161	169	176	183	
12	b	N+H+	8	10	12	16	21	27	46	60	60	60	60
13	b	N+H+	11	14	22	34	37	54	83	98	98	98	98
14	a	N-H+	9	9	11	13	14	16	19	23	26	27	27
15	a	N-H+	10	10	12	15	17	21	25	25	25	25	25
16	a	N-H+	9	9	13	16	17	17	22	22	22	22	22
17	a	N-H+	8	9	11	13	15	17	18	18	18	18	18
18	a	N-H+	9	10	16	20	21	22	28	28	28	28	28
19	a	N-H+	9	9	11	13	15	18	23	27	28	28	28
20	a	N-H+	9	9	11	13	15	18	25	25	25	25	25
21	a	N-H+	9	10	13	17	17	19	24	26	30	30	30
22	a	N-H+	9	10	11	13	16	18	21	22	22	22	22
14	b	N-H+	10	10	12	14	15	17	24	32	36	43	43
15	b	N-H+	7	9	11	13	14	17	20	20	20	20	20

Table A.2 (continued)

id_tree	id_sh	tr	DOY										
			119	126	133	141	147	155	161	169	176	183	
16	b	N-H+	10	10	12	16	16	23	31	31	31	31	31
17	b	N-H+	7	8	9	11	13	15	17	17	17	17	17
18	b	N-H+	9	9	11	14	15	17	18	18	18	18	18
19	b	N-H+	8	9	11	14	15	19	21	27	27	27	27
20	b	N-H+	8	9	11	15	16	22	26	26	26	26	26
21	b	N-H+	9	15	21	28	29	32	51	57	61	61	61
22	b	N-H+	8	10	11	12	14	18	21	21	21	21	21
23	a	N+H-	9	11	15	20	24	28	39	39	39	39	39
24	a	N+H-	10	16	24	30	33	39	43	43	43	43	43
25	a	N+H-	9	11	17	27	29	41	53	57	57	57	57
26	a	N+H-	10	12	15	22	24	30	35	37	37	37	37
27	a	N+H-	13	18	25	36	38	42	46	46	46	46	46
28	a	N+H-	9	17	19	29	30	46	63	65	65	65	65

Table A.2 (continued)

id_tree	id_sh	tr	DOY										
			119	126	133	141	147	155	161	169	176	183	
29	a	N+H-	5	7	10	17	19	19	29	45	45	45	45
30	a	N+H-	9	12	21	25	26	31	34	36	36	36	36
31	a	N+H-	11	15	24	36	40	49	55	55	55	55	55
32	a	N+H-	10	11	13	15	17	22	24	24	24	24	24
33	a	N+H-	8	9	11	15	15	20	21	21	21	21	21
34	a	N+H-	8	10	11	19	21	30	40	50	53	54	54
23	b	N+H-	11	18	26	38	41	51	78	81	81	81	81
24	b	N+H-	10	12	15	21	21	27	37	37	37	37	37
25	b	N+H-	7	8	12	14	14	14	18	19	19	19	19
26	b	N+H-	10	13	23	28	30	33	37	41	41	41	41
27	b	N+H-	6	10	14	17	18	20	22	24	24	24	24
28	b	N+H-	7	8	10	14	16	21	30	32	32	32	32
29	b	N+H-	7	10	14	17	19	24	33	33	33	33	33

Table A.2 (continued)

id_tree	id_sh	tr	DOY										
			119	126	133	141	147	155	161	169	176	183	
30	b	N+H-	8	10	15	22	24	32	38	42	42	42	
31	b	N+H-	11	18	32	62	73	103	166	167	167	167	
32	b	N+H-	11	17	24	29	33	39	44	44	44	44	
33	b	N+H-	10	14	21	27	28	42	56	56	56	56	
34	b	N+H-	9	11	12	15	15	22	26	33	33	34	
35	a	N-H-	12	19	29	43	50	63	80	80	80	80	
36	a	N-H-	9	10	11	14	16	20	22	22	24	25	
37	a	N-H-	7	9	12	14	14	16	20	20	20	20	
38	a	N-H-	7	7	9	11	13	14	15	15	16	18	
39	a	N-H-	10	11	15	19	20	27	36	36	38	38	
40	a	N-H-	7	7	10	14	15	16	18	18	18	18	
41	a	N-H-	9	11	17	20	22	24	26	26	28	28	
42	a	N-H-	10	12	14	17	17	17	20	20	20	20	

Table A.2 (continued)

id_tree	id_sh	tr	DOY										
			119	126	133	141	147	155	161	169	176	183	
43	a	N-H-	8	10	10	13	15	19	22	22	23	23	23
44	a	N-H-	9	11	13	19	20	25	29	29	30	30	30
35	b	N-H-	12	16	20	25	26	34	41	41	41	41	41
36	b	N-H-	9	11	11	12	13	15	17	20	22	22	22
37	b	N-H-	9	9	12	16	17	23	30	30	30	30	30
38	b	N-H-	9	12	17	22	24	29	32	36	36	36	36
39	b	N-H-	10	11	21	27	28	36	48	49	54	54	54
40	b	N-H-	8	9	12	13	18	20	22	22	22	22	22
41	b	N-H-	8	11	19	30	32	39	54	54	54	54	54
42	b	N-H-	9	10	12	15	15	17	19	19	19	19	19
43	b	N-H-	9	13	20	28	31	44	54	54	54	54	54
44	b	N-H-	7	9	12	14	16	17	21	21	21	21	21

Table A.3: Class of aphid abundance observed on two growing shoots (id_sh: a, b) on 44 peach trees (id_tree) subjected to different fertilization and irrigation treatments (tr: N+H+, N-H+, N+H-, N-H-). The observation dates are reported DOY 2013.

id_tree	id_sh	tr	DOY													
			127	134	141	147	155	161	169	176						
1	a	N+H+	2	3	3	4	4	5	2	0						
2	a	N+H+	1	1	3	3	5	5	4	0						
3	a	N+H+	0	0	1	1	2	4	4	0						
4	a	N+H+	2	3	3	4	4	4	2	0						
5	a	N+H+	0	0	1	1	2	4	3	0						
6	a	N+H+	1	0	0	0	1	2	4	4						
7	a	N+H+	1	2	3	3	3	4	4	0						
8	a	N+H+	1	1	2	2	3	4	4	0						
9	a	N+H+	2	3	3	3	4	5	5	0						
10	a	N+H+	2	3	3	3	4	4	4	0						
11	a	N+H+	0	0	1	0	2	3	4	2						

