CHAPTER NINE

Ecological Complexity in Plant Virus Host Range Evolution

Michael J. McLeish, Aurora Fraile, Fernando García-Arenal[1]
Centro de Biotecnología y Genómica de Plantas UPM-INIA, and E.T.S.I. Agrícola, Alimentaria y de Biosistemas, Campus de Montegancedo, Universidad Politécnica de Madrid, Madrid, Spain
[1]Corresponding author: e-mail address: fernando.garciaarenal@upm.es

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Abstract

The host range of a plant virus is the number of species in which it can reproduce. Most studies of plant virus host range evolution have focused on the genetics of host–pathogen interactions. However, the distribution and abundance of plant viruses and their hosts do not always overlap, and these spatial and temporal discontinuities in plant virus–host interactions can result in various ecological processes that shape host range evolution. Recent work shows that the distributions of pathogenic and resistant genotypes, vectors, and other resources supporting transmission vary widely in the environment, producing both expected and unanticipated patterns. The distributions of all of these factors are influenced further by competitive effects, natural enemies, anthropogenic disturbance, the abiotic environment, and herbivory to mention some. We suggest the need for further development of approaches that (i) explicitly consider resource use and the abiotic and biotic factors that affect the strategies by which viruses exploit resources; and (ii) are sensitive across scales. Host range and habitat specificity will largely determine which phyla are most likely to be new hosts, but predicting which host and when it is likely to be infected is enormously challenging because it is unclear how environmental heterogeneity affects the interactions of viruses and hosts.

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1. INTRODUCTION

Host range, defined as the number of host species used by a pathogen, is a simple metric that is central to understanding pathogen epidemiology and pathogenicity. Host range conditions the transmission dynamics and survival of pathogens and is predicted to be a major factor in their evolution. The host range of a pathogen affects how diversification and speciation occur, the incorporation of new species into the number of hosts a pathogen can infect, shifting among hosts in response to biotic and abiotic environmental variation, or the ability to persist in the environment between epidemics (Duffy et al., 2007; García-Arenal and Fraile, 2013; Moury et al., 2017; Parker and Gilbert, 2004; Weaver and Barrett, 2004; Woolhouse and Gowtage-Sequeria, 2005). From an anthropocentric viewpoint, host range determines pathogen reservoirs and inoculum sources, disease emergence and reemergence, population thresholds for disease invasion, and/or critical community size for disease persistence (Elena et al., 2014b; Frank, 1996; Haydon et al., 2002; Lajeunesse and Forbes, 2002; Woolhouse et al., 2001, 2005). Pathogens have been typically classified by host range either as specialists (which in nature can only infect, multiply efficiently, and be transmitted, in hosts from one or a few taxonomically related species) or as generalists or multihost pathogens (which infect, multiply, and are transmitted efficiently in hosts from different species, often from unrelated taxa). However, a pathogen host range is generally unknown in absolute terms and should not be treated as a fixed value in natural systems because ecological factors (broadly the distribution, abundance, and interaction of species) shape the range of host species with which a pathogen comes into contact (Arnold and Lutzoni, 2007). Moreover, this simple metric is difficult to estimate, largely because identifying all "nonhosts" is practically impossible. Changes in host–pathogen associations can lead to a number of evolutionary outcomes (de Vienne et al., 2013). A pathogen can shift, or change between hosts, with the number of hosts, i.e., host range, being unchanged. Alternatively, a pathogen species can expand its host range (host breadth expansion), or contract it (host breadth contraction) in response to ecological or other changes (Janz et al., 2001; Mitchell and Power, 2003; Ng and Perry, 2004; Petanidou et al., 2008; Sexton et al., 2017).

The ecological and evolutionary causes of specialization or generalization have fascinated biologists for decades (Futuyma and Moreno, 1988). Host range is determined by different sets of factors, some extrinsic to the pathogen, related to its ecology and epidemiology, and others intrinsic to

the pathogen, such as genetic traits that determine its fitness in different hosts. Explanations of host range evolution have focused mostly on this second set of intrinsic, genetic factors. Plant viruses are largely generalists (García-Arenal and McDonald, 2003; Power and Flecker, 2003), as is true for many pathogens of humans and other animals (McDonald and Linde, 2002; Woolhouse et al., 2001).

Despite this evidence and the fact that a large host range provides more opportunities for pathogen transmission and survival, it is widely considered that evolution should favor specialization. The argument is that the evolution of host range breadth will be constrained by selection for specialization in host traits (Futuyma and Moreno, 1988): since the host provides the major environmental component of the pathogen, differential host-associated selection would limit the fitness of generalists in any one host, so that in any one host generalists would be outcompeted by specialists (Elena et al., 2009, 2014b; García-Arenal and Fraile, 2013; Kirchner and Roy, 2002; Woolhouse et al., 2001). This hypothesis is based on the concept of trade-offs in adaptation to different hosts, so that an increase of pathogen fitness in one host would result in a fitness decrease in others. As across-host fitness trade-offs will constrain host range expansion (Asplen et al., 2012), which is relevant for disease emergence or for developing sustainable disease control strategies, the analysis of across-host trade-offs has received much attention (Elena et al., 2014b; García-Arenal and Fraile, 2013). However, both theory and empirical evidence of across-host trade-offs derive from analyses of single-host–single-pathogen interactions in homogeneous environments. There is only limited empirical or experimental evidence about how environment modulates host range in plant viruses, and a need for studies that consider, jointly with genetic factors, the ecology of host range evolution.

Here we review what is known about the genetics of plant virus host range evolution, and how it may be modulated by environmental interactions, and then examine the ecology of host range evolution when increasing levels of biological complexity are considered. We also identify analytical and conceptual developments that specifically address some of the challenges ecology and plant virology face.

2. GENETICS OF HOST RANGE EVOLUTION IN PLANT VIRUSES

The search for intrinsic determinants of virus host ranges has led to analyses aimed at identifying virus traits that correlate with host range.

For example, for viruses infecting mammals, feature selection techniques have been used to identify sequences in viral genomes that are associated with the capacity to infect particular host species (Aguas and Ferguson, 2013). Variability in these alleles was then used to predict adaptation to new hosts. Similarly, locus-by-locus analyses of molecular variance have allowed the identification of the genes in potato virus Y (PVY) involved in a host jump (Vassilakos et al., 2016). The relationship between host range breadth and several viral traits (including the type of nucleic acid of the genome, the nonsegmented or segmented structure of the genome, and the nature of horizontal and vertical transmission) was analyzed for a set of 480 plant viruses with host ranges reported in the VIDE data base (Brunt et al., 1996). It was found that viruses with single-stranded genomes (either RNA or DNA), three-segmented genomes, and nematode transmitted had broader host ranges (Moury et al., 2017). It was also found that seed transmission was associated with host range breadth, but the nature of the association varied with the genome. A third important finding was that the widest host ranges were largely constrained to the taxonomic level of family in plant viruses (Moury et al., 2017). Specificity of infection is a major trait in host–pathogen interactions, and genetic studies of host range evolution have mostly focused on the coevolution of hosts and viruses, as developed in the rest of this section.

2.1 Genetic Models of Host Range Evolution

A major trait of host–pathogen interactions determining host range is genetic specificity, i.e., only a subset of pathogens can infect and multiply in a certain host, and for a particular host–pathogen interaction often only a subset of pathogen genotypes can infect and multiply in each host genotype (Agrawal and Lively, 2002, 2003; Antonovics et al., 2013; Zhan et al., 2014). The outcome (infection vs noninfection) of the host–pathogen interaction can be integrated into coevolutionary models that consider infection/noninfection as the result of the interaction between resistance and infectivity loci in the host and pathogen, respectively. Coevolutionary models differ in the underlying infection matrices (Dybdahl et al., 2014) and derive from two general ones—the gene-for-gene (GFG) and the matching-alleles (MA) models—initially proposed to explain plant–pathogen and invertebrate–pathogen interactions, respectively (Agrawal and Lively, 2002, 2003).

In the GFG model, there is a hierarchy of alleles determining resistance in the host or infectivity in the pathogen: some resistance alleles are intrinsically

more effective than others, conferring resistance to a larger set of pathogen genotypes, and similarly, some infectivity alleles in the pathogen are intrinsically better than others, allowing infection of a larger set of host genotypes. In contrast, no such hierarchy of resistance/infectivity alleles occurs in MA systems. Instead a particular resistance allele is better at resisting a subset of pathogen genotypes and less effective at resisting the rest, and a pathogen infectivity allele is better at infecting a subset of host genotypes and worse on the rest. GFG and MA models correspond to different mechanisms of host–pathogen interaction: in the GFG model, infection occurs when the host genotype does not recognize the pathogen genotype, while in the MA model successful infection requires molecular matches between host and pathogen (Dybdahl et al., 2014). The above assumptions condition the evolution of resistance and infectivity loci. Notably, the GFG model predicts that pathogens will evolve to infect all host genotypes (universal infectivity), while this is not possible under the MA model (Agrawal and Lively, 2002, 2003; Frank, 1996).

