HAL
open science

Can biological control be a strategy to control vector-borne plant viruses?

Sacha Roudine, Cécile Le Lann, Sophie Bouvaine, Anne Le Ralec, Joan van Baaren

To cite this version:

Sacha Roudine, Cécile Le Lann, Sophie Bouvaine, Anne Le Ralec, Joan van Baaren. Can biological control be a strategy to control vector-borne plant viruses?. Journal of Pest Science, In press, 10.1007/s10340-022-01587-0 . hal-03950921

HAL Id: hal-03950921

https://hal.science/hal-03950921v1

Submitted on 22 Jan 2023

HAL is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.

Copyright

Can biological control be a strategy to control vector-borne plant viruses?

This is a post-peer-review, pre-copyedit version of an article published in Journal of Pest Science. The final authenticated version is available online at: https://doi.org/10.1007/s10340-022-01587-0

Sacha Roudine[1], *Cécile Le Lann*[1], *Sophie Bouvaine*[2], *Anne Le Ralec*[3], *Joan van Baaren*[1]

[1]University of Rennes, CNRS, ECOBIO (Ecosystèmes, Biodiversité, Evolution)-UMR 6553, Rennes, France
[2]Natural Resources Institute, University of Greenwich, Central Avenue, Chatham Maritime, Kent, ME4 4TB, UK
[3]UMR IGEPP, INRAE-Agrocampus Ouest-Université de Rennes 1, F-35600 Le Rheu, France

Abstract
Plant viruses transmitted by vector pests are one of the most important worldwide threats to global food production and security. Biological control strategies to enhance natural enemies (parasitoids and predators) have mainly focused on their ability to reduce pest density. In contrast, few studies have examined how natural enemies affect the spread and the incidence of viruses in a crop, although those results could be used as levers for a more sustainable management of viral diseases. Vector-borne plant viruses can be classified in three categories based on their transmission mode: non-persistently transmitted viruses, semi-persistently transmitted viruses and persistently transmitted viruses, whereas vector density, fitness and movement were identified as main drivers of virus spread in a crop, their relative contributions to virus epidemiology may also depend on both the transmission mode and the presence of natural enemies. The first part of the review focuses on virus transmission dynamics in relation to vector activity and density. Because we identify different patterns for each type of plant viruses, control strategies that lead to changes in vector traits, should be adapted to the targeted virus. However, biological control of insect vectors has been rarely adapted to the mode of transmission of the target virus. Thus, the last part of the review explores the conditions required for natural enemies (parasitoids and predators) to prevent epidemics outbreaks of each type of plant viruses. Briefly, if combined with other practices, biological control of vectors to keep virus incidence below the economic threshold is a promising approach for persistently transmitted viruses but might be more difficult to achieve with non-persistently transmitted viruses and semi-persistently transmitted viruses.

Keywords
Non-persistently transmitted viruses; persistently transmitted viruses; virus outbreaks; conservation biological control; predators; parasitoids

Key message

- There is a knowledge gap on biological control as a way to mitigate the spread of vector-borne viruses in crops, specifically whether biological control of insect vectors could maintain virus damage below the economic threshold.

- The effects of biological control on virus spread are not easy to predict because natural enemies (predators and parasitoids) can affect different insect vector characteristics simultaneously (fitness, behaviour) but not with the same magnitude and direction.

- We reviewed, for each transmission mode, the conditions required for natural enemies to prevent virus outbreaks in crops for each transmission mode.

- If combined with other practices, biological control of vectors is a promising approach to keep persistently transmitted virus incidence below the economic threshold.

- Additional studies in greenhouse and in field conditions are needed to quantify the impact of natural enemies on virus incidence and damage.

Introduction

Plant viruses are one of the most important worldwide threats to global food production and security. Despite the implementation of control strategies, plant viruses are responsible for major yield losses, destroying between 10 and 15% of crop production (Loebenstein 2008). Plant viruses are obligate parasites that depend on a host to survive and replicate. A majority (about 70%) of plant-infecting viruses are dependent on vectors (vector-borne viruses) for their transmission from one host plant to another (Hogenhout et al. 2008). Phloem-feeding hemipterans are the most important vectors, being responsible for the transmission of 55% of the vector-borne plant viruses (Hogenhout et al. 2008). The main hemipteran insect vectors include aphids (*Aphididae*), whiteflies (*Aleyrodidae*), planthoppers (*Delphacidae*) and leafhoppers (*Cicadellidae*) (Hogenhout et al. 2008).

Vector-borne plant viruses have been classified into three categories based on the time between the acquisition access period, when the insect feeds and acquires the virus, and the inoculation access period, when the insect becomes able to transmit the virus (Nault 1997). This classification differentiates among persistently transmitted viruses, semi-persistently transmitted viruses and non-persistently transmitted viruses. Firstly, persistently transmitted viruses have long acquisition and inoculation access periods, ranging from hours to days, to ensure effective virus acquisition and inoculation. They are ingested by the vector and circulate through the vector digestive system to the salivary glands, involving a close and highly specific interaction between the virus and its vector. They are characterized by a long retention period, during which the vector retains the ability to transmit the virus, which lasts from hours to the whole vector's lifespan. Depending on the capacity of the virus to replicate in vector cells, they are divided in persistently transmitted propagative viruses, which are capable of replication inside the vector, or persistently transmitted circulative viruses, which do not replicate inside the vector. About 47% of vector-borne plant viruses are persistently transmitted, mainly by planthoppers and thrips (for persistently transmitted propagative viruses) and whiteflies, aphids and leafhoppers (for persistently transmitted circulative viruses) (Hogenhout et al. 2008). Most known persistently transmitted viruses do not replicate in their insect vectors (141 persistently transmitted circulative virus species *vs* 47 persistently transmitted propagative virus species) (Hogenhout et al. 2008). In

contrast, non-persistently transmitted viruses have short acquisition and inoculation access periods, ranging from seconds to minutes. After acquiring the virus on an infected source plant, the retention time usually last from few minutes to few hours during which the vector must probe another host plant to transmit it efficiently. In non-persistent transmission, virus particles bind to receptors located on vectors stylets. Non-persistently transmitted viruses are transmitted mainly by transient vectors (mainly aphids) that land on a crop and make brief exploratory probes with their stylets to distinguish host from non-host plants. About 43% of plant vector-borne viruses are non-persistently transmitted (Hogenhout et al. 2008). Finally, semi-persistently transmitted viruses, are generally recognized as intermediate between non-persistently and persistently transmitted viruses. Semi-persistently transmitted virus particles are located in the insect foregut but are not internalized in the insect tissues and are mainly transmitted by aphids and whiteflies (Ng and Zhou 2015). Acquisition and inoculation usually require intermediate feeding time (Li et al. 2016). Only 10% of vector-borne plant viruses are semi-persistently transmitted (Hogenhout et al. 2008) but some of them, including beet yellows virus and cauliflower mosaic virus, are responsible for important crop damage (Ng and Perry 2004).

Transmission by vectors is a complex process involving interactions between the virus, the vector and the host plant (Irwin and Thresh 1990). For all transmission modes, the spread between host plants depends on the density of infective vectors, the number of interactions between vectors and host plants and the probability of transmitting the virus per plant visit (Fabre et al. 2003; Kirchner et al. 2011). This number of interactions is not only a function of the overall abundance of infective vectors but rather related to vector spatial occupancy rate on host plants (Power 1991) as only one vector individual suffices to transmit the virus in most cases. The probability of virus transmission per plant visit also affect patterns of infection and depends on the virus type (Madden et al. 2000). Indeed, the probabilities of non-persistently transmitted virus and semi-persistently transmitted virus transmission are high in the early stages of host plant selection by a vector, whereas the probability of persistently transmitted virus transmission increases when the vector settles on the host plant. (Caraco et al. 2001). Thus, predicting vector-borne virus dynamics is complex as vector population dynamics, vector behaviours, infected host spatial distribution and transmission modes are key parameters for understanding patterns of infection in fields.

Until now, management strategies to reduce transmission have mainly targeted vector–plant interactions to reduce the transmission by limiting vector density through insecticides use (Perring et al. 1999). Seed treatments with neonicotinoid compounds was the main method to prevent viral disease outbreaks of crops including maize, soybean, barley, wheat or beet but was banned in Europe in 2018 (Jeschke et al. 2011). Many insects transmitting viruses have developed resistance against insecticides that are currently used (Foster et al. 2007; Bass et al. 2015). Insecticides also have detrimental effects on biodiversity, ecosystem functioning and human health (Koureas et al. 2012; Chagnon et al. 2015). Insecticides use may even increase, rather than suppress the spread of virus transmission by inducing higher vector dispersion between plants (Budnik et al. 1996) and may be ineffective in preventing non-persistently transmitted viruses, as it may not kill transient vectors fast enough to prevent virus spread (Zafaroni et al. 2021).

Other more environmentally friendly management methods include cultural control and the use of genetic resources (resistant or tolerant plant cultivars) to control viral disease. The

most common cultural control approaches rely on the use of certified seeds and the reduction of the number of infected plants in the field through rogueing of symptomatic host plants (cultivated or weeds) (Jeger et al. 2004). This strategy is only likely to be effective if carried out on a large spatial scale, which is rarely feasible. The breakdown of plant resistance by virus populations and the time needed to bring new resistant cultivars to the market also restrict genetic resource applications (Mc Namara et al. 2020). Neither insecticides, nor cultural approaches alone are sufficient to maintain virus incidence below the economic threshold.

In this context, there is an urgent need to assess the effect of alternative management strategies (Jactel et al. 2019). A complementary way of limiting virus spread can be to take advantage of ecological services provided by biodiversity, such as conservation biological control. In the environment, pathosystems are included in complex food webs. For instance, insect vectors interact with their natural enemies (predators and parasitoids), which in turn affect the transmission of plant viruses (Jeger et al. 2012). Mortality induced by the consumption of vectors by natural enemies (*i.e.* consumptive effects) can be a major biotic factor shaping vector population dynamics (Pimentel 2005), minimizing the spread of plant viruses. Natural enemy timing in relation to vector population build up in a crop, natural enemy abundance, searching efficiency and foraging behaviour (Ekbom et al. 1992; van der Werf 1995; Driesche and Hoddle 2009) are additional factors that determine the efficacy of biological control in limiting virus outbreaks. However, the effect of natural enemies on the dynamics of plant viruses is not limited to vector consumption as natural enemies can also mediate changes in vector morphology, physiology or behaviour that can in turn affect the spread of plant viruses (Finke 2012) **(***i.e.* non-consumptive effects). Both consumptive and non-consumptive effects should therefore be considered to evaluate the effect of natural enemies on virus incidence and impacts.

Research on the link between natural enemy-vector dynamics and the link between vector-virus dynamics is extensive, but one of the levers in the management of viral diseases, the biological control, has been poorly studied. The link between natural enemies and viruses can only be understood through natural enemy-vector-virus interactions, and therefore the mode of transmission. Here, we review the data available on the control of vector-borne plant viruses by vector natural enemies to identify the key epidemic parameters for each type of virus considering their transmission modes. First, we will focus on virus transmission dynamics in relation to vector activity and density, by identifying the different patterns of each type of plant viruses. Based on these patterns, we aim in the second part to understand the mechanisms by which natural enemies can affect the incidence of plant viruses. Finally, we identify the conditions required for natural enemies to prevent virus outbreaks in crops by maintaining virus incidence below economic threshold, including natural enemy timing and the magnitude of vector population reduction to achieve. We use this information to propose options for the management of plant viruses.

