Emerging themes and approaches in plant virus epidemiology

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Abstract

Plant diseases caused by viruses share many common features with those caused by other pathogen taxa in terms of the host-pathogen interaction, but there are also distinctive features in epidemiology, most apparent where transmission is by vectors. Consequently, the host-virus-vector-environment interaction presents a continuing challenge in attempts to understand and predict the course of plant virus epidemics. Theoretical concepts, based on the underlying biology, can be expressed in mathematical models, and tested through quantitative assessments of epidemics in the field; this remains a goal in understanding why plant virus epidemics occur and how they can be controlled. To this end, this review identifies recent emerging themes and approaches to fill in knowledge gaps in plant virus epidemiology. We review quantitative work on impact of climatic fluctuations and change on plants, virus and vectors under different scenarios where impacts on the individual components of the plant-virus-vector interaction may vary disproportionately; the continuing sometimes discordant debate on host resistance and tolerance as plant defence mechanisms, including aspects of farmer behaviour and attitudes to disease management that may affect deployment in crops; disentangling host-virus-vector-environment interactions as these contribute to temporal and spatial disease progress in field populations, computational techniques for estimating epidemiological parameters from field observations, and the use of optimal control analysis to assess disease control options. We end by proposing new challenges and questions in plant virus epidemiology.

Introduction

Epidemiology is the study of the rates of temporal and spatial change of disease in populations and the determining factors underlying change. Of necessity, epidemiology is a quantitative science. For plant viruses, transmission is a key determining factor of disease dynamics and, in most cases, depends on the interactions of viruses, host plants, and vectors, subject to the biotic and abiotic environment (Jeger 2020). The complexity of these interactions makes field-based studies difficult to interpret without supporting experimental studies, either laboratory or microcosm-based, that provide information on the parameters involved in transmission. Problems then arise in scaling up information to the field. The expectation has been that mathematical models based on the known or assumed biology can make a bridge between the specific information provided by laboratory or microcosm studies and field observations on disease dynamics; whether to provide a greater understanding, to test hypotheses, to make predictions on future change, or to improve disease management through deployment of host resistance and tolerance, phytosanitation, and vector control (Jeger et al. 2004).

In practice, there are few examples of such bridges being made and models used, largely because of the broader biotic and abiotic environment of plant and vector populations, including the impact of climatic fluctuations and the spatial component of both within- and between-spread, factors which add further layers of ecological complexity (Jeger et al., 2022). Any approach to model plant virus epidemics needs to recognise when assumptions previously made are no longer tenable (Jones et al. 2014a), and when an epidemiological approach must be subsumed into an overall systems approach based upon modelling constraints for a particular crop (Chavez et al., 2022). There is a need to consider wild plants as a major biotic factor in the epidemiology of viruses in crops. Much activity may occur in wild plant populations but has been rarely modelled (Fabre et al. 2012, 2015: Djidjou-Demasse et al. 2017). How to address these challenges is an emerging theme in plant virus epidemiology.

Thematic areas for which gaps in current modeling effort in plant virus epidemiology were identified by Jeger et al. (2017) are shown in Box 1. Although directed mainly at modeling, these areas are relevant for all aspects of quantitative epidemiological research and disease management. These areas of identified gaps are re-examined in this review in which we provide an update on recent work and discuss emerging themes as both challenges and opportunities in plant virus epidemiology and disease management. We review recent work on climatic influences on plant virus diseases, where predictions can be made on the impact of climate change, different strategies for disease control including plant responses to virus infection and disease development, and the different approaches that can be taken in disentangling the virus-vector-host interaction. These approaches will be illustrated by the extent to which spatial and landscape aspects can be included in epidemiological analysis, the assessment of the consequences of virus manipulation of hosts and vectors in the field, and the widespread occurrence of co-infections in plants. We also note where new applications of statistical and computational techniques have been made in quantitative epidemiology and finish by proposing new challenges, questions, and opportunities for research.

Climate change and fluctuations

More than half of current human infectious diseases, including those caused by viruses and vector-borne pathogens, have been aggravated by climate change (Mora et al. 2022). Largely detrimental effects have been noted on the emergence, transmission, and consequences on viruses of animals and plants (Dash et al. 2021). For plant viruses, elevated CO_2, increasing temperature, changes in water availability and extreme events will have effects on viruses through changes in plant hosts and vectors (Trebicki 2020). Specific mechanisms for these effects include temperature-sensitive interactions between viruses and host plants, such as the effects of warming on plasmodesmata and systemic cell to cell movement of viruses (Amari et al. 2021).

Impacts of climate change on disease in wild plant populations and communities are also expected (Jeger 2022a). There is a need to consider the crop ecosystem, including managed, wild, and invasive plants more generally. Interactions between wild hosts, crop plants and climate should be studied dynamically – with time (Burdon and Zhan, 2020). In some cases, e.g., *Candidatus* Phytoplasma solani affecting grapevine ('bois noir') and solanaceous crops ('stolbur'), these cultivated crops suffer major damage and loss with infection but are essentially incidental hosts (Quaglimo et al. 2013), being dead ends for the phytoplasma. Bindweed (*Convolvulus arvensis*) and stinging nettle (*Urtica dioica*) are wild hosts for the pathogen and reproductive hosts for the planthopper vector, *Hyalesthes obsoletus (*Cixiidae) and possibly other invasive vectors.

A variety of papers have dealt with the effects of climate change on virus diseases but without explicitly considering a vector. Sardanyés et al. (2022) modelled temperature effects on pepino mosaic virus strains which are mostly seed, mechanically, or water transmitted, although there is some evidence for bumble bees, whiteflies, and *Olpidium* as vectors (Blystad et al. 2015). Virus replication rates were modelled as functions of temperature and incorporated into a Logistic model for a single strain or a Lotka-Volterra model for competition between strains. The time trajectories for virus load within a single plant in both single and mixed infections are shown in Figure 1. This appears to be the first quantitative study on temperature effects on within-cell virus dynamics where there is mixed infection. Similarly Gutiérrez-Sánchez (2023, this issue) has linked modelling with experimental data to look at likely climate change effects on seed transmission and viability and future infection risk. : Environmental conditions predicted under climate change determine infection risk by modulating plant virus vertical transmission and viability of infected seeds.

Effects of climate on population dynamics of insect pests and virus vectors have been noted in extensive multi-year studies in rice and potato (Yamamura et al. 2006; Gutierrez-Illan et al. 2020) with both seasonal and between-year variations. In cases where there is predominantly vector transmission, the effects of temperature on transmission need to be modelled. Gamarra et al. (2020) developed a temperature driven model for potato yellow vein virus transmission by the whitefly *Trialeurodes vaporariorum*, incorporating both a phenology component for the vector and a non-linear dichotomous response for transmission efficiency based on laboratory experiments at constant and fluctuating temperatures. Maps were generated using risk indices derived from the model, evaluated against the current distribution of the virus, and used to predicted areas of high risk where the virus had yet to be reported. Following subsequent surveillance, a first report of the virus was made in western Panama, predicted to be a high-risk area. Simulated maps to 2050 showed a predicted lowering of virus incidence in tropical regions but an increase in temperate regions, a shift in distribution due to projected climate change under a Representative Concentration Pathway (RCP 6.0) scenario with the Community Climate System Model (CCSM).

With plant viruses, there is the need to consider the integrated effects of climate change on hosts, pathogen, and vector, as done for tomato leaf curl disease, *Solanum lycopersicum*, and *Bemisia tabaci* (Ramos et al. 2018, 2019). Three of the four best predictor variables (annual mean temperature, annual precipitation, and mean diurnal temperature range) were common for host and vector, but only the first two were closely aligned (Figure 2). Also, effects of climate change on associated biota such as natural enemies as found in studies of elevated CO_2 and biocontrol of aphid and whitefly vectors need to be integrated (Sun et al. 2011). Other examples based on climatic niche correlative models for the much studied and globally important vector *B. tabaci* are summarized in Table 1.

Other host-virus-vector systems considered include: pineapple wilt viruses/mealybug vectors and IPCC projections (Wei et al. 2020); sugarcane mosaic virus in maize/aphid vectors with socio-economic classifications and climate change models (Li et al. (2022); maize viruses, leaf and planthopper vectors and time-lagged incidences in relation to environmental fluctuations (Reynaud et al. 2009); cucumber mosaic virus, aphid vectors and water deficits under abiotic stress Bergès et al. (2021); and population development rates for potato pathogens and pests, including potato viruses and aphids used as proxies (van der Waals et al. 2021). As well as the monitoring of vector populations, surveillance of plant virus disease has been found important for identifying climate change signals, including remote sensing for tomato yellow leaf curl disease (Oh et al., 2019) and maize streak disease (Chivasa et al. 2020).

Disease management

Disease management, including the control of virus diseases and virus vectors, can rarely be considered as isolated from other plant pest and agronomic practices. This can be illustrated in studies on seed health in vegetatively propagated crops (Thomas-Sharma et al. 2017, Buddenhagen et al. 2022), modelling aphid control in intercropping systems (Allen-Perkins and Estrada, 2019), possible conflicts between management strategies based on insecticide resistance models and epidemiological models (Sisterson, 2022), and management decisions on whether to rehabilitate or renovate perennial crops due to virus disease, ageing, and other performance factors (Somarriba et al., 2021).

Field practices

The effectiveness of virus disease control depends on variation in farming practices and environment. Within- and between-field processes that potentially affect maize lethal necrosis (MLN) disease dynamics, together with management practices that can be used to control the causal viruses, maize chlorotic mottle virus (MCMV) and sugarcane mosaic virus (SCMV), were modelled by Hilker et al. (2017) (see also de Groote et al. 2021). Long-term (cross-season) dynamics of MCMV and SCMV, and MLN prevalence, was modelled for different management strategies and epidemiological scenarios. A baseline parameterization was compared with scenarios where coinfected seed caused increased vertical transmission and where there was exogenous infection. In general, crop rotation practiced in large farms was an effective means of controlling MLN, but eradication was not possible with exogenous infection. The potential of vector control (aphids for SCMV and assumed to be thrips species, but unproven, for MCMV) and rogueing of infected plants was evaluated using a mathematical model for co-infection (Chapwanya et al. 2021). Rogueing was proposed as a viable alternative to crop rotation for smallholder farms, but without considering the behaviour and attitudes of the grower.