Table A.3 (continued)

id_tree	id_sh	tr	DOY										
			127	134	141	147	155	161	169	176			
12	a	N+H+	2	1	2	3	3	5	3	0			
13	a	N+H+	2	2	4	4	4	5	3	0			
1	b	N+H+	2	3	4	4	4	5	3	0			
2	b	N+H+	1	2	2	3	4	4	3	0			
3	b	N+H+	2	2	3	3	4	4	4	0			
4	b	N+H+	2	2	3	3	4	5	3	0			
5	b	N+H+	1	0	2	2	3	4	5	0			
6	b	N+H+	1	1	3	3	4	4	4	1			
7	b	N+H+	1	2	2	2	3	4	4	0			
8	b	N+H+	2	3	3	3	4	5	4	0			
9	b	N+H+	1	1	2	1	2	4	5	0			
10	b	N+H+	1	0	1	0	2	3	4	0			
11	b	N+H+	1	1	2	3	2	3	4	3			

Table A.3 (continued)

id_tree	id_sh	tr	DOY													
			127	134	141	147	155	161	169	176						
12	b	N+H+	0	0	1	1	3	3	4	0						
13	b	N+H+	1	2	1	1	3	4	4	0						
14	a	N-H+	0	1	0	0	0	2	1	0						
15	a	N-H+	2	2	3	3	4	5	3	0						
16	a	N-H+	1	0	2	3	3	4	3	0						
17	a	N-H+	0	0	1	2	3	3	3	0						
18	a	N-H+	0	0	1	0	2	4	3	0						
19	a	N-H+	0	0	2	1	2	2	4	4						
20	a	N-H+	1	2	3	3	3	4	4	0						
21	a	N-H+	2	1	0	2	1	1	0	0						
22	a	N-H+	1	0	1	1	2	3	3	0						
14	b	N-H+	1	2	3	2	0	2	3	2						
15	b	N-H+	1	2	3	3	3	4	3	0						

Table A.3 (continued)

id_tree	id_sh	tr	DOY										
			127	134	141	147	155	161	169	176			
16	b	N-H+	2	2	3	3	4	4	4	2	0		
17	b	N-H+	0	0	1	1	2	3	3	0			
18	b	N-H+	0	0	2	3	4	3	2	0			
19	b	N-H+	0	0	1	1	2	3	4	4			
20	b	N-H+	0	0	2	2	3	4	4	0			
21	b	N-H+	2	2	3	3	3	2	0	0			
22	b	N-H+	1	0	2	3	3	4	3	0			
23	a	N+H-	2	2	2	3	4	4	3	0			
24	a	N+H-	2	2	3	3	4	4	3	0			
25	a	N+H-	2	2	3	3	3	4	3	0			
26	a	N+H-	1	0	1	0	1	3	3	0			
27	a	N+H-	2	1	2	3	3	4	3	0			
28	a	N+H-	1	1	3	3	3	3	3	0			

Table A.3 (continued)

id_tree	id_sh	tr	DOY													
			127	134	141	147	155	161	169	176						
29	a	N+H-	2	2	3	3	4	5	3	0						
30	a	N+H-	0	0	0	1	3	3	3	1						
31	a	N+H-	3	3	3	3	4	4	3	0						
32	a	N+H-	2	2	3	3	4	3	2	0						
33	a	N+H-	0	1	2	3	3	4	3	0						
34	a	N+H-	2	1	1	1	1	0	1	1						
23	b	N+H-	1	0	0	1	2	4	4	0						
24	b	N+H-	1	0	1	2	3	4	4	0						
25	b	N+H-	1	0	1	1	3	3	3	0						
26	b	N+H-	2	2	2	1	2	3	3	2						
27	b	N+H-	1	1	1	2	3	3	3	0						
28	b	N+H-	1	1	0	1	2	3	4	0						
29	b	N+H-	2	3	3	3	4	4	3	0						

Table A.3 (continued)

id_tree	id_sh	tr	DOY										
			127	134	141	147	155	161	169	176			
30	b	N+H-	1	1	2	3	3	3	3	3	3	1	
31	b	N+H-	3	3	3	3	4	4	4	4	0	0	
32	b	N+H-	2	2	3	3	3	4	4	3	0	0	
33	b	N+H-	1	0	3	3	4	4	4	4	0	0	
34	b	N+H-	2	1	1	0	1	1	1	1	2	2	
35	a	N-H-	1	1	3	3	4	4	4	4	0	0	
36	a	N-H-	1	1	0	0	1	2	3	3	3	3	
37	a	N-H-	1	0	2	2	3	4	2	0	0	0	
38	a	N-H-	1	0	0	0	2	2	1	0	0	0	
39	a	N-H-	1	1	2	3	3	4	4	2	2	2	
40	a	N-H-	1	2	3	4	4	4	3	0	0	0	
41	a	N-H-	1	0	1	1	3	4	4	0	0	0	
42	a	N-H-	1	0	3	2	4	4	3	0	0	0	

Table A.3 (continued)

id_tree	id_sh	tr	DOY													
			127	134	141	147	155	161	169	176						
43	a	N-H-	1	1	1	2	2	3	3	0						
44	a	N-H-	0	1	2	2	2	3	4	0						
35	b	N-H-	2	1	3	3	3	3	3	0						
36	b	N-H-	1	1	0	1	1	1	3	1						
37	b	N-H-	2	2	3	3	4	4	3	0						
38	b	N-H-	1	1	0	1	1	1	1	0						
39	b	N-H-	1	1	2	2	3	3	4	3						
40	b	N-H-	2	1	3	3	4	2	2	0						
41	b	N-H-	2	2	3	3	3	4	4	0						
42	b	N-H-	0	0	0	1	3	3	3	0						
43	b	N-H-	2	2	3	3	4	4	4	0						
44	b	N-H-	0	0	0	0	2	3	3	0						

Appendix B

Supplementary Information - Chapter 2

B.1 Model processes and parameters

The processes in equation (2.1) in Chapter 2 are reported below. Details of state variables and parameters are reported in Table B.1.

$$\begin{aligned}\Theta_S &= \sigma_C S \left[\left(1 + \frac{S}{\nu} \right) \left(1 + \frac{C_S}{S l_C} \right) \right]^{-1} \\ \Gamma_S &= \varphi_C \frac{C_S N_S}{S} S \\ \Upsilon_C &= \left(\frac{C_S}{S} - \frac{C_R}{R} \right) (SR)^q \cdot (S^q + R^q)^{-1} \\ \Delta_C &= \alpha \frac{C_S}{S} A \\ \Upsilon_N &= \left(\frac{N_R}{R} - \frac{N_S}{S} \right) (SR)^q \cdot (S^q + R^q)^{-1} \\ \Lambda_S &= \varphi_N \frac{C_S N_S}{S} S\end{aligned}$$