Originally these models were developed to explain host genotype × pathogen genotype interactions, and so have been applied to the study of plant–virus interactions and plant virus host range evolution (reviewed in Fraile and García-Arenal, 2010; García-Arenal and Fraile, 2013). However, they can also be related to the evolution of host range at the interspecies level, a GFG-like system allowing host range expansion, and thus the evolution of generalists, and an MA-like determining host shifts and thus the evolution of specialists. Thus, the GFG and MA models have been used to interpret host range evolution in bacteriophages. The study of the interaction among large sets of bacteria and bacteriophage species suggests that the evolution of generalism would be due to a GFG-like interaction and results in a nested structure of infection networks, while the evolution of specialism will be driven by MA-like interactions and results in a modular structure of infection networks (Flores et al., 2011; Weitz et al., 2013). A similar analysis of infection network structure has been recently published based on a 37 virus × 28 plant species infectivity matrix. The matrix was significantly nested, but also included significant modules that corresponded to viruses infecting three particular plant families, indicating two groups of viruses, one built of generalists and the other of specialists on different host families (Moury et al., 2017).

Another important issue in which the predictions of GFG and MA models differ is that fitness penalties (costs) associated with host resistance and viral infectivity are required to maintain polymorphisms at resistance

and infectivity loci in the host or pathogen population, respectively, if the interaction is according to the GFG model, but not if it is according to the MA one (Agrawal and Lively, 2002, 2003; Frank, 1996). Costs of infectivity will hinder host range expansions in GFG systems, while adaptation to a new host in an MA system will result in a host shift. It should be noted that pure GFG or MA systems may not exist in nature, and that the assumptions of MA models may be relaxed to allow for partial resistance/infectivity incorporating partial costs of resistance/infectivity (Agrawal and Lively, 2003). A good example of such intermediate systems in plant–virus interactions is that of PVY and recessive resistance in solanaceous hosts (Moury et al., 2014). In any case, the mechanistic assumptions of the MA model, and the costs of extended infectivity in the GFG model both result in the pathogen fitness varying across all its potential hosts. These fitness differences lead to the concept of adaptive trade-offs among hosts: because fitness is environment dependent and the host is a major component of a pathogen's environment, a pathogen cannot simultaneously maximize its fitness in all its potential hosts. Rather, it will become adapted to one or a few related hosts in which fitness will be maximal, i.e., it will evolve to specialization. If adaptation to a particular host implies a fitness cost in other hosts, an adaptive trade-off among hosts will be generated. As a corollary, a generalist would evolve to maximize its fitness across its hosts, but in each individual host its fitness would be lower than the corresponding maxima. Evolution would thus favor specialization rather than generalization.

2.2 Evidence for Across-Host Fitness Trade-Offs

Translating these general concepts about host range evolution to viruses, the hypothesis that generalism incurs a cost is supported by the observation that many plant and animal virus emergences result when a virus jumps from original (reservoir) hosts to a taxonomically related host (Elena et al., 2014b; Longdon et al., 2014). It is further supported by evidence that generalist plant viruses do not infect all hosts with equal efficiency. An analysis of the prevalence of 5 generalist viruses in 21 wild plant species showed significant host–virus associations. The more prevalent viruses were the more host selective (lack of homogeneity of the prevalence of a pathogen among hosts), indicating host selectivity (i.e., host specialization) as a successful strategy in generalist viruses (Malpica et al., 2006). Host selectivity by barley yellow dwarf viruses (BYDV) across different grass species was likewise reported (Power et al., 2011). Together, these results suggest that the multiplication

and transmission of generalist viruses vary across hosts with possible adaptive trade-offs, as experimentally demonstrated in some cases (Betancourt et al., 2011; Power et al., 2011; Sacristán et al., 2005).

The nature of these trade-offs and how they affect transmission across host species is an important research area. The plant–virus literature abounds with experimental evidence of fitness penalties associated with the capacity to infect a new host, expressed as a reduced fitness in the primary host. We refer the reader to recent reviews on this subject (Elena et al., 2009, 2014b; García-Arenal and Fraile, 2013; Miyashita et al., 2016). The mechanistic causes of across-host fitness trade-offs have also been extensively analyzed. The simplest cause is antagonistic pleiotropy in which the phenotypic effect of mutations depends on the environment, and mutations that have a positive fitness effect on one given host are deleterious in another one (Whitlock, 1996). Evidence of antagonistic pleiotropy has been reported for several RNA viruses belonging to different taxa, such as PVY, turnip mosaic virus, soybean mosaic virus, hibiscus chlorotic ringspot virus, pepper mild mottle virus (PMMoV), tomato mosaic virus, or pelargonium flower break virus (e.g., Ayme et al., 2006; Fraile et al., 2011; Ishibashi et al., 2012; Janzac et al., 2010; Jenner et al., 2002; Liang et al., 2002; Montarry et al., 2012; Moury and Simon, 2011; Poulicard et al., 2010, 2012; Rico et al., 2006; Wang and Hajimorad, 2016). Antagonistic pleiotropy may be particularly relevant in plant viruses as an unavoidable consequence of their small genomes, which are highly compact, and encode multifunctional proteins. This genomic architecture makes it more difficult to improve one function without jeopardizing another. Epistatic interactions among mutations involved in host adaptation are another cause of across-host trade-offs across hosts (e.g., Ashby et al., 2014a), best studied in plant viruses through experimental evolution approaches (Hillung et al., 2015; Lalic and Elena, 2012a,b). For the role of antagonistic pleiotropy, epistatic, and higher order interactions of adaptive mutations in host range evolution, we refer the reader to an excellent recent review (Bedhomme et al., 2015).

Under a trade-off scenario, the evolution of generalism requires high rates of transmission among heterologous hosts (Wilke et al., 2006): if among-host transmission is infrequent, the virus will adapt to the current host; however, if heterologous transmission is frequent, the viral population behaves as if the fitness landscape were constant and equivalent to the average of each host. This prediction has been confirmed by experiments in which RNA viruses such as vesicular stomatitis virus, eastern encephalitis virus, or Venezuelan encephalitis virus were passaged alternating between

different cells, in cell culture, or between different hosts (e.g., Coffey et al., 2008; Cuevas et al., 2003). Also, in a phylogenetic study of 23 bat species (Streicker et al., 2010), it was shown that rabies was transmitted at higher intensities between closely related, rather than distantly related species, and to a lesser extent, between geographically adjacent species. The work suggests that cross-species trade-offs were steeper between distantly related hosts, and is consistent with higher rates of transmission being necessary for adaptation to novel hosts. Thus, to explain the evolution of generalism under assumptions of genetic models, it is necessary to call on ecological parameters, such as transmission rates.

2.3 Environmental Heterogeneity and Across-Host Fitness Trade-Offs

Most evidence of across-host fitness trade-offs derives from experiments in which few virus–host interactions are tested. For instance, a classical experiment is to assay the effects of both a virus genotype that overcomes host resistance [a resistance-breaking (RB) genotype] and its non-RB parent in one resistant and one susceptible genotype of a single-host species. When experiments involve more host species, results are more complex and predictions about host range evolution become more difficult. For example, the assay of 20 single-nucleotide substitution mutants of tobacco etch virus (TEV) on 8 different plant host species showed that the distribution of mutational fitness effects depended on the host, and was more similar to taxonomically related host of the family Solanaceae, which include the natural hosts of this virus (Lalic et al., 2011). Most mutations in TEV genotypes had deleterious effects on the virus fitness in the solanaceous hosts, to which the virus was adapted, but a larger fraction of TEV mutations were classified as beneficial to the virus in hosts from unrelated families, in which the fitness of the wild-type TEV genotype was low. In another study, seven genotypes of PMMoV with coat protein mutations determining RB of different alleles at the *L* resistance locus of pepper were assayed in five susceptible pepper genotypes (Moreno-Pérez et al., 2016). These coat protein RB mutations had pleiotropic effects on within-host virus multiplication, but the sense and magnitude of these fitness effects depended on the specific mutation and on the susceptible host genotype and followed no evident pattern. These two studies underscore the fact that mutations for host adaptation are not associated with trade-offs in all instances of cross-species infection. If trade-offs exist at all, they will depend on the specific mutation and on the specific host. A corollary of this conclusion

is that in heterogeneous host populations, such as those a virus will encounter in nature, the evolution of host range will be difficult to predict on the basis of such trade-offs.

In nature, virus populations are also heterogeneous and host plants are often coinfected by different virus species and genotypes (Malpica et al., 2006)—a fact for which deep sequencing analyses of the virome of individual plants or plant populations are providing compelling evidence (Mascia and Gallitelli, 2016). Coinfection results in virus interactions that span the range between synergism and antagonism, and that may determine the evolution of viral traits such as within-host multiplication, virulence, and host range (Elena et al., 2014a; Tollenaere et al., 2016). Costs associated with host range expansion may be affected by coinfection (Fraile et al., 2011). When the effects of RB mutations in PMMoV CP were assayed in coinfections combining different virus genotypes, it was shown that the sense and magnitude of the pleiotropic effects of these mutations on virus fitness depended on the type (single or multiple) of infection, and on the partner genotype in coinfection (Moreno-Pérez et al., 2016). This quite unexpected result unveiled the modulation of cross-host trade-offs by an environmental factor (multiple or single infection) not considered before.

