

Evolution and ecology of plant viruses

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Abstract | The discovery of the first non-cellular infectious agent, later determined to be tobacco mosaic virus, paved the way for the field of virology. In the ensuing decades, research focused on discovering and eliminating viral threats to plant and animal health. However, recent conceptual and methodological revolutions have made it clear that viruses are not merely agents of destruction but essential components of global ecosystems. As plants make up over 80% of the biomass on Earth, plant viruses likely have a larger impact on ecosystem stability and function than viruses of other kingdoms. Besides preventing overgrowth of genetically homogeneous plant populations such as crop plants, some plant viruses might also promote the adaptation of their hosts to changing environments. However, estimates of the extent and frequencies of such mutualistic interactions remain controversial. In this Review, we focus on the origins of plant viruses and the evolution of interactions between these viruses and both their hosts and transmission vectors. We also identify currently unknown aspects of plant virus ecology and evolution that are of practical importance and that should be resolvable in the near future through viral metagenomics.

Arthropods

These invertebrate animals have exoskeletons, segmented bodies and paired jointed appendages. Arthropods belong to the phylum Euarthropoda that includes insects, arachnids, myriapods and crustaceans.

Viral metagenomics (or viromics) studies are shifting the focus from examining individual virus–host–vector systems towards characterizing the diversity and biology of viruses in the context of entire environments. The genetic diversity of viruses dwarfs that of the cellular kingdoms of life^{1–5}, and the present and past ubiquity of viruses suggest that, rather than being entirely pathological agents of destruction, viruses have likely been performing key functions in global ecosystems since the dawn of life³.

All viral contributions to plant ecosystem function must derive from the complex interactions between viruses, plants and transmission vectors that developed during the rise of terrestrial plants, fungi and arthropods over the past 450 million years^{6,7}. However, it is still uncertain for even the best-studied interactions — that is, for viral pathogens of crops — how viruses affect the diversity of plant species in natural environments. Specifically, their host ranges, their prevalence in uncultivated, alternative hosts and the mutualistic–antagonistic interactions with different hosts are almost entirely unknown^{8,9}.

Just as discoveries made with plant viruses laid the foundations of virology and yielded crucial insights into numerous fundamental aspects of cellular and molecular biology (BOX 1), understanding the ecological roles of plant viruses will likely be pivotal in obtaining an accurate and robust understanding of global ecosystems.

In this Review, we address the evolutionary origins of plant viruses in relation to their hosts and transmission

modes, and examine how these viruses adapt to new host species in changing environments. We suggest ways in which their capacity to adapt might foster the stability of ecosystems and even provide direct benefits to host species, and we highlight ongoing challenges of plant virus ecology in a bid to fully comprehend the diversity and ecological roles of plant viruses.

What are plant viruses?

When attempting to understand the polyphyletic origins of plant viruses¹⁰ it is perhaps helpful to realize first that the very concept of ‘plant viruses’ is problematic. Many contemporary plant viruses infect the non-plant organisms that ferry them between plants and most, if not all, purely plant-infecting viruses have likely evolved from viruses that once infected non-plant organisms. The evolutionary history of eukaryotic viruses encompasses three main themes¹¹: co-evolution and co-divergence of viruses with their hosts (for example, picorna-like viruses), evolution facilitated by horizontal gene transfers between viral groups that infect distinct hosts (for example, bunyaviruses) and parallel evolution of viral groups with related genetic elements (for example, circoviruses and geminiviruses). Inter-family gene transfers have been common, with many transfers occurring between viruses that now exclusively infect hosts in different eukaryotic kingdoms^{3,12–15}.

The closest non-plant-infecting relatives of contemporary plant viruses (green nodes in FIG. 1) tend to infect fungi (brown nodes in FIG. 1) or arthropods (pink nodes

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Box 1 | History of plant virology