Grower behaviour

The likelihood of an individual grower adopting rogueing, or any other disease management strategy, depends on the prevalence of disease. Trade-offs in perceived costs vs. benefits in deciding on control options have been included in models of human disease for some time (Funk et al., 2010; Chang et al., 2020). The simplest class of models allows awareness of disease to spread concurrently with the pathogen, leading to heterogeneous risks of infection (Kiss et al, 2009), a situation analogous to grower awareness of the spread of a plant disease. More complex models allow for behavioural changes by individuals. The most notable examples for human diseases have focused on take-up of prophylactic vaccination (Bauch & Earn, 2004), social distancing (Del Valle et al., 2013) and face masks (Karlsson & Rowlett, 2020).

Fewer modelling studies of this type have focused on plant diseases, although some models do represent control behaviour which depends on an individual growers' assessment of the likely profitability of each action (Milne et al., 2016). The single example specifically targeting a plant virus epidemic (McQuaid et al., 2017a) uses a relatively complex, spatially explicit simulation model of cassava brown streak (McQuaid et al., 2017b) as an underpinning model. Arguably this complexity obscures how the different components interrelate. The key feedback is that decisions made by any one grower affect disease prevalence and so in turn future decisions made by other growers.

A recent study by Murray Watson et al. (2022) attempts to resolve these coupled trade-offs in a deliberately simplified way by integrating aspects of game theory into a simple model of a clean seed system, again using cassava diseases as a motivation. The long-term proportion of growers deploying clean seed depends on the epidemiological and logistical parameters affecting its effectiveness and cost. However, the predictions of the model also depend on how the behavioural component of the model is formulated; in terms of whether growers are assumed to behave according to rational or strategic-adaptive expectations, as well as how precisely growers estimate the current level of risk posed by disease.

Basic epidemiological theory tells us that successful disease management within a population of growers relies on a sufficiently large fraction adopting control (Jeger, 2000). When insecticide sprays are used to control vectored diseases by reducing vector population densities, there can also be issues spatiotemporal synchrony in management, since control is most likely to be successful if done "area-wide" (Bassanezi et al., 2013). Milne et al. (2020) extended modelling behaviour to account for this type of co-operative control scheme, focusing on the bacterial disease huanglongbing, a major threat to commercial citrus production. They used models of "opinion dynamics" (Moussaïd, 2013) to understand the impact of social forces on growers' decisions. Bate et al. (2021) took a somewhat similar approach, using coalition theory (Mesterton-Gibbons et al 2011) to model voluntary participation in a regional biosecurity scheme. These types of idea around how the choices made by individual growers can directly account for the behaviour of others have not yet been applied to virus disease epidemics.

Resistance and tolerance

Resistance has long been considered the major means of controlling plant virus epidemics, with much recent work proposing the use of tolerance as an alternative or complementary disease management strategy. “Tolerance as a disease management strategy has been claimed to be as widespread as host resistance although problems remain in the strict definition of tolerance and how it can be assessed” (Jeger 2020). Tolerance has been defined as a limited symptom development or reduction in plant vigor or yield despite a normal virus accumulation as in a susceptible cultivar, or alternatively as a limited reduction in plant fitness (fecundity, reproduction period); whereas there is limited virus accumulation and symptom development for a resistant variety but a possible penalty in terms of reduced vigor and yield. A comprehensive review reconciling these viewpoints, but also pointing out the ambiguities and some of contradictions that remain has been made (Pagán & Garcia-Arenal, 2020).

There has been limited modelling of tolerance for plant viruses (Cronin et al. 2014; Lazaro et al. 2017; Moore et al. 2011; Sisterson & Stenger 2018; Zeilinger & Daugherty 2013; van den Bosch et al 2006). Van den Bosch et al. (2006) proposed an epidemiological model to compare different forms of plant defence mechanisms, including tolerance, at the field and within-plant levels, and how deployment in cultivars affected virus evolution using an adaptive dynamics approach. The model structure proposed was motivated by African cassava mosaic virus (ACMV) disease and coupled a between-plant vector transmission component and a within-plant virus multiplication component. It was found that titre-reducing and symptom-reducing defence mechanisms impose selection on the virus, leading to an increase in within-plant virus multiplication. If symptom reduction is seen as an expression of tolerance, then the model predicts selection for an increased virus titre. However, the crop considered here was cassava and comparison of defense mechanisms for cassava mosaic disease and hence symptom reduction, sometimes termed “mortality tolerance”, was considered rather than a reduction in fitness, measured as plant fecundity.

Recent experimental work has placed more emphasis on fecundity or reproductive stage stress tolerance to a range of biotic and abiotic stressors. For example, tolerance in *Arabidopsis thaliana* challenged with either cucumber mosaic virus (CMV) or turnip mosaic virus (TuMV) (Montes and Pagán, 2019). Tolerance of CMV was associated with resource allocation from growth to reproduction; for TuMV, it was associated with the time to and length of the reproductive period. A trade-off in tolerance between the two viruses was found, carrying potential implications for disease management in crops. The emphasis on reproductive stage tolerance offers many opportunities to link the effects of biotic and abiotic stressors on plant genetics, physiology, and disease ecology (Jeger 2023). This will require a whole life history approach. For example, with annual plants and indeterminate flowering: seed germination and seedling emergence occur on shorter time scales than vegetative plant growth, flowering may occur at any time during the growth period, which also corresponds to the pollination period, at the end of the growth and pollination period, seeds drop, and eventually the plant dies, and only seeds that survive the overwintering period start a new cycle if there is no seedbank. The challenge is then to disentangle the interactions of reproductive stage tolerance with plant virus epidemiology.

As described for controls which are more effective when growers co-operate, the use of resistance or tolerance as strategies for controlling plant virus disease carries implications beyond the choices made by individual farmers and extends to whole farming communities. This was modelled by Murray-Watson and Cunniffe (2022a), using tomato yellow leaf curl virus (TYLCV) as a case study. Disease has relatively little effect on the yields of those growers who use tolerant crop varieties, but – by increasing the prevalence of disease in the system – can significantly affect the yields of those who do not deploy tolerance. In this sense, therefore, deployment of tolerant varieties can be viewed as “selfish”. In contrast, resistant crop varieties benefit not only those who grow them, but also those who do not, since overall levels of disease are reduced. The distinct effects of resistance and tolerance lead to divergent consequences when modelling grower behavior. Resistant varieties can be associated with other growers “free riding”, i.e., gaining the benefit in terms of reduced disease due to control enacted by other growers, without themselves incurring the costs of the resistant variety. Murray-Watson and Cunniffe (2022b) extended the set of strategies adopted by growers to allow for planting an unimproved, a resistant, or a tolerant crop. Additionally, growers’ use of resistant or tolerant varieties could be subsidized by a “social planner” to determine whether and how socially optimal outcomes could be promoted. Subsidizing a tolerant crop incurs a recurrent cost to the planner, since when use of tolerant crop becomes established, continued use of this crop becomes necessary via a feedback mechanism. Subsidizing a resistant crop, however, provides widespread benefits by reducing the prevalence of disease across the community of growers, including those that do not control. A reduction in the level of subsidy required for resistant crop occurs because only a subset of growers need to use it for the benefits to be felt across the community of growers, with other growers “free riding” upon the control efforts of others.

Disentangling the virus-vector-host-environment interaction

The disease triangle concept has been extended to vector-borne diseases by many authors, e.g., Islam et al. (2020). However, the disease triangle concept and its extension to include a vector is essentially static. The more fluid concept of the “ecological trinity” was proposed earlier in the 1930’s (Jeger, 2008; 2020) by the American entomologist Walter Carter. He developed the concept of the ecological trinity of viruses, hosts, and vectors within a particular environment based on interactions of viruses and vectors with crops, weeds and other wild or volunteer hosts as influenced by the environment and cropping practices. Epidemics then result from disturbance to previously stable situations in which neither host nor virus had gained permanent ascendancy.

Transmission

Embedded in any concept of the virus-vector-host interaction is the importance of transmission and how the retention and movement of plant viruses leads to classification of transmission mode (Whitfield et al. (2015). The classification can be made in terms of stylet retention (also described as non-persistent transmission), foregut retention (semi-persistent transmission) and circulative movement (including both persistent-circulative and persistent-propagative transmission). With some systems there is also the possibility of transovarial, transstadial, and venereal movement of viruses in the vector population.

To represent these transmission possibilities a SEIR (Susceptible, Exposed, Infectious, Removed) model for a plant virus epidemic was proposed linked with a vector population model in which compartments of non-viruliferous, viruliferous but non-inoculative, and inoculative vectors were defined, including migration terms (Jeger et al. 1998; Madden et al. 2000). For plant diseases, "susceptible" equates to the disease-free state (healthy) and "exposed" equates to the latent state (infected but not yet infectious). Virus acquisition occurs when nonviruliferous vectors probe/feed on infectious plants; virus inoculation occurs when viruliferous vectors probe/feed on healthy plants. In this modelling framework, parameter values where known, relevant to transmission mode, can be used.

Basic reproduction number

From this basic model the basic reproduction number can be derived, the average number of new infections arising from the introduction of one infected unit into an otherwise healthy population during the unit's period of infectiousness. In the case of vector-borne diseases there are two cycles, one in the vector and one in the plant, with the basic reproduction number represented in squared form as R_0^2; if > 1 an epidemic will develop (van den Bosch et al., 2008). Given the complexity of host-virus-vector models, the next generation method is often used to derive R_0^2 using classical mathematical methods (van den Bosch and Jeger, 2017). The basic reproduction number is now a standard epidemiological concept and tool for assessing disease management actions.

Spatial dynamics of vectored plant virus diseases

Selecting an appropriate model framework to track spatial aspects of plant virus disease epidemics remains a key challenge (Cunniffe et al., 2015a), since various classes of epidemiological model are available which can account for spatial effects (Cunniffe and Gilligan, 2020). Early work for spread at relatively small scales, such as within individual fields or orchards, often involved detailed simulations (e.g., Ferriss and Berger, 1993). These simulations tracked the movements made by, and infective status of, individual vectors. This class of model is still used (Ferriss et al., 2020; Kho et al., 2020), and sits within the broader class of individual-based models (DeAngelis and Grimm, 2014). However, even for plant virus diseases, individual based models often concentrate only upon the disease status of the plant host (Gibson et al. 1996; Atallah et al. 2015; Varghese et al. 2020). This is particularly the case for diseases of fruit trees, for which the number of individual hosts within a given production setting such as a block, grove or orchard is not too large. This type of compartmental individual-based modelling approach focusing on the disease status of individual *plants* is routinely used for diseases which are not vectored (e.g., Adrakey et al.