$$\begin{aligned}
\Delta_N &= \alpha \frac{N_S}{S} A \\
\Omega_S &= \frac{\lambda^\eta}{\lambda^\eta + t^\eta} \frac{C_S}{S} \frac{N_S}{S} S \\
\Psi &= \begin{cases} \theta A & \text{if } \theta \cdot A \leq \kappa \Omega_S \left(1 - \beta_1 \frac{\frac{D}{S}^{\delta_1}}{\pi_1^{\delta_1} + \frac{D}{S}^{\delta_1}}\right) \\ \kappa \Omega_S \left(1 - \beta_1 \frac{\frac{D}{S}^{\delta_1}}{\pi_1^{\delta_1} + \frac{D}{S}^{\delta_1}}\right) & \text{otherwise} \end{cases} \\
\Gamma_R &= \varphi_C \frac{C_R}{R} \frac{N_R}{R} R \\
\Theta_R &= R \left[\left(1 + \frac{R}{\nu}\right) \left(1 + \frac{N_R}{R \iota_N}\right) \right]^{-1} \\
\Lambda_R &= \varphi_N \frac{C_R}{R} \frac{N_R}{R} R \\
\Omega_R &= \frac{\lambda^\eta}{\lambda^\eta + t^\eta} \frac{C_R}{R} \frac{N_R}{R} R \\
E_C &= \varepsilon_C \alpha \frac{C_S}{S} A \\
E_N &= \varepsilon_N \alpha \frac{N_S}{S} A \\
\Xi &= \begin{cases} \xi \theta \left(1 - \beta_2 \frac{\frac{D}{S}^{\delta_2}}{\pi_2^{\delta_2} + \frac{D}{S}^{\delta_2}}\right) - \mu & \text{if } \theta \cdot A \leq \kappa \Omega_S \left(1 - \beta_1 \frac{\frac{D}{S}^{\delta_1}}{\pi_1^{\delta_1} + \frac{D}{S}^{\delta_1}}\right) \\ \xi \kappa \Omega_S \left(1 - \beta_1 \frac{\frac{D}{S}^{\delta_1}}{\pi_1^{\delta_1} + \frac{D}{S}^{\delta_1}}\right) \frac{1}{A} \left(1 - \beta_2 \frac{\frac{D}{S}^{\delta_2}}{\pi_2^{\delta_2} + \frac{D}{S}^{\delta_2}}\right) - \mu & \text{otherwise} \end{cases}
\end{aligned}$$

Table B.1: Model variables and parameters.

Variable	Dim.	Description	Source
S	g	Shoot structural dry mass	
R	g	Root structural dry mass	
C_S	g	Shoot carbon substrate	
C_R	g	Root carbon substrate	
N_S	g	Shoot nitrogen substrate	
N_R	g	Root nitrogen substrate	
D	DU	Plant induced defences	
A	ind.	Aphid population	
Parameter	Value	Description	Source
C and N assimilation			
σ_C	0.1	Assimilation rate of C	Thornley (1998)
σ_N	[1.2; 12; 120] 10^{-3} ^a	Assimilation rate of N	Zaffaroni et al. (2020)
ν	1000	Shoot (root) mass halving substrate assimilation due to self shading (competition)	Thornley (1998)
l_C	0.1	Semi-saturation C concentration	Thornley (1998)
l_N	0.01	Semi-saturation N concentration	Thornley (1998)
C and N substrates allocation to plant growth			
φ_C	0.50	Unit of substrate C per unit of structural dry mass	Thornley (1998)
φ_N	$2.50 \cdot 10^{-2}$	Unit of substrate N per unit of structural dry mass	Thornley (1998)
k	[18.2; 182; 1820] ^b	Maximum rate of substrate utilization	Zaffaroni et al. (2020)
η	73	Switch-off function of plant growth: steepness	Zaffaroni et al. (2020)
λ	169	Switch-off function of plant growth: date of equal partitioning between growth and reserves	Zaffaroni et al. (2020)
Transport			
q	0.86	Plant architecture scaling parameter	Zaffaroni et al. (2020)
Defences development			
α	0.02	Allocation of substrates to defences per unit of aphid	Zaffaroni et al. (2020)
ε_C	$5 \cdot 10^{-2}$	Conversion efficiency of C substrate in defences	Schoonhoven et al. (2005)
ε_N	1	Conversion efficiency of N substrate in defences	Schoonhoven et al. (2005)
Aphid			
θ	$1.12 \cdot 10^{-3}$	Maximum food intake per aphid	Day and Irzykiewicz (1953)
ξ	171	Maximum conversion efficiency of ingested food into descendants	Saguez et al. (2005)
μ	0.04	Aphid natural mortality rate	Gange et al. (1999)
μ_P	[0; 0.0625; 0.125] ^c	Aphid natural mortality rate	Gange et al. (1999)
π_1	$8.52 \cdot 10^{-3}$	Switch-on function of defences protected phloem fraction: defences concentration at which defences effect is half-saturated	Zaffaroni et al. (2020)
δ_1	0.65	Switch-on function of defences protected phloem fraction: steepness	Zaffaroni et al. (2020)
π_2	0.05	Switch-on function of defences induced mortality/repulsion rate: defences concentration at which defences effect is half-saturated	Zaffaroni et al. (2020)
δ_2	118	Switch-on function of defences induced mortality/repulsion rate: steepness	Zaffaroni et al. (2020)
β_1	1	Switch-on function of defences protected phloem fraction: asymptotic value	Zaffaroni et al. (2020)
β_2	28	Switch-on function of defences induced mortality/repulsion rate: asymptotic value	Zaffaroni et al. (2020)

^aValues refer to low, intermediate and high fertilization treatments, respectively.