Although plant virology as a discipline is only ~120 years old, historic literature indicates that humans have been aware of plant virus diseases for over a millennium. A poem written in 752 AD by the Japanese empress Koken describes a striking yellow leaf pattern that was subsequently confirmed to be caused by a geminivirus–satellite disease complex¹⁴⁹. Another, perhaps more famous, early description of plant virus diseases is tulip breaking disease¹⁵⁰. The potyvirus tulip breaking virus causes an elegant swirled colour pattern in tulip flowers, a highly valued commodity during the Dutch ‘tulip mania’ in the seventeenth century, but first described in 1576. Aside from inspiring poetry and instigating a speculative bubble that destroyed the Dutch economy, plant viruses are responsible for major scientific breakthroughs, starting with the science of virology itself. The exact date that the first virus was discovered is difficult to pinpoint, but priority of discovery is generally given to Russian scientist Dimitri Ivanovski, who first documented in 1892 the presence of a filterable, submicroscopic infectious agent in tobacco plants¹⁵¹. Dutch scientist Martinus Beijerinck was the first to use the term ‘contagium vivum fluidum’ or ‘virus’ for the mosaic disease-causing infectious agent in 1898 (REF.¹⁵²). The discovery of this agent, later called tobacco mosaic virus, then led to a series of scientific firsts, including confirmation of the nature and structure of viruses via paracrystallization¹⁵³, electron microscopy¹⁵⁴ and X-ray crystallography¹⁵⁵, and, finally, confirmation of the universality of the genetic code¹⁵⁶. More recently, plant viruses were integral to uncovering the mechanism of RNAi or gene silencing, a discovery which revolutionized genetics in the late 1990s^{157,158}. Initially recognized as a plant antiviral defence strategy, gene silencing or RNAi is today known to be an almost universal mechanism of gene regulation and pathogen defence, functionally analogous across kingdoms¹⁵⁹. Now, as we head for the third decade of the twenty-first century, plant viruses are yet again at the forefront of a conceptual revolution. Driven by advances in metagenomics, the relatively unbiased sampling and sequencing of viral genomes within entire environments is changing the perspective of viruses from agents of disease to vital components of global ecosystems.

RNA-dependent RNA polymerases

These enzymes catalyse the synthesis of RNA from an RNA template. RNA-dependent RNA polymerases are essential to the replication of viruses that have no DNA stage.

Movement proteins

Some plant viruses encode these proteins to facilitate cell-to-cell movement of viral particles and/or uncoated viral nucleic acids. They frequently function by increasing the size exclusion limits of plasmodesmata.

in FIG. 1). In fact, even when discounting virus genera that contain both plant viruses and viruses that infect fungi or arthropods (for example, members of several genera in the *Partitiviridae* and *Reoviridae* families), most viral genera that contain only plant viruses have genes and/or genomic structures that are homologous with arthropod and/or fungal viruses^{16,17}. For example, the RNA-dependent RNA polymerases of secoviruses are most closely related to those of dicistroviruses, iflaviruses, marnaviruses, picornaviruses and unclassified viruses infecting plant-parasitic nematodes and other invertebrates¹⁸.

Viruses of fungi, commonly referred to as mycoviruses, were only discovered in the early 1960s¹⁹, and have remained far less studied than animal and plant viruses²⁰. However, the discovery of new fungal virus genomes through viral metagenomics^{21,22}, through

transcriptome mining^{23,24} or as integrated sequences within fungal genomes^{25,26} has revealed that these viruses share several ancestors with plant viruses¹⁷. For example, plant-infecting ourmiaviruses are apparently evolutionarily related to members of the *Narnaviridae* family, which replicate in the mitochondria²⁷ of fungi and plants²⁸, and whose genomes encode only an RNA-dependent RNA polymerase. Ourmiaviruses potentially originated after an ancestral narnavirus acquired genes that encode movement proteins and capsid proteins through recombination from a presently unknown plant virus²⁹. These additional genes may have enabled ourmiaviruses to escape from mitochondria so that they could systemically infect plants.

The capacity of some fungal viruses to replicate in plant cells³⁰ and the capacity of some plant viruses to replicate in fungal cells (in vitro and in vivo)^{31,32} suggest that a pool of viral groups and/or species exists, possibly some that are presently defined as plant viruses, that in fact infect plants and fungi. The strong association between plant and fungal viruses may be in part due to the complex and evolutionarily ancient symbiotic associations, ranging from facilitating nutrient uptake to stress tolerance, of terrestrial plants with fungi^{33–36}.