2017; Cunniffe et al. 2014, 2015; Hyatt-Twynam et al. 2017; Neri et al. 2014), as well as for vector-borne bacterial diseases, e.g., citrus greening (Craig et al. 2018; Parnell et al. 2015; Parry et al. 2014). The influence of vectors on transmission is subsumed into a dispersal kernel, a statistical representation of how the rate at which an infected host causes infection of susceptible hosts falls off with distance (Fabre et al. 2021).

When larger scale predictions are required, a simple approach is to use essentially the same idea, with the disease status of an entire field or farm tracked as a simple binary variable. Disease transmission can then occur between entities either via a dispersal kernel (e.g., Murray-Watson et al. 2022) or an explicit network parameterised to represent certain types of movement, for example movements of planting material or seed (e.g., Andersen et al. 2019). When more finely resolved information on the level of disease within each field or farm is required, transmission can still be captured via a dispersal kernel, but with the dynamics of disease within each "node" also modelled. This can be done relatively simplistically, via an increase in within-node prevalence at a predetermined rate (Holt and Chancellor, 1997) or by allowing the dynamics within each node to follow an internal compartmental model (McQuaid et al., 2017; Picard et al. 2018). The approach can also be adopted at very large scales (Gilligan et al., 2007). Often the host distribution is then further approximated by discretisation to a lattice of a certain size (Godding et al., 2022), an approach which has proved useful in modelling landscape scale spread of various plant diseases (Cunniffe et al. 2016).

Conditional vector preference

There has been much recent work over the last two decades on vector preference: how the landing and feeding behaviour of virus vectors depends on the disease status of both the host (healthy or infected) and the vector (viruliferous or non-viruliferous) and whether the virus can manipulate the plant and vector to its own advantage (Mauck & Chesnais, 2020; Eigenbrode et al. 2018; Zhao et al. 2022). In some cases, there may be an environmental influence on vector preference such as water stress (Del Cid et al., 2018).

Following on from previous vector preference models (Roosien et al. 2013, Shaw et al. 2017, Shoemaker et al. 2019), Cunniffe et al. (2021) developed a model that explored the epidemiological and ecological consequences of virus manipulation of host and vector in plant virus transmission, while echoing the original models (Jeger et al. 1998; Madden et al. 2000) by allowing distinct features of different transmission types to be represented. The assumptions made in developing the model are listed in Box 2. The epidemiological model was structured in compartments. Parameters were defined for the flying, settling, and feeding behaviours of vectors and combined in the model with the plant-virus-vector interaction (Table 1 of Cunniffe et al. 2021). A distinction was made between the preference parameters for viruliferous and non-viruliferous vectors, at least for persistently transmitted viruses, and these are shown in Table 2.

A basic reproductive number was derived from the model equations, which shows the importance of the bias of a nonviruliferous vector for an infected plant and the number of healthy plants visited by a vector once virus has been acquired. This basic reproduction number has a direct heuristic interpretation of the successive terms in the expression:

Average number of vectors per plant in absence of virus × average infectious period (time units) of a single infected plant × average number of plants visited by a vector (per unit time) × probability of virus acquisition by a single vector during a single visit × average period (time units) a vector remains viruliferous × average number of plants visited per vector (per unit time) × probability of inoculation by a single viruliferous vector

An online version of the model can be accessed via

https://plantdiseasevectorpreference.herokuapp.com/explanation

Models were parameterised to ensure a default value of R_0 = 2 for both nonpersistent and persistent transmission so that the dynamics of healthy and infected hosts and non-viruliferous and viruliferous vectors could be directly compared and used as a baseline (Figure 3: A-C for nonpersistent transmission, D-E for persistent transmission). For certain sets of parameters, the model has multiple stable biologically plausible equilibria, where which of two locally stable equilibria in disease incidence is attained depends on the initial conditions. Even without conditional vector preference, the outcome can depend on the initial disease incidence (Figure 3A). When vector population dynamics are introduced, there is a rich dynamical behaviour with again bistability, and stable or unstable outcomes in disease incidence as birth rate changes, whenever infected hosts are more able to support vector reproduction (Figure 3F-G).

Importantly, the consequences of vector preference and manipulation in terms of crop loss and economic returns have been modelled using data for three viruses – pea enation mosaic virus, bean leaf roll virus, and potato leaf roll virus (Eigenbrode & Gomulkiewicz 2022). In each case, the effect on performance of a single insecticide spray was greater with than without vector manipulation. For the psyllid vector of huanglongbing, additional returns for multiple sprays diminished more with than without vector manipulation.

Evolution of conditional vector preference

Conditional vector preferences occur when viruliferous and non-viruliferous vectors show contrasting preferences for infected versus uninfected hosts. The question is whether evolution shaped these preferences in a way to promote vector performance and/or virus spread (Mauck et al. 2018). The evolution of conditional vector preferences has been addressed by Gandon (2018), making reference to barley yellow dwarf virus (BYDV), TYLCV, and potato leaf roll virus (PLRV), but with most relevance for animal systems. More specifically, the author explored a relatively simple epidemiological model akin to Roosien et al. (2013), itself a simple adaptation of the classical Ross model of 1911 for vector-borne diseases. Vector fecundity depends on whether it feeds on infected or uninfected hosts. Extreme preferences for uninfected or infected hosts may drive the vector to extinction, hence the vector should avoid rare and low-quality hosts. If preferences are controlled by the vector (as opposed to the virus), and if infected hosts are of relatively low quality, evolution may select for increasing preferences against infected hosts, leading to ultimate extinction of both the vector and the virus. Other evolutionary outcomes are possible as well, depending on whether the vector, the virus or both control preferences. For instance, intermediate preferences may evolve if there is a trade-off between the virus ability to drive viruliferous vectors toward uninfected hosts, and its ability to make infected hosts attractive to vectors. The main thrust of Gandon (2018) was to model vector preference and parasite manipulation in animal systems but could be adapted for plant viruses.

Epidemiology of Co-Infecting Plant Viruses

Co-infection of plant hosts by two or more viruses is common in agricultural crops and natural plant communities. It has long been recognised that some diseases are associated with multiple pathogens including viruses and mollicutes, such as the corn stunt disease, but models for this disease previously concentrated only on one pathogen component (Vandermeer and Power, 1990). Standard methods of analysis are not sufficient to investigate interactions within and among plants across different viruses or virus strains, which adds further levels of complexity. Co-infection has been shown to interact with vector preferences in cases where two viruses have the same or different vectors (Table 3). However, the results reported are difficult to generalise due to differences in vector taxonomies, behaviours, reproductive systems, and transmission modes.

As already described, maize lethal necrosis is a disease arising from co-infection with maize chlorotic mottle virus and a potyvirus such as sugarcane mosaic virus (Hilker et al. 2017, de Groote et al. 2021). Analysis of field surveys of MLN and the individual viruses, MCMV and SCMV, in a range of surveys suggest the prevalence of MLN is given by the product of MCMV and SCMV prevalence (Mahuku et al. 2015) (Figure 4) indicating independent transmission of the two viruses. This result may reflect the differences in vectors and transmission type between the co-infecting viruses.

Independence and interaction between co-infecting viruses

In wild rather than crop populations where natural mortality and regrowth occurs, the probability of co-infection by non-interacting pathogens was shown to be greater than the product of their individual incidences (Hamelin et al. 2019) (Figure 5A) unless host natural mortality can be neglected. This deviation from independence raises questions on the validity of statistical tests performed to detect interactions between pathogens responsible for long-lasting diseases. Hamelin et al. (2019) provided a novel method to test for interactions among pathogens. The method was tested with data on strains of anther smut, human papillomavirus, tick-transmitted bacteria, and *Plasmodium*. For plant viruses, the authors reanalysed the data set for barley and cereal yellow dwarf viruses (B/CYDV) from Seabloom et al. (2009) and found, with this method controlling for host mortality, that the five virus species co-occurred more often than expected by chance (Figure 5B).

Vector transmission and co-infecting plant viruses

A variety of studies have investigated the dynamics of co-infection but track only the disease status of infected and co-infected plants. Much less attention has been paid to the role of vector transmission in co-infection, i.e., acquisition and inoculation and their synergistic and antagonistic interactions. A vector-explicit model for co-infection was proposed for one vector species and one plant species with potential co-infection by two viruses (Allen et al. 2019). This model included both vector and host plant components. The basic reproduction number provides conditions for successful invasion of a single virus. The main question asked in this study is what determines invasion of a co-infecting plant virus? A new invasion threshold was proposed which provides conditions for successful invasion of a second virus.

Two special cases were considered. In the first case, one virus depends on an autonomous virus for successful transmission with only one of the viruses invading in the absence of the other. The equilibrium prevalence for the vector and host and the corresponding invasion reproduction numbers were derived as functions of acquisition of the established virus and inoculation of both the established and invading virus by the vector. In the second case, both viruses are unable to invade alone but can both establish themselves when initial prevalence is high. This case leads to interesting dynamics in which the outcome depends on the initial prevalence of each virus and can lead to bistability (Figure 6), with a disease-free equilibrium and a co-infected equilibrium as a function of the initial frequencies of the two viruses.

Recently, McLaughlin et al. (2022) reported experiments on transmission, infection, and replication of tomato yellow leaf curl virus (TYLC) and tomato mottle virus (ToMoV) in tomato: data on acquisition and co-inoculation by *B. tabaci* were found to be fundamental in disentangling the vector-virus-host interaction and the spread of single and co-infections.

Interaction between vectors

In some cases, co-infection may occur when the co-infecting viruses have different vector species. In these cases, there may be interactions between vectors due to differing life history characteristics and transmission mode and efficiencies. In the case of competition between two vectors, a Lotka-Volterra model was used in deriving a basic reproduction number for a single virus (van den Bosch and Jeger, 2017) but not for the case where they each transmit a different virus. Often, particularly important for non-persistently transmitted viruses are interactions between transient and resident aphids (Zaffaroni et al. 2021). Transient aphids probe several plants per day, and so are important vectors of viruses both within and between fields, whereas resident aphids complete their life cycle on a single plant host, and so tend to lead to plant host damage via herbivory rather than by their vectoring activity. Many agronomic practices, most notably spraying with pesticides, have more pronounced effects on resident aphid populations. Under mild assumptions about how transient aphids can be dissuaded from probing plants that are already heavily infested with resident aphids, this in turn means that pesticide application can potentially have the counter-intuitive effect of increasing the amount of disease.