Most published work on across-host fitness trade-offs considers the effects of host adaptation mutations on virus multiplication within the infected host, i.e., on the reproductive component of viral fitness. However, in order to fully understand the evolution of host range, it is necessary to consider potential costs on other fitness components. This is because the evolution of organisms may be constrained by conflicting trade-offs between different fitness components (Alizon et al., 2008; Goldhill and Turner, 2014; Kochin et al., 2010), and one such trade-off predicted by life history evolution models is that between increased reproduction and extended survival. When these theoretical considerations are applied to viruses, the reproduction–survival trade-off would be established between the intracellular (within-host multiplication) and extracellular (survival outside the host) stages of the virus life cycle (Goldhill and Turner, 2014). Unlike in other organisms, reproduction/survival trade-offs may not actually occur in viruses, as it is unclear what mechanistic basis could exist for it; virus survival in the extrahost environment is related to particle stability, which need not be related mechanistically to within-host multiplication (Goldhill and Turner, 2014). However, a trade-off between survival and reproduction does seem to be a general feature in bacteriophages (de Paepe and Taddei, 2006; Dessau et al., 2012; Keen, 2014), although apparently not so in viruses infecting eukaryotes (Handel et al., 2013, 2014;

McGee et al., 2014; Roche et al., 2014). A mechanistic link between reproduction and survival would perhaps not be infrequent in plant viruses, as in many systems host range mutations occur in the coat protein gene (Fraile and García-Arenal, 2010). Indeed, in PMMoV it has been shown that selection for an increased host range selects for altered particle stability and survival, i.e., for a trait unrelated to the plant–virus interaction (Fraile et al., 2014). The analysis of nine genotypes of PMMoV with host adaptation mutations in the coat protein showed that mutations had pleiotropic effects on particle stability (analyzed as a proxy for survival), whose sense and magnitude depended on the specific mutations, and was not correlated with within-host multiplication or host range breadth (Bera et al., 2017). These results do not support a reproduction–survival trade-off, but indicate, as do other studies discussed in this subsection, that across-host fitness trade-offs may not be general and depend on environmental conditions.

We should stress that experimental evidence of across-host trade-offs that are modulated by the environment is in line with analyses that incorporate ecological factors into genetic models of host–pathogen coevolution. Factors such as asynchrony in host and pathogen life cycles, or spatial structure in host and parasite populations or in disease severity also mitigate fitness costs (Ashby et al., 2014b; Brown and Tellier, 2011; Débarre, 2015; Tellier and Brown, 2011). All of these models predict that environmental heterogeneity may result in the maintenance of polymorphisms for host–pathogen specificity in the absence of fitness costs, and highlight the need to consider ecological factors for understanding host range evolution.

3. ECOLOGICAL EFFECTS ON HOST RANGE EVOLUTION

Most models of disease spread require assumptions about the breadth of a pathogen's host range, which are often inaccurate estimates of a pathogen's potential to move among hosts. Inaccurate estimates of host range will negatively affect how we interpret intrinsic (genetic) interactions between the host and the pathogen (Elena, 2017; Laine and Barrès, 2013; Roossinck and García-Arenal, 2015), discussed in Section 2, and the ability to predict the spread of disease (Dallas et al., 2017; García-Arenal and Fraile, 2013; Moury et al., 2017; Schulze-Lefert and Panstruga, 2011). A major concern motivating plant virologists to study host range evolution is the need to understand the process of viral emergence (Elena et al., 2014b; Woolhouse and Gowtage-Sequeria, 2005), which by definition first requires an encounter between the virus and the new host, then adaptation to the

new host to ensure between-host transmission (usually by insects or other vectors), and finally optimization of between-host transmission. All three steps require nonexclusive molecular and ecological processes. Transmission is arguably the central process determining disease dynamics and the host ranges of viruses will strongly influence how transmission proceeds. In the previous sections of this review, we focused on the more abundant literature on molecular mechanisms that invoke evolutionary processes that are not independent of ecological processes. Critical knowledge gaps in the relationship between a virus and its hosts, and the influence the ecosystem has on this association, have been acknowledged in the field of plant virology (Malmstrom et al., 2011). In this section, the focus is on ecological processes that are implicitly spatial in nature—the area organisms occupy and the interactions within that space. Thus, we use the conceptual frameworks of ecology to describe spatial relationships and then introduce approaches for quantifying the effects of biological complexity on host range evolution. Unfortunately, most empirical examples explicitly examining scale relationships are from outside the plant virology community. Pioneering work in the field of ecology largely related to spatial relationships, which influences the interpretation of pattern process, is revisited in the first subsection. Specific examples from virology in relation to the ecological concepts presented are discussed at the end of this subsection. The relevance of observations made at different scales is then examined in Sections 3.2 and 3.3 in respect to trait variation and the biological complexity inherent in natural host–pathogen systems. We conclude by summarizing ecological interactions in terms of resource specialization and show that the conceptual frameworks used in ecology have widespread applications in plant virology. Although some of the working definitions presented here will continue to be the subject of debate, the essence of much of the early work by ecologists on scaling is still highly pertinent to future plant virology research. These tools will be necessary for establishing accurate biotic and abiotic contexts for developing realistic predictive models of disease spread in natural systems.

3.1 Spatial Scale and Host Range

Each pathogen and host has a limited spatial distribution and abundance. Generally, abiotic environmental variation constrains spatial distributions and ecological interactions at wide spatial scales because of variation among organisms in physiological, phenological, and behavioral traits. Environmental variation, or heterogeneity, is therefore linked to differences in

encounter rates between pathogen and hosts, transmission, and the spread of disease at smaller scales. Observations made at different scales and by different studies may not match because researchers mistakenly treat the processes that produce the patterns they analyze as scale insensitive (Peterson et al., 1998; Ricklefs, 1987; Wiens, 1989). As Levin (1992) put it in reference to localized random disturbance: "[a]s the scale of description is increased beyond the scale of individual disturbances, variability declines, and predictability correspondingly increases." Put another way, the patterns of processes of interest at local scales are averaged over at regional scales, due to a negative variability–area relationship (Peterson et al., 1998). To further compound these discrepancies, there is often limited information about the distribution of pathogens, including plant viruses, in natural environments (Malmstrom et al., 2011; Roossinck and García-Arenal, 2015). Specifically, estimations of the number of hosts a pathogen can infect are prone to sampling bias because the accuracy of this parameter is dependent on the distribution and abundance of hosts (Dallas et al., 2017). Although appearing rather absolute under controlled conditions, host range in natural populations and communities is expected to vary widely (Bascompte, 2010; Bäumler and Fang, 2013; Leventhal et al., 2015; Roossinck, 2015; Woolhouse et al., 2001). This might be especially so when novel encounters occur at high rates in an agricultural ecosystem, or under anthropogenic disturbance, which promote ecological changes such that evolutionary constraints in host range may not explain disease risk (Elena et al., 2014b; Hoberg and Brooks, 2015). Host range is therefore central to quantifying the extent of species interactions, provides information about the potential of a pathogen to spread, and is subject to spatial dependencies. The potential for a pathogen to expand its host range has been examined from either a "phylogenetic relatedness" perspective, where the magnitude of shifts between hosts is measured by the genetic distances between them, or an ecological and environmental point of view, which may be assessed by the degree of niche conservatism (e.g., host range conservatism). Both these approaches rely on considering parameters of the community and environment in which the organism persists.

There is a long tradition of ecologists describing biotic processes on the basis of spatial patterns (Turner, 1989). It is widely recognized that different ecological processes (e.g., predation, herbivory, disturbance) shape species interactions at different scales. It is also the multiplicity of these processes among species that make the prediction of disease spread from spatial patterns alone, unrealistic (Real and McElhany, 1996). Fig. 1 presents a

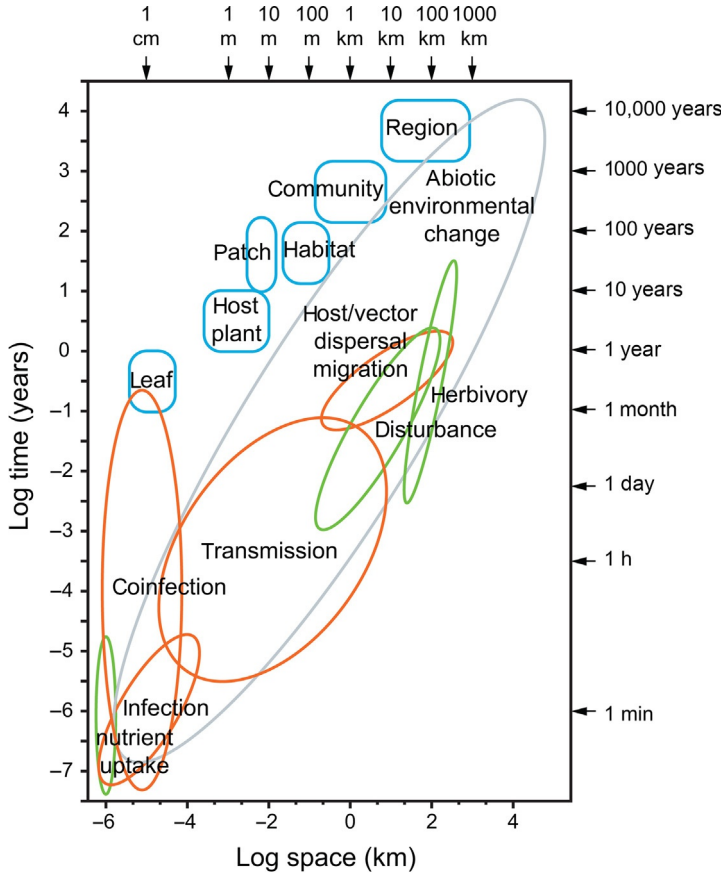

Fig. 1 A hypothetical scheme showing spatial and temporal scale relationships among physical properties and processes of a natural system. The following *color ellipses* represent: *orange*, species interactions; *green*, ecological processes; *blue*, properties of host plants; *gray*, climate and weather variation. *Modified from Peterson, G., Allen, C.R., Holling, C.S., 1998. Ecological resilience, biodiversity, and scale. Ecosystems 1, 6–18.*