Further, the hypothesis that many plant and vertebrate viruses may have originated from arthropod viruses is supported by the astonishing diversity of viruses that has been discovered during arthropod transcriptome studies^{37,38}. Although some viruses that presently infect plants may have been co-evolving with their arthropod vectors since arthropods first evolved, it is also plausible that some viruses that used to infect arthropods more recently switched to infecting plants. For example, flock house virus (in the *Nodaviridae* family) infects arthropods but can also systemically infect plants when it is complemented with the movement proteins of either tobacco mosaic virus or red clover necrotic mosaic virus (both of which are plant viruses)³⁹.

More viral sequence data will undoubtedly help to refine hypotheses about the evolutionary origins of different plant viruses. For example, recent viral metagenomic and metatranscriptomic studies^{37,38} have discovered numerous viral sequences (grey dots with red outlines in FIG. 1) and revealed previously unknown relationships between many viral groups (red edges in FIG. 1). More data will also indicate how common it is that a particular virus species infects hosts in different kingdoms and whether transmission between hosts in different kingdoms is epidemiologically and/or ecologically relevant.

Further, the refinement of techniques to determine exactly when homologous genome fragments of distantly related viruses last shared a common ancestor would help to clarify the deep and frequently complex evolutionary relationships of different virus families. For example, there is strong evidence that many tobamoviruses (other than those that infect brassicas) likely co-evolved and co-diverged with their hosts since angiosperms first arose^{40–42}. Assuming that the most recent common ancestor of all tobamoviruses existed ~130 million years ago, it has been possible to infer the dates for the most recent common ancestors of

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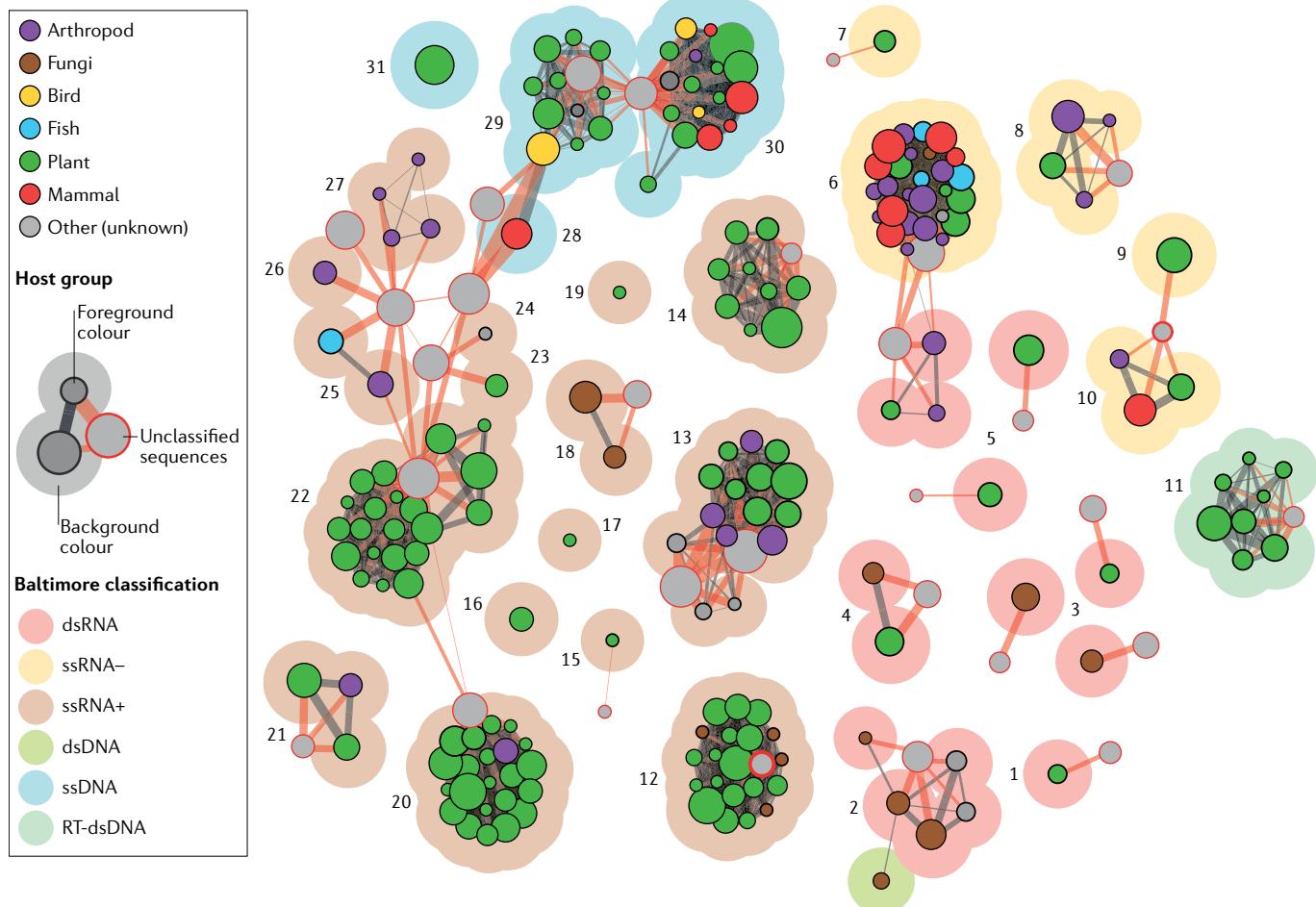