Advances in statistical and computational techniques

Optimal control theory

Two areas seem to be highly relevant in plant virus epidemiology. The first relates to the optimisation of disease management practices. Such techniques have been used to evaluate the choices farmers make when selecting planting material for the next season's cassava crop (Bokil et al. 2019). In a similar vein, Hamelin et al. (2021) used dynamic optimal control theory to evaluate the use of clean seed, motivated in part by work on MLN referred to at several points in this review. More specifically, the authors showed that depending on epidemiological and economical parameters, controlling plant virus with clean seeds may or may not be economically viable, and when viable, may or may not lead to disease eradication. Subsidizing clean seeds may help in switching from unviable to viable control but cannot lead to disease eradication. The only way to achieve disease eradication in this case is additionally to use control methods that decrease horizontal transmission of the pathogen.

A key limitation of optimal control theory is that the underpinning mathematics rapidly becomes rather complex, making its use intractable for more detailed models. However, Bussell et al. (2019) recently proposed a methodology to allow optimal control theory to be applied to models which attempt to capture significant biological detail. Essentially, the machinery of optimal control theory is applied to a simplified "approximate" model, carefully calibrated to adequately reflect the results of the "full" model of interest over a range of parameterisations. Optimal strategies identified in the approximate model can then be "lifted" back to the full model, informing disease control in the situation of particular interest. Although this approach has to date not yet been applied to virus disease epidemics, but to an oomycete pathogen (Bussell and Cunniffe 2020; 2022), the technique promises much for vectored virus disease in terms of going beyond the simplified models considered by, e.g., Hamelin et al. (2021), and accounting for the various ways in which virus disease epidemics are distinctive.

Estimation of epidemiological parameters

The second area refers to advances in computational techniques which allow for estimation of epidemiological parameters from field data. Such techniques in principle allow for a link between estimates made in the laboratory or microcosm experiments and estimates made in the field. Certain parameters required by mathematical models, for example the delay between first infection of a plant host and the emergence of symptoms, can be estimated from the results of designed experiments involving individual plants. Experiments can also be used to obtain relatively detailed information concerning the preferences of vector species, as well as their vital dynamics (Wamonje et al. 2020; Tungadi et al. 2017, 2020). These parameters are particularly important in models which focus predominantly on the behaviour of individual vectors (Donnelly et al. 2019). However, parameters controlling the rate of spread of disease often must be inferred by fitting the output of an epidemiological model to data. Often the key uncertainty is around the dispersal kernel, which tends to be only very loosely characterised for many pathogens (Fabre et al. 2021). For virus diseases it reflects the probability of vector-borne transmission linking pairs of plants at a certain distance.

Often this is done in an explicitly Bayesian framework, which allows prior knowledge to constrain model fitting. However, this requires a likelihood function to be written for the model of interest, which in turn tends to require information on epidemiological transitions which are not recorded, and in many cases never could be, e.g., the time at which a plant was first infected. While some methods based on approximations to the likelihood function which do not require this type of information have been developed (Pleydell et al. 2018), the calculations rapidly become complex. A more general method, introduced into plant disease epidemiology by Gibson and Austin (1996, 1997a, b) relies on "data augmentation", which in this context means treating unknown/unknowable parameters as additional parameters to be estimated, by using Markov Chain Monte Carlo (MCMC) techniques to draw samples from the relevant posterior distribution. These methods work particularly well for field data consisting of successive snapshots from different surveys (Neri et al. 2014; Parry et al. 2015). The example shown in Figure 7 is from Cunniffe et al. (2014) and shows pairwise posterior distributions as sampled via MCMC for key parameters in an individual based model of Bahia bark scaling fitted to data from a small experimental plot.

There is an extensive literature on the principles and use of MCMC techniques in plant disease epidemiology, including viruses and other vector-borne diseases: e.g., spatiotemporal dynamics of plum pox disease (Pleydell et al. 2018); temporal dynamics and emergence of *Xylella fastidiosa* (Soubeyrand et al. 2018); disease mapping of citrus Huanglongbling (Luo et al. 2012); and diagnosis/detection of tomato viruses and bacteria (Mohanty et al. 2016, Hernandez & Lopez 2020).

However, there are limits on the size of the system that can be adequately represented in this way. Data augmentation becomes infeasible for models with large numbers of individuals and/or complex transitions between states. However, most models – including very complex models – can be simulated relatively easily. This is the motivation for Approximate Bayesian Computation (ABC), which uses statistics drawn from simulation results as a proxy for the likelihood function (Jabot et al. 2013; Toni et al., 2009). By using the fraction of simulation results that are sufficiently close to the data as an estimator of the likelihood for any given set of parameters, estimation can be done without ever writing down any mathematics. The challenge, however, is to identify the "correct" summary of experimental results to use in the comparison between model results and data. An example for plant virus disease is shown in Figure 8; it shows two snapshots of the disease status in a citrus grove infected by citrus tristeza virus (reproduced from Marcus 1984). As shown by Minter and Retkute (2019), an ABC algorithm using a spatial statistic based on the minimum distance between newly infected trees in the 1982 snapshot and infected trees in the 1981 snapshot can be used to drive model fitting.

There is also an extensive literature on ABC techniques, widely used in ecological and evolutionary studies (Beaumont 2010), and in epidemiological studies: e.g., within-field dynamics of banana bunchy top disease (Varghese et al. 2020); colonisation history of the fungal pathogen causing South American Leaf Blight (Barrès et al. 2012); and pollen dispersal (Soubeyrand et al. 2013).

Challenges and opportunities

Quantitative epidemiological analysis, including mathematical models, has given insights into how a changing environment, the host-virus-vector association, and vector life history, behaviour, and population dynamics interact at the systems level in plant virus epidemiology. A broader perspective and synthesis are needed to account for the ecological context and the evolutionary implications of these interactions. How do new evolved strains emerge (Antia et al. 2003) and what are the consequences for host ranges, crop losses, and natural wild populations?

Epidemiological analysis

Much progress has been made on integrating vector life history parameters with epidemiological parameters, although the difficulties in scaling up from laboratory/microcosm experiments to field observations remains a challenge. The complex spatial dynamics of virus disease means that the 'mean-field' assumption of randomness in host-vector association is untenable, especially when vector preference is conditional on the vector-virus association. At the field level, 'one-off' observations on the size of vector populations and the association with virus incidence have been made, but rates of change in each variable need to be assessed to better describe time delays, vector and epidemic dynamics, and options for disease management.

Innovative field-based research and further modelling is required to determine the epidemiological significance of the different forms of vector preference. Further research is required on further aspects of vector behaviour: for example, on the energetic costs associated with number of vector flights per individual feed with respect to virus transmission and vector preference, and on competitive and other interactive effects in relation to co-infection, vector preference and transmission.

Does a link between vector preference, transmission type, and natural enemies lead to increased virus fitness? Tritrophic plant-virus-vector-parasitoid relationships potentially add a further level of signaling mechanisms (Jeger et al. 2012) in which the vector shows preference for either healthy or infected host plants, the host plant uses a "cry-for-help" signal to attract parasitoids, the parasitoid induces an "alarm signal" initiating vector movement, and once virus has been acquired the vector switches to a preference for healthy plants. The first element in increasing virus fitness through transmission is then conditional vector preference with non-viruliferous vectors preferring infected plants and viruliferous vectors preferring healthy plants. Parasitoids may then be attracted by the "cry for help" signal from infected plants infested with an insect vector. The alarm signal among vectors may encourage the movement of vectors from infected plants. If viruliferous vectors then show a preference for healthy plants, a virtuous circle has been completed, thereby increasing virus fitness. A further element that needs to be accounted for is that natural enemies, as well as affecting vector population dynamics and behaviour, may affect vector developmental rates. This was modelled by Keissar et al. (2020) who counter-intuitively showed that slowing down development rate increased disease prevalence due to an apparent competition between infected and uninfected vectors.

Ecological context

There have been many recent reviews on plant virus ecology (Jones, 2014b, Aranda and Freitas-Astúa 2017, Lefeuvre et al. 2019, Shates et al. 2019). Often, most concern has been with molecular virology, diversity, and evolution, and disease in (semi-)natural plant populations, and do not always make clear the link with epidemiology, where the interchange between crops and wild plants mediated by vectors contributes to a complex ecology that merits further study (Jeger 2022).

Multiple infections, plant fitness effects and life history traits, transmission, and movement ecology of vectors in heterogeneous environments are major drivers of plant-virus-vector systems and require a higher level of analysis than provided by molecular virological studies if forecasting models of disease risk are to become a reality (McLeish et al. 2020). These authors consider that the next major step in plant virus epidemiology will come from the synthesis of high throughput sequencing systems ecology and remote sensing. Such a synthesis is wide and ambitious, but the interrogation of intensive data sets is receiving much attention both in genomic studies, field observations and environmental monitoring and as the authors suggest may prove to be a major development in plant disease epidemiology.

Research in disease ecology has stressed the interactions between host composition and structure, diversity, and infection risk (Seabloom et al. 2013). In studies on B/CYDV in grassland communities, they found that niche differentiation arising from nutrient treatment was an important factor in virus species distribution and assemblages. The spatial structure of virus species in these grassland communities, especially pairs, was found to be aggregated resulting from shared vectors and their distribution (Kendig et al. 2017). The prevalence and diversity of potyvirus species was studied in natural riparian forests in Spain (Rodríguez-Nevado et al. 2020). A novel generalist virus was found accounting for the highest proportion of infected plants and was best predicted by host abundance and species richness. These ecological factors together with virus prevalence largely determined selection and genetic diversity in the virus population.

A mathematical model was proposed describing the joint effect of a mycorrhizal mutualist and a viral pathogen (Rúa and Umbanhowar, 2015) one of the few models exploring cross-taxa and cross-functional group interactions. Where there was low plant productivity due to limited resource availability the pathogen depended on the mutualist for persistence; when plant productivity was high under some circumstances the mutualist may go extinct. Cyclical virus dynamics were only found with the presence of the mutualist but were not consistently associated with high viral pathogenicity.

Environmental conditions may affect the ecology of insect vectors if they affect host preferences. High temperature tolerance of insects affects population dynamics under extreme temperature events, and this would include aphid vectors of plant viruses. The cowpea aphid *Aphis craccivora* showed an ecological niche switch from cotton to soybean under high temperatures, showing that heat tolerance was host associated (Zhaozhi et al., 2017). The aphid is known as a vector of soybean viruses and has been reported as transmitting cotton leaf roll viruses, although the main vector is predominantly *A. gossypii*.