hypothetical scheme of processes and the scales they may be applied to in terms of virus infection dynamics. For example, transmission and replication cycles of viruses occur at different spatial scales, and persistence strategies might be distinct between each scale (Handel et al., 2014). Generally, biophysical processes might dominate at small (and fast) scales, while competition for resources might occur at larger and slower scales. Disease pandemics might occur at still larger and even slower scales. At the largest scale, climatic and biogeographic factors are important to variation in and evolution of ecological processes across continents and over geological time. The working

definitions used to define scales of ecological interaction (taken from Leibold et al., 2004) are helpful for consistency among studies. A *patch* is generally describing a discrete area of habitat and used interchangeably with *locality*. A locality comprises a *local* community. A region is a large area of habitat with multiple localities. A set of local communities linked by the movement ecology (how, why, when, and where they move; Nathan et al., 2008) of multiple interacting species are a metacommunity. Although the term "dispersal" is somewhat analogous to the movement ecology of an organism, the latter term is arguable more appropriate because plant virus transmission and seed dispersal often involve multiple species within and between communities. At the metacommunity scale, spatial variation in the distributions and abundances of host species and their viruses reveal whether they interact strongly with one another or not (Seabloom et al., 2015). Natural communities are filled with species that might share a given habitat at a given time and affect evolutionary processes among them (Laine, 2006; Thrall et al., 2002). It is entirely feasible that disease risk and host range evolution proceeds according to the community the population persists in.

Identifying key disease risk factors, or patterns, requires knowledge of the host range and distribution and abundance of species to determine the spatial scales at which processes such as pathogen transmission occurs. A pathogen can occupy multiple sites that are subject to changes in community composition, which determine species interactions, the distribution of host traits, and the possible extent of a pathogen's host range. For example, transmission at an ecosystem scale is subject to connectivity among crops and changes in host density (Margosian et al., 2009). Ecological processes are also dependent on the relationships between species traits and spatial scale. For instance, the size of the environment terrestrial mammals occupy has been explained by body mass (Fisher et al., 2011). So-called habitat selection was likewise species specific in mammalian hosts of rabies, but with the density of conspecifics and plant species composition of each community affecting the occupancy of a habitat by each host differently (Tardy et al., 2014). The latter example shows that spatial variation in species interactions can be determined by community composition. A different type of question is whether ecological processes are connected across scales, as is often assumed, or form discontinuities between them. Cross-scale analysis of habitat structure of reefs and the complexity of fish assemblages demonstrated scale-related discontinuities in fish body size distributions (Nash et al., 2013). To date it is unknown whether transmission is relevant to a particular scale or how it might connect other processes between them. Although these types of

spatial dependencies have been recognized in the field of ecology since the 19th century (Connor and McCoy, 2001), they are only now being incorporated into a conceptual understanding of pathogen systems (Laine, 2006; Perkins et al., 2013; Thrall et al., 2016). By considering a high level of habitat complexity in multispecies systems, we have to assume that the co-occurrence of pathogens and hosts, and the potential for host range evolution, will be subject to scale patterns. The extent to which species richness changes in relation to area is called a species–area relationship and is a central concept in ecology (Connor and McCoy, 2001). Spatial dependencies influence ecological processes (and how they are interpreted) because "you will find more species if you sample a larger area" (Rosenzweig, 1995). When the abundance of a species is a positive function of sampling area, local extinction is more likely in small areas, and rates of colonization are higher in larger areas. None, some, or all of the host and pathogen species might be present in the area being studied. The composition of species in a given area is also subject to change over time. Species interactions at a local scale may not be representative of either their host range or generalizable ecological patterns. Incorporating explicit statements about scale is important to contextualize observations. As the dimensions of ecological interactions are often not self-evident, observations may or may not be consistent with general expectations.

Virus studies conducted at different scales illustrate the context-specific nature of transmission patterns. For instance, transmission patterns of West Nile virus by three avian host species at roughly a 24-km^2 scale varied among sites as a result of differences in how the mosquito vector fed on bird hosts (Hamer et al., 2011); thus, avian and vector traits associated with selective feeding by vectors on avian hosts had strong local effects on infection prevalence. Although this study did not directly address host range effects on variation in transmission, it demonstrates local effects that might alter perceptions of host range evolution. At still larger scales (ca., hundreds of square kilometers, 30-year period), transmission of canine distemper virus was seen first to be limited to exchanges between two host species (domestic dogs to lions), but later data indicated that other species were required to explain transmission patterns (Viana et al., 2015). Although the scale at which transmission occurs between hosts might be evident, the scope of the ecological interactions may not be. Within the plant virus field a most studied system is that of barley and cereal yellow dwarf viruses (B/CYDV) in annual and perennial grasses. In this system, Borer et al. (2010) addressed the effects of spatial scale on resource use by host species

and on infection. The authors were careful to consider the spatial scale at which aphid-vector movement ecology was relevant to virus transmission. A key finding of the study was identifying the spatial scales (100–1000 m^2) at which local community context and infection differences were strongest. These examples highlight the difficulty in estimating the spatial and temporal scales at which species interactions become relevant to the analysis of host range and transmission dynamics, because the scale and nature of the interactions are specific to each study system. Knowing the host range of a virus is difficult to estimate without prior knowledge of the system and the movement ecology of the interacting species. The measurements encompassed at any scale, including those related to host range and transmission, must also be sensitive to an organism's responses to spatial heterogeneity.

Local ecological effects that produce an evolutionary response in a pathogen or host are considered to be features of ecological opportunity (Yoder et al., 2010). Much in the same way ecological opportunity might lead to diversification in herbivorous insects by shifting hosts (Winkler et al., 2009), viruses may also confront varying opportunities to shift hosts due to ecological factors (Parker and Gilbert, 2004; Pedersen and Davies, 2009). Ecological opportunity describes the concept that lineages exposed to a variety of available resources will diversify to utilize a different portion of those resources (Losos, 2010). For example, ecological interactions of humans with wild and domestic animal reservoirs possibly provide pathogens an opportunity to emerge (e.g., Woolhouse and Gaunt, 2007). The resources made available to viruses are a product of the ecological community they belong to because extinction, evolution, and colonization by hosts, vectors, and other viruses are subject to environmental heterogeneity. Local effects (e.g., predators, competitors, temperature, soil nutrients) will cause variation in host–pathogen interactions between local communities. For instance, Malmstrom et al. (2006) showed that clipping (to simulate herbivory) influenced water use of exotic *Bromus* species competing with native grasses. Virus infection reduced survivorship of the native grasses and this effect was intensified under competition. However, clipping ameliorated the resource-depleting effects of competition and survivorship of the native grasses increased. Ecological disturbance that upsets community equilibria (Fraile et al., 2017) also provides opportunity for novel encounters. Generally, when species coexist in the same environments or are closely related, the opportunity to interact is more likely than between spatially and taxonomically distant species (Moury et al., 2017).

Recent studies also show that local adaptation of host species at relatively small spatial scales might be common (Fournier-Level et al., 2011; Hendrick

et al., 2016; Kawecki and Ebert, 2004; Müller et al., 2017). Local adaptation represents a source of heterogeneity in fitness effects on plant viruses and traits affecting vector behavior. For instance, the aggregation of fungal pathogens that cause powdery mildew in an herbaceous perennial has been related to the genetic structure of host populations, providing evidence of selection for resistance at local spatial scales (Laine, 2006). However, in pathogen systems, local adaptation can theoretically arise by several mechanisms (Richardson et al., 2014). These mechanisms ultimately rely on host susceptibility and competence, but will be shaped by habitat preferences of pathogens, hosts, and vectors. A hosts' competence to transmit a virus might depend on within-host mechanisms related to virus and vector multiplication (Parker et al., 2015; Seabloom et al., 2009) and/or on feeding preferences by vectors (Borer et al., 2010). An analysis of the effect of compositional changes among communities (relative surface of crops and unmanaged land) on the prevalence of PVY and its aphid vectors showed that percentage of crop land negatively impacted aphid species richness and positively affected virus prevalence. Effects occurred at a small spatial scale ($\leq$1500 m) (Claflin et al., 2017a), which may be related to aphid dispersal ranges. It is clear that differences in infection patterns exist at the local community scale because species interactions are relatively variable at this scale compared to larger scales. The potential for host range variation may either be favored or inhibited by these ecological factors that have follow-on effects on emergence given specific characteristics of the community to which they belong.