Fig. 1 | The many origins of plant viruses. A network of shared genes between plant viruses and other viruses. In the network, virus genera are represented with nodes of different sizes depending on the number of sequences in the database. The colours of nodes indicate the most frequent host from which sequences were isolated (see key). The background colours represent the Baltimore classification¹⁶⁰ (that is, the type of genetic material of the virus genera). Groups of genera with significant similarities are connected with edges of increasing width depending on the protein similarity. The protein similarity network (minimum E -value of 1×10^{-10} , minimum bit-score of 100 and Markov Cluster Algorithm (MCL) inflation parameter of 2 as described in vConTACT¹⁶¹) is based on all reference sequences described in the virus–host database¹⁶² (accessed on October 2018, dataset detailed online: [Plant viruses dataset](#)). Importantly, all shown genera either contain plant viruses or viruses that express proteins with substantial similarity to those expressed by a plant virus. Whereas some genus clusters contain only plant viruses (for example, the Potyviridae and the Caulimoviridae family clusters), others contain genera that infect hosts from distinct kingdoms. As a general trend, plant viruses cluster with either fungal viruses (brown nodes) or arthropod viruses (purple nodes). Interestingly, the addition to this analysis of the flurry of recently discovered ‘omics viruses (grey dots circled in red^{37,38}) link several clusters that otherwise appear unrelated (see red edges). For clarity, these new sequences are grouped into clusters that display similar patterns of gene sharing, although many (if not most) of these still unclassified sequences represent new genus-level or family-level taxonomic groupings. ds, double-stranded; RT, reverse transcriptase; ss, single-stranded.

homologous genes that encode RNA-dependent RNA polymerase and capsid proteins in tobamovirus and, for example, viruses infecting charophyte algae¹⁴.

Despite the complexity of the evolutionary pathways that have yielded the present diversity of plant viruses, even highly divergent and apparently unrelated plant viruses share features that differentiate them from other viruses. Among these features is the frequent occurrence of multipartite genomes, which are packaged as individual genome components into separate viral particles during transmission. Other viruses with multiple genome components, called segmented viruses, differ from multipartite viruses in that all of their genome components are packaged into a single viral particle. Whereas almost all known multipartite viruses infect plants⁴³, segmented viruses infect both plants and animals (for example, reoviruses and togaviruses) or just animals (for example, orthomyxoviruses).

Moreover, many nominally monopartite plant viruses associate with self-replicating satellite nucleic acid molecules (for example, begomoviruses and nanoviruses associate with members of the *Alphasatellitidae* family⁴⁴) or satellite viruses, which they *trans*-replicate (for example, tobacco necrosis satellite virus) and with which they are frequently co-transmitted⁴⁵. These viruses and their satellites could represent intermediate stages in the evolution of multipartitism. If the satellites eventually were to stop self-replication and/or self-encapsulation and become *trans*-replicated and/or *trans*-encapsulated by the helper virus, they would essentially become components of multipartite genomes.

Why plant viruses in particular show multipartitism remains unknown. Similar to segmented viruses, multipartite viruses can undergo a simple form of genetic recombination called genome-component reassortment. Although reassortment and other forms of genetic recombination can potentially uncouple the fate of genome regions carrying deleterious mutations from that of the remainder of the genome⁴⁶, such processes can also undermine the gene coadaptation within viral genomes⁴⁷.