The consequences of such host switching have been little explored in plant virus epidemiology.

Evolutionary implications

In the paper on the evolution of conditional vector preferences, Gandon (2018) assumed, for simplicity, that the manipulation of infected vectors is fully governed by the pathogen in the vector, and that the pathogen in the infected host can affect only the behaviour of uninfected vectors. However, a pathogen strain making hosts more attractive to vectors may attract vectors carrying a pathogen strain that would otherwise drive the vector to uninfected hosts. Research exploring further the evolution of vector preferences should account for such indirect interactions between pathogen strains.

Future work should also address the evolution of conditional mutualism. Specifically, conditional mutualism occurs when infected plants have lower fecundity than uninfected plants under favourable conditions, and higher fecundity than uninfected plants under unfavourable conditions such as water stress (Hily et al., 2016). Hamelin et al. (2017) explored the evolution of unconditional mutualism, a situation in which infected plants' fecundity is greater than uninfected plant fecundity. The authors showed, among other results, that mutualism may evolve from and evolutionarily exclude parasitism under certain conditions. However, it would be interesting to extend this type of approach to conditional (environment-dependent) mutualism in plant viruses.

Further synthesis and questions

This review has attempted to identify emerging themes and approaches in plant virus epidemiology. Perhaps an overarching theme is how these themes are linked and how experimental, observational, and modelling studies can contribute to these linkages. For example, the deployment of tolerance as a disease management option or as a natural phenomenon in wild plant populations has received much attention recently (Jeger 2022b). Linking across the other themes in this review, we can ask how virus epidemiology interacts with the various forms of tolerance in plants? Can direct damage from vector feeding be disentangled from that resulting from virus infection when there is plant tolerance to the vector as pest as well as to the virus? How are interactions of virus epidemiology with reproductive stage stress tolerance manifested where there are other biotic and abiotic stressors in both wild plants and crops? Do interactions of vector preference with tolerance impact upon reproductive fitness? Does deploying tolerant varieties affect vector population dynamics, and how does this in turn feed into disease dynamics? How is tolerance to single viruses affected when there is coinfection with multiple viruses? Perhaps most importantly, and applicable to all themes, how can mathematical models with the theoretical framework(s) outlined here be fitted to observations from the field and/or designed experiments to allow us to disentangle the significant complexity underpinning these – and other – interactions? Finally, the choices made by growers of necessity are based on making linkages across all aspects of disease epidemiology and management. Can the choices made by individual growers directly account for the behaviour of others, and be applied to virus disease epidemics in future studies?

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Box 1 Thematic areas for gaps in modeling effort in plant virus epidemiology (Jeger et al. 2017)
Theme 1. Identifying the consequences of large-scale climatic fluctuations, including global warming, for plant virus epidemics and shifts in virus and vector distributions.
Theme 2. Basing control of plant virus epidemics on locale-specific conditions, including crop, landscape and farmer heterogeneity, and interactions; and by so doing contribute to improved methods of disease control.
Theme 3. Disentangling the interactions between viruses, vectors, host plants, and the biotic and abiotic environment presents major challenges for experimental and epidemiological studies, where typically pairwise interactions are the norm. Some advances have been made by modelers in meeting these challenges, but more can realistically be achieved, by: a. Integrating vector population dynamics and ecology into epidemiological models in a more realistic way, specifically, by recognizing that virus transmission and transmission type may affect vector life history parameters, and flight, landing, and feeding behavior; and b. Developing evolutionary models for viruses, vectors, and the virus-vector interaction based on fitness trade-offs and other population genetic approaches. Can viruses manipulate vectors, natural enemies, and host plants to enhance

their fitness? How best to characterize virus-virus interactions within plants as synergistic, neutral, or antagonistic?
Theme 4. Advances in statistical and computational techniques should facilitate a greater interrogation of observational data, estimation of epidemiological parameters, and evaluating their relative importance in determining epidemic outcomes.

Box 2 Assumptions made in the vector preference model of Cunniffe et al. (2022)
1. Vectors are attracted by plant cues (visual or olfactory) to land on infected plants
2. Whether vectors settle and feed for an extended period, or only probe and then depart, depends on the plant's infection status.
3. The strength of vector preference can differ for viruliferous and non-viruliferous vectors, i.e., preference is conditional on vector status as well as plant infection status.
4. The proportion of probes that leads to vectors settling for an extended feed affects the number of plants visited by vectors per unit of time, and so the overall transmission rate.
5. Whether vectors probe or feed has different effects on transmission for non-persistent vs persistent viruses.
6. The fecundity of the vector can be affected by the plants it feeds on, with vectors that predominantly feed on infected plants potentially having either a higher or lower birth rate.
7. The loss rate of the vector, from additional mortality or movement away from the plant population, may be affected by the number of plants visited per extended feed.
8. The flight duration of a vector may depend on whether it is viruliferous or non-viruliferous.

Table 1. Bemisia papers

Authors	Crop/virus	Geographical relevance	Comments

Aregbesola, et al. 2020. J. Pest Sci. 93: 1225-1241.	Cassava viruses/cassava colonising groups – East and Southern Africa (SSA-ESA)	Tanzania	Life history (oviposition, fecundity, survival, developmental time) was studied in relation to temperature in controlled environments and field experiments. A phenology model was developed which could be used for pest risk mapping under climate change.
Bradshaw et al. 2019. PLoS ONE 14: e0221057.	Multiple crops and viruses/biotypes MEAM1 and MED	North-west Europe	A set of 49 indices was developed based on current climate to compare the UK (no outdoor populations recorded) with France (established populations). Climate projections (2-4 C warmer) suggest establishment in UK outdoor crops in summer months, with a clear south-north gradient for these indices.
Kriticos et al. 2020. Scientific Rep. 10: 22049.	Cassava viruses/Sub-Saharan Africa (SSA) groups	East Africa	Historical changes in climate suitability for SSA sub-groups were analysed using the CLIMEX niche model corroborated with a 13-year time series of *B. tabaci* abundance. Modelled climatic suitability improved significantly over the almost 40 years of experienced cassava virus pandemics in East Africa.
Ramos et al. 2018. PLoS ONE 13: e0198925.	*Solanum lycopersicum* viruses/MEAM1 and MED groups	Global	Levels of risk to open field tomato production were assessed using species distribution and global climate models. Projections to 2050 showed an extension in area of 180% in high-risk areas, but a reduction of 67% and 27% in medium and low-risk areas respectively. Projections to 2070 showed an extension of 164 (high risk) and reductions of 49 and 64% (medium and low risk).
Ramos et al. 2019. Agric. Systems 173: 524-535.	Tomato yellow leaf curl virus (TYLCV)/ MEAM1 and MED groups	Global	Distribution of TYLCV in areas suitable for open field tomato production and *B. tabaci*. Under climate change projections for 2050 and 2070, large regions are predicted to be at risk from TYLCV in areas suitable for both open

			field tomato production and *B. tabaci*. Where there are predicted optimal conditions for tomato and suitable conditions for *B. tabaci*, there will be a medium risk of TYLCV establishment

Table 2. Four parameters are labelled according to whether the vector is non-viruliferous (-) or viruliferous (+)

$\boldsymbol{\nu}_-$	Bias of non-viruliferous vector to land on infected plants
$\boldsymbol{\nu}_+$	Bias of viruliferous vector to land on infected plants
$\boldsymbol{\omega}_-$	Probability that a non-viruliferous vector settles to feed on a susceptible plant
$\boldsymbol{\omega}_+$	Probability that viruliferous vector settles to feed on a susceptible plant
$\boldsymbol{\epsilon}_-$	Bias of non-viruliferous vector to settle to feed on an infected plant
$\boldsymbol{\epsilon}_+$	Bias of a viruliferous vector to settle to feed on an infected plant
$\boldsymbol{\phi}_-$	The average number of plants visited by a non-viruliferous vector per unit of time (derived)
$\boldsymbol{\phi}_+$	The average number of plants visited by a viruliferous vector per unit of time (derived)

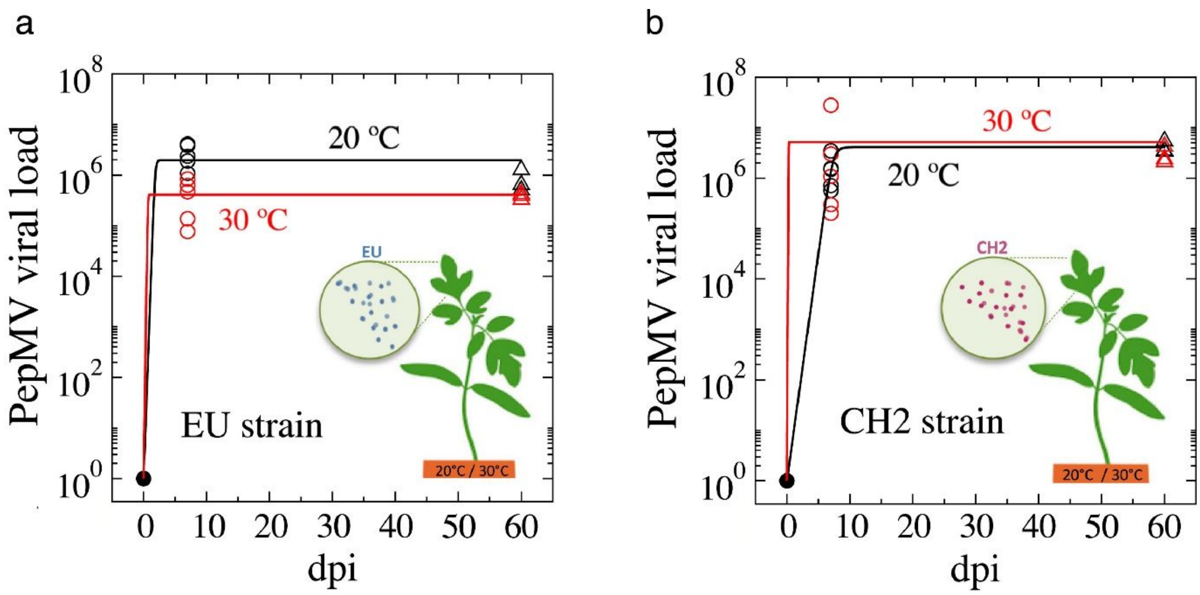
Table 3 Vector preference with co-infecting viruses