Continental-scale studies demonstrate that heterogeneity in resource availability to hosts differs among communities and might be an important influence in the evolution of a diversity of virulence and resistance mechanisms (Young et al., 2017). For example, variation in ecological disruption to communities that support viruses infecting wild pepper (chiltepin) over its distribution in Mexico corresponded to differences in infection risk (Fraile et al., 2017). Rodelo-Urrego et al. (2013) showed that landscape variation over Mexico affected the genetic structure of begomoviruses infecting chiltepin populations, and suggested that the spatial patterns reflected vector movement ecology. Another large-scale study considered the relationship of pathogen richness with host species occupancy of habitat types, their geographic range sizes, and native-introduced range status in Europe and North America (Mitchell et al., 2010). The study established that accumulation of pathogen species on exotic hosts was slow compared to other ecological processes such as host shifting. Plant species adapted to higher nitrogen availability in their home ranges tend to experience elevated pathogen attack

there and release from pathogens in exotic ranges (Blumenthal et al., 2009). These examples relate how variation in ecological and evolutionary processes that arise due to spatial separation might be most dramatic at larger scales. Differences in how host range evolution may proceed depending on the community are associated with processes propagating from distinct scales.

Process incompatibilities between sites also occur between scales. As the number of biotic interactions increases with the size of a study area, discontinuities among ecological processes that occur at different scales should become stronger. Wiens (1989) referred to these discontinuities as "domains" of scale relevant to particular ecological phenomena. For instance, a study conducted at the global scale on the distributions of three emerging pathogens from different kingdoms (*Batrachochytrium dendrobatidis*, *Borrelia burgdorferi*, and West Nile virus) showed that biotic (the reduction of host species diversity) processes affected ecological interactions at local scales (10^2–10^3 km^2), while abiotic (climate) processes affected ecological interactions at regional scales ($>10^4$ km^2), but the scale differential between processes also depended on the host–pathogen system (Cohen et al., 2016). The ecological processes of niche differentiation (in which competing species utilize the environment differently, e.g., anthropogenic disturbance) and ecological sorting (in which species abundances relate directly to environmental conditions, e.g., climate) can produce different patterns of spatial and temporal species richness similar to the previous example. However, it is unclear to what degree these processes are independent of one another. For example, the processes that result in variation among vertical soil profiles of mycorrhizal fungi communities might involve either deterministic factors affecting population dynamics at the microhabitat scale or stochastic factors generated by climate (Bahram et al., 2015). Together, these studies show that specific processes can be relevant to distinct scale domains, and may also interfere with one another to influence on how species occupy a space. Critically, to make predictions about the state (e.g., infection risk status) of a community, ecosystem, or region, it is important to understand how pattern–process relationships are distributed across scales (de Bello et al., 2013; Holling, 2001; Nash et al., 2014; Stoffels et al., 2005).

In summary, spatial scales are critical to realizing the extent of interactions that pathogens potentially encounter, and provide essential clues about the ways in which processes relate to one another. Dependencies between ecological processes at different scales remain a future challenge to research in ecology in general and are also pertinent to understanding host range evolution (Nash et al., 2013, 2014; O'Dwyer and Cornell, 2017). To achieve accurate predictions of disease spread, it is necessary to identify processes

appropriate to particular domains of scale, and those that connect them. We must also be aware that processes are expected to vary among study systems and sites of comparable scale. Host range evolution by a particular plant virus is therefore likely to depend on molecular exchanges, local-scale species encounters, be influenced by the biotic community and regional effects of biodiversity, and likely to vary widely at regional and continental scales. At the very least, any steps taken to developing spatially explicit hypotheses (spatial pattern) will improve our ability to compare process relating to different study systems, and how processes at different scales relate to one another.

3.2 Effects of Ecological Community on Host Range Evolution

In biological communities it is necessary to identify complex species interactions to understand transmission, infection, and host range evolution. The paucity of studies on biologically complex host–pathogen systems reflects the challenge of accurately quantifying transmission in any multispecies community (Caron et al., 2015; Fenton et al., 2015; Johnson et al., 2015b). Studies of single-host–single-parasite interactions (Agrawal and Lively, 2002; Thrall et al., 2016) often assume homogeneous, randomly mixed populations of hosts and pathogens (Perkins et al., 2013), thus ignoring the effect biological complexity has on host range evolution. The likelihood and intensity of emerging disease outbreaks (Bedhomme et al., 2012; Rottstock et al., 2014; Strauss et al., 2015), the evolution of virulence in plant generalists (Betancourt et al., 2013), causes of widespread and persistent diseases (Johnson et al., 2015a), and pathogen species invasions (Mitchell et al., 2010) all require an understanding of multispecies interactions. For instance, species that dominate transmission (Streicker et al., 2013) and changes to communities that affect reservoir competence (Swei et al., 2011) influence a pathogen's movement among hosts. Community ecology offers plant virologists a way forward in understanding how these processes occur in biologically complex systems and how a virus's relationship with a community influences its host range.

An ecological community has been described as any "association of interacting populations, usually defined by the nature of their interaction, or by the place in which they live" (Ricklefs and Miller, 1999). Due to practicalities, plant virology studies have tended to define relatively few species associations as communities, and assume negligible host range effects. It is important to move beyond these "reduced" types of communities to those that clearly represent a higher biological complexity. Most experimental

studies typically use model species, or those that are relatively easy to maintain either under controlled conditions in the greenhouse or under natural conditions. The most recent syntheses on host–pathogen biology advocate community-level approaches that address much wider biological organization and a higher order of ecological complexity (Alexander, 2015; Johnson et al., 2015a,b; Parker et al., 2015; Seabloom et al., 2015; Suzán et al., 2015; Thrall et al., 2016; Tollenaere et al., 2016; Viana et al., 2014; Young et al., 2017). In particular, knowledge about the ecology of viruses in wild plant and animal communities is critical to understanding the influence host range has on viral diseases in crops and humans (Alexander et al., 2014; Elena et al., 2014a; McLeish et al., 2017; Murray and Daszak, 2013; Roossinck and García-Arenal, 2015; Simpson et al., 2012). Biologically complex wild communities present many unique practical and empirical challenges for which the field of community ecology has developed robust methods to accommodate. Recent developments emphasize the distribution of traits, as opposed to species, in biological systems (Cavender-Bares et al., 2009; McGill et al., 2006). Groupings of species that share a trait or traits may be considered together as one functional group in the analysis of species interactions (Hooper and Vitousek, 1997; Leibold and McPeek, 2006). Species interactions within communities can therefore be quantified at a "higher" level of biological organization. Interactions are described in terms of traits such as a pathogen species specificity for a host-related trait (Gómez et al., 2010; Thrall et al., 2007), host disease resistance and tolerance (Barrett et al., 2009), and pathogen virulence (e.g., Alizon et al., 2013; Kirchner and Roy, 2002). Considering interactions between traits and phylogenetic relatedness of hosts and parasites/pathogens has also proven useful in generalizing about patterns of host range variation and pathogen diversification in communities (Schulze-Lefert and Panstruga, 2011). Parasitic and trophic interactions have also been investigated to understand their impact on transmission (Dáder et al., 2012) and herbivory (Mauck et al., 2015).

Johnson et al. (2015a) provide three motivations for the adoption by disease researchers of community ecology-based approaches: (i) to manage ecological complexity; (ii) to identify drivers of heterogeneities in hosts and pathogens; and (iii) to understand cross-scale processes. These motivations clearly point to translating analogous processes in community ecology to disease systems. Community ecology emphasizes four general processes and interactions among them: ecological drift, selection, dispersal, and speciation, all of which contribute to community dynamics (Vellend, 2010).

Stochastic processes, those with no deterministic effects on species abundances, produce *ecological drift* in community composition and are especially important in smaller communities. Ecological drift changes how interactions within a community occur and the frequency of species moving among habitats within those communities (Orrock and Watling, 2010). Evolutionary change in plant viruses can be linked to selection and speciation processes. Similarly, dispersal can be linked to transmission, as ecological drift can be used as a proxy for community composition changes. Species *coexistence* in communities (indefinite persistence of a defined set of species within a given space) largely depends on *selection*, due to fitness "trade-offs" under different conditions. Trade-offs may occur due to differences in host, pathogen, or vector densities, or in abiotic factors (Handel et al., 2014). *Dispersal* connects habitats and communities, and *speciation* adds species to communities. Each of these four processes connects species traits in the context of an ecological community and can be used to investigate a number of ecological and evolutionary mechanisms. Advocacy of community ecology is appropriate because local-scale processes, such as possible adaptive trade-offs of viruses across hosts, can be linked to those at larger scales such as transmission and emergence (Borer et al., 2013), as stressed in Section 2.2. In any multispecies community identifying connectivity between source and target populations of transmission within and between natural communities is challenging (Alexander et al., 2014). Pathogen reservoirs are expected to range from single-host populations to complex communities of mixed maintenance and nonmaintenance populations (Caron et al., 2015; Haydon et al., 2002; Murray and Daszak, 2013; Viana et al., 2014). Host range may affect the potential of pathogens to persist in species-rich reservoir communities. The movement ecology of vectors and pathogens, and direct and indirect factors affecting transmission, is therefore required to determine how disease spreads within and between reservoir communities and new hosts.