Another advantage of multipartite or segmented genomes over monopartite or unsegmented genomes is the ability of dynamic changes in the relative copy numbers of the genes encoded by different genome components. This extra layer of gene regulation could, for example, enable multipartite or segmented viruses to respond more rapidly to environmental changes than viruses with monopartite or unsegmented genomes^{48,49}.

Whatever the advantages or constraints imposed by having multiple genome components, they should be valid regardless of whether viruses infect plants, animals, bacteria, archaea or fungi. The main advantage of multipartite genomes over segmented genomes in plants possibly relates to the size of viral particles. By packaging each genome component into a separate particle, multipartite viruses can increase their genome sizes without concomitantly increasing their particle sizes, which may make multipartitism particularly appealing for plant viruses that traffic between cells through plasmodesmata. Indeed, another feature of currently characterized plant viruses is the presence of movement proteins that increase the size exclusion limits of plasmodesmata to

enable the cell-to-cell movement of viral particles and/or uncapsidated viral genomes^{50,51}. Although this hypothesis of multipartitism as a response to plasmodesmata-imposed viral particle size constraints is appealing, supporting evidence is lacking⁵². Therefore, along with the many unresolved evolutionary factors impacting long-distance intra-plant virus movements⁵³, the evolutionary underpinnings of multipartitism in plant viruses remain a mystery.

Transmission and spread

Unlike animal viruses, plant viruses are only rarely transmitted by direct contact between infected and uninfected individuals^{54–56}. Although transmission modes can involve parasitic plants, natural root grafts or contaminated soil or water⁵⁷, the most effective transmission modes are through vectors, pollen or seeds.

Hypothetically, for any given plant virus, the relative effectiveness of these transmission modes should depend on how strongly it harms the host. At one extreme, the survival of virus species that are exclusively transmitted through seed or pollen would demand that they minimally affect the reproductive success of infected plants or that they provide infected plants with some advantage over uninfected plants^{58,59}. At the other extreme, vector-transmitted viruses could be far more antagonistic, needing to only ensure that infected plants do not either die too fast or die in such large numbers that the pool of potential uninfected hosts runs dry.

The relative frequencies across all plant viruses of vertical transmission through seeds or pollen versus horizontal transmission through vectors remain unknown. It is also unknown how common it is for viruses to be transmissible by both vectors and seeds or pollen. Modern plant virology has mainly focused on antagonistic virus–crop interactions, and this has undoubtedly contributed to a general underestimation of the role of vertical transmission in plant virus epidemiology and ecology. Nonetheless, vector-mediated transmission appears to be more common than other transmission modes and is therefore probably the most epidemiologically and ecologically important mode of plant virus transmission⁶⁰.

Whereas plant virus vectors include arachnids, fungi⁶⁰, nematodes and some protists (plasmodesmata)^{60,61}, insects, belonging mostly to the order Hemiptera, transmit more than 70% of known plant viruses⁶². Hemipterans are particularly well suited to transmitting plant viruses because of their needle-like mouthparts that they use for sucking sap and/or the contents of plant cells.

Insects transmit plant viruses in two distinct ways⁶³. The simpler of the two ways is variously called non-persistent, semi-persistent or non-circulative. It involves specific and reversible interaction of viral particles with molecular components of mouthparts, with imbibed sap promoting the binding of virions whereas excreted saliva promotes their release⁶⁴. The second transmission mode is called circulative or persistent and the insect needs to acquire a virus only once for it to be able to transmit the virus during the remainder of its lifespan. In such cases, ingested viral particles traverse the gut epithelia and enter the haemocoel before moving to, and entering, the salivary glands. Sometimes, this circulative

Brassicaceae

Brassica is a genus in the mustard family (Brassicaceae) of plants, which includes cabbage, lettuce and cauliflower.

Angiosperms

Angiosperms are also known as flowering plants and are the most diverse group of land plants. While both gymnosperms and angiosperms produce seeds, angiosperms are characterized by the presence of flowers, an endosperm within the seeds and the inclusion of seeds within fruits.

Plasmodesmata

These microscopic channels traverse plant cell walls enabling intercellular trafficking of macromolecules.

Plasmodesmata

This class of plant parasites comprises organisms in the orders Plasmodesmata and Phagomixida. They have long been recognized as a basal group to fungi, but recent molecular phylogenetic analysis suggests that they are more closely related to protozoa in the phylum Ciliophora.