^bValues refer to low, intermediate and high irrigation treatments, respectively

^cValues refer to no, intermediate and high pesticide treatments, respectively

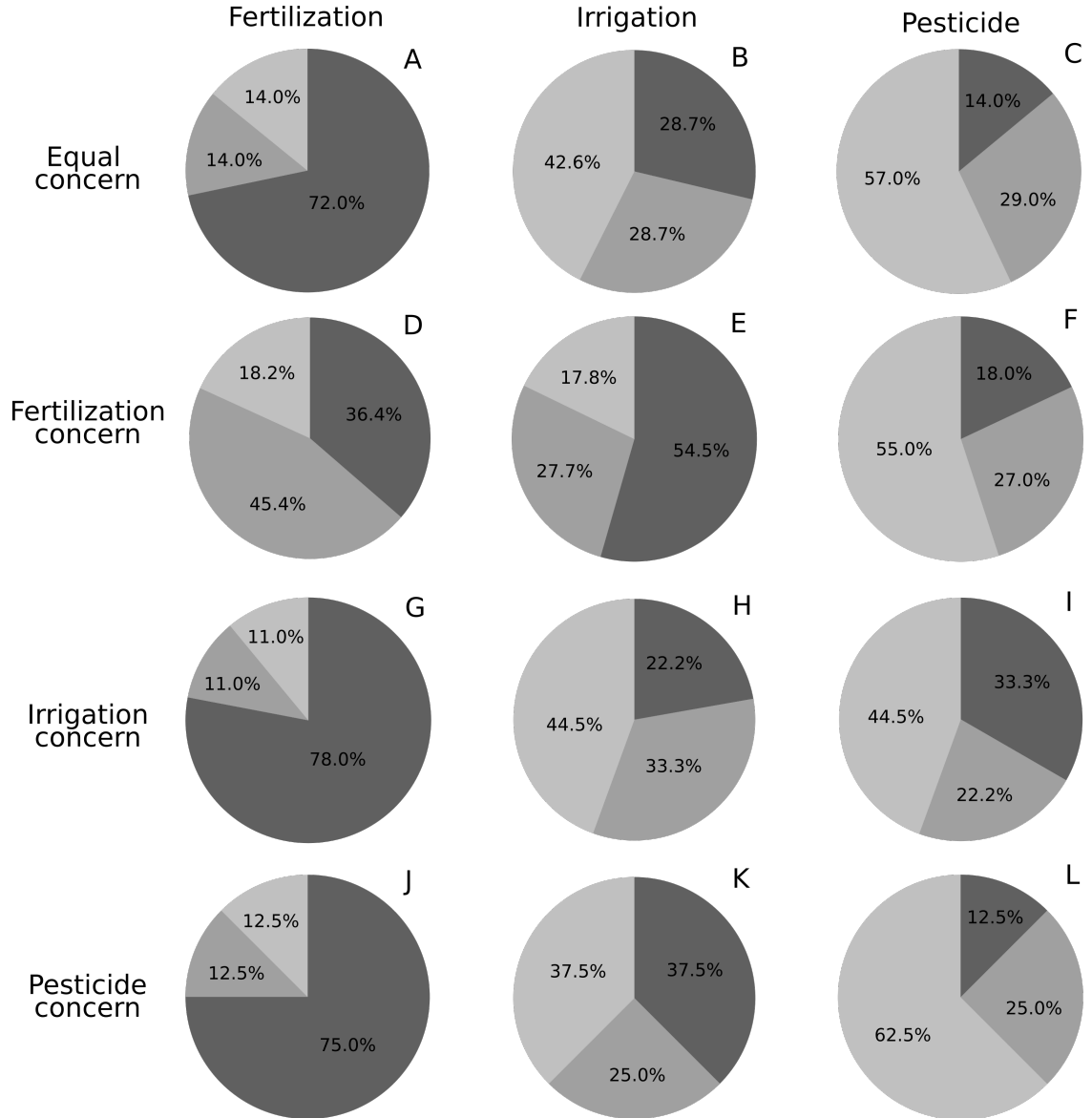


Figure B.1: Response of the percentage of Pareto-optimal scenarios characterized by low (light grey), intermediate (grey) and high (dark grey) fertilization, irrigation or pesticide applications to weights combinations (w_n, w_h and w_p). In the equal concern case (A-B-C) $w_n = w_h = w_p = 0.33$, in the fertilization concern case (D-E-F) $w_n = 0.9, w_h = w_p = 0.05$, in the irrigation concern case (G-H-I) $w_h = 0.9, w_n = w_p = 0.05$, and in the pesticide concern case (J-K-L) $w_p = 0.9, w_n = w_h = 0.05$.

Appendix C

Supplementary Information - Chapter 3

C.1 Scaling of the interference strength parameter V_i ($i = 1, 2$)

To assure that the value of parameter V_1 in equation (3.4) in Chapter 3 is biologically relevant when the scaling parameter β_1 changes, we assumed that, at the “reference” value of aphids hosting capacity $h = h_R$, the value of the interference function is the same independently from the interference mechanism (*i.e.* the value of β_1), all other things being equal. It follows that:

$$f(N_C; \alpha_1, V_1 = A, \beta_1 = a, h = h_R) = f(N_C; \alpha_1, V_1 = B, \beta_1 = b, h = h_R) \quad (\text{C.1})$$

$$\frac{1}{1 + \left(A \frac{N_C}{h_R^a}\right)^{\alpha_1}} = \frac{1}{1 + \left(B \frac{N_C}{h_R^b}\right)^{\alpha_1}} \quad (\text{C.2})$$

which solved results in the expression:

$$B = \frac{A}{h_R^{a-b}} \quad (\text{C.3})$$

By considering $a = 1$ and consequently $A = \nu_1$ (i.e. the strength in case of direct interference), we obtained the value of the strength parameter ($B = V_1$) for a general case where $b = \beta_1 \neq 1$ (corresponding to equation (3.5) in Chapter 3):

$$V_1 = \frac{\nu_1}{h_R^{1-\beta_1}} \quad (\text{C.4})$$

Similar considerations were done for the strength parameter of the emigration interference (V_2).

C.2 Response of R_0 to μ under different parametrizations

We randomly extracted 100,000 sets of model parameters from a range $[0.5P; 2P]$, where P is the default value of the parameters, except for the parameters $\alpha = 1$, $\nu = 3.5$ (weak interference) or $\nu = 10.5$ (strong interference). For each parameters set, we investigated whether the response of R_0 to variation of μ (μ varying between 0.04 and 0.15) is monotonic or non monotonic. In the weak interference scenario, the response is monotonic in 44% and non-monotonic in 56% of the considered parametrizations. In the strong interference scenario, the response is monotonic in 0.01 % and non monotonic in 99% of the considered parametrizations. Some examples of parametrizations

corresponding to non-monotonic responses of R_0 to varying μ are reported in figure C.1.