Individual pairs of species may interact directly through processes such as predation, competition, and symbioses. With the introduction of a third species, the fitness dependencies between the first pair of species change. Indirect interactions between them arise because they depend on the relative density of the third one. As such, these ecological processes influence viral transmission within communities via complex pathways. For example, the presence of parasitoids caused a reduction of aphid vectors that coincided with an increase in bean yellow mosaic virus infection due to a decrease in aphid resting times, thereby increasing their movement and thus virus transmission among plants (Hodge et al., 2011). Differences in the timing

of transmission between cucumber mosaic virus (CMV) and cucumber aphid-borne yellows virus depended on the introduction of a parasitoid of their aphid vector (Dáder et al., 2012). The endosymbiotic bacterium *Hamiltonella* that is hosted by the whitefly *Bemisia tabaci*, which vectors tomato yellow leaf curl virus (TYLCV), has been implicated in the recent spread of TYLCV in China (Su et al., 2013). Regardless of the density of whiteflies, transmission frequency of TYLCV was significantly different when the bacterial symbiont was present in the vector. In all the above cases, virus transmission was dependent on the diversity of species and the distribution of their traits in the community.

In a community ecology context, a number of theoretical models have been invoked to explain the coexistence of species (Begon et al., 2002; Seabloom et al., 2015). Two models commonly used to explain disease transmission (Keesing et al., 2006) rely either on host contact rate as a function of population density (a density-dependent model) or on frequency of contact (in frequency-dependent models). The difference is essential that the contact rate is held constant in the frequency-dependent model regardless of the density of the population (Begon et al., 2002). In a two-host community of voles and mice, for example, frequency-dependent models best explained cowpox virus transmission within each species (Begon et al., 1999). However, the study also revealed negligible transmission between the two-host species, and an unexplained increase of infection in one of the host species, which suggested interactions with a third host occurred during the experiment. Gilbert et al. (2001) used a model parameterized with empirical data from wild populations of hosts (grouse, hare, and deer) of louping ill virus and its tick vectors, to predict virus persistence under changing host combinations and densities. The model was conditioned on density-dependent assumptions and showed that the addition of host species was related to increased virus persistence. However, the authors acknowledge that their model assumed that species densities were in equilibrium over time when this was not held up by the empirical data, and that model parameters (i.e., density-dependent constraints) were the same across sites when they were not. The study shows that density-dependent models of transmission are applicable only on a case-by-case basis. The probability of contact between an infectious and susceptible host in both models is assumed to be spatially homogeneous and constant. Other studies on transmission of zoonotic diseases in multihost systems have dispensed with the assumption of these models and instead focus on traits to average over species interactions when host heterogeneity becomes considerable. For example, asymmetrical infections between host species and differential vector and virus

amplification suggest apparent competition between hosts (e.g., Alexander and Holt, 1998; Malmstrom, 1998). Apparent competition occurs when competition between two coexisting species occurs indirectly, where the interaction of a natural enemy mediates their abundances (Holt, 1977). Power and Mitchell (2004) relate this process to spillover and pathogens moving between reservoir and nonreservoir species. Likewise, Malmstrom et al. (2005a,b, 2006) have shown that displacement of native perennial grasses by exotic annual grasses in California grasslands is at least in part mediated by apparent competition due to the effects of infection by B/CYDV: the exotic grasses were more competent hosts and amplified aphid-vector populations, so that virus prevalence in natives was increased by their presence. In turn, infection substantially reduced plant growth, survivorship, and fecundity in natives, whose low-density population structure was more susceptible to disease-induced losses than were the exotics. Apparent competition between native and exotic grasses due to the effect of fungal seed pathogens has also been reported (Mordecai, 2013). These studies explain dynamics in terms of traits and their effects on species abundances, densities, and incidence and speculate about how changes to host diversity might have influenced these observations.

Disease dynamics in species diverse systems have generally related community parameters, such as richness and diversity, to disease prevalence (Johnson et al., 2013). Species diversity is typically described as a function of species richness (the number of species) and often also the species relative abundance (evenness) (Hurlbert, 1971; Mendes et al., 2008). Diversity is therefore a metric of community structure. Biodiversity ("biological variation," Gaston, 2000) has been identified as a key factor that shapes disease risk. Hypotheses propose that biodiversity may either increase or decrease the risk of infection (Keesing et al., 2006; Lacroix et al., 2014a; Ostfeld and Keesing, 2000). However, given the evidence that species distributions are shaped by multiple sources of variation at different scales, it is difficult to predict disease risk from metrics of biodiversity alone (Randolph and Dobson, 2012; Strauss et al., 2015). It seems essential that species identity and/or specific traits be considered as well. For instance, infection prevalence in three species of rodents (gerbils) depended on both the effect of their richness and the specificity traits of the microbial symbionts (Kedem et al., 2014). The work of Borer et al. (2009) illustrates how community parameters can be used in multispecies communities to assess their influence in plant virus infection patterns. Indirect effects of herbivores on the prevalence of B/CYDV in annual invasive grasses were quantified by evaluating virus prevalence in areas with and without a history of grazing by large herbivores.

Herbivory was associated with greater relative abundance of exotic annual grasses, which have greater competence for virus transmission than other resident species. The elevated relative abundance of competent hosts (an altered community composition) in grazed areas increased B/CYDV infection risk. This work underscores the value of assessing community interactions as a function of species traits and host diversity on infection patterns. Virus diversity within individual hosts in natural systems can also affect transmission in communities.

Coinfecting pathogens that share hosts have been defined as within-species communities that also represent another change in the scale of interactions and processes. Within-host processes have consequences for ecological community processes. Host range, variation in vector transmission efficiency, and within-host accumulation of pathogens in coinfected hosts are hypothesized to affect plant virus distributions (Elena et al., 2014a; Mascia and Gallitelli, 2016; Wintermantel et al., 2008). Coinfection communities of the aphid-transmitted viruses *Alfalfa mosaic virus* (AMV), *Beet western yellows virus* (BWYV), *CMV*, and *Watermelon mosaic virus* (WMV), and the thrips-transmitted *Tomato spotted wilt virus* (TSWV) in wild plant species were sampled from within an agroecosystem in Central Spain (Malpica et al., 2006). This study of the prevalence of infection by the 5 virus species in 21 wild plant species found that 18% of infected plants were infected by more than one virus. In another study, coinfections by BYDV and CYDV in a perennial grass species were common; among the 17% of plants found to have infection, 70% were infected by more than one virus (Seabloom et al., 2009). Community ecology approaches offer means of elucidating within-plant–pathogen interactions and the generation of novel genetic combinations and diversity, and host-mediated mechanisms (Tollenaere et al., 2016). Within-host interactions might have a role in viral host range evolution because coinfection has positive and negative influences on transmission (Pedersen and Fenton, 2007). For example, Seabloom et al. (2009) demonstrated spatial and temporal heterogeneity within communities of luteoviruses and poleroviruses. The study suggested that the community composition of vector species with different host ranges, and their relative degree of specialization, influences disease prevalence and affects cross-protection mechanisms. Thus, coinfections represent model systems for understanding small community dynamics (Seabloom et al., 2015), but also for investigating how processes at different scales interact. Vector ecology connects within-host processes with transmission and the structuring of viral reservoirs of vector-borne diseases in biologically complex communities.

Vector and host heterogeneity determine the intensity of interspecific transmission, which underlies mechanisms involved in host range evolution. Recapture experiments with whiteflies indicated bimodal ranges of movement with primarily dispersal flights of less than 500 m, with occasional migration flights of 2.5–10 km (Byrne, 1999). The study provided evidence for the spatial scale at which transmission can occur and how vector behavior might influence pathogen distributions in communities. For instance, the spatial scale of whitefly movement in another study was similar to that of genetic structure of their vectored begomoviruses (Rodelo-Urrego et al., 2013) and suggests the scales of virus and host evolution might be relatively similar. However, the diversity of vector species present in a community will affect transmission because of variation in their feeding behavior and movement ecology. For example, aphid movement increased with the initial density of aphid populations, but their community composition, not richness per se, increased the rate of PVY transmission (Claflin et al., 2017b). Analyses of models parameterized with experimental data on the transmission of BYDV and PVY suggest a positive correlation between population growth rate of aphid vectors and the rate of disease spread (Shaw et al., 2017). Together these studies show that vector movement ecology and diversity might have a strong effect on prevalence and rate of infection at scales between 500 m and 10 km. These examples are evidence that transmission dynamics results in infection heterogeneity at larger scales. This patchwork of virus distributions is embedded in a heterogeneous community of hosts with differences in their susceptibility and competence to transmit infection.