Hemiptera

This order of insects includes insects such as aphids, cicadas, leafhoppers and planthoppers. Most hemipterans feed on plant sap with their sucking and piercing mouthparts.

Haemocoel

This is the body cavity in arthropods wherein haemolymph (plasma with haemocytes) circulates.

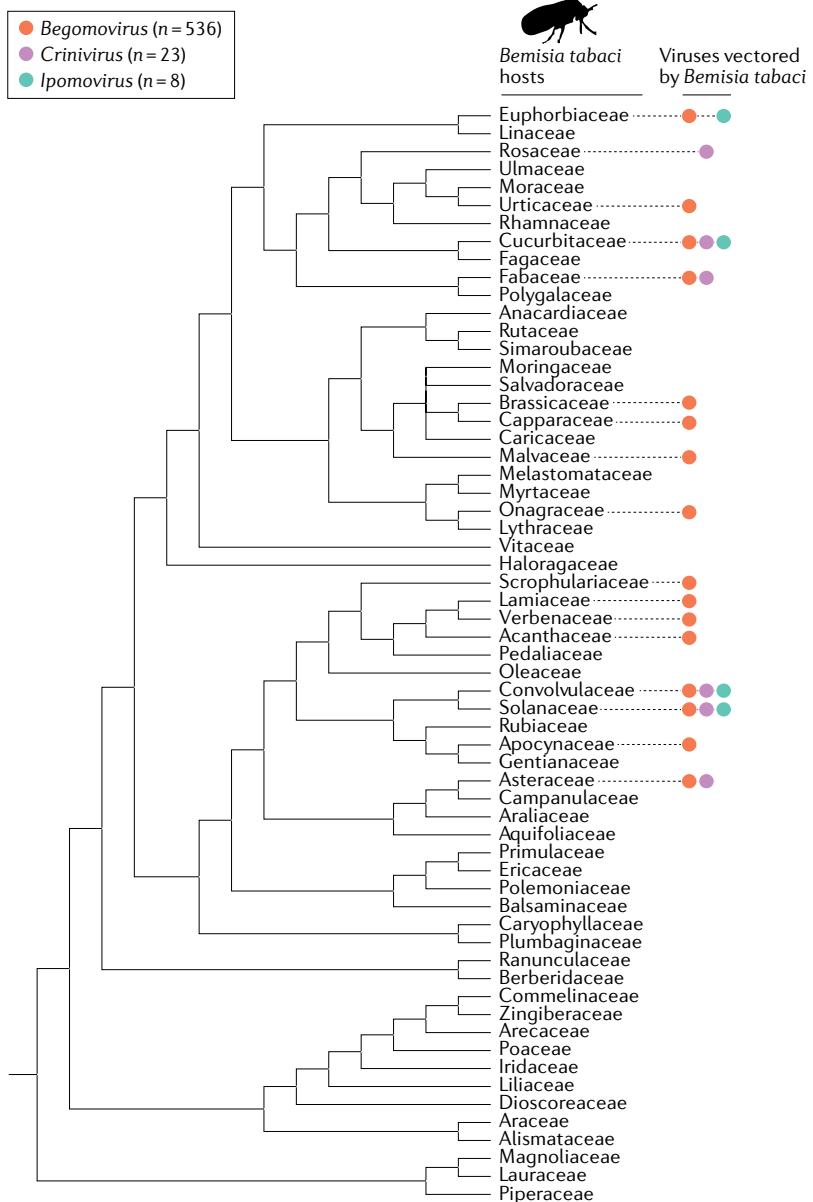


Fig. 2 | Overlap between the plant hosts of a whitefly vector species and transmitted viruses. The taxonomic tree of the plant families that the whitefly *Bemisia tabaci*¹⁶³ feeds on and which of these families contains species that are infected with viruses in the genera *Begomovirus*, *Crinivirus* and *Ipomovirus* that this insect transmits (as defined in the virus–host database¹⁶²). The number of unique plant species–virus species pairs is indicated in brackets for each virus genus. Notably, this figure represents only the known (and therefore the minimal) host ranges of *B. tabaci* and the viruses in the three transmitted genera. Despite this bias, it is evident that the known natural virus host ranges (that is, plants from which viruses have been sampled in ‘the wild’) span much of the *B. tabaci* range. Also striking is that a single vector transmits such a large number of distinct virus species¹⁶⁴. However, it is unlikely (although certainly not impossible) that an individual begomovirus, crinivirus or ipomovirus has a natural host range that includes all of the plant species in all of these families. New plant virus species are now being discovered at a rate that surpasses our capacity to biologically characterize them. As a consequence, a decreasing proportion of described plant viruses have either identified vector species or known host ranges. Metagenomic plant virus studies that focus on unbiased sampling of insects and plants within ecosystems should, in the near future, be powerful enough to reveal the host ranges and vector species of many poorly characterized virus species.

process can also involve interactions of virus particles with insect endosymbionts, or the uncoating and replication of viruses in insect cells, in which case the viruses might accurately be defined as being both insect and plant viruses^{65,66}.

Many insect-transmitted plant viruses have likely also evolved mechanisms to manipulate vector behaviour. They either make infected plants more attractive to sap-feeding insects than uninfected plants and/or ensure that infected plants produce chemicals that, upon ingestion by insects, promote insect behaviours that are most conducive to transmission⁶⁷. In fact, several lines of evidence indicate that viruses that are non-persistently transmitted might manipulate host plants in ways that encourage vectors to feed for shorter periods, whereas viruses that are persistently transmitted might manipulate plants to encourage vectors to feed for longer periods⁶³. Further, as is exemplified by pepper cryptic virus 1 (a deltapartitivirus), some plant viruses that are not transmitted by insects may manipulate their hosts to discourage potentially destructive insects such as aphids from feeding on them, presumably to promote host survival⁶⁸.