Authors	Host plant	Viruses	Vector(s)	Comments
Srinivasan and Alvarez, 2007	*Solanum tuberosum*	Potato virus Y Potato leafroll virus	*Myzus persicae* *Macrosiphum euphorbiae*	Alatae and apterae preferentially settled on co-infected rather than single infected or non-infected plants
Gautam et al. 2020a	*Cucurbita pepo*	Cucurbit leaf crumple virus Cucurbit yellow stunting disorder virus Tomato yellow leaf curl virus	*Bemisia tabaci* MEAM1	A wide and complex range of effects on settling preferences, acquisition, inoculation, and vector virus load between singly- and co-infected plants were noted, but no effects on vector fitness.
Gautam et al. 2020b	*Capsicum annuum*	Cucumber mosaic virus Tomato spotted wilt orthoptovirus	*Myzus persicae* *Frankliniella fusca*	Vector preferences were not greatly different between co-infected and singly infected plants. Overall co-infection in pepper plants did not enhance vector(s) fitness although in singly infected plants, vector fitness was enhanced.
Lightle and Lee, 2014	*Rubus idaeus*	Raspberry leaf mottle virus Raspberry latent virus	*Amphorophora agathonica*	Aphid fecundity only increase on co-infected plants. After 24 h, aphids preferred to settle on RLMV-infected over healthy plants, but on healthy over RbLV plants. There were no differences in settling between healthy and co-infected plants.
Peñaflor et al, 2016	*Glycine max*	Soybean mosaic virus Bean pod mottle virus	*Aphis glycines* *Epilachna varivestis*	Single infection by either virus increased palatability for *E. varivestis* but co-infected plants were no more palatable than healthy plants. SMV infection increased aphid feeding preference (nom-conducive for nonpersistent

				transmission), but this effect was reduced with co-infection.
Salvaudon et al.2013	*Cucurbita pepo*	Watermelon mosaic virus Zucchini yellow mosaic virus	*Aphis gossypii*	ZYMV replicated at similar rates in single and co-infected plants, whereas WMV replication was reduced in the presence of ZYMV. ZYMV enhanced aphid recruitment to infected plants; whereas WMV did not, although it was readily transmitted from co-infected plants.
Ban et al. 2021	*Nicotiana tabacum*	Tomato yellow leaf curl virus Tomato yellow leaf curl China virus	*Bemisia tabaci* MEAM1	Plants infected by the two viruses showed amplified symptoms, but vector performance and preferences were not affected compared with singly infected plants
Zhao and Rosa, 2020	*Emilia sonchifolia*	Tomato spotted wilt orthoptovirus Impatiens necrotic spot orthoptovirus	*Frankliniella occidentalis*	Thrips prefer to oviposit on TSWV and INSV co-infected plants compared with singly infected or healthy plant providing the opportunity for acquisition by nymphs. However, inoculation generally favoured one of the two viruses rather than co-inoculation of both.

Figure 1 Viral load (days post inoculation) of two strains of pepino mosaic virus in experiments at two temperatures (Sardanyés et al. 2022, reproduced under the terms of a CC BY-NC-ND licence).

A: Single infections



B: Mixed infections

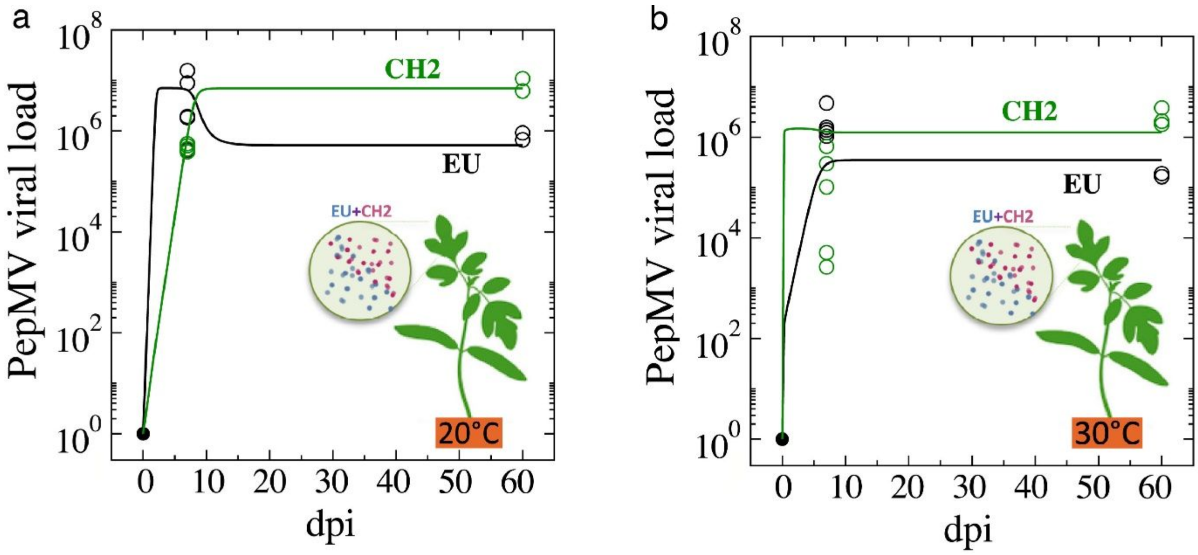
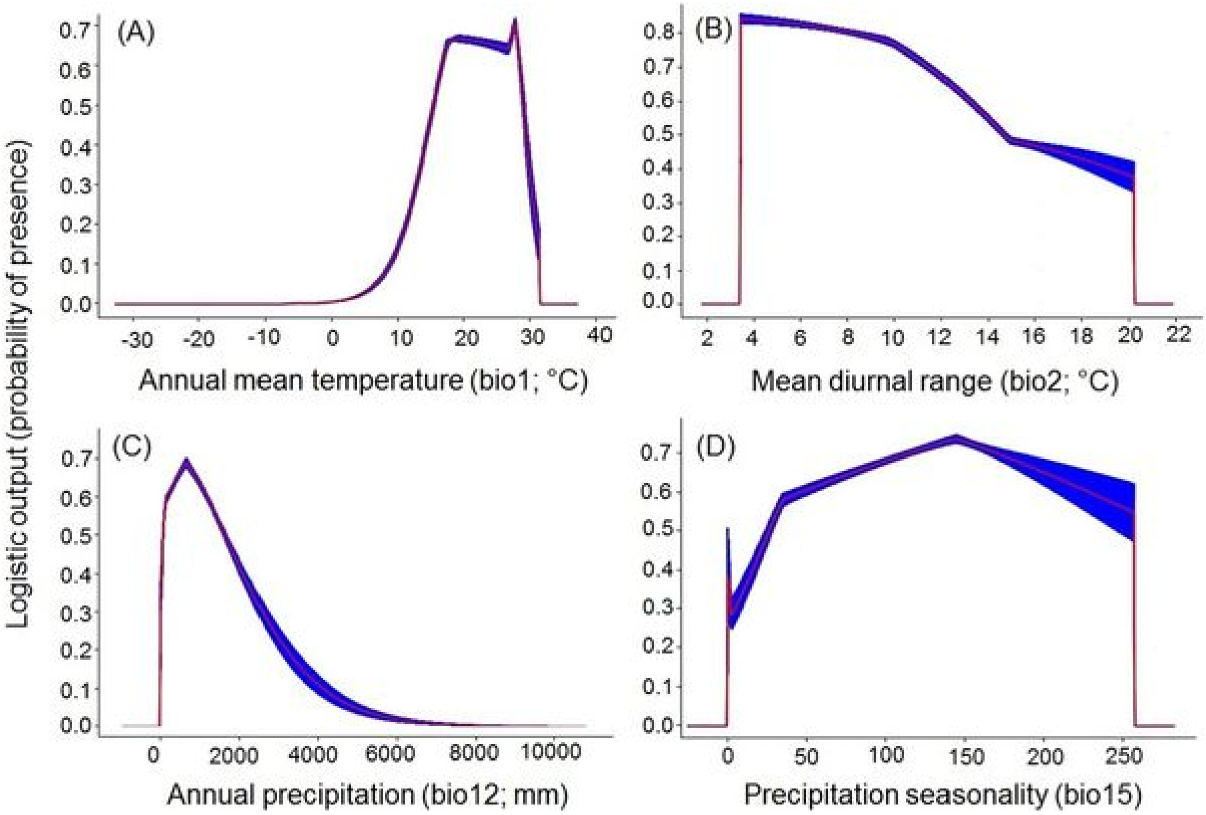


Figure 2. Response curves for probability of presence in relation to the best environmental predictor variables (Ramos et al. 2018, reproduced under the terms of the Creative Commons Attribution licence). Blue shaded areas represent the coefficient of variation in response.