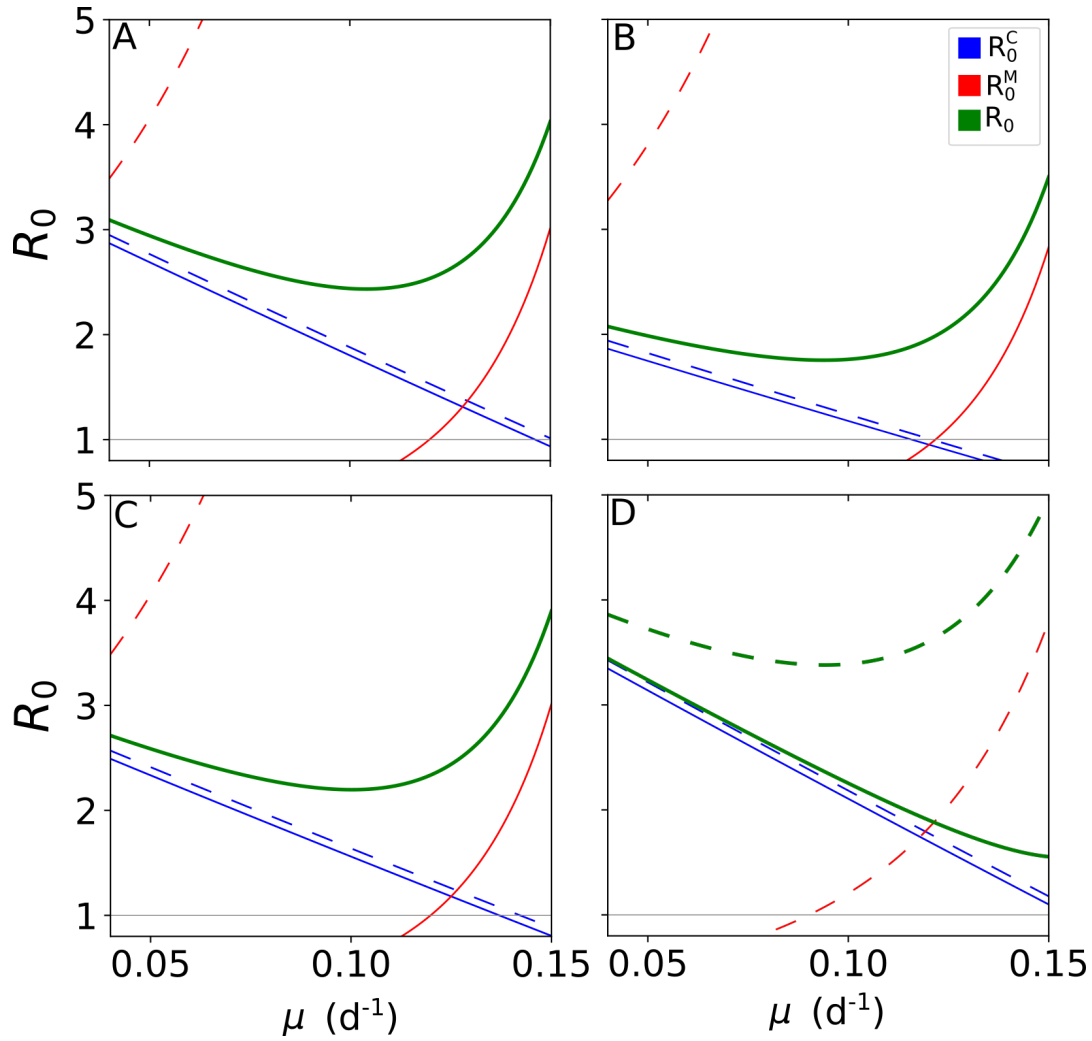


Figure C.1: Example of parametrizations resulting in non monotonic responses of R_0 to varying value of μ (A,B,C) in the strong ($\nu = 10.5$) and (D) in the weak ($\nu = 3.5$) interference scenarios. Dashed lines identify weak interference scenario and continuous lines strong interference scenarios (note: the value of R_0^C is insensitive to the value of ν , so dashed and continuous blue lines overlap).

C.3 Non dimensionalization

The system of differential equations corresponding to the model, after integrating the assumption in Chapter 3 (N_P constant and $\lambda = M\tau$), and after including the dynamics of the population size of colonizer ($N_P = X_C + Z_C$) and migrant ($N_M = X_M + Z_M$) aphids, can be rewritten as

$$\begin{cases} \dot{I} = (\Lambda_C \delta_C Z_C N_P + \Lambda_M \delta_M f(N_C) Z_M N_P) \left(\frac{N_P - I}{N_P} \right) - (\rho + \theta) I \\ \dot{N}_C = r N_C \left(1 - \frac{N_C}{h} \right) - \mu N_C \\ \dot{Z}_C = \Lambda_C \epsilon_C \frac{I}{N_P} (N_C - Z_C) - (\gamma + \mu) Z_C \\ \dot{N}_M = \tau (M - g(N_C) N_M) \\ \dot{Z}_M = \pi \tau M + \Lambda_M \epsilon_M f(N_C) \frac{I}{N_P} (N_M - Z_M) - (\gamma + \tau g(N_C)) Z_M \end{cases} \quad (\text{C.5})$$

Where

$$f(N_C) = \frac{1}{1 + \left(\frac{\nu_1}{h_R^{1-\beta_1}} \frac{N_C}{h^{\beta_1}} \right)^{\alpha_1}} \quad (\text{C.6})$$

$$g(N_C) = 1 + \left(\frac{\nu_2}{h_R^{1-\beta_2}} \frac{N_C}{h^{\beta_2}} \right)^{\alpha_2} \quad (\text{C.7})$$

The equation of \dot{S} , \dot{X}_C and \dot{X}_M have been omitted because they can be derived from $S = N_P - I$, $X_C = N_C - Z_C$ and $X_M = N_M - Z_M$, respectively. To highlight the colonizer aphids carrying capacity, the equation for N_C can be rewritten as

$$\dot{N}_C = r' \left(1 - \frac{N_C}{\kappa} \right) \quad (\text{C.8})$$

where

$$r' = r - \mu \quad \kappa = h \left(1 - \frac{\mu}{r} \right) \quad (\text{C.9})$$

By making the transformation

$$I = N_P \hat{I} \quad (\text{C.10})$$

$$N_C = \kappa \hat{N}_C \quad (\text{C.11})$$

$$Z_C = \kappa \hat{Z}_C \quad (\text{C.12})$$

$$N_M = M \hat{N}_M \quad (\text{C.13})$$

$$Z_M = M \hat{Z}_M \quad (\text{C.14})$$

$$t = \frac{\hat{t}}{\rho + \theta} \quad (\text{C.15})$$

and writing

$$f(\hat{N}_C) = \frac{1}{1 + \left(\frac{\nu_1 \hat{N}_C}{\hat{h}_R^{1-\beta_1} \hat{h}^{\beta_1}} \right)^{\alpha_1}} = f(\cdot) \quad (\text{C.16})$$

$$g(\hat{N}_C) = 1 + \left(\frac{\nu_2 \hat{N}_C}{\hat{h}_R^{1-\beta_2} \hat{h}^{\beta_2}} \right)^{\alpha_2} = g(\cdot) \quad (\text{C.17})$$

where \hat{h}_R , \hat{h}_1 and \hat{h}_2 are the non dimensional version of the parameter h_R , h_1 and h_2 , that is

$$h_R = \kappa \hat{h}_R \quad (\text{C.18})$$

$$h_1 = \kappa \hat{h}_1 \quad (\text{C.19})$$

$$h_2 = \kappa \hat{h}_2 \quad (\text{C.20})$$

we obtained a nondimensionalized version of the original model equations:

$$\begin{cases} \dot{\hat{I}} = \left(i_C \hat{Z}_C + i_M f(\cdot) \hat{Z}_M \right) (1 - \hat{I}) - \hat{I} \\ \dot{\hat{N}}_C = q \hat{N}_C (1 - \hat{N}_C) \\ \dot{\hat{Z}}_C = a_C (\hat{N}_C - \hat{Z}_C) \hat{I} - m \hat{Z}_C \\ \dot{\hat{N}}_M = e (1 - g(\cdot)) \hat{N}_M \\ \dot{\hat{Z}}_M = u + a_M f(\cdot) (\hat{N}_M - \hat{Z}_M) \hat{I} - (d + eg(\cdot)) \hat{Z}_M \end{cases} \quad (\text{C.21})$$