The complexity of such interactions suggests long-term co-evolution of plant viruses with specific combinations of vectors and host species⁶⁹. In contrast to viruses that are non-persistently transmitted by arthropods and which commonly have relaxed vector associations (for example, cucumber mosaic virus⁷⁰), most of the known plant viruses that are persistently transmitted by arthropods are transmitted effectively by only one or a small number of closely related insect species, with the host ranges of these viruses generally corresponding to the host ranges of their vector species⁷¹.

Plant viruses commonly have a host range that includes several species from one or more different plant families; however, they rarely are transmitted by more than a few very closely related insect species⁷¹ (an example of begomoviruses, criniviruses and ipomoviruses, all transmitted by the hemipteran *Bemisia tabaci*, is provided in FIG. 2).

Given the strong vector restriction of many persistently transmitted viruses, it is likely that adaptation to being transmitted by a new vector species may be a more difficult evolutionary challenge than adaptation to infecting a new plant host. Consistent with this hypothesis is the finding that the capsid protein phylogenies of some plant virus families, such as the geminiviruses, mirror those of their vector species far more closely than those of their host species (FIG. 3). In fact, strict vector specificity and the congruence of vector and plant virus host ranges imply that the primary evolutionary imperative of most plant viruses that are persistently transmitted by one or a small number of vectors is not maintenance of optimal virus–host interactions but maintenance of optimal virus–vector interactions. The roles of viral capsids as structural proteins and as mediators of specific interactions between plant viruses and their vectors may impose strong constraints on the evolution of these proteins. These constraints may severely reduce the probability of viruses evolving to be persistently transmitted by new vectors.

Although the inherent genetic plasticity of viruses may predispose them to infect new host species and to eventually emerge as pathogens, it is likely that many emergence events have been facilitated by the existence of arthropod vectors with very broad host ranges (that is, polyphagous vectors)^{72–74}. Accordingly, several emerging crop diseases are attributed to plant viruses that are vectored by invasive polyphagous arthropods (for example, begomoviruses; FIG. 3). During their invasive spread, these vectors provide the plant viruses that they transmit with an expanded menu of potential hosts, some of which may be important crop species. Such new host encounters are a vital first step in expansion of the virus host range and the emergence process.

Host-range evolution

Many viruses rapidly adapt to changes in host genotypes⁷⁵ after new host encounters, which, in some cases, results in epidemics. Within a host, viruses evolve towards optimal virus–host interactions that enable efficient viral replication and movement inside the host. To maximize the overall fitness of a virus, evolution must constantly tweak the efficiencies of viral replication, movement inside the host and transmission inside the heterogeneous host populations in which the virus circulates.