1- Vector activity and density as determinants of virus transmission dynamics

In the absence of inoculum in the propagation material, the initial virus infection in a field depends on viruliferous vectors migrating from external sources (primary spread). A second cycle of spread arises when vectors proliferate and disperse within the field after acquiring the virus from infected plants (secondary spread). Depending on the host plant, vectors can be

classified as "colonizing" (or residents), which spend the majority of their cycle on a single host plant, or "non-colonizing" (or transient), which visit a large number of plants over a given period of time **(Figure 1**). Non-colonizing vectors land on a plant and make an exploratory probing. When the plant is rejected as unpalatable, vectors quickly disperse to another host plant. Non-colonizing vectors contribute more than colonizing vectors to the spread of viruses that are transmitted quickly during brief probes because of their high mobility between host plants, despite generally lower transmission efficiency than colonizing vectors (Raccah et al. 1985; Maling et al. 2008 ; Galimberti et al. 2020). Colonizing vectors are also reported to be important to overall spread of certain non-persistently transmitted viruses (Kaya et al. 2014), in particular when they are induced to change their host in response to biotic (e.g. low plant quality, high vector density) or abiotic stress (e.g. lower temperature). For instance, Maling et al. (2008) showed that if the number of flights made by colonizing vectors each day increase (from 0 to 0.2 flight per vector), yellow mosaic virus incidence is likely to double. Non colonizing vectors are often the main vectors of non-persistently transmitted viruses and some semi-persistently transmitted viruses, while colonizing vectors are the main vectors of persistently transmitted viruses and some semi-persistently transmitted viruses that require longer feeding times (Fereres and Moreno 2009).

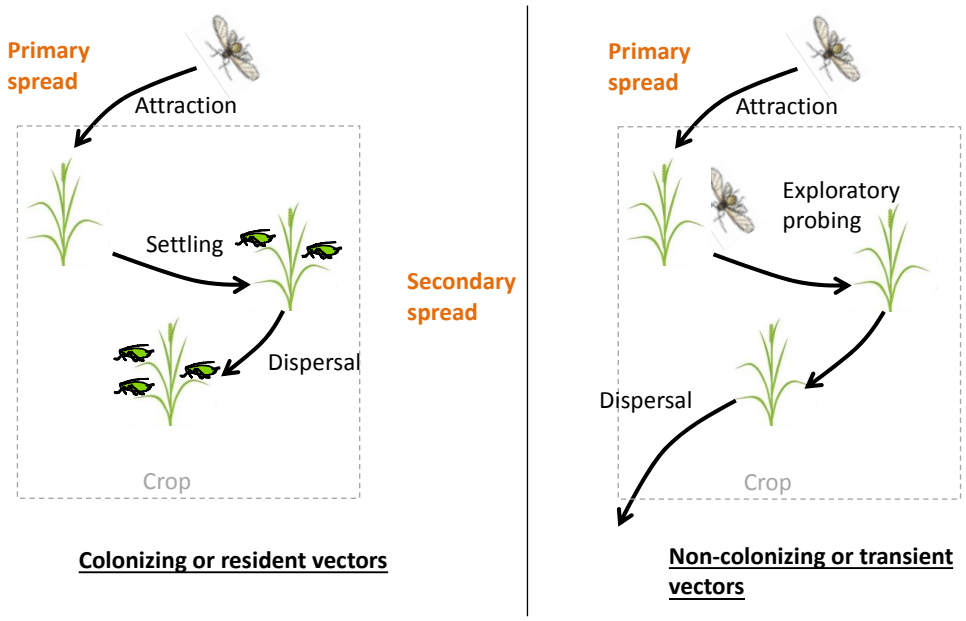

Fig. 1 Patterns of virus spread in relation to vector colonisation. The vector is an aphid but the principles illustrated apply to other vector species. Vectors are attracted by chemical or visual cues. Once hosts are located, vectors must interact with the plant for virus transmission. The inoculation occurs during short probing or sustained feeding, ensuring primary spread if vectors are viruliferous. Non-colonizing vectors make an exploratory probing and when the plant is rejected as unpalatable, vectors quickly disperse to another host plant, facilitating transmission. Colonizing vectors settle in the crop and spend most of their cycle on a single host plant. In response to abiotic stresses (e.g., climatic conditions) or biotic stresses (e.g., host defences, host nutritional status, chemical cues, crowded conditions) vectors disperse to other susceptible hosts, facilitating secondary transmission.

A- Primary spread

Vector immigration is a key parameter in plant virus epidemiology. Its effect on virus spread depends on the proportion of lost vectors (by death or emigration) replaced by immigrants and the proportion of viruliferous vectors in the immigration pool (Madden et al. 2000). In systems with a continuous flow of migrants from external sources, the rate of virus spread is very high regardless of the mode of transmission because new plant infections will result both from infected plants inside the field and from vectors carrying the virus from outside the field (Roy et al. 2021; Zaffaroni et al. 2021). At relatively low vector immigration rate, virus incidence can reach disease saturation, which hinders the effect of natural enemies. Holt et al. (1999), using tomato leaf curl disease as a model, showed that a vector immigration rate of approximately 0.1 individuals per plant per day is sufficient to cause full infection of a crop and to make virus incidence unaffected by the vector mortality within the field.

The proportion of viruliferous vectors in the immigration pool affect virus primary spread in a field differently depending on the transmission mode. The epidemiology of persistently transmitted viruses is affected by the proportion of viruliferous immigrants rather than the number of migrants landing on a crop because of the long times needed for a vector in the field to acquire and inoculate the virus to a healthy plant (Madden et al. 2000). As an example, the epidemic model developed by Matsukura et al. (2017) for southern rice black-streaked dwarf virus, predicted a high contribution of the density of viruliferous immigrant vectors (a planthopper) to the virus spread in a field. Similar results were observed with barley yellow dwarf viruses (BYDV). Here, a proportion of viruliferous aphid migrants below 5% resulted in less than 20% of infected plants (Thackray et al. 2009). By contrast, non-persistently and semi-persistently transmitted virus spread are less influenced by the proportion of viruliferous immigrants because of the short times needed for acquisition and inoculation (Madden et al. 2000). In their epidemiological model, Zaffaroni et al. (2021) showed that even with a small proportion of viruliferous immigrants, the spread of non-persistently transmitted viruses can be continuous in the crop. When immigrants are a continuous source of vectors, we can hypothesize that management strategies to reduce the proportion of viruliferous immigrants closest to 0% reduce persistently transmitted virus primary spread within a field but will probably have less effects on non-persistently and semi-persistently transmitted virus spread. However, epidemiological models for non-persistently or semi-persistently transmitted viruses rarely include immigration parameters such as the fraction of viruliferous immigrant vectors. Thus, their contributions to virus spread in a field remain largely unknown. Arrival of migrants in a field could lead to potential introduction of inoculum, but whether this introduction result in epidemics will depend partly on whether vectors go on to colonize new plants (Irwin and Thresh 1990).

B- Secondary spread

The basic reproductive number R_0, defined as the number of infections arising from a newly single infected plant in a healthy population, has been used to provide an estimation of the number of secondary infections that will occur in the plant population following the introduction of the virus (Van den Bosch and Jeger 2017). This parameter mainly depends on vector density per plant, vector mortality or lifespan and vector mobility, estimated as the number of plants visited per unit of time (Holt et al. 1997). In the first mathematical model to predict secondary spread within a field, a constant "contact rate" parameter was used to estimate the number of

plants visited per unit of time (Chan and Jeger 1994; Holt et al. 1997). This constant parameter did not incorporate any transmission characteristics and vector feeding period. In particular, no account was taken of how the probability of transmission could be affected by the feeding period duration. In order to predict and compare more accurately secondary spread dynamics of the four types of viruses, these three characteristics (vector density, mobility and lifespan) were later included in epidemiological models (Jeger et al. 1998; Grilli and Holt 2000; Madden et al. 2000; Shaw et al. 2017)

In general, the incidence of all virus types increases with higher vector density following a logistic growth. However, this increase is always more pronounced for semi-persistently transmitted viruses and persistently transmitted circulative viruses. Indeed, in their theoretical works, Madden et al. (2000) showed that vector densities resulting in a R_0 of 1 (threshold for virus persistence within a crop), were 0.044, 0.072, 0.310 and 1.070 vectors per plant respectively for semi-persistently transmitted viruses, persistently transmitted circulative viruses, non-persistently transmitted viruses and persistently transmitted propagative viruses. The greater number of vectors required for non-persistently transmitted viruses and persistently transmitted propagative viruses can be linked to the short infectious period for non-persistently transmitted viruses (lost after several probing) and to the dependence upon the vector for persistently transmitted propagative virus replication. Similarly, in their modelling work on potato viruses, Kho et al. (2020) showed that, with the same initial vector density, the spread of a persistently transmitted circulative virus, the potato leafroll virus (PLRV), was seven times faster than the spread of a non-persistently transmitted virus, the potato virus Y(PVY). This suggest that lower vector densities are required for the spread of persistently transmitted circulative viruses as they can be transmitted to different plants multiple times by a single vector. Thus, as high vector densities are required for the spread of non-persistently transmitted viruses and persistently transmitted propagative viruses, strategies to reduce resident vector density may be more effective on these two types of viruses.

Secondary spread in a field is also dependent on vector movements from one plant to another. The link between vector mobility and disease dynamics depends on the mode of transmission considered. A small change in vector mobility between host plants is expected to have stronger impact on the incidence of non-persistently and semi-persistently transmitted viruses than persistently transmitted viruses (Madden et al. 2000). Indeed, the faster the acquisition and transmission times are, the stronger the association between virus spread and vector mobility is. With the short acquisition and transmission times of non-persistently and semi-persistently transmitted viruses, a short visit time per plant associated to higher vector-host plant encounters is not a constraint and will favour the spread of the virus (Ng and Falk 2006). In particular, non-persistently transmitted virus spread is largely dependent on the rapid dispersion of vectors from infected plant after probing because if vectors settle on the plant and feed from the phloem following viral acquisition, the viruliferous status of the vector may be lost (Jeger et al. 1998). Conversely, for viruses with a long acquisition and retention time (*i.e.* persistently transmitted viruses), the potential increase in virus spread associated with high vector mobility between host plants might be negated by the decreased feeding time available per visit (Crowder et al. 2019). For instance, reducing vector feeding period on host plant can limit persistently transmitted virus spread within a field (Grilli and Holt 2000). Several theoretical

studies have shown that management strategies aiming at altering vector movement, by reducing the number of plants visited, are more effective in controlling plant viruses than strategies targeting vector abundances, in particular with non-persistently transmitted viruses (Crowder et al. 2019; Zaffaroni et al. 2021).

Finally, vector life history traits such as lifespan exert an influence on secondary spread. We expect that a reduction of vector lifespan arising for example from the presence of natural enemies in a crop has less impact on the epidemics of non-persistently or semi-persistently transmitted viruses than persistently transmitted viruses (Madden et al. 2000). As persistently transmitted viruses require a long period of acquisition and latency, reducing vector lifespan could reduce the likelihood of transmission of these viruses. For example, by comparing the effect of parasitoids on the dynamics of cucumber mosaic virus and cucurbit aphid-borne yellows virus, transmitted by aphids respectively in a non-persistent and persistent manner, Dáder et al. (2012) showed that a parasitism-induced reduction in aphid lifespan only affects persistently transmitted viruses spread. Thus, management strategies that aim to reduce the lifespan of vectors might reduce persistently transmitted virus secondary spread but might have no or reduced effect on non-persistently and semi-persistently transmitted viruses. In conclusion, vector density, movement and fitness are all drivers of virus spread in a field, but their contribution to virus epidemiology will depend on each mode of transmission.

Because of the differential effects of vector characteristics on primary and secondary spread patterns, control strategies should be adapted to the targeted virus, in particular if they lead to changes in vector density, movement or life history traits. The sensitivity of the epidemiology of each type of virus to vector characteristics is summarized in **Figure 2**. Control strategies to prevent virus outbreaks, including biological control, usually affect these different vector characteristics simultaneously but not with the same magnitude and direction. Therefore, the effect of these strategies on virus dynamics is not easy to predict (Crowder et al. 2019). It is even more complicated because virus infection might affect higher trophic levels through vector and natural enemy fitness and behaviour (Moiroux et al. 2018).