A. *Bemisia tabaci*



B. *Solanum lycopersicum*

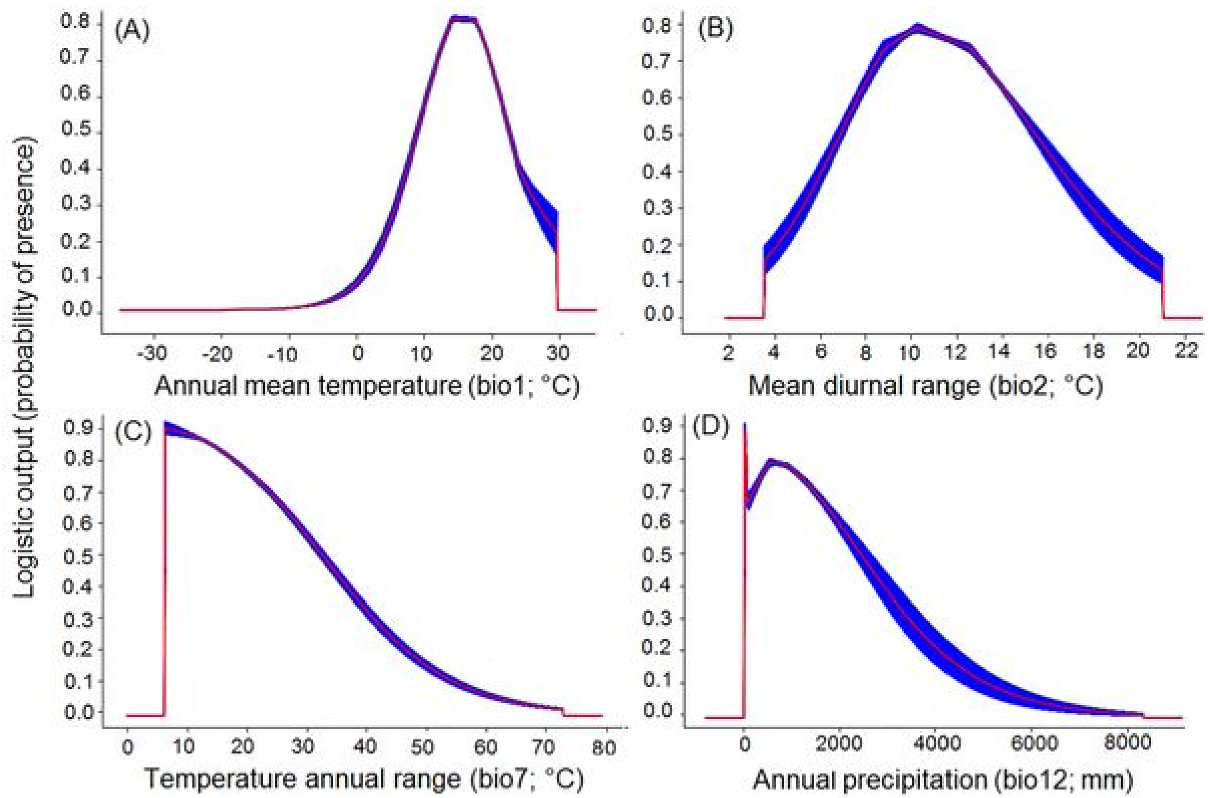


Figure 3**.** Selected results from the vector preference model of Cunniffe *et al.* (2021, reproduced under the terms of the Creative Commons Attribution licence**).** (A)-(C) Effects of vector preference parameters controlling landing bias (ν), settling bias (ϵ) and the probability of feeding (ω) on the basic reproduction number (R_0) and the terminal disease incidence ($I_\infty/(S_\infty+I_\infty)$) when the model is parameterised for non-persistent transmission (NPT). The baseline parameterisation, for which $R_0 = 2$, is marked with a black dot. The model exhibits bistability for a small range of values of the landing bias parameter (marked in grey in panel A). (D) & (E) show the results of the model when parameterised for persistent transmission (PT), when conditional vector preference is possible. Here the responses of viruliferous and non-viruliferous vectors are distinguished (for example ν_+ is the landing bias shown by viruliferous vectors, whereas ν_- viruliferous is the corresponding response for non-viruliferous vectors). (F) & (G) show the responses of the final incidence to vector birth rate (σ) for both classes of transmission as the relative birth rate on infected plants (β) varies (the different coloured lines). Whenever vectors can reproduce more rapidly on infected plants ($\beta > 1$), this induces bistability (dotted lines) for both classes of transmission. In these cases, disease can spread even when $R_0 < 1$, so long as there is sufficient infection initially present in the system, since infected plants lead to larger vector population densities, promoting spread of disease.

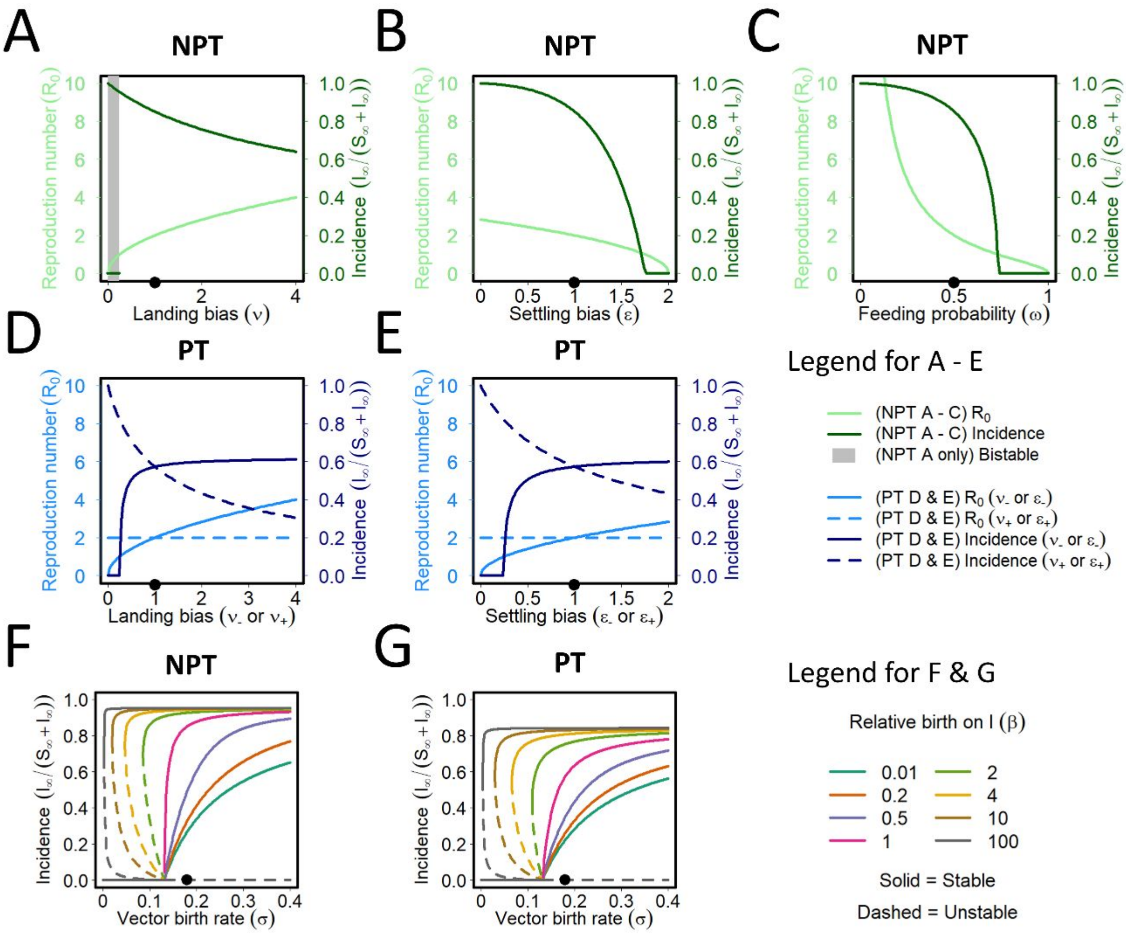


Figure 4. Field survey data on maize chlorotic mottle virus (MCMV) and sugarcane mosaic virus (SCMV) in maize lethal necrosis (MLN) reported by Mahuku et al. (2015). The best-fitting linear response (*N* = −1.28 + 1.08*SM)* is shown with a solid black line, where MLN is represented by *N*, MCMV by *M*, and SCMV by *S*. The dotted line corresponding to the assumption of independence (*N* = *SM*) is contained within the 95% confidence interval (Hilker et al. 2017, republished under the CC BY-NC-ND 4.0 international license).

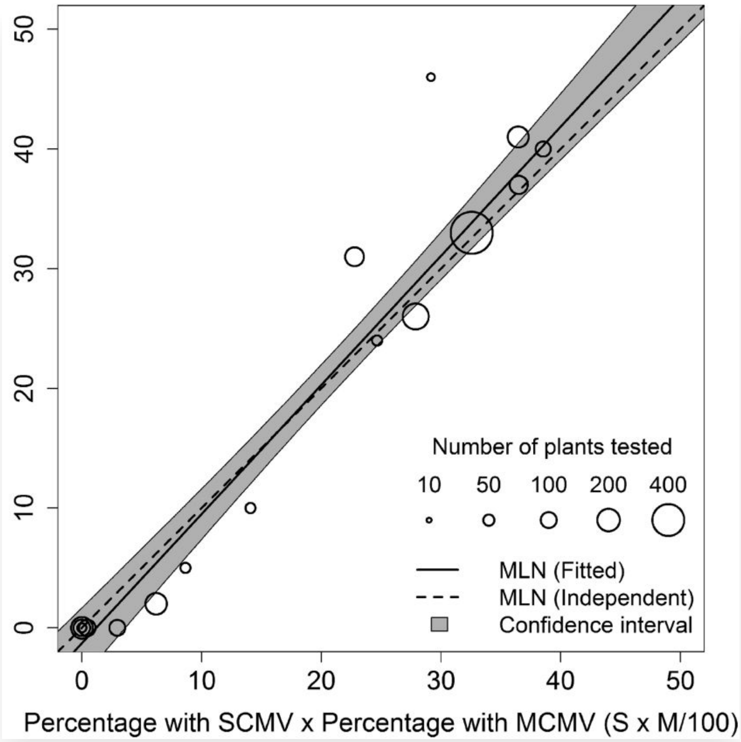


Figure 5. A. Dynamics of a co-infection model (Hamelin et al. 2019 Supporting Information, made available under the Creative Commons CC0 public domain dedication) in which pathogens have no interactions. J_1 and J_2 are the proportion of hosts infected with a single pathogen 1 or 2; $J_{1,2}$ is the proportion of co-infected hosts; $I_1 = J_1 + J_{1,2}$ and $I_2 = J_2 + J_{1,2}$ are the net incidences of pathogen 1 or pathogen 2, respectively. The proportion of co-infections, $J_{1,2}$, is not equal to the product of the pathogens' net incidences, $P=I_1 + I_2$. This deviation from statistical independence is due to host mortality, and therefore mostly concerns pathogens causing long-lasting infections. B. The Binomial model assumes non-interacting pathogens are statistically independent, while the Non-interacting Similar Pathogens (NiSP) model does not make this assumption, which is especially strong in plant viruses making long-lasting infections in their hosts. Although the NiSP model is a better fit to the data than the Binomial model, there is evidence of lack of goodness of fit, and so our test indicates these pathogens interact (or are epidemiologically different). Data from Seabloom et al. 2013.