When confronted with complex environments containing multiple host plant species or genotypes, plant viruses can adopt a continuum of evolutionary strategies ranging from generalization to specialization. On the one hand, a virus species may undergo adaptive radiation, which enables species to adapt to heterogeneous habitats. Adaptive radiations occur when either different lineages within a species or different species within a given biome become adaptively specialized to more effectively use particular resources within that biome, while concomitantly becoming maladapted to use alternative resources⁷⁶. This type of specialization can minimize competition between the different lineages or species and result in highly polymorphic viral populations. On the other hand, generalist viruses that infect multiple host species (such as cucumber mosaic virus^{77,78}) have access to a larger array of resources but compete with other viruses for these resources. Such a situation should yield a low-diversity viral population dominated by one or a few of the best-adapted viral genotypes⁷⁶.

Obviously, whether a virus evolves as a specialist or as a generalist will strongly depend on the feeding preferences of its vectors^{79,80}. Viruses that have generalist vectors or several vectors should have more opportunities to infect different plant species. In this situation, the virus may evolve as a generalist or as a specialist. By contrast, viruses that have specialist vectors will encounter a limited number of potential hosts and might therefore be expected to evolve as specialists. For example, although aphids are quite specific with regards to which plants they feed on, they probe every plant that they encounter, thus facilitating the transmission of non-persistent viruses such as the generalist cucumber mosaic virus⁸¹. Conversely, the acquisition by aphid vectors of phloem-restricted viruses requires that the insects feed on virus-infected tissues. Possibly as a consequence of this requirement, viruses such as barley yellow dwarf

virus have specialized for transmission by a particular aphid species and, by extension, for infecting the plant species that its aphid vector feeds on⁸².

In keeping with the old adage that a ‘jack of all trades is a master of none’, it has been suggested that evolution should favour specialists because of expected trade-offs that limit the fitness of generalists in their alternative hosts^{83,84}. One of the reasons for such trade-offs, called antagonistic pleiotropy, might arise when mutations that are beneficial in one host are deleterious in another host⁸⁵. A fitness trade-off might also arise if neutral mutations accumulate by random genetic drift in genes that are dispensable in the current host but are useful in other hosts⁸⁶. Given the compactness of viral genomes, in particular those of small RNA and single-stranded DNA viruses, which have many multifunctional proteins, this second mechanism seems less plausible than the first. However, our current knowledge of viruses in their natural ecosystems^{87–89} is still too limited to satisfactorily establish how each of these mechanisms explains the observed host ranges of plant viruses. Although metagenomic studies are providing valuable information on the prevalence of different viruses in different host species, this information is not yet linked to quantitative measures of viral fitness in each host.

To compensate for this lack of observational data, experimental evolution in controlled greenhouse conditions is being increasingly used to study aspects of host-range expansion^{83,84,90,91}. Of note, in these experimental studies, evolution in a single new host always increases fitness in that host but, in most cases, concomitantly decreases fitness in the original host. At the molecular level, multiple cases of convergent evolution have been described, often involving the same mutations in several independent experiments^{83,91–93}. This suggests that trade-offs may occur as a consequence of a limited number of alternative evolutionary pathways towards adaptation to a novel host, such that adaptive mutations in a novel host are maladaptive in the original host.

By contrast, if viruses are alternatively passaged through several host species, they will frequently evolve as ‘no-cost generalists’⁸⁵. These generalist viruses are as fit as specialist viruses in all hosts, without paying the fitness cost predicted by the ‘jack of all trades but master of none’ hypothesis. In other words, under fluctuating host conditions, ‘master of all’ viruses can evolve. Although the evolution of no-cost generalists during such experiments has been frequently observed in animal viruses^{94–96}, only a single instance has been reported for plant viruses, in which lineages of tobacco etch virus were evolved in Solanaceae hosts⁹⁷. Whereas single-host virus lineages evolved as specialists and displayed the expected fitness trade-offs, lineages alternatively passaged between hosts evolved to be as fit as the single-host lineages in their corresponding hosts, thus behaving as true no-cost generalists. Interestingly, all generalist lineages fixed convergent mutations. The fact that some of these mutations were also detected in single-host lineages suggests that non-epistatic host species-specific mutations were responsible for the observed fitness increases in the alternative hosts.