The trait-based perspective of community ecology is insightful because the distribution of traits relating to resource use (e.g., generalism) and host range evolution might be distinct from host species distributions (Barrett et al., 2015; Pilosof et al., 2014; Poisot et al., 2011). Intraspecific variation in virulence or resistance, or phenotypic plasticity in species more generally, is arguable better treated in terms of trait distributions instead of individual species. This is important to realize because host range evolution might be largely modulated by intra- and interspecific variation in traits associated with host resource use, not purely the number of hosts (abundance) or species (richness) that a community comprises. Therefore, virus host jumps might be both a function of phylogenetic relatedness among hosts (Alexander, 2015; Parker et al., 2015) and convergence on host traits that have evolved independently in multiple lineages (e.g., Tatineni et al., 2011; Vassilakos et al., 2016). Cronin et al. (2010) tested the hypothesis that reservoir potential was related to

host physiological phenotype by assaying six grass species for susceptibility to BYDV infection, competence for transmission, and capacity to sustain vector populations. Short-lived hosts, with nutrient-rich and high metabolism tissues, ranged higher for these epidemiological traits, while phylogenetic relatedness or geographic provenance of the hosts had no explanatory effects. Subsequent model-based analyses determined that differences in resistance were important in explaining the higher reservoir potential of hosts with faster developmental tempo (Cronin et al., 2014). The relationship between the developmental traits of hosts and reservoir potential has also been addressed in an experimental study of the interaction of CMV with short- and long-lived genotypes of *Arabidopsis thaliana* (Hily et al., 2014). Results showed that host life span was not related to host competence in transmitting the virus, rather the short-lived host was more susceptible to infection than the long-lived host, and better able to sustain vector populations. The lower defenses of the short-lived host resulted in higher reservoir potential for infection, but this was balanced by the longer infectious period of the long-lived host. The balance depended on the density and genetic composition of the host population. The relationship between a host's life span and its reservoir potential illustrates how host traits might better predict transmission potential than might pathogen host ranges. Knowledge of trait distributions is thus a useful "shortcut" for summarizing complex biological interactions.

3.3 Habitat-Specific Host Range: Specialization of Generalist Viruses

Host range is a virus trait that only partly defines its specificity in host use, in addition to the qualities of the host that have been discussed earlier. In host–pathogen systems, the definitions of specialist and generalist are often given in terms of the number of hosts a pathogen uses to fulfill the needs of reproduction. There has been much interest in these forms of resource use across the whole of evolution and ecology (Agrawal and Lively, 2002; Ehrlich and Raven, 1964; Futuyma and Moreno, 1988; Hubbell, 2001; Hutchinson, 1957; Sexton et al., 2017). Hence, the conceptual framework goes much deeper than describing the host range of a pathogen or vector and rather emphasizes factors that determine resource use. Typically, the host range of a pathogen is enumerated as the number of hosts, rather than that of host-related resources. As such, distinctions between the host itself, the host's traits, and the host's environment tend to be ignored. For example, B/CYDV distributions in six grass species across a 700-km latitudinal gradient varied with host species identity, soil nutrient availability, and vector

host species preferences (Seabloom et al., 2013). The differences in environmental conditions, hosts, or vectors associated with resource availability at each site explained variation in the virus community compositions. Distinguishing such traits that are associated with the breadth of resource use is tantamount to understanding the process of adaptation to a subset of possible environments: i.e., ecological specialization (Poisot et al., 2011). Similarly, niche theory (Hutchinson, 1957) explains the relationship between the resource breadth of a species (an *n*-dimensional hypervolume with axes representing environmental variables) and its resource exploitation strategy. A narrow range of resources used by a consumer reflects a small niche breadth (Ackermann and Doebeli, 2004). In other words, specialists are expected to exhibit highest fitness variation across habitats due to relatively specific requirements, whereas generalists do not. Host range has often been used analogously with niche breadth (e.g., Fellous et al., 2014) when examining variation in performance among available hosts. A major step in the development of niche theory has been the introduction of the "realized niche" concept, which refers to the subset of the available resources that a species actually uses, or the fundamental niche. The fundamental niche emphasizes genetic potentials in the face of environmental variation (Futuyma and Moreno, 1988; Soberón and Peterson, 2005). For a pathogen, the fundamental niche may include resources such as susceptible host tissue and the host genotype (Al-Naimi et al., 2005). The niche may also include ecological interactions and result in changes to the expression of those genes (MacArthur, 1970). Competition, predation, mutualism, herbivory, and parasitism affecting any phase of host range evolution (Coyte et al., 2015; Gilbert et al., 2001; Hily et al., 2016; Malmstrom et al., 2006; Orrock and Watling, 2010; Roossinck, 2015; Thrall et al., 2007) restrict the number of dimensions available in the hypervolume of an individual's niche. Thus, as the number of resources available for specialization (dimensionality of the niche) increases, the types of traits expected to be important to host range evolution at either local- or wider-scale processes (competition and local extinction vs colonization and patch connectivity) are expected to be different (Kneitel and Chase, 2004).

Pathogen host range restrictions have generally been assessed either at the species or population level, as a consequence of trade-offs in the performance and adaptation of pathogens to certain host genotypes (see Section 2.2). As pointed out earlier, fitness trade-offs are questionable and might not be a strong determinant in plant virus host range evolution (Egas et al., 2004; Elena, 2017; Lambrechts et al., 2006) because host genotypes that can be infected by a specific pathogen genotype vary between environments

(Wolinska and King, 2009). Preferences for particular hosts that result from differential costs between them (i.e., gene by gene) or their environments (i.e., gene by environment) might give rise to trade-offs that are hypothesized to result in specialization on a host (Agrawal, 2000) or habitat (Kassen, 2002). Broad host ranges are possibly maintained by selection across heterogeneous environments, while narrow host ranges are hypothesized to evolve in environmentally homogeneous and stable habitats (Kassen, 2002). Theoretically, species interactions are shaped by differentiation of the niches they are associated with, in response to spatial patchiness of suitable habitat. For instance, Seabloom et al. (2010), studying four different B/CYDV species, related within-host virus diversity to coinfection rates across a latitudinal gradient, with differences in the strength of niche differentiation between communities. The study showed that the ability of the four viruses to coexist was largely determined by their traits associated with niche assembly—where a species trait determines whether it is able to establish in a given locality (see Chase, 2007). The strength of such trait-based mechanisms was conditional on environmental factors including vector ecology. In other studies, it was shown that the prevalence and interactions of BYDV and CYDV in one host species were dependent on availability of limiting resources such as nitrogen and phosphorus (Lacroix et al., 2014b) that are determinants of their realized niche. Together, the distributions and abundances of traits, along with other processes such as intra- and interspecific competitive interactions, determine habitat quality for a pathogen. To explain the evolution of variation, maintenance, and stability of species niche breadths (e.g., variation in host range), genetic correlations with fitness in different environments must also be considered in terms of the amount of gene flow between them. Variation in habitat quality, due to environmental heterogeneity, makes host range evolution subject to spatial and temporal variation in gene flow, which in turn depends on the movement ecology of pathogens and hosts. For example, patterns of host range breadth among populations of the pathogenic fungus *Colletotrichum cereale* that infects exotic grasses in North America suggested that low levels of gene flow and environmental isolation affected the degree of specialization (Crouch et al., 2008). These studies demonstrate that not all host species of a pathogen are genetically or ecologically equivalent. Host specificity is a trait that is not only a measure of host range, but how closely hosts are genetically related to one another (Poulin and Mouillot, 2003), and so provides information about the lability of host traits influencing the degree of specialization. Therefore, the distinction between the niche breadth and host range of either "generalist" or

"specialist" pathogens is not trivial. Differences in host specificity of plant viruses might be explained by variation in habitat quality and gene flow over their geographic range, which determines the availability of resources at a given space in time.

Differences in the breadth of resource use need not be explained as a function of species traits in a given environment, or by differential restrictions to resources depicted in high-dimensional hypervolumes. If fitness trade-offs are not strong among available host species (Bedhomme et al., 2012; Elena, 2017), the primary forces acting on pathogen–host range evolution might be driven by stochastic fluctuations in community composition, local extinctions and speciation, and the movement ecology of pathogens (e.g., dispersal characteristics of vectors). These processes have been examined under the neutral theory of ecology (Hubbell, 2001), which assumes functional equivalence between species at the community level (Hubbell, 2005). For example, neutral models of community assembly have been used to examine pathogen community composition in the absence of fitness trade-offs (Seabloom et al., 2015) and applied to cases of specialized and generalized resource use (Pandit et al., 2009). These studies suggest that, even in the absence of trade-offs, environmental heterogeneity and habitat connectivity have a part in the evolution of a species resource breadth. For instance, "generalist" and "specialist" plant viruses show particular resource use characteristics (Malpica et al., 2006) that might restrict or promote host range evolution (Woolhouse et al., 2005). In a multihost system, habitat heterogeneity and host range phenotypes of a suite of 11 generalist plant virus species strongly affected the prediction of infection prevalence (McLeish et al., 2017). Infection prevalence only became conditional on host diversity when habitat heterogeneity was considered. Predictive models were conditioned on spatial scale with a negative or positive prevalence–diversity relationship depending on host diversity of a given habitat. In this study, the realized host range estimates for each plant virus further altered the prevalence–diversity relationships. The changes to the realized host ranges of each plant virus across habitats were consistent with definitions of facultative generalism (Shipley et al., 2009). Facultative generalism has been reported in mammalian consumers (e.g., Sokos et al., 2015), but is also consistent with observations of facultative alterations of virulence in *Plasmodium* parasites (Taylor et al., 1998). Theoretically, facultative generalists have the broadest fundamental niches. This resource use strategy differs from obligate generalists essentially because preferences among resources change with time and become deterministic rather than being driven purely by resource encounter frequencies, as occurs in obligate generalists (Glasser, 1982). For example, preferences