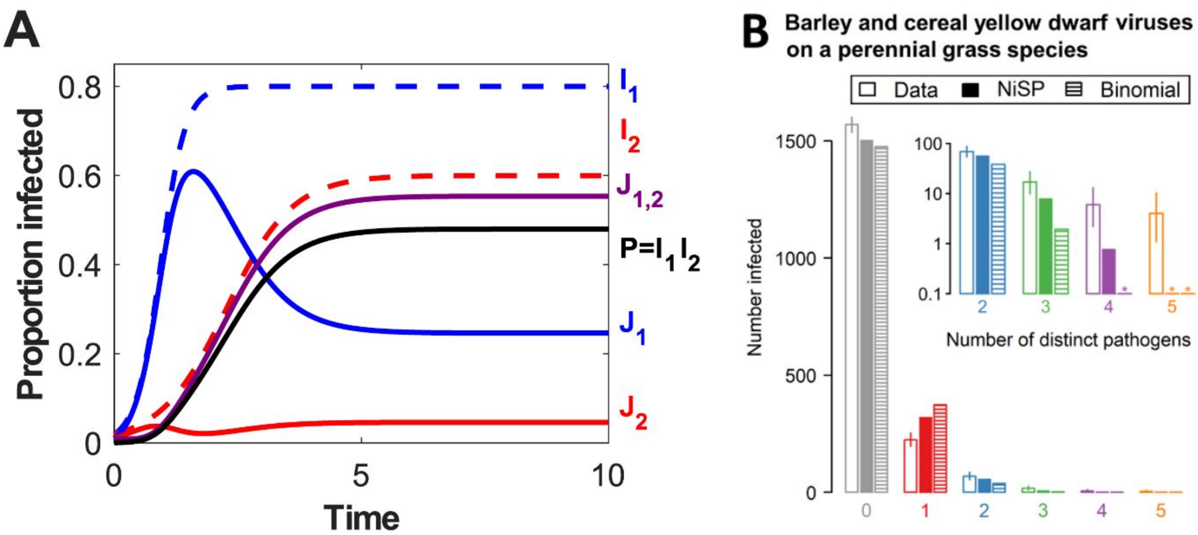


Figure 6. Dynamics of coinfection with two viruses (Allen et al. 2019, republished under an Open Access Creative Common CC BY license). In this special case, neither virus can invade in the absence of the other. A. Prevalence of co-infection with virus A and virus B as a function of the initial frequencies of the two viruses. The black dots represent the endemic co-infection equilibrium in the shaded area and the disease-free equilibrium in the white area. B. In time plots of coinfection, the blue cross and red asterisk indicate initial conditions in different basins of attraction and show convergence either to the disease-free state or to the co-infection equilibrium.

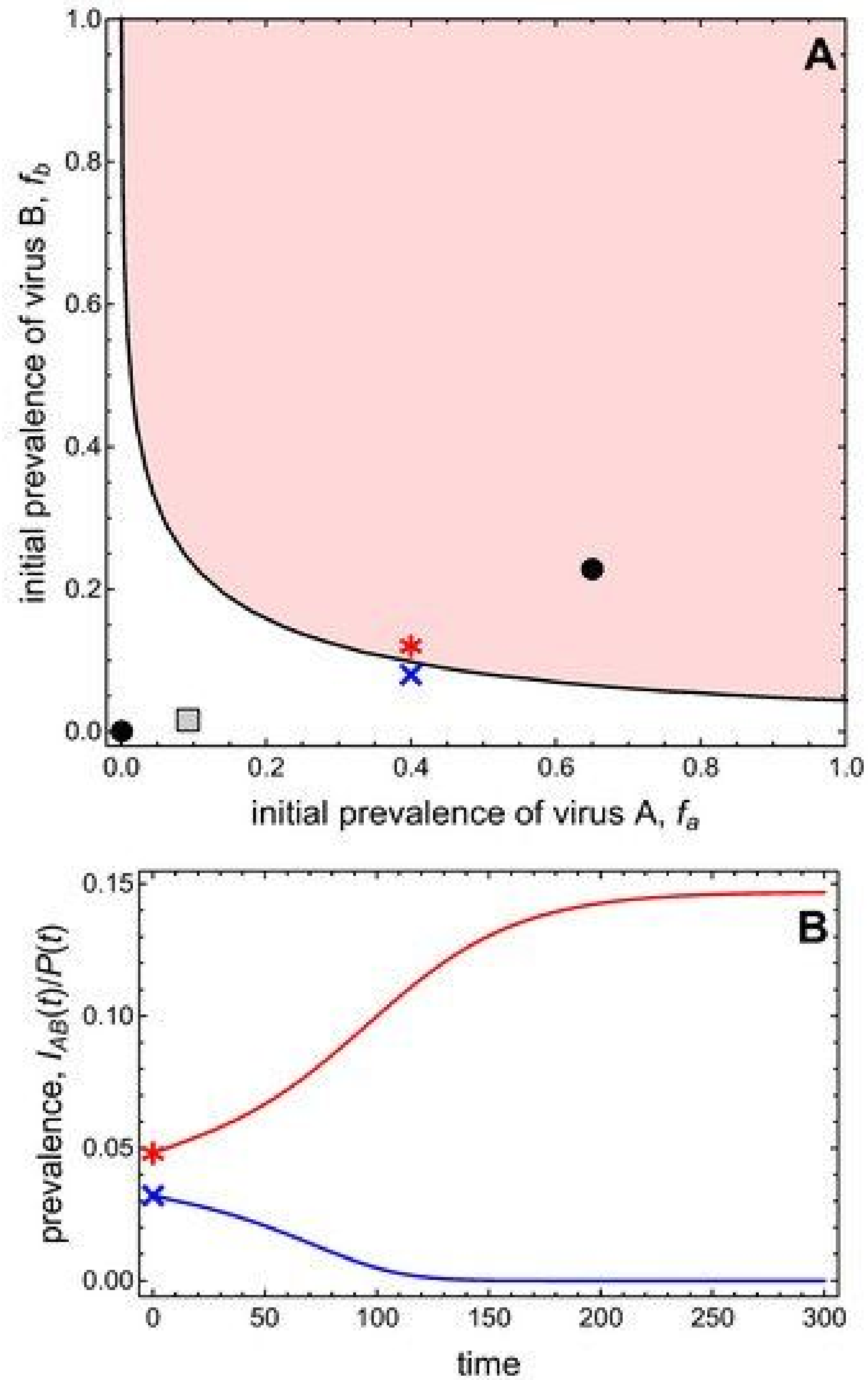


Figure 7. Estimation of epidemiological parameter (pairwise distributions) from experimental data of Bahia bark scaling of citrus using a stochastic model and Monte Carlo Markov Chain (MCMC) techniques (Cunniffe et al. 2014, republished under the terms of the Creative Commons Attribution license).

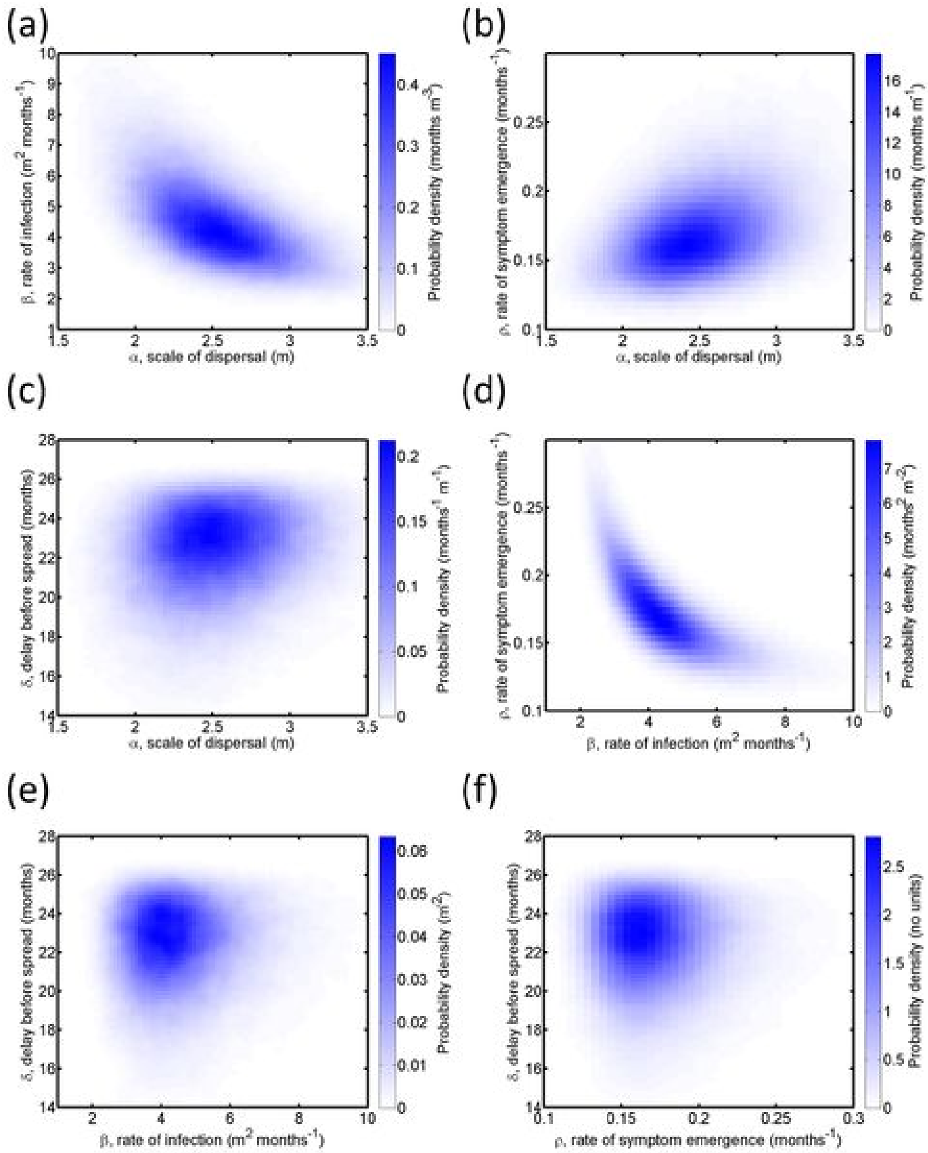
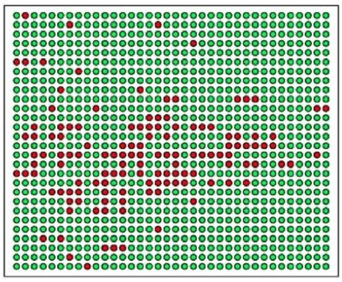


Figure 8. Spread of citrus tristeza virus in an orchard (data from Marcus et al. 1984, re-analysed by Mintner and Retkute 2019, republished under the Creative Commons CC-BY-NC-ND license). Green circles represent healthy trees, red circles infected trees.

Infected in 1981



Infected in 1982