Viruses can affect their vectors directly after acquisition or indirectly (plant mediated). Direct effects of viruses on their vectors are expected to occur mainly for viruses that are internalized by the vectors (persistently transmitted viruses) (Eigenbrode et al. 2018). After acquisition, viruses can manipulate the settling, probing and feeding behaviour of their vectors (Moreno-Delafuente et al. 2013; Ingwell et al. 2012) which can greatly increase virus spread in a crop (Shaw et al. 2017). Indirect effects of viruses on their vectors can arise when plant defence responses are manipulated by the virus, modifying the behaviour or the fitness of vectors.

Different manipulation strategies, reviewed by Shi et al. (2021) are expected depending on the mode of transmission. For non-persistently and semi-persistently transmitted viruses, infection tends to induce plant defence responses that increase vector dispersal and repeated landing. By contrast, for persistently transmitted viruses, virus infection tends to reduce plant defence response and manipulates plant quality inducing an increase of vector fitness and ensuring long-term feeding. Virus infection can also alter positively or negatively natural enemy effects on vector population. For example, it has been reported that faster development of vectors on infected plants can reduce predation risks (Belliure et al. 2008). By contrast, infected plants can attract natural enemies (Mauck et al. 2015) or viruliferous vectors can be more

vulnerable to natural enemy attacks (de Oliveira et al. 2014) favouring biological control. These effects should be considered in vector management strategies, particularly when virus presence may reduce natural enemy abundance and biological control.

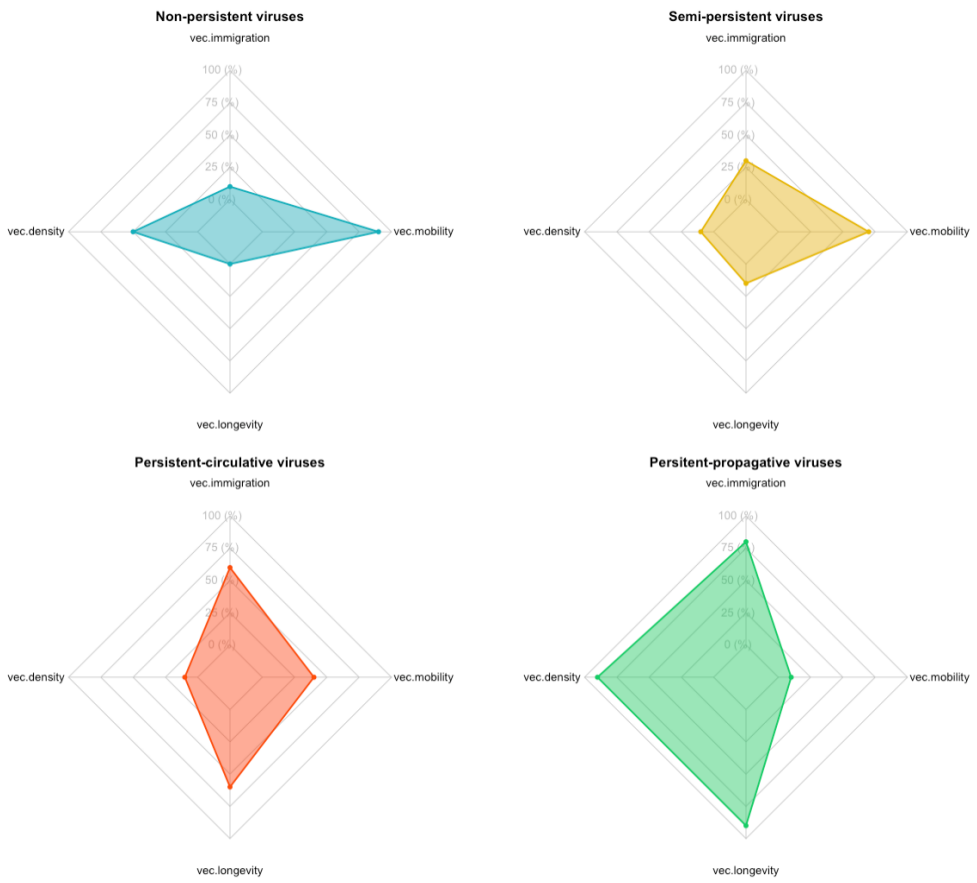

Fig. 2 Relative influence of vector characteristics on virus epidemiology. The graphs were built based on the figures presented in Madden et al. 's (2000) paper, which correspond to the sensitivity of models to different vector characteristics depending on the mode of transmission. Vector characteristics include vector density (vec.density), vector longevity (vec.longevity), vector movement (vec.mobility) and the proportion of viruliferous immigrants (vec.immigration). The percentages are derived from each figure in Madden et al. (2000); a high percentage means that the characteristic considered strongly influences epidemiology of the virus, i.e. a small change in this characteristic can greatly alter the spread of the virus.

2- Effects of natural enemies on vector-borne viruses

A- Consumptive and non-consumptive effects

We limited this paper to the enhancement of predators and parasitoids of insect vector. In field conditions, it refers to Conservation Biological Control, i.e. the implementation of practices that maintain and enhance the reproduction, survival, and efficacy of natural enemies like predators and parasitoids (Landis et al. 2000). We added, when pertinent, a few references about inundative biological control, i.e., the release in large number of natural enemies (mainly in greenhouses).

There is a general relationship between pest abundance and damage to cultivated plants and thus an 'economic threshold' above which the benefit of control measures (generally insecticides) justifies the associated cost (Pedigo et al. 1986). This decision rule for appropriate control measures has also been adapted for natural enemies (Zhang and Swinton 2009) and can be applied to virus epidemics (Jones 2004). For virus disease, the threshold is the virus incidence threshold above which economic losses are sufficient to justify control measures.

Most studies that have explored the effect of conservation biological control in maintaining pest populations at acceptable levels have focused on the ability of natural enemies to reduce pest density (van Lenteren 2000; Pimentel 2005). Mortality induced by natural enemies may keep pest populations below the economic thresholds, where there is little or no risks of yield losses (Albajes and Alomar 1999; Boivin et al. 2012). However, only few studies have attempted to quantify vector reduction required to have an effect on the incidence of vector-borne viruses in a crop, whether or not it is below the economic threshold. For example, Jeger et al. (2004) showed that the density of the whitefly *Bemisia tabaci* must be reduced to less than 15% of its natural density, to eradicate the African cassava mosaic virus (a persistently transmitted circulative viruses). For the other modes of transmission no study is available addressing this threshold with the exception of the theoretical work of Madden et al. (2000) which defines thresholds of vectors per plant needed to prevent virus outbreaks. Based on the Holt et al. 1997 model for the African cassava mosaic virus (without considering transmission characteristics), Okamoto and Amarasekare (2012) investigated the consequences of introducing a biocontrol agent in the model. Interestingly, they showed that vector eradication is not a prerequisite for reducing virus incidence and that natural enemies can successfully reduce long-term incidence even though they may only slightly reduce vector densities but whether this reduction is sufficient to keep virus incidence below the economic threshold was not assessed there.

The magnitude of direct consumptive effects of natural enemies on virus spread will depend in part on natural enemy trophic specialisation. It can be hypothesized that given the very low number of vectors required for the spread of persistently transmitted circulative viruses within a crop (Madden et al. 2000), species that exert a strong numerical response on vector densities could be effective in maintaining virus incidence below the economic threshold, by keeping colonising vector populations at very low levels. Specialists, that feed on a narrow range of prey type, are expected, in some cases, to exert a stronger numerical response than generalist that exploit a wide range of prey species (Schmidt et al. 2003; Driesche and Hoddle 2009) but whether this has consequences for viral incidence has never been tested.

The magnitude of consumptive effects of natural enemies on virus spread will also depend on the timing of natural enemy settlement in the target crop. Due to the very quick development cycle of vectors, natural enemy activity may reduce vector density, but may not bring vector densities below the economic threshold, once they expand exponentially in the crop (Landis et al. 2000; Raymond et al. 2015). Generalists predators regulate vector populations early in the season because of their ability to maintain themselves in an environment through the presence of alternative preys/hosts and their ability to prey on vectors when they are in low density (Symondson et al. 2002; Mesmin et al. 2021). On the other hand, due to the tight interaction with one or few prey species, specialists might not establish before their host or prey in a crop, making unlikely to maintain vector density below the economic threshold if only

specialists are present. However, some specialists (in particular parasitoids) can exhibit very rapid development cycles (Godfray 1994) and could trigger a rapid response on vector densities via mortality and a decrease in reproduction induced by parasitic castration (Digilio et al. 2000; Beckage and Gelman 2004).

Through non-consumptive effects, natural enemies can impact various vector traits, including physiological, morphological or behavioural traits. In particular, natural enemy-induced defensive responses represent a cost to the fitness and the survival of vectors which can affect vector population growth (Nelson et al. 2004). Natural enemies can reduce fecundity and survival of vectors via reduced feeding time on the plant arising when vectors flee to a new habitat to avoid natural enemies (Losey and Denno 1998; Peckarsky and McIntosh 1998). These non-consumptive effects can in turn indirectly affect the virus transmission process. For example, predators can limit the spread of wheat dwarf virus in barley fields by altering the feeding behaviour of the leafhopper *Psammotettix alienus* with delayed and reduced acquisition and inoculation phases (Tholt et al. 2018). Non-consumptive effects of natural enemies are amplified for aphids as they exhibit a polymorphism whereby individuals are either winged or apterous. The winged morphs are produced in response to adverse abiotic and biotic conditions to ensure the colonization of new hosts. Thus, when exposed to parasitoids or predators, aphids increase their rate of dispersal by producing winged offspring (Weisser et al. 1999 ; Sloggett and Weisser 2002), which might increase virus spread within a crop.

Several mechanisms affect the magnitude and the direction of non-consumptive effects on virus spread: vector previous exposure to risk, foraging strategies of natural enemies and compensatory reproduction. Vectors that had been previously exposed to predator cues increased dispersal, reduced feeding time and suffer less predation than unexposed aphids (Tamai and Choh 2019). The effect of foraging strategies on non-consumptive effects is well illustrated by the review of Preisser et al. (2007) which found that predator hunting mode (e.g., actively hunting, sit-and-pursue, sit-and-wait) will determine the strength and intensity of non-consumptive effects on prey; with the 'sit and pursue' hunting mode triggering the stronger non-consumptive effects. Vectors may also respond to natural enemy risk through compensatory reproduction, which can in turn offset the direct consumption of vectors by natural enemies (Lee et al. 2022).

There is cumulating evidence in controlled conditions that natural enemies alter movements of vectors which in turn affect vector feeding duration and plant-to-plant movement with various consequences on virus spread (Jeger et al. 2012; Claflin et al. 2015). The magnitude and the direction of non-consumptive effects on virus spread is likely to differ depending on transmission mode and whether vector movement results in reduced feeding duration or increased vector-host encounter rates. The few studies that have attempted to separate the indirect and direct effects of natural enemies on virus spread revealed that, in controlled conditions, interactions that altered vector behaviours (non-consumptive effects) may contribute more to virus spread than those affecting vector fitness and density (Crowder et al. 2019; Lee et al. 2022). However, in more complex conditions (like in greenhouses or fields), the exact opposite is observed with virus spread being more strongly influenced by vector life history traits (Clark et al. 2019).

B- An overview of studies on the effect of natural enemies on non-persistently transmitted virus and semi-persistently transmitted virus spread

To date, only seven studies have investigated the relationship between natural enemies and non-persistently transmitted virus or semi-persistently transmitted virus incidence, including eight natural enemy-vector interactions (**Table 1**). Vectors are exclusively aphids. The studies were all carried out under controlled conditions, which allowed a better understanding of the mechanisms by which natural enemies impact virus spread but may be not representative of what happen in field conditions. The only study available for semi-persistently transmitted viruses was a field-conducted experiment (Landis and Van der Werf 1997). Among the eight interactions tested, two clearly showed a positive effect of natural enemies on the proportion of infected plants, four showed no significant effect of the presence of natural enemies and two studies had opposite results depending on the experimental conditions: duration of the trial (Dáder et al. 2012) and the presence of non-host plants (Claflin et al. 2015) (see **Table 1**).