The composite parameters are given by

$$i_C = \frac{\Lambda_C \delta_C \kappa}{\rho + \theta} \quad i_M = \frac{\Lambda_M \delta_M M}{\rho + \theta} \quad q = \frac{r'}{\rho + \theta} \quad (\text{C.22})$$

$$a_C = \frac{\Lambda_C \epsilon_C}{\rho + \theta} \quad m = \frac{\gamma + \mu}{\rho + \theta} \quad u = \frac{\pi \tau}{\rho + \theta} \quad (\text{C.23})$$

$$a_M = \frac{\lambda_M \epsilon_M}{\rho + \theta} \quad d = \frac{\gamma}{\rho + \theta} \quad e = \frac{\tau}{\rho + \theta} \quad (\text{C.24})$$

C.4 Basic reproduction number

Considering the infected subsystem (*i.e.* the equations that describe the production of new infections and changes in state among infected individuals) of the nondimensional system in equation (C.21), linearized around the infection-free steady state, and assuming that $u = 0$, which is equivalent to assume that only aviruliferous migrant aphids immigrate into the system, we

determined the basic reproduction number of the disease, R_0 , using the next generation method (Diekmann et al., 2010).

Matrix F describes the production of new infections and matrix V describes changes in state.

$$F = \begin{bmatrix} 0 & i_c & i_m f(\cdot) \\ a_C \hat{N}_C & 0 & 0 \\ a_M f(\cdot) \hat{N}_M & 0 & 0 \end{bmatrix} \quad V = \begin{bmatrix} -1 & 0 & 0 \\ 0 & -m & 0 \\ 0 & 0 & -(d + eg(\cdot)) \end{bmatrix} \quad (\text{C.25})$$

$$F(-V)^{-1} = \begin{bmatrix} 0 & \frac{i_C}{m} & \frac{i_m f(\cdot)}{d+eg(\cdot)} \\ a_C \hat{N}_C & 0 & 0 \\ a_M f(\cdot) \hat{N}_M & 0 & 0 \end{bmatrix} \quad (\text{C.26})$$

The basic reproduction number is the dominant eigenvalue of matrix $F(-V)^{-1}$. Note that, from equation (C.21), *i*) the value of \hat{N}_C at the infection-free steady state is $\hat{N}_C = 1$ when $q > 0$ and $\hat{N}_C = 0$ when $q < 0$, and *ii*) the value of \hat{N}_M at the infection-free steady state is $\hat{N}_M = \frac{1}{g(\cdot)}$.

$$R_0 = \frac{i_C a_C}{m} + \frac{i_M f(\cdot)^2 a_M}{g(\cdot)(d + eg(\cdot))} = R_0^C + R_0^M \quad \text{if } q \geq 0 \quad (\text{C.27})$$

$$R_0 = \frac{i_M f(\cdot)^2 a_M}{g(\cdot)(d + eg(\cdot))} = R_0^M \quad \text{if } q < 0 \quad (\text{C.28})$$

where R_0^C considers the virus transmission by colonizer aphids and R_0^M considers the virus transmission by migrant aphids.

The basic reproduction number can be rewritten with biological parameters, considering that at the infection-free steady state the value of $N_C = \kappa$

if $r > \mu$, or $N_C = 0$, otherwise, and the value of $N_M = \frac{M}{g(\kappa)}$. The interference functions $f(\cdot)$ and $g(\cdot)$ are equal to 1 if $N_C = 0$.

$$R_0 = \frac{1}{\rho + \theta} \left(\frac{\Lambda_C^2 \delta_C \epsilon_C \kappa}{\gamma + \mu} + \frac{\Lambda_M^2 \delta_M \epsilon_M f(\kappa)^2 M}{g(\kappa)(\gamma + \tau g(\kappa))} \right) \text{ if } r > \mu \quad (\text{C.29})$$

$$R_0 = \frac{\Lambda_M^2 \delta_M \epsilon_M M}{(\rho + \theta)(\gamma + \tau)} \text{ if } r < \mu \quad (\text{C.30})$$

C.5 Epidemic equilibrium

The equilibrium values of \hat{N}_C come from a solution of

$$0 = q\hat{N}_C(1 - \hat{N}_C) \quad (\text{C.31})$$

When $q = r - \mu < 0$, the equilibrium value of \hat{N}_C is 0, when $q = r - \mu \geq 0$ the equilibrium value of \hat{N}_C is 1. The equilibrium value of \hat{N}_M comes from the solution of

$$0 = e(1 - g(\cdot)\hat{N}_M) \quad (\text{C.32})$$

$$\hat{N}_M = \frac{1}{g(\cdot)} \quad (\text{C.33})$$

$\hat{\mathbf{N}}_C = \mathbf{0}$

If the equilibrium values of \hat{N}_C is 0, it follows that the equilibrium value of \hat{Z}_C is 0, $f(\cdot) = 1$, $g(\cdot) = 1$ and consequently the equilibrium value of $\hat{N}_M = 1$. The equilibrium values of \hat{I} and \hat{Z}_M come from a solution to

$$0 = \left(i_M \hat{Z}_M\right)(1 - \hat{I}) - \hat{I} \quad (\text{C.34})$$

$$0 = u + a_M(1 - \hat{Z}_M)\hat{I} - (d + e)\hat{Z}_M \quad (\text{C.35})$$

The second equation indicates

$$\hat{Z}_M = \frac{u + a_M \hat{I}}{a_M \hat{I} + d + e} \quad (\text{C.36})$$

Back subbing into the first equation

$$0 = \left(i_M \frac{u + a_M \hat{I}}{a_M \hat{I} + d + e}\right)(1 - \hat{I}) - \hat{I} \quad (\text{C.37})$$

- $u = 0$

If $u = 0$, which is equivalent to assume that all the aphids entering the system are non-viruliferous, the equation (C.37) can be rewritten as

$$0 = \hat{I} \left(\left(\frac{i_M a_M}{a_M \hat{I} + d + e} \right) (1 - \hat{I}) - 1 \right) \quad (\text{C.38})$$

which means there is one root when $\hat{I} = 0$ and one root comes from the solution to

$$\frac{i_M a_M}{a_M \hat{I} + d + e} = \frac{1}{(1 - \hat{I})} \quad (\text{C.39})$$

After algebraic manipulations, recalling that $R_0^M = \frac{i_M a_M}{d+e}$ when $f(\cdot) = g(\cdot) = 1$, the solution to equation (C.39) is

$$\hat{I} = \frac{R_0^M - 1}{R_0^M + a_M/(d+e)} \quad (\text{C.40})$$

which, for $R_0^M > 1$, is always in the biologically-meaningful interval $[0,1]$.