among hosts by vector-borne viruses develop in conjunction with their movement ecology. Viral gene products associated with host and vector manipulation alter transmission by changing host traits that affect the attractiveness, settlement, or feeding behavior (Fereres, 2015; Fereres and Moreno, 2009; Groen et al., 2017; Mauck et al., 2016). This type of manipulation has been observed in the homopteran and thrips vectors of viruses such as CMV (Mauck et al., 2010; Ziebell et al., 2011), BYDV and potato leafroll virus (Bosque-Pérez and Eigenbrode, 2011; Fereres et al., 1989), TYLCV (Fang et al., 2013), PVY (Boquel et al., 2012), and TSWV (Stafford et al., 2011). Overall, neutral model predictions highlight the contribution of stochastic factors to pathogen distributions in the environment, which are necessary to understand the evolution of host range. Resource exploitation strategies, like facultative generalism, imply conditional responses to environmental heterogeneity and the existence of niche differences among hosts and pathogens. Exploitation strategies often involve "trade-offs" for limited resources such as host species, and the ability of a pathogen species to exploit more than one resource or evolve to exploit another (Barrett and Heil, 2012) is often necessary to enable their coexistence.

In summary, selection for wider host ranges might involve both direct and indirect mechanisms. To understand the ecology and evolution of host range, it is critical to identify the traits responsible for specialist and generalist interactions involving molecular (Genin, 2010), species (Philibert et al., 2011), or environmental (Hily et al., 2016) factors. If many plant viruses infect more than one host species, then it is valuable to relate host range to the distribution of resources available to the viruses (e.g., as shaped by the distribution of resistance and tolerance traits), the vectors (e.g., diversity of the vector's host plants), and hosts (e.g., nutrients, soil moisture, pollinators) in the environment. Generally, a pathogen's host range or its habitat specificity will determine which phyla are more likely to be new hosts (Clark and Clegg, 2017). Given the ecological complexity of natural communities, establishing the niche dimensions of plant viruses is a useful approach to establishing factors important to host range evolution under specific conditions. The factors that restrict ecological interactions include biotic processes such as competition (e.g., coinfection), predation (e.g., on vectors), and herbivory (e.g., on hosts), and abiotic variables influencing environmental heterogeneity. Ecological interactions among species that mediate host range evolution can therefore be described between genes, between genes and the environment, and via deterministic and stochastic processes that govern changes to community composition.

4. FUTURE DIRECTIONS

Biological complexity in plant–virus interactions in natural environments inevitably produces analytical challenges such as multivariate data sets with dissimilar probability distributions (Warton et al., 2015), taxonomic anomalies (Blüthgen et al., 2008; Novotny et al., 2002), and spatial or temporal scale dependencies (Nash et al., 2014; O'Dwyer and Cornell, 2017; Peterson et al., 1998). The ability to generate larger data sets using data-mining or high-throughput approaches has spawned sophisticated methods for combining geographical, phenotypic, and phylogenetic variables to understand the role the environment has in structuring species interactions and communities. For example, the "metacommunity" approach is used to model interactions among local communities (Leibold et al., 2004) linked by dispersal at larger spatial scales (Declerck et al., 2013; Suzán et al., 2015). The conceptual overlap between ecology and epidemiology is complemented by niche modeling approaches that seek to determine the distribution of viruses using their niche requirements (Escobar and Craft, 2016). The availability of comprehensive climate and environmental data, and improved techniques for ecological niche modeling has been increasingly utilized in understanding environmental limitations on virus and host distributions at global (Samy and Peterson, 2016), interregional (Fraile et al., 2017), and regional (Sallam et al., 2016) scales. It therefore will be possible to separate the environmental conditions suited to a host and the viruses that infect it, and these conditions need not overlap because the physiological requirements of the pathogen need not correspond to those of hosts.

Network-based approaches quantify ecological interactions within communities by connecting species based on interdependencies among them and examine the architecture of this connectivity to understand ecological and evolutionary processes (Bascompte, 2010). Connecting hundreds of species in this way has been useful for interpreting the distribution of traits that are embedded in biologically complex interactions (Andreazzi et al., 2017; Lewinsohn et al., 2006). For instance, networks have been used to model microbial competition involved in stabilizing gut communities (Coyte et al., 2015), to infer the probability of transmission (May and Lloyd, 2001; Newman, 2002), to explore disease resistance among hosts (Jousimo et al., 2014), to compare antagonistic and mutualistic interactions (Olesen et al., 2007; Thébault and Fontaine, 2010; Welti and Joern, 2015), and to identify

the causes of the spread of infectious disease in heterogeneous environments (Craft et al., 2011; Leventhal et al., 2015). Network approaches have also been used to identify keystone species that might have important structural or functional roles in a community (Jordano et al., 2003; Macfadyen et al., 2011). For instance, the presence of generalist species with high levels of connectivity in ecological networks implied that generalist species act as "hubs" that potentially stabilize communities (Ings et al., 2009; Solé and Montoya, 2001; Toju et al., 2017). Network analysis has also been used to compare the niche and "neutral" concepts explaining host–parasite interactions (Canard et al., 2014). Niche and neutral theories (Hubbell, 2001) have evolved as competing hypotheses used to explain the structure of natural communities and provide scope for comparing "null" or stochastic expectations against deterministic analyses (Leibold and McPeek, 2006). The most recent developments in the prediction of species distributions integrate quantitative parameters, derived from community ecology approaches, with variables related to regional processes such as climate data (Staniczenko et al., 2017; Vayssier-Taussat et al., 2014). These analytical frameworks have previously been treated separately due to scale incompatibilities. The need for scale-sensitive methods in the analysis of nonlinear dynamical systems is arguably driving the forefront of development in elucidating on distribution and abundance patterns and species interactions (Bjørnstad, 2015; Bokler, 1993; O'Dwyer and Cornell, 2017; Sugihara and May, 1990). Neutral theory (Hubbell, 2001) also represents a suitable candidate for cross-scale spatial modeling (Rosindell and Cornell, 2013). A number of techniques to resolve relationships between discontinuities in processes at different scales have been available for some time (outlined in Purse et al., 2015; Stow et al., 2007).

A substantial step in understanding large- and small-scale ecological processes in disease systems has come with the advent of high-throughput sequencing technologies. Information on the host range of a virus is essential to predicting avenues for transmission, the influence of maintenance species on virus persistence, and the effective implementation of disease control strategies. Bulk sampling approaches for the detection of the virus (or viruses) are becoming increasingly available (Ho and Tzanetakis, 2014; Massart et al., 2017) and can be used to expand the scope of measurements of ecological interactions evaluated in a study (Vayssier-Taussat et al., 2014). Metagenomics approaches are commonly used for the purposes of identification (Massart et al., 2017), detection (Massart et al., 2014), and discovery (Massart et al., 2017) of plant viruses and these can be improved in combination with traditional techniques (Blouin et al., 2016). Metagenomic

approaches supersede the prerequisite of amplifying cDNA/DNA of a target virus (Blawid et al., 2017; Picard et al., 2017; Roossinck et al., 2015). The taxonomic scope, spatial, and temporal scales of investigative studies can be considerably enlarged using an approach that pools samples from identified host plants gathered from larger areas known as "ecogenomics" (Roossinck et al., 2010). Metagenomics has also resulted in considerable development and revision of how virus taxonomy is conducted (Simmonds et al., 2017). (For additional discussion of metagenomics applications with plant viruses, see Claverie et al., 2018.)

In conclusion, the application of spatially and temporally sensitive as well as high-throughput approaches that integrate biological complexity in plant virus systems is still in its infancy. Readily available approaches can be used to understand how the distribution and abundance of hosts, vectors, and plant viruses result in particular ecological interactions at different scales. The ability to rapidly gather data over many host and vector taxa will go a long way to unlocking the influence species interactions, and the environment, have on the evolution of host range in the enormous numbers of known and unknown plant viruses.

We trust that this review of the role of ecological complexity in plant virus host range evolution will convince the reader this recent field of research addresses a topic with an exciting future. Understanding the role of ecological complexity in plant–virus interactions is complementary to earlier studies of the molecular genetics, population genetics, and evolutionary factors that shape virus host ranges. Unlocking scale relationships among these factors will provide scope for future lines of research. The time is ripe for collaboration with plant community ecologists at well-inventoried sites to examine viral dynamics in community assembly at the global scale.

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FURTHER READING

Laine, A.L., 2008. Temperature-mediated patterns of local adaptation in a natural plant–pathogen metapopulation. Ecol. Lett. 11, 327–337.