The effect of natural enemies on non-persistently transmitted viruses under controlled conditions seems to be driven by their effects on vector plant-to-plant movement. When natural enemies promote movement between host plants, the spread of non-persistently transmitted viruses is increased (Roitberg and Myers 1978; Dáder et al. 2012). The magnitude of these effects appears to be primarily dependent upon the escape response of the vector. For example, when the interaction involves the pea aphid, *Acyrthosiphon pisum*, that shows a typical drop and move escape response, natural enemies positively affect virus spread (Roitberg and Myers 1978; Hodge et al. 2011). Conversely, the effect of natural enemies on non-persistently transmitted viruses involving *Myzus persicae* or *Aphis gossypii,* which remain on the plant and walk away on the same plant or aggregate, tend to be null (Belliure et al. 2011; Garzón et al. 2015). To a lesser extent, these non-consumptive effects partially depend on the foraging strategies of their natural enemies. Belliure et al. (2011) showed that coccinellid adults induce higher disturbance of *M. persicae* colonies than syrphid larvae resulting in a stronger antipredator behaviour of vectors and a higher spread of the broad bean wilt virus-1 in the presence of coccinellids. Predatory larvae, that are very mobile while exhibiting low movement capacity, are likely to cause less disturbance and less prey movement than natural enemies that tend to fly away, such as ladybeetles (Lommen et al. 2008). Natural enemies tend to enhance or have no significant effect on the initial spread of non-persistently transmitted viruses under controlled conditions, but the outcome seems to be largely variable with each natural enemy-vector interaction. The lack of studies for semi-persistently transmitted viruses makes it impossible to draw general conclusions.

C- An overview of studies on the effect of natural enemies on persistently transmitted virus spread

Eleven studies have investigated the relationship between natural enemies and persistently transmitted virus incidence, including 10 persistently transmitted circulative viruses and only one persistently transmitted propagative virus **(Table 1).** Thirteen natural enemy-vector-virus- interactions have been studied (exclusively in controlled or greenhouse conditions). The majority of the vectors studied were aphids, but two other vector groups were also represented: thrips (*Frankliniella occidentalis*) and whiteflies (*Bemisia tabaci*). Five studies clearly showed a negative effect of the presence of natural enemies on virus incidence. However, three studies revealed a positive effect of the presence of natural enemies on the proportion of infected plants and an equal number of studies were inconclusive (non-significant overall effect).

Finally, two studies had opposite results depending on the duration of the trial (Smyrnioudis et al. 2001; Dáder et al. 2012). Unlike non-persistently transmitted viruses, the anti-predator behaviour of vectors such as higher movement between host plants, does not seem to be a determinant of the effect of natural enemies on persistently transmitted virus spread (see **Table 1**).

In general, when natural enemies alter the feeding duration of vectors by triggering escape behaviours, the spread of persistently transmitted viruses is reduced even if vector dispersal from plant-to-plant increases (Smyrnioudis et al. 2001; Long and Finke 2015). In fact, when vector dispersal occurs in response to natural enemies, feeding might be too short to efficiently transmit the virus. When a natural enemy simultaneously reduces the abundance of a vector and increases its plant-to-plant dispersal without affecting feeding duration, its overall effect on persistently transmitted virus spread will depend on the ratio between consumptive and non-consumptive effects. For example, using a structural equation modelling approach, Clark et al. (2019) demonstrated that the magnitude of the effect of the predatory ladybug *Hippodamia convergens* on the abundance of the pea aphids was greater than the effect of the predator on aphid movement resulting in an overall reduction of the pea enation mosaic virus (PEMV) incidence in small cages. Moreover, the acquisition or inoculation times of persistently transmitted viruses, which vary greatly from one pathosystem to another (from a few minutes to a few hours) (Grilli and Holt 2000; Bragard et al. 2013), may explain to some extent the variations in responses to natural enemies depending on the pathosystem considered. Despite a low transmission efficiency, it has been shown that some persistently transmitted viruses, including PEMV (Demler et al. 1996) and more recently BYDV (Jiménez et al. 2020) can be transmitted after a short inoculation period lasting only a few minutes. Natural enemies tend to reduce or leave unaffected the initial spread of persistently transmitted circulative viruses under controlled conditions, but whether these effects could be observed under field conditions is not yet explored. There are not enough studies to draw conclusions on persistently transmitted propagative viruses.

Tab. 1 Review of publications reporting the effect of natural enemies (parasitoids **Pa** and predators **Pr**) on the spread of each virus type. Significant vector suppression by natural enemies (consumptive effects), significant host-to-host movement induced by natural enemies and anti-predator behaviour of vectors are mentioned to help understand the overall effect of natural enemies on virus spread. This overall effect is mentioned **+**: increase of virus spread, **-**: decrease of virus spread and **NS**: no significant effect. For each transmission mode, studies are displayed according to the overall effect of natural enemies.

	Crop	Virus	Vector	Natural enemies	Experimental conditions	Trial duration	Consumptive effects	Host-to-host movement	Antipredator behaviour	Effect on virus spread	Reference
Non-persistently transmitted	Potato	PVY	*Rhopalosiphum padi*	*Hippodamia convergens* **(Pr)**	Insect cages	3 hours	**No**	**No**	**Low** (Passive[1])	Non-host plant: - Monoculture: +	*Claflin et al. 2015*
	Fava bean	BYMV	*Acyrthosiphon pisum*	*Coccinella californica* **(Pr)**	Insect cages	3 days	**Yes**	**Yes**	**High** (Dropping[2])	**+**	*Roitberg and Myers 1978*
	Fava bean	BYMV	*Acyrthosiphon pisum*	*Aphidius ervi* **(Pa)**	Insect cages	24 hours	**Not measured**	**Not measured**	**High** (Dropping[2])	**+**	*Hodge et al. 2011*
	Cucumber	CMV	*Aphis gossypii*	*Aphidius colemani* **(Pa)**	Insect cages	2 days	**No**	**Yes**	**Low** (Passive[3])	**+**	Dáder et al. 2012
						7 days	**No**	**No**	**Low** (Passive[3])	**NS.**	
	Pepper	BBWV-1	*Myzus persicae*	*Sphaerophoria rueppellii* **(Pr)**	Insect cages	24 hours	**Yes**	**No**	**Low**[4]	**NS.**	*Belliure et al. 2011*
				Adalia bipunctata **(Pr)**	Insect cages	24 hours	**Yes**	**No**	**Low**[4]	**NS.**	
	Cucumber	CMV	*Aphis gossypii*	*Chrysoperla carnea* **(Pr)**	Insect cages	1-5 days	**Yes**	**No**	**Low**[4]	**NS.**	Garzón et al. 2015
				Adalia bipunctata **(Pr)**	Insect cages	1-5 days	**Yes**	**No**	**Low**[4]	**NS.**	
Semi-persistently transmitted	Sugar beet	BYV	*Myzys persicae*	Soil predators **(Pr)**	Field (exclusion cages)	Season-long (4 months)	**Yes**	**Not measured**	**Low**[4]	-	*Landis and Van der Werf 1997*
Persistent transmitted propagative	Impatiens	TSWV	*Frankliniella occidentalis*	*Amblyseius cucumeris* **(Pr)**	Greenhouse	2-6 weeks	**Yes**	**Not measured**	Unknown	-	*Bennison et al. 2002*
Persistent transmitted circulative	Wheat	B/CYDV	*Rhopalosiphum padi*	*Chrysoperla plorabunda* **(Pr)** *Coleomegilla maculata* **(Pr)** *Nabis americoferus* **(Pr)** *Geocoris punctipes* **(Pr)**	Insect cages	2 days	**Yes**	**Yes**	**Low** (Passive[1])	-	*Long and Finke 2015*
	Zucchini	ToLCNDV	*Bemisia tabaci*	*Amblyseius swirskii* **(Pr)**	Greenhouse	20 days	**Yes**	**Not measured**	Unknown	-	*Tellez et al. 2017*
	Pea	PEMV	*Acyrthosiphon*	*Hippodamia convergens* **(Pr)**	Insect cages	8 days	**Yes**	**Yes**	**High**	-	*Clark et al. 2019*

		pisum						(Dropping[2])		
Wheat	BYDV	*Rhopalosiphum padi*	*Aphidius rhopalosiphi* **(Pa)**	Insect cages	7 days	**Not measured**	**No**	**Low** (Passive[1])	-	*Smyrnioudis et al. 2001*
					14 days	**Not measured**	**No**	**Low** (Passive[1])	-	
			Coccinella septempunctata **(Pr)**	Insect cages	7 days	**Not measured**	**Yes**	**Low** (Passive[1])	+	
					14 days	**Not measured**	**Yes**	**Low** (Passive[1])	**NS.**	
Cucumber	CABYV	*Aphis gossypii*	*Aphidius colemani* **(Pa)**	Insect cages	7 days	**No**	**No**	**Low** (Passive[3])	**NS.**	Dáder et al. 2012
					14 days	**Yes**	**No**	**Low** (Passive[3])	**-**	
Potato	PLRV	Myzus persicae	*Coccinella septempunctata* **(Pr)**	Insect cages	2 days	**No**	**Not measured**	**Low**[4]	**NS.**	*Sewell et al. 1990*
Cucumber	CABYV	*Aphis gossypii*	*Chrysoperla carnea* **(Pr)**	Insect cages	7-14 days	**Yes**	**Yes**	**Low**[4]	**NS.**	Garzón et al. 2016
			Adalia bipunctata **(Pr)**	Insect cages	7-14 days	**Yes**	**Yes**	**Low**[4]	**NS.**	
Oat	B/CYDV	*Rhopalosiphum padi*	*Coleomegilla maculata* **(Pr)**	Insect cages	2 days	**No**	**Yes**	**Low** (Passive[1])	**+**	*Bailey et al. 1995*
Faba bean	PEMV	*Acyrthosiphon pisum*	*Aphidius ervi* **(Pa)**	Insect cages	24h	**Not measured**	**Yes**	**High** (Dropping [2])	**+**	*Hodge and Powell 2008*
Pea	PEMV	*Acyrthosiphon pisum*	*Hippodamia convergens* **(Pr)**	Insect cages	5 days	**Yes**	**Yes**	**High** (Dropping [2])	**+**	*Lee et al. 2022*

[1]*personal observation* [2]*Losey and Denno 1998* [3]*Rodrigues and Bueno 2001* [4] *Nelson and Rosenheim 2006*

3- Conservation biological control and management of vector-borne viruses in agroecosystems

A- From controlled conditions to field studies

On Web of Science using search terms "parasitoid*"or "predat*" and "plant virus", 44% of the studies (see **Table 1**) make an explicit claim that natural enemies increase the spread of plant viruses mainly in laboratory conditions. Some studies reported that virus transmission can increase when vectors are parasitized (Christiansen-Weniger et al. 1998). While these studies are very informative, particularly in terms of understanding the mechanisms by which natural enemies can affect the incidence of plant viruses, most of them are short-term experiments (from hours to days) using a limited number of natural enemies and vector taxa in controlled environments. In short-term cage experiment studies, non-consumptive effects of natural enemies mainly represent what happens at the beginning of the natural enemy-vector interaction. High natural enemy pressure may trigger early vector movement shortly after their release in the cage and favour the spread of viruses, the intensity of disturbance will then determine the overall effect of the natural enemy studied (Smyrnioudis et al. 2001). The relative importance of consumptive and non-consumptive effects might change if the duration of the monitoring increases. For example, the presence of *Coccinella septempunctata* increased the spread of BYDV at 7 days but had no significant effect after 14 days (Smyrnioudis et al. 2001). An increase in the duration of the monitoring is particularly relevant for parasitoids (Ingerslew and Finke 2020). Indeed, unlike predators, there is no immediate consumption of the prey and the death of the parasitized host may only occur after a long period of time. Thus, the presence of *Aphidius colemani* had no significant effect on the spread of the cucurbit aphid-borne yellows virus (CABYV) after 7 days but resulted in a reduced spread of the virus after 14 days of trial when the effects on vector abundance begin to be observed (Dáder et al. 2012).