- **$u > 0$**

If $u > 0$, which is equivalent to assume that migrant aphids can bear the disease from outside the system, $\hat{I} = 0$ is not a solution of equation (C.37), so it is acceptable to divide equation (C.37) by \hat{I} , leading to

$$\left(\frac{i_M u}{\hat{I}(a_M \hat{I} + d + e)} + \frac{i_M a_M}{a_M \hat{I} + d + e} \right) (1 - \hat{I}) - 1 = 0 \quad (\text{C.41})$$

$$\frac{i_M u}{\hat{I}(a_M \hat{I} + d + e)} + \frac{i_M a_M}{a_M \hat{I} + d + e} = \frac{1}{1 - \hat{I}} \quad (\text{C.42})$$

$$b_1(\hat{I}) = v(\hat{I}) \quad (\text{C.43})$$

The function $b_1(\hat{I})$ is: *i*) always positive for $\hat{I} \in (0, 1]$; *ii*) always decreasing; *iii*) has $b_1(\hat{I}) \rightarrow +\infty$ as $\hat{I} \rightarrow 0^+$. The function $v(\hat{I})$ is always positive for $\hat{I} \in [0, 1)$; *ii*) is always increasing; *iii*) has $v(0) = 1$ and has $v(\hat{I}) \rightarrow +\infty$ as $\hat{I} \rightarrow 1^-$. Taken together the properties of $b_1(\hat{I})$ and $v(\hat{I})$, we can conclude that there is always a single biologically meaningful root with $\hat{I} \in (0, 1)$ (irrespective of the values of parameters) (figure C.2A).

$$\hat{N}_C = 1$$

If the equilibrium values of \hat{N}_C is 1, it follows that $f(\cdot) = \frac{1}{1 + \left(\frac{\nu_1}{\hat{h}_R^{1-\beta_1} \hat{h}^{\beta_1}} \right)^{\alpha_1}} =$

$f, g(\cdot) = 1 + \left(\frac{\nu_2}{\hat{h}_R^{1-\beta_2} \hat{h}^{\beta_2}} \right)^{\alpha_2} = g$, and the equilibrium value of $\hat{N}_M = \frac{1}{g}$.

The equilibrium value of \hat{I} , \hat{Z}_C and \hat{Z}_M come from a solution to

$$0 = (i_C \hat{Z}_C + i_M f \hat{Z}_M)(1 - \hat{I}) - \hat{I} \quad (C.44)$$

$$0 = a_C(1 - \hat{Z}_C)\hat{I} - m\hat{Z}_C \quad (C.45)$$

$$0 = u + a_M f \left(\frac{1}{g} - \hat{Z}_M \right) \hat{I} - (d + eg)\hat{Z}_M \quad (C.46)$$

The second and third equations indicates

$$\hat{Z}_C = \frac{a_C \hat{I}}{a_C \hat{I} + m} \quad (C.47)$$

$$\hat{Z}_M = \frac{ug + a_M f \hat{I}}{g(a_M f \hat{I} + d + eg)} \quad (C.48)$$

Back subbing into the first equation

$$0 = \left(i_C \frac{a_C \hat{I}}{a_C \hat{I} + m} + i_M f \frac{ug + a_M f \hat{I}}{g(a_M f \hat{I} + d + eg)} \right) (1 - \hat{I}) - \hat{I} \quad (C.49)$$

• **$u = 0$**

If $u = 0$, equation (C.49) can be rewritten as

$$0 = \hat{I} \left(\left(\frac{i_C a_C}{a_C \hat{I} + m} + \frac{i_M a_M f^2}{g(a_M f \hat{I} + d + eg)} \right) (1 - \hat{I}) - 1 \right) \quad (C.50)$$

which means there is one root when $\hat{I} = 0$ and others come from the solutions to

$$\left(\frac{i_c a_c}{a_c \hat{I} + m} + \frac{i_M a_M f^2}{g(a_M f \hat{I} + d + eg)} \right) = \frac{1}{(1 - \hat{I})}, \quad (\text{C.51})$$

The exact equilibrium value of \hat{I} can be found by solving equation (C.51)

$$\left(\frac{i_c a_c / m}{a_c \hat{I} / m + 1} + \frac{i_M a_M f^2 / (g(d + eg))}{a_M f \hat{I} / (d + eg) + 1} \right) = \frac{1}{(1 - \hat{I})} \quad (\text{C.52})$$

$$\frac{R_0^C}{(a_c \hat{I} / m) + 1} + \frac{R_0^M}{a_M f \hat{I} / (d + eg) + 1} = \frac{1}{1 - \hat{I}} \quad (\text{C.53})$$

After algebraic manipulation equation (C.53) can be written as

$$a_2 \hat{I}^2 + a_1 \hat{I} + a_0 = 0 \quad (\text{C.54})$$

$$p(\hat{I}) = 0 \quad (\text{C.55})$$

Where

$$a_2 = -\left(R_0^C \frac{a_M f}{d + eg} + R_0^M \frac{a_C}{m} + \frac{a_C a_M f}{m(d + eg)} \right) \quad (\text{C.56})$$

$$a_1 = \left(\frac{a_M f}{d + eg} (R_0^C - 1) + \frac{a_C}{m} (R_0^M - 1) - (R_0^C + R_0^M) \right) \quad (\text{C.57})$$

$$a_0 = R_0^C + R_0^M - 1 \quad (\text{C.58})$$

Since $a_2 < 0$, when $a_0 > 0$, which corresponds to $R_0^C + R_0^M = R_0 > 1$, the quadratic function $p(\hat{I})$ in equation (C.54) has two roots, one posi-

tive and one negative. Since $p(1) = a_2 + a_1 + a_0 = -\left(\frac{a_C a_M f}{m(d+eg)} + \frac{a_M f}{d+eg} + \frac{a_C}{m} + 1\right) < 0$ the positive root must have $\hat{I} < 1$ for the intermediate value theorem, thus it is in the biologically meaningful interval $[0, 1]$.

We can conclude that equation (C.54) has one biologically meaningful solution, expressed as

$$\hat{I} = \frac{-a_1 - \sqrt{a_1^2 - 4a_2a_0}}{2a_2} \quad (\text{C.59})$$

- $u > 0$

If $u > 0$, $\hat{I} = 0$ is not a solution of equation (C.49), so it is acceptable to divide equation (C.49) by \hat{I} , leading to

$$\begin{aligned} & \left(\frac{i_C a_C}{a_C \hat{I} + m} + \frac{i_m f u}{\hat{I}(a_M f \hat{I} + d + eg)} + \frac{i_M a_M f^2}{g(a_M f \hat{I} + d + eg)} \right) (1 - \hat{I}) - 1 = 0 \\ & \left(\frac{i_C a_C}{a_C \hat{I} + m} + \frac{i_m f u}{\hat{I}(a_M f \hat{I} + d + eg)} + \frac{i_M a_M f^2}{g(a_M f \hat{I} + d + eg)} \right) = \frac{1}{1 - \hat{I}} \\ & b_2(\hat{I}) = v(\hat{I}) \end{aligned} \quad (\text{C.60})$$

The function, $v(\hat{I})$ has the same properties as before, whereas the function $b_2(\hat{I})$ is: *i*) always positive on the interval $\hat{I} \in (0, 1)$; *ii*) always decreasing; *iii*) has $b_2(\hat{I}) \rightarrow +\infty$ as $\hat{I} \rightarrow 0^+$. Taken together with properties of $v(\hat{I})$ this mean there is always a single biologically meaningful root with $\hat{I} \in (0, 1)$ (irrespective of the values of parameters) (figure C.2B).