Both consumptive and non-consumptive effects influence virus spread, but their relative importance is difficult to evaluate outside of controlled conditions (Ingerslew and Finke 2020; Lee et al. 2022). Although difficult to measure, non-consumptive effects of natural enemies in the field are unlikely to affect vector populations over the long-term (Ingerslew and Finke 2020; Sheriff et al. 2020) and might have less influence on virus spread than in controlled conditions. In particular, in natural conditions, vectors disturbed by a natural enemy who fall to the ground have a much lower survival rate than in experimental conditions. Vectors that fall to the ground are exposed to other ground predators and to the risk of desiccation and starvation (Dill et al. 1990).

Studies focusing on the management of viruses via biological control in agro-ecosystems are lacking. Based on the theoretical works and the experimental studies mentioned in **Table 1**, we have synthetized the probability that natural enemies successfully prevent virus outbreaks in the field by maintaining damage below the economic threshold for each transmission mode (**Figure 3)**.

B- Can biological control prevent non-persistently transmitted virus outbreaks in crops?

Both colonizing and non-colonizing vectors species contribute to the overall spread of non-persistently transmitted viruses (Atiri et al. 1986; Nebreda et al. 2004) and could therefore act in combination to increase virus transmission. Given the short duration of the exploratory probing, the short acquisition and inoculation access periods and the low fraction of viruliferous

immigrants required for virus spread, it is unlikely that natural enemies present in the crop maintain virus incidence below the economic threshold, via consumptive or non-consumptive effects on non-colonizing vectors (**Figure 3**). However, to our knowledge, no field studies have been carried out to test this claim. Moreover, for some non-persistently transmitted virus pathosystems, colonizing vectors are the main transmitters, that is the case for example for, *cucumber mosaic virus* (CMV) in melon crops which is mainly transmitted by the melon-colonizing aphid species *Aphis gossypii* (Garzo et al. 2004). When colonizing vectors are involved in primary or secondary spread of non-persistently transmitted viruses in a crop, natural enemies could contribute to reduce vector density with consequences for virus spread. Indeed, given that the number of vectors per plant required to maintain an epidemic is intermediate for non-persistently transmitted viruses (Madden et al. 2000), generalist predators present early in the crops might have the potential to maintain virus incidence below economic threshold.

Non-persistently transmitted virus transmission is enhanced by vector movements between host plants. Several studies conducted under controlled conditions showed that natural enemies can favour their spread through non-consumptive effects on vectors (see **table 1**). While several studies report that greenhouse pests can be controlled by releasing natural enemies (Pilkington et al. 2010; Calvo et al. 2012), no studies have explicitly investigated if inundative biological control can sufficiently mitigate the risk of the secondary spread of non-persistently transmitted viruses in greenhouses. For example, CMV, that can be transmitted by more than 80 aphid species, has been described as the virus of greatest economic importance of several plant cultivated in greenhouses including cucurbit, lettuce, pepper and tomato (Gallitelli 2000). To our knowledge, the impact of the release of natural enemies in the greenhouse on CMV spread has not been studied, although the presence of natural enemies could act in synergy with other agronomic levers such as CMV-resistant varieties or the use of sticky traps.

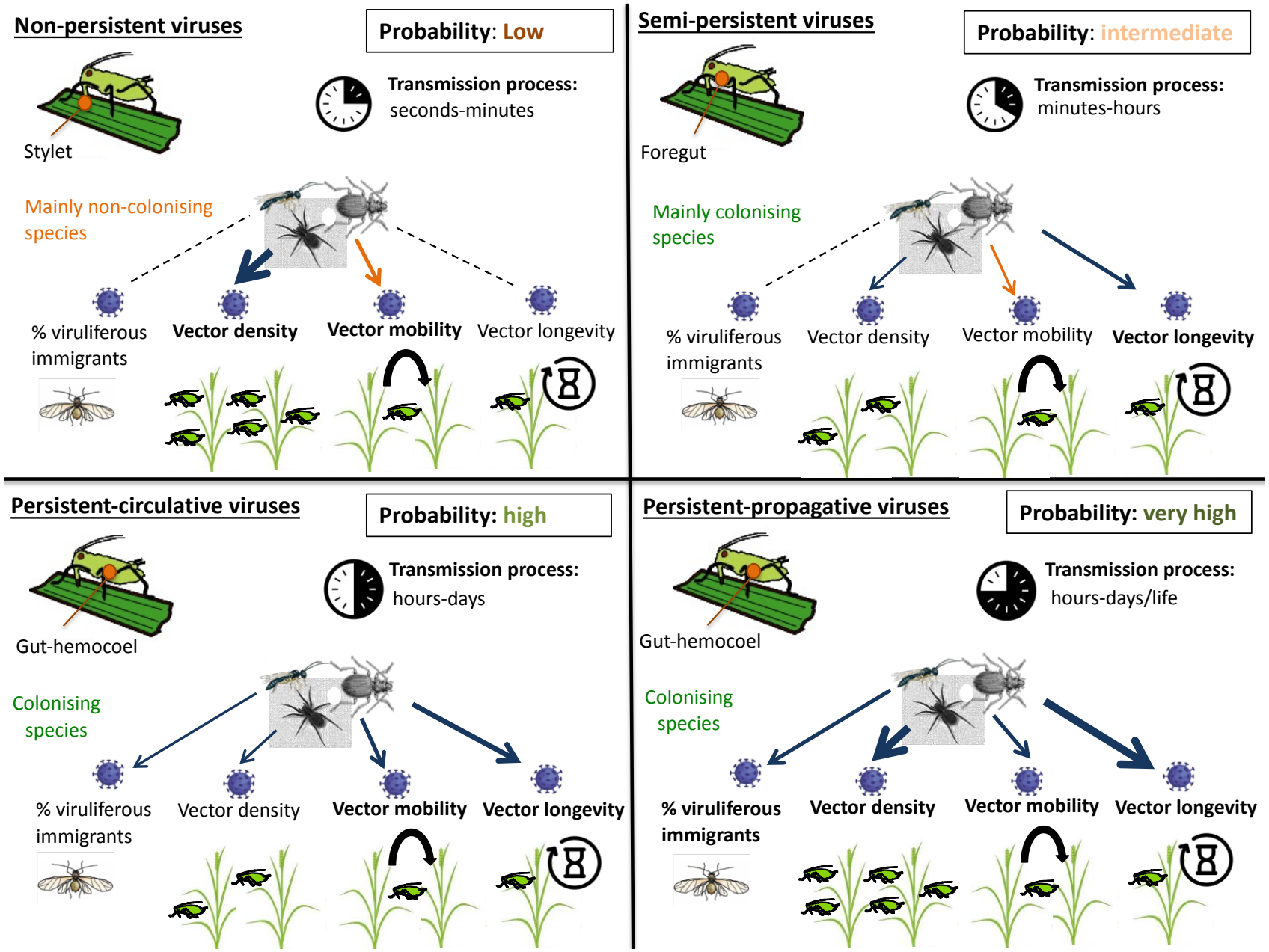
Non-persistent viruses
Probability: Low
Transmission process:
seconds-minutes
Stylet
Mainly non-colonising species
% viruliferous immigrants
Vector density
Vector mobility
Vector longevity
Semi-persistent viruses
Probability: intermediate
Transmission process:
minutes-hours
Foregut
Mainly colonising species
% viruliferous immigrants
Vector density
Vector mobility
Vector longevity
Persistent-circulative viruses
Probability: high
Transmission process:
hours-days
Gut-hemocoel
Colonising species
% viruliferous immigrants
Vector density
Vector mobility
Vector longevity
Persistent-propagative viruses
Probability: very high
Transmission process:
hours-days/life
Gut-hemocoel
Colonising species
% viruliferous immigrants
Vector density
Vector mobility
Vector longevity

Fig 3. Biological control and the epidemiology of the four types of vector-borne viruses. Vector characteristics that can influence the efficiency of natural enemies to affect virus spread are detailed in the figure. For each characteristic an arrow is shown, whose size is proportional to the probability that natural enemies maintain virus spread below the economic threshold via this characteristic. An orange arrow indicates that natural enemies may increase virus spread through the given vector parameter. A blue arrow indicates that natural enemies may reduce virus spread through the given vector parameter. A dotted arrow indicates that natural enemies may have no effect on virus epidemiology through the given vector parameter. Characteristics in bold are those that will play a major role in explaining the link between natural enemies and virus spread. A global probability that natural enemies reduce the spread of each type of virus is given. Other key parameters are also mentioned: the location of viral particles and the time frames of the stages during the transmission process (Acquisition, retention and inoculation).

In greenhouses, special attention should be given to the selection of natural enemies that effectively consume their prey without inducing great vector movements (non-consumptive/consumptive ratio). In particular, the density of released natural enemies will need to be assessed experimentally to determine the optimal number of individuals that will cause the least possible disturbance while consuming the highest number of vectors. Finally, the intensity of the target vector's escape behaviour in response to natural enemies could also be a determining factor in the overall effect of natural enemies in non-persistently transmitted virus spread in greenhouses.

C- Can conservation biological control prevent semi-persistently transmitted virus outbreaks in crops?

For the same reasons as for non-persistently transmitted viruses, the activity of natural enemies in crops is unlikely to affect the primary spread of some semi-persistently transmitted viruses which are spread by non-colonizing vectors. The very low densities of vectors per plant needed to prevent virus outbreaks (Madden et al. 2000), reduce the probability that natural enemy activity keep virus incidence below economic threshold (Figure 3). Nevertheless, the only available study for a semi-persistently transmitted virus, the beet yellows virus (BYV), one of the most important diseases of sugar beet, seems to show that natural enemies can reduce its secondary spread in the field (Landis and Van der Werf 1997). Using soil predator exclusion cages, the authors showed a 2-fold increase of the incidence of the virus four months after the start of the experiment when predators were excluded.

Many semi-persistently transmitted viruses, including BYV, are spread by insects that colonize the plant and prolonged phloem feeding increases virus acquisition and/or inoculation (Zhou et al. 2018). Thus, the probability that semi-persistent virus incidence is kept below the economic threshold by natural enemies is higher than that for viruses that are transmitted quickly during brief probes.

D- Can conservation biological control prevent persistently transmitted virus outbreaks in crops?

Persistently transmitted propagative viruses differ from all other types of viruses (including persistently transmitted circulative viruses) in the number of vectors required to maintain the epidemic (Jeger et al. 1998; Madden et al. 2000). Indeed, viruses that replicate in vector tissues require a high number of vectors to maintain the spread of a virus and are not very sensitive to variations in the vector population around the epidemic threshold (Jeger et al. 1998). For this reason**,** the reduction of the number of viruliferous vectors due to natural enemy activity might be enough to reduce the virus secondary spread and maintain virus incidence below the economic threshold. For instance, the only study on the topic showed that the release of the predatory mite *Amblyseius cucumeris* in a greenhouse maintained the number of Western flower thrips *(Frankliniella occidentalis*) per plant at low level, reducing by 50% the spread of a tospovirus, the tomato spotted wilt virus (Bennison et al. 2002) and reducing symptoms intensity on infected plants. Tospoviruses are transmitted by thrips and infect many important food crops and ornamental plants. In greenhouses, the release of biological agents at the beginning of the crop before the thrips reach the economic threshold is recognised as an effective control method to prevent persistently transmitted propagative virus outbreaks (Reitz et al. 2020). Moreover, Jeger et al. (1998), showed in their modelling work that the lifespan of vectors, which can be strongly affected by the presence of natural enemies, only affects the epidemiology of persistently transmitted propagative viruses. Likewise, their spread is not affected by vector movements

between host plants (Madden et al. 2000). For these reasons, it is likely that natural enemies could play a major role in preventing persistently transmitted propagative virus outbreaks (**Figure 3**) but there is a lack of field studies to support these claims and make this method of control widely accepted.

Biological control to maintain persistently transmitted circulative virus spread below the economic threshold might be more complicated to achieve because of the vector density per plant thresholds required to avoid virus outbreaks in a crop (Jeger et al. 1998; Madden et al. 2000). However, the only study to have measured the effect of natural enemies on persistently transmitted circulative virus epidemiology, under semi-controlled conditions (greenhouses), showed that the pre-release of a predatory mite (*Amblyseius swirskii)* reduced the secondary spread of the tomato leaf curl New Delhi virus (ToLCNDV) through the consumption of the eggs of its vector, the tobacco whitefly (*Bemisia tabaci*) (Tellez et al. 2017).

Unlike non-persistently transmitted viruses and some semi-persistently transmitted viruses, natural enemy activity might reduce the primary spread of both types of persistently transmitted viruses (**Figure 3**). Indeed, given the long acquisition and inoculation access periods required for persistently transmitted viruses to be effectively transmitted, the presence of natural enemies already established in the crop is likely to limit primary spread as long as the fraction of virus-carrying immigrants remains low (<10%) (Madden et al. 2000).

Conclusion and future research prospects

This review highlights the need for additional modelling studies to explore, for each transmission mode, the conditions required for natural enemies to prevent virus outbreaks. Existing modelling studies (Finke 2012; Jeger et al. 2012; Crowder et al. 2019) emphasize the need for quantifying both the consumptive and non-consumptive effects of natural enemies and considering complex ecological interactions including virus infection effect on vector and natural enemy fitness and behaviour. Future modelling studies should focus on levels of vector reduction required to affect virus spread and economic losses and determine whether the action of natural enemies could achieve these levels for each transmission mode. Defining these thresholds could provide levers for farmers and introduce new decision rules, in particular if modelling works seek to determine whether several agronomic levers used in addition to biological control could allow these thresholds to be reached.

Theoretical works and some empirical studies conducted under controlled conditions indicate that natural enemies could greatly affect vector-borne virus spread, but their effects on virus spread under field conditions remain largely unknown. This review highlights the need for additional empirical studies under field conditions that explore, for each transmission mode, the contribution of natural enemies on reduction of virus spread or economic losses. This contribution could be assessed by field exclosure experiments with cages used to exclude natural enemies (Rusch et al. 2013; Gagic et al. 2017). To validate the hypotheses highlighted in this review, regarding the different probabilities of success of natural enemies on mitigating virus spread for each type of transmission, studies could be designed to investigate, in the same crop, the effect of natural enemy exclusion on the spread of different viruses. These studies could be carried out on crops simultaneously affected, for example, by persistently transmitted and non-

persistently transmitted viruses and transmitted by the same vector, as is the case in potato crop with *Myzus persicae* transmitting PLRV and PVY or in cucumber crop with *Aphis gossypii* transmitting CMV and CABYV.

These studies under field conditions should, as far as possible, take into account the complexity of ecological interactions including temporal dynamic and structure of vector-natural enemy interactions, as the success of the implementation of a biological control strategy will rely on our understanding of this greater level of complexity. This includes field experiments with cages set at different periods of crop growing season to assess the contribution of early versus late natural enemy response on virus spread (Mesmin et al. 2021). This also includes efforts to assess the contribution of different natural enemy communities. In semi-controlled conditions (greenhouses), introducing a combination of different natural enemy species is possible in order to determine the optimal community assembly that could maintain virus incidence below economic threshold. In greenhouses, the optimal natural enemy density can also be assessed by varying the number of individuals released in the non-exclusion zone. In field experiments, selective cages could be used to differentiate the contribution of ground-dwelling predators from flying predators or parasitoids (Schmidt et al. 2003). Consequences of natural enemy community structure on virus spread are not well known (Long and Finke 2015) although results could serve as levers for the promotion of conservation biological control.

Although landscape composition is expected to affect natural enemy-vector-virus interaction, landscape effects on vector-borne viruses have received little attention (Carrière et al. 2014; Claflin et al. 2017). Landscape composition could affect virus incidence through both direct and indirect effects on vector populations. Landscape composition is known to directly increase or decrease vector abundance through variation in host crop area (Bosem Baillod et al. 2017). It may also directly modify the proportion of viruliferous vectors through the presence of infected host plants near the field (reservoir) (Fabre et al. 2005). The distance between these host plants and the field plays a key role, especially for the spread of non-persistently transmitted viruses and some semi-persistently transmitted viruses. Indeed, these two types of viruses can be vectored by non-colonising species coming from surrounding areas that can quickly lose their ability to transmit the virus after several probes (Carrière et al. 2014). Landscape composition could also affect virus incidence, indirectly by decreasing vector abundance via their natural enemies (Gagic et al. 2017) but to what extent the landscape-dependent decline in vector abundance induced by natural enemies translates into a decline in virus incidence has never been measured. Future empirical studies on natural enemy-vector-virus interactions must therefore integrate landscape characteristics in order to assess natural enemy contribution to virus spread reduction.

Replacing the use of insecticides will not be possible via enhancement of predators and parasitoids alone. Conservation biological control might cause a relatively small decrease in virus incidence without affecting economic losses. But when integrated with other measures that act in an additive way, the likelihood that damages remain below the economic threshold could be maximised. Several cultural practices have been shown to be effective in limiting the incidence of vector-borne viruses and rely mainly on maintaining spatial and temporal discontinuities in host plants (review in Jones (2004)). The maintenance of temporal discontinuities includes removing

of weeds and volunteers crop plants, crop rotation with non-host plants and manipulation planting date to minimise virus carry over between successive crops by breaking infection and vector cycle. The creation of spatial discontinuities relies on non-host plant diversification within (intercropping) or around the field (barrier crop). These plant diversification strategies have been shown to reduce virus spread through a bottom-up regulation of vector populations (Waweru et al. 2021; Grauby et al. 2022). When vectors are searching for a new host, mixed cropping with non-host plants or tall non-host cover crop around the field could constitute physical barriers and causes vector confusion due to non-host plant chemical cues (Xu et al. 2017) which can lower vector incidence and reduce the period they spend on host plants (Alvarez-Baca et al. 2022). These plant diversification strategies are particularly effective for non-persistently transmitted viruses and some semi-persistently transmitted viruses as landing on non-host plants could in addition decrease the proportion of viruliferous vectors that can lost their viruliferous status only after one or few explanatory probing (Hooks and Fereres 2006; Claflin et al. 2015).

Future studies should investigate the design of multifunctional agricultural practices that incorporate several control levers. For example, non-host plants used for intercropping could be selected for their potential to attract beneficial organisms which can help reducing vector populations. Similarly, species used for barrier crop could be selected according to their ability to serve as both a barrier and a reservoir for natural enemies (habitat/resources) (Schoeny et al. 2019) using tall flowering covers for example (Damien et al. 2017).

References

Albajes R, Alomar Ò (1999) Current and potential use of polyphagous predators. In: Albajes R, Lodovica Gullino M, van Lenteren JC, Elad Y (eds) Integrated Pest and Disease Management in Greenhouse Crops. Springer Netherlands, Dordrecht, pp 265–275

Alvarez-Baca JK, Montealegre X, Lann CL, et al (2022) Effect of a cover crop on the aphid incidence is not explained by increased top-down regulation. PeerJ 10:e13299. https://doi.org/10.7717/peerj.13299

Atiri GI, Enobakhare DA, Thottappilly G (1986) The importance of colonizing and non-colonizing aphid vectors in the spread of cowpea aphid-borne mosaic virus in cowpea. Crop Prot 5:406–410. https://doi.org/10.1016/0261-2194(86)90073-6

Bailey SM, Irwin ME, Kampmeier GE, et al (1995) Physical and biological perturbations: Their effect on the movement of apterous *Rhopalosiphum padi* (Homoptera: Aphididae) and localized spread of Barley Yellow Dwarf Virus. Environ Entomol 24:24–33. https://doi.org/10.1093/ee/24.1.24

Bass C, Denholm I, Williamson MS, Nauen R (2015) The global status of insect resistance to neonicotinoid insecticides. Pestic Biochem Physiol 121:78–87. https://doi.org/10.1016/j.pestbp.2015.04.004

Beckage NE, Gelman DB (2004) Wasp parasitoid disruption of host development: Implications for new biologically based strategies for insect control. Annu Rev Entomol 49:299–330. https://doi.org/10.1146/annurev.ento.49.061802.123324

Belliure B, Amorós-Jiménez R, Fereres A, Marcos-García MÁ (2011) Antipredator behaviour of *Myzus persicae* affects transmission efficiency of Broad bean wilt virus 1. Virus Res 159:206–214. https://doi.org/10.1016/j.virusres.2011.04.019

Belliure B, Janssen A, Sabelis MW (2008) Herbivore benefits from vectoring plant virus through reduction of period of vulnerability to predation. Oecologia 156:797–806. https://doi.org/10.1007/s00442-008-1027-9

Bennison J, Maulden K, Barker I, et al (2002) Reducing spread of TSWV on ornamentals by biological control of western flower thrips

Boivin G, Hance T, Brodeur J (2012) Aphid parasitoids in biological control. Can J Plant Sci 92:1–12. https://doi.org/10.4141/cjps2011-045

Bosem Baillod A, Tscharntke T, Clough Y, Batáry P (2017) Landscape-scale interactions of spatial and temporal cropland heterogeneity drive biological control of cereal aphids. J Appl Ecol 54:1804–1813. https://doi.org/10.1111/1365-2664.12910

Bragard C, Caciagli P, Lemaire O, et al (2013) Status and prospects of plant virus control through interference with vector transmission. Annu Rev Phytopathol 51:177–201. https://doi.org/10.1146/annurev-phyto-082712-102346

Budnik K, Laing MD, da Graça JV (1996) Reduction of yield losses in pepper crops caused by potato virus Y in KwaZulu-Natal, South Africa, using plastic mulch and yellow sticky traps. Phytoparasitica 24:119–124. https://doi.org/10.1007/BF02981406

Calvo FJ, Bolckmans K, Belda JE (2012) Biological control-based IPM in sweet pepper greenhouses using *Amblyseius swirskii* (Acari: Phytoseiidae). Biocontrol Sci Technol 22:1398–1416. https://doi.org/10.1080/09583157.2012.731494

Caraco T, Duryea MC, Glavanakov S, et al (2001) Host spatial heterogeneity and the spread of vector-borne infection. Theor Popul Biol 59:185–206. https://doi.org/10.1006/tpbi.2000.1517

Carrière Y, Degain B, Hartfield KA, et al (2014) Assessing transmission of crop diseases by insect vectors in a landscape context. J Econ Entomol 107:1–10. https://doi.org/10.1603/EC13362

Chagnon M, Kreutzweiser D, Mitchell EAD, et al (2015) Risks of large-scale use of systemic insecticides to ecosystem functioning and services. Environ Sci Pollut Res 22:119–134. https://doi.org/10.1007/s11356-014-3277-x

Chan M-S, Jeger MJ (1994) An analytical model of plant virus disease dynamics with roguing and replanting. J Appl Ecol 31:413–427. https://doi.org/10.2307/2404439

Christiansen-Weniger P, Powell G, Hardie J (1998) Plant virus and parasitoid interactions in a shared insect vector/host. Entomol Exp Appl 86:205–213. https://doi.org/10.1046/j.1570-7458.1998.00282.x

Claflin SB, Jones LE, Thaler JS, Power AG (2017) Crop-dominated landscapes have higher vector-borne plant virus prevalence. J Appl Ecol 54:1190–1198. https://doi.org/10.1111/1365-2664.12831

Claflin SB, Thaler JS, Power AG (2015) Predators, host abundance, and host spatial distribution affect the movement of wingless non-colonizing vector *Rhopalosiphum padi* (L.) and PVY prevalence in an oat/potato system. Arthropod-Plant Interact 9:301–309. https://doi.org/10.1007/s11829-015-9370-3

Clark RE, Basu S, Lee BW, Crowder DW (2019) Tri-trophic interactions mediate the spread of a vector-borne plant pathogen. Ecology 100:. https://doi.org/10.1002/ecy.2879

Crowder DW, Li J, Borer ET, et al (2019) Species interactions affect the spread of vector-borne plant pathogens independent of transmission mode. Ecology 100:e02782. https://doi.org/10.1002/ecy.2782