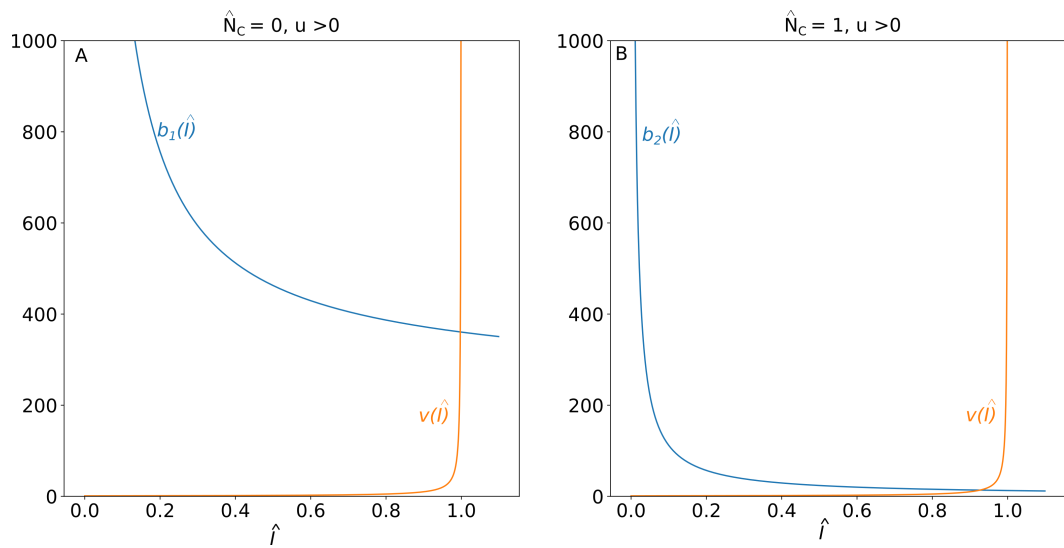


Figure C.2: Graphical representation of A) functions $b_1(\hat{I})$ and $v(\hat{I})$ and B) functions $b_2(\hat{I})$ and $v(\hat{I})$. The intersection of the two functions identify the equilibrium value of \hat{I} . $\nu = 7.0$, $\pi = 0.1$, other parameters are set to default value.

The model behaviour at equilibrium is summarize in Table 3.3 in Chapter 3. Moreover, for 5000 sets of parameter values randomly selected from the range $[0.8P; 1.2P]$ where P is the default value of the parameters, in all cases the result of the numerical simulation matches the results of the mathematical analysis.

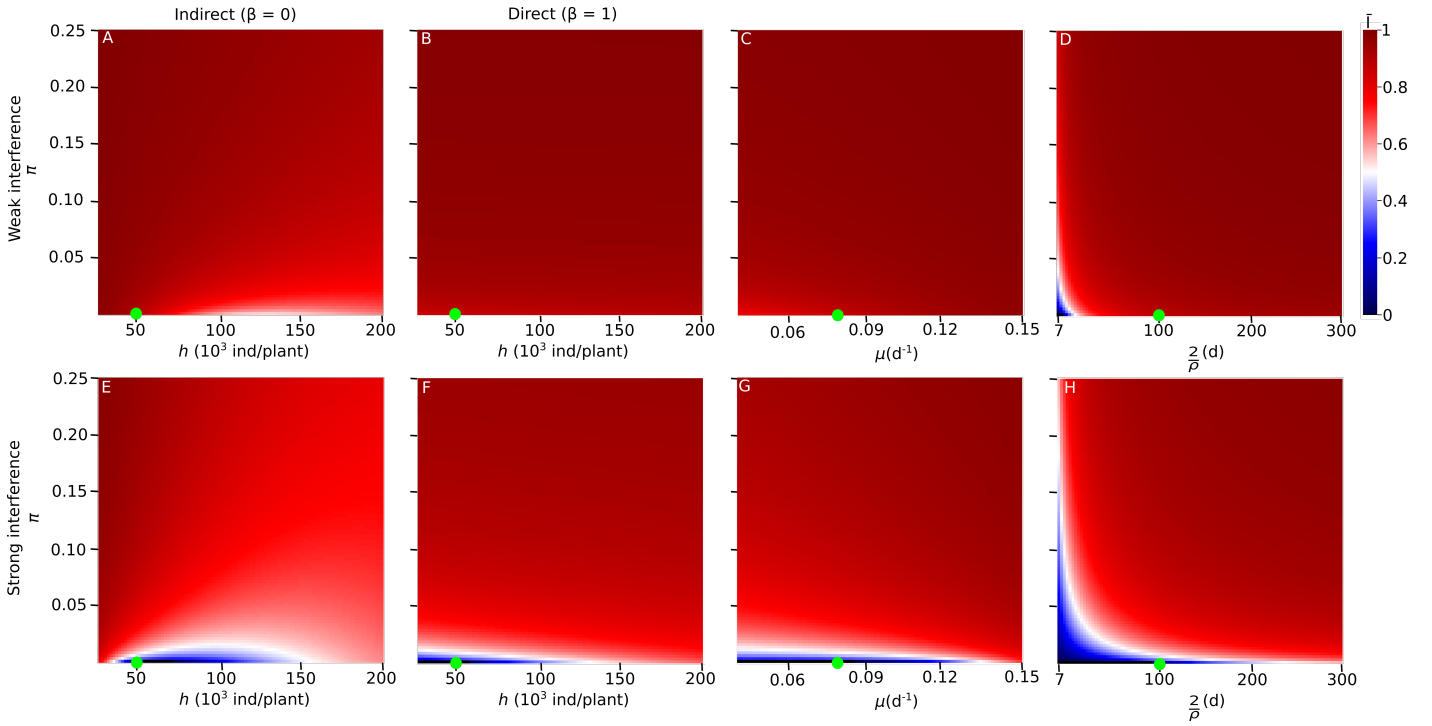


Figure C.3: Response of the density of infected plants at the equilibrium (\bar{I}) to changes in fraction of viruliferous migrant aphids entering the system (π) and aphids hosting capacity (h), both in the indirect ($\beta = 0$, A-E) and direct ($\beta = 1$, B-F) interference scenarios, colonizer aphid mortality (μ , C-G) and surveillance interval ($2/\rho$, D-H). The upper row refers to a weak interference scenario ($\nu = 3.5$) and the bottom row to a strong interference scenario ($\nu = 10.5$). Other model parameters are set to default values.