Dáder B, Moreno A, Viñuela E, Fereres A (2012) Spatio-temporal dynamics of viruses are differentially affected by parasitoids depending on the mode of transmission. viruses 4:3069–3089. https://doi.org/10.3390/v4113069

Damien M, Le Lann C, Desneux N, et al (2017) Flowering cover crops in winter increase pest control but not trophic link diversity. Agric Ecosyst Environ 247:418–425. https://doi.org/10.1016/j.agee.2017.07.015

de Oliveira CF, Long EY, Finke DL (2014) A negative effect of a pathogen on its vector? A plant pathogen increases the vulnerability of its vector to attack by natural enemies. Oecologia 174:1169–1177. https://doi.org/10.1007/s00442-013-2854-x

Demler SA, de Zoeten GA, Adam G, Harris KF (1996) Pea Enation Mosaic Enamovirus: properties and aphid transmission. In: Harrison BD, Murant AF (eds) The plant viruses: polyhedral virions and bipartite rna genomes. Springer US, Boston, MA, pp 303–344

Digilio MC, Isidoro N, Tremblay E, Pennacchio F (2000) Host castration by *Aphidius ervi* venom proteins. J Insect Physiol 46:1041–1050. https://doi.org/10.1016/S0022-1910(99)00216-4

Dill LM, Fraser AHG, Roitberg BD (1990) The economics of escape behaviour in the pea aphid, *Acyrthosiphon pisum*. Oecologia 83:473–478. https://doi.org/10.1007/BF00317197

Driesche R van, Hoddle M (2009) Control of pests and weeds by natural enemies: an introduction to biological control. John Wiley & Sons

Eigenbrode SD, Bosque-Pérez NA, Davis TS (2018) Insect-borne plant pathogens and their vectors: ecology, evolution, and complex interactions. Annu Rev Entomol 63:169–191. https://doi.org/10.1146/annurev-ento-020117-043119

Ekbom BS, Wiktelius S, Chiverton PA (1992) Can polyphagous predators control the bird cherry-oat aphid (*Rhopalosiphum padi*) in spring cereals?: A simulation study. Entomol Exp Appl 65:215–223. https://doi.org/10.1111/j.1570-7458.1992.tb00674.x

Fabre F, Dedryver CA, Leterrier JL, Plantegenest M (2003) Aphid abundance on cereals in autumn predicts yield losses caused by Barley yellow dwarf virus. Phytopathology® 93:1217–1222. https://doi.org/10.1094/PHYTO.2003.93.10.1217

Fabre F, Plantegenest M, Mieuzet L, et al (2005) Effects of climate and land use on the occurrence of viruliferous aphids and the epidemiology of barley yellow dwarf disease. Agric Ecosyst Environ 106:49–55. https://doi.org/10.1016/j.agee.2004.07.004

Fereres A, Moreno A (2009) Behavioural aspects influencing plant virus transmission by homopteran insects. Virus Res 141:158–168. https://doi.org/10.1016/j.virusres.2008.10.020

Finke DL (2012) Contrasting the consumptive and non-consumptive cascading effects of natural enemies on vector-borne pathogens. Entomol Exp Appl 144:45–55. https://doi.org/10.1111/j.1570-7458.2012.01258.x

Foster SP, Devine G, Devonshire AL (2007) Insecticide resistance. Aphids Crop Pests 261–285

Gagic V, Kleijn D, Báldi A, et al (2017) Combined effects of agrochemicals and ecosystem services on crop yield across Europe. Ecol Lett 20:1427–1436. https://doi.org/10.1111/ele.12850

Galimberti A, Alyokhin A, Qu H, Rose J (2020) Simulation modelling of potato virus Y spread in relation to initial inoculum and vector activity. J Integr Agric 19:376–388. https://doi.org/10.1016/S2095-3119(19)62656-0

Gallitelli D (2000) The ecology of Cucumber mosaic virus and sustainable agriculture. Virus Res 71:9–21. https://doi.org/10.1016/S0168-1702(00)00184-2

Garzo E I, Duque M, Fereres A (2004). Transmission efficiency of different non-persistent viruses infecting melon by four aphid species. Spanish Journal of Agricultural Research, 2(3), 369-376.

Garzón A, Budia F, Medina P, et al (2015) The effect of *Chrysoperla carnea* (Neuroptera: Chrysopidae) and *Adalia bipunctata* (Coleoptera: Coccinellidae) on the spread of cucumber mosaic virus (CMV) by *Aphis gossypii* (Hemiptera: Aphididae). Bull Entomol Res 105:13–22. https://doi.org/10.1017/S0007485314000534

Garzón A, Budia F, Morales I, et al (2016) Do *Chrysoperla carnea* and *Adalia bipunctata* influence the spread of Cucurbit aphid-borne yellows virus and its vector *Aphis gossypii*? Ann Appl Biol 169:106–115. https://doi.org/10.1111/aab.12284

Godfray HCJ (1994) Parasitoids: behavioral and evolutionary ecology. Princeton University Press

Grauby S, Ferrer A, Tolon V, et al (2022) Can mixed intercropping protect cereals from aphid-borne viruses? An experimental approach. Insects 13:521. https://doi.org/10.3390/insects13060521

Grilli MP, Holt J (2000) Vector feeding period variability in epidemiological models of persistent plant viruses. Ecol Model 126:49–57. https://doi.org/10.1016/S0304-3800(99)00194-5

Hodge S, Hardie J, Powell G (2011) Parasitoids aid dispersal of a nonpersistently transmitted plant virus by disturbing the aphid vector. Agric For Entomol 13:83–88. https://doi.org/10.1111/j.1461-9563.2010.00493.x

Hodge S, Powell G (2008) Complex interactions between a plant pathogen and insect parasitoid via the shared vector-host: consequences for host plant infection. Oecologia 157:387–397. https://doi.org/10.1007/s00442-008-1083-1

Hogenhout SA, Ammar E-D, Whitfield AE, Redinbaugh MG (2008) Insect vector interactions with persistently transmitted viruses. Annu Rev Phytopathol 46:327–359. https://doi.org/10.1146/annurev.phyto.022508.092135

Holt J, Colvin J, Muniyappa V (1999) Identifying control strategies for tomato leaf curl virus disease using an epidemiological model. J Appl Ecol 36:625–633. https://doi.org/10.1046/j.1365-2664.1999.00432.x

Holt J, Jeger MJ, Thresh JM, Otim-Nape GW (1997) An epidemilogical model incorporating vector population dynamics applied to african cassava mosaic virus disease. J Appl Ecol 34:793–806. https://doi.org/10.2307/2404924

Hooks CRR, Fereres A (2006) Protecting crops from non-persistently aphid-transmitted viruses: A review on the use of barrier plants as a management tool. Virus Res 120:1–16. https://doi.org/10.1016/j.virusres.2006.02.006

Ingerslew KS, Finke DL (2020) Non-consumptive effects stabilize herbivore control over multiple generations. PLOS ONE 15:e0241870. https://doi.org/10.1371/journal.pone.0241870

Ingwell LL, Eigenbrode SD, Bosque-Pérez NA (2012) Plant viruses alter insect behavior to enhance their spread. Sci Rep 2:578. https://doi.org/10.1038/srep00578

Irwin ME, Thresh JM (1990) Epidemiology of Barley Yellow Dwarf: a study in ecological complexity. Annu Rev Phytopathol 28:393–424. https://doi.org/10.1146/annurev.py.28.090190.002141

Jactel H, Verheggen F, Thiéry D, et al (2019) Alternatives to neonicotinoids. Environ Int 129:423–429. https://doi.org/10.1016/j.envint.2019.04.045

Jeger M, Chen Z, Cunningham E, et al (2012) Population biology and epidemiology of plant virus epidemics: from tripartite to tritrophic interactions. Eur J Plant Pathol 133:3–23. https://doi.org/10.1007/s10658-011-9913-0

Jeger MJ, Holt J, Van Den Bosch F, Madden LV (2004) Epidemiology of insect-transmitted plant viruses: modelling disease dynamics and control interventions. Physiol Entomol 29:291–304. https://doi.org/10.1111/j.0307-6962.2004.00394.x

Jeger MJ, van den Bosch F, Madden LV, Holt J (1998) A model for analysing plant-virus transmission characteristics and epidemic development. Math Med Biol J IMA 15:1–18. https://doi.org/10.1093/imammb/15.1.1

Jeschke P, Nauen R, Schindler M, Elbert A (2011) Overview of the status and global strategy for neonicotinoids. J Agric Food Chem 59:2897–2908. https://doi.org/10.1021/jf101303g

Jiménez J, Arias-Martín M, Moreno A, et al (2020) Barley yellow dwarf virus can be inoculated during brief intracellular punctures in phloem cells before the sieve element continuous

salivation Phase. Phytopathology® 110:85–93. https://doi.org/10.1094/PHYTO-07-19-0260-FI

Jones RAC (2004) Using epidemiological information to develop effective integrated virus disease management strategies. Virus Res 100:5–30. https://doi.org/10.1016/j.virusres.2003.12.011

Kaya K, Gazel M, Serce CU, et al (2014) Potential vectors of Plum pox virus in the Eastern mediterranean region of Turkey. Entomol Gen 35:137–150. https://doi.org/10.1127/0171-8177/2014/0019

Kho J-W, Kim KH, Lee D-H (2020) Comparing the plant virus spread patterns of non-persistently transmitted virus and persistently transmitted virus using an individual-based model. J Asia-Pac Entomol 23:371–379. https://doi.org/10.1016/j.aspen.2020.01.001

Kirchner S m., Döring T f., Hiltunen L h., et al (2011) Information-theory-based model selection for determining the main vector and period of transmission of Potato virus Y. Ann Appl Biol 159:414–427. https://doi.org/10.1111/j.1744-7348.2011.00501.x

Koureas M, Tsakalof A, Tsatsakis A, Hadjichristodoulou C (2012) Systematic review of biomonitoring studies to determine the association between exposure to organophosphorus and pyrethroid insecticides and human health outcomes. Toxicol Lett 210:155–168. https://doi.org/10.1016/j.toxlet.2011.10.007

Landis DA, Van der Werf W (1997) Early-season predation impacts the establishment of aphids and spread of beet yellows virus in sugar beet. Entomophaga 42:499–516. https://doi.org/10.1007/BF02769810

Landis DA, Wratten SD, Gurr GM (2000) Habitat management to conserve natural enemies of arthropod pests in agriculture. Annu Rev Entomol 45:175–201. https://doi.org/10.1146/annurev.ento.45.1.175

Lee BW, Clark RE, Basu S, Crowder DW (2022) Predators affect a plant virus through density and trait-mediated indirect effects on vectors. Food Webs 33:e00251. https://doi.org/10.1016/j.fooweb.2022.e00251

Li J, Liang X, Wang X, et al (2016) Direct evidence for the semipersistent transmission of Cucurbit chlorotic yellows virus by a whitefly vector. Sci Rep 6:36604. https://doi.org/10.1038/srep36604

Loebenstein G (2008) Plant virus diseases: economic aspects. In: Mahy BWJ, Van Regenmortel MHV (eds) Encyclopedia of Virology (Third Edition). Academic Press, Oxford, pp 197–201

Lommen STE, Middendorp CW, Luijten CA, et al (2008) Natural flightless morphs of the ladybird beetle *Adalia bipunctata* improve biological control of aphids on single plants. Biol Control 47:340–346. https://doi.org/10.1016/j.biocontrol.2008.09.002

Long EY, Finke DL (2015) Predators indirectly reduce the prevalence of an insect-vectored plant pathogen independent of predator diversity. Oecologia 177:1067–1074. https://doi.org/10.1007/s00442-014-3194-1

Losey JE, Denno RF (1998) Positive predator–predator interactions: enhanced predation rates and synergistic suppression of aphid populations. Ecology 79:2143–2152. https://doi.org/10.1890/0012-9658(1998)079[2143:PPPIEP]2.0.CO;2

Madden LV, Jeger MJ, van den Bosch F (2000) A theoretical assessment of the effects of vector-virus transmission mechanism on plant virus disease epidemics. Phytopathology® 90:576–594. https://doi.org/10.1094/PHYTO.2000.90.6.576

Maling T, Diggle AJ, Thackray DJ, et al (2008) An epidemiological model for externally sourced vector-borne viruses applied to bean yellow mosaic virus in lupin crops in a mediterranean-type environment. Phytopathology® 98:1280–1290. https://doi.org/10.1094/PHYTO-98-12-1280

Matsukura K, Watanabe T, Matsumura M (2017) An epidemic model of a rice virus transmitted by a migratory planthopper. J Pest Sci 90:669–682. https://doi.org/10.1007/s10340-016-0811-2

Mauck KE, Smyers E, De Moraes CM, Mescher MC (2015) Virus infection influences host plant interactions with non-vector herbivores and predators. Funct Ecol 29:662–673. https://doi.org/10.1111/1365-2435.12371

Mc Namara L, Gauthier K, Walsh L, et al (2020) Management of yellow dwarf disease in Europe in a post-neonicotinoid agriculture. Pest Manag Sci 76:2276–2285. https://doi.org/10.1002/ps.5835

Mesmin X, Maret M, Vincent M, et al (2021) Biological control at work: demonstrating the complementary effects of natural enemies on two contrasting pests and the damage they cause. J Pest Sci. https://doi.org/10.1007/s10340-021-01426-8

Moiroux J, Chesnais Q, Spicher F, et al (2018) Plant virus infection influences bottom-up regulation of a plant-aphid-parasitoid system. J Pest Sci 91:361–372. https://doi.org/10.1007/s10340-017-0911-7

Moreno-Delafuente A, Garzo E, Moreno A, Fereres A (2013) A plant virus manipulates the behavior of its whitefly vector to enhance its transmission efficiency and spread. PLOS ONE 8:e61543. https://doi.org/10.1371/journal.pone.0061543

Nault LR (1997) Arthropod transmission of plant viruses: a new synthesis. Ann Entomol Soc Am 90:521–541. https://doi.org/10.1093/aesa/90.5.521

Nebreda M, Moreno A, Pérez N, et al (2004) Activity of aphids associated with lettuce and broccoli in Spain and their efficiency as vectors of Lettuce mosaic virus. Virus Res 100:83–88. https://doi.org/10.1016/j.virusres.2003.12.016

Nelson EH, Matthews CE, Rosenheim JA (2004) Predators reduce prey population growth by inducing changes in prey behavior. Ecology 85:1853–1858. https://doi.org/10.1890/03-3109

Nelson EH, Rosenheim JA (2006) Encounters between aphids and their predators: the relative frequencies of disturbance and consumption. Entomol Exp Appl 118:211–219. https://doi.org/10.1111/j.1570-7458.2006.00378.x

Ng JC, Zhou JS (2015) Insect vector–plant virus interactions associated with non-circulative, semi-persistent transmission: current perspectives and future challenges. Curr Opin Virol 15:48–55. https://doi.org/10.1016/j.coviro.2015.07.006

Ng JCK, Falk BW (2006) Virus-vector interactions mediating nonpersistent and semipersistent transmission of plant viruses. Annu Rev Phytopathol 44:183–212. https://doi.org/10.1146/annurev.phyto.44.070505.143325

Ng JCK, Perry KL (2004) Transmission of plant viruses by aphid vectors. Mol Plant Pathol 5:505–511. https://doi.org/10.1111/j.1364-3703.2004.00240.x

Okamoto KW, Amarasekare P (2012) The biological control of disease vectors. J Theor Biol 309:47–57. https://doi.org/10.1016/j.jtbi.2012.05.020

Peckarsky BL, McIntosh AR (1998) Fitness and community consequences of avoiding multiple predators. Oecologia 113:565–576. https://doi.org/10.1007/s004420050410

Pedigo LP, Hutchins SH, Higley LG (1986) Economic injury levels in theory and practice. Annu Rev Entomol 31:341–368. https://doi.org/10.1146/annurev.en.31.010186.002013

Perring TM, Gruenhagen NM, Farrar CA (1999) Management of plant viral diseases through chemical control of insect vectors. Annu Rev Entomol 44:457–481. https://doi.org/10.1146/annurev.ento.44.1.457

Pilkington LJ, Messelink G, van Lenteren JC, Le Mottee K (2010) "Protected Biological Control" – Biological pest management in the greenhouse industry. Biol Control 52:216–220. https://doi.org/10.1016/j.biocontrol.2009.05.022

Pimentel D (2005) Environmental and economic costs of the application of pesticides primarily in the United States. Environ Dev Sustain 7:229–252. https://doi.org/10.1007/s10668-005-7314-2

Power AG (1991) Virus spread and vector dynamics in genetically diverse plant populations. Ecology 72:232–241. https://doi.org/10.2307/1938917

Preisser EL, Orrock JL, Schmitz OJ (2007) Predator hunting mode and habitat domain alter nonconsumptive effects in predator–prey interactions. Ecology 88:2744–2751. https://doi.org/10.1890/07-0260.1

Raccah B, Gal-On A, Eastop VF (1985) The role of flying aphid vectors in the transmission of cucumber mosaic virus and potato virus Y to peppers in Israel. Ann Appl Biol 106:451–460. https://doi.org/10.1111/j.1744-7348.1985.tb03135.x

Raymond L, Ortiz-Martínez SA, Lavandero B (2015) Temporal variability of aphid biological control in contrasting landscape contexts. Biol Control 90:148–156. https://doi.org/10.1016/j.biocontrol.2015.06.011

Reitz SR, Gao Y, Kirk WDJ, et al (2020) Invasion biology, ecology, and management of western flower thrips. Annu Rev Entomol 65:17–37. https://doi.org/10.1146/annurev-ento-011019-024947

Rodrigues SMM, Bueno VHP (2001) Parasitism rates of *Lysiphlebus testaceipes* (Cresson) (Hym.: Aphidiidae) on *Schizaphis graminum* (Rond.) and *Aphis gossypii* Glover (Hem.: Aphididae). Neotrop Entomol 30:625–629. https://doi.org/10.1590/S1519-566X2001000400017

Roitberg BD, Myers JH (1978) Effect of adult coccinellidae on the spread of a plant virus by an aphid. J Appl Ecol 15:775–779. https://doi.org/10.2307/2402774

Roy B, Dubey S, Ghosh A, et al (2021) Simulation of leaf curl disease dynamics in chili for strategic management options. Sci Rep 11:1010. https://doi.org/10.1038/s41598-020-79937-0

Rusch A, Bommarco R, Jonsson M, et al (2013) Flow and stability of natural pest control services depend on complexity and crop rotation at the landscape scale. J Appl Ecol 50:345–354. https://doi.org/10.1111/1365-2664.12055

Schmidt MH, Lauer A, Purtauf T, et al (2003) Relative importance of predators and parasitoids for cereal aphid control. Proc R Soc Lond B Biol Sci 270:1905–1909. https://doi.org/10.1098/rspb.2003.2469

Schoeny A, Lauvernay A, Lambion J, et al (2019) The beauties and the bugs: A scenario for designing flower strips adapted to aphid management in melon crops. Biol Control 136:103986. https://doi.org/10.1016/j.biocontrol.2019.05.005

Sewell GH, Storch RH, Manzer FE, Forsythe HY (1990) The relationship between coccinellids and aphids in the spread of potato leafroll virus in a greenhouse. Am Potato J 67:865–868

Shaw AK, Peace A, Power AG, Bosque-Pérez NA (2017) Vector population growth and condition-dependent movement drive the spread of plant pathogens. Ecology 98:2145–2157. https://doi.org/10.1002/ecy.1907

Sheriff MJ, Peacor SD, Hawlena D, Thaker M (2020) Non-consumptive predator effects on prey population size: A dearth of evidence. J Anim Ecol 89:1302–1316. https://doi.org/10.1111/1365-2656.13213

Shi X, Zhang Z, Zhang C, et al (2021) The molecular mechanism of efficient transmission of plant viruses in variable virus–vector–plant interactions. Hortic Plant J 7:501–508. https://doi.org/10.1016/j.hpj.2021.04.006

Sloggett JJ, Weisser WW (2002) Parasitoids induce production of the dispersal morph of the pea aphid, *Acyrthosiphon pisum*. Oikos 98:323–333. https://doi.org/10.1034/j.1600-0706.2002.980213.x

Smyrnioudis IN, Harrington R, Clark SJ, Katis N (2001) The effect of natural enemies on the spread of barley yellow dwarf virus (BYDV) by *Rhopalosiphum padi* (Hemiptera: Aphididae). Bull Entomol Res 91:301–306. https://doi.org/10.1079/BER2001110

Symondson WOC, Sunderland KD, Greenstone MH (2002) Can generalist predators be effective biocontrol agents? Annu Rev Entomol 47:561–594. https://doi.org/10.1146/annurev.ento.47.091201.145240

Tamai K, Choh Y (2019) Previous exposures to cues from conspecifics and ladybird beetles prime antipredator responses in pea aphids *Acyrthosiphon pisum* (Hemiptera: Aphididae). Appl Entomol Zool 54:277–283. https://doi.org/10.1007/s13355-019-00623-3

Tellez M del M, Simon A, Rodriguez E, Janssen D (2017) Control of Tomato leaf curl New Delhi virus in zucchini using the predatory mite *Amblyseius swirskii*. Biol Control 114:106–113. https://doi.org/10.1016/j.biocontrol.2017.08.008

Thackray DJ, Diggle AJ, Jones R a. C (2009) BYDV PREDICTOR: a simulation model to predict aphid arrival, epidemics of Barley yellow dwarf virus and yield losses in wheat crops in a Mediterranean-type environment. Plant Pathol 58:186–202. https://doi.org/10.1111/j.1365-3059.2008.01950.x

Tholt G, Kis A, Medzihradszky A, et al (2018) Could vectors' fear of predators reduce the spread of plant diseases? Sci Rep 8:8705. https://doi.org/10.1038/s41598-018-27103-y

Van den Bosch F, Jeger MJ (2017) The basic reproduction number of vector-borne plant virus epidemics. Virus Res 241:196–202. https://doi.org/10.1016/j.virusres.2017.06.014

van der Werf W (1995) How do immigration rates affect predator/prey interactions in field crops? Predictions from simple models and an example involving the spread of aphid-borne viruses sugar beet

van Lenteren JC (2000) Success in biological control of arthropods by augmentation of natural enemies. In: Gurr G, Wratten S (eds) Biological Control: Measures of Success. Springer Netherlands, Dordrecht, pp 77–103

Waweru BW, Rukundo P, Kilalo DC, et al (2021) Effect of border crops and intercropping on aphid infestation and the associated viral diseases in hot pepper (*Capsicum* sp.). Crop Prot 145:105623. https://doi.org/10.1016/j.cropro.2021.105623

Weisser WW, Braendle C, Minoretti N (1999) Predator-induced morphological shift in the pea aphid. Proc R Soc Lond B Biol Sci 266:1175–1181. https://doi.org/10.1098/rspb.1999.0760

Xu Q, Hatt S, Lopes T, et al (2017) Antipredator response of aphids to ladybeetles: Effect of intercropping on aphid dispersal. In: Communications in Agricultural and Applied Biological Sciences. Universitéit Gent, Belgium

Zaffaroni M, Rimbaud L, Mailleret L, et al (2021) Modelling interference between vectors of non-persistently transmitted plant viruses to identify effective control strategies. PLoS Comput Biol 17:e1009727. https://doi.org/10.1371/journal.pcbi.1009727

Zhang W, Swinton SM (2009) Incorporating natural enemies in an economic threshold for dynamically optimal pest management. Ecol Model 220:1315–1324. https://doi.org/10.1016/j.ecolmodel.2009.01.027

Zhou JS, Drucker M, Ng JC (2018) Direct and indirect influences of virus–insect vector–plant interactions on non-circulative, semi-persistent virus transmission. Curr Opin Virol 33:129–136. https://doi.org/10.1016/j.coviro.2018.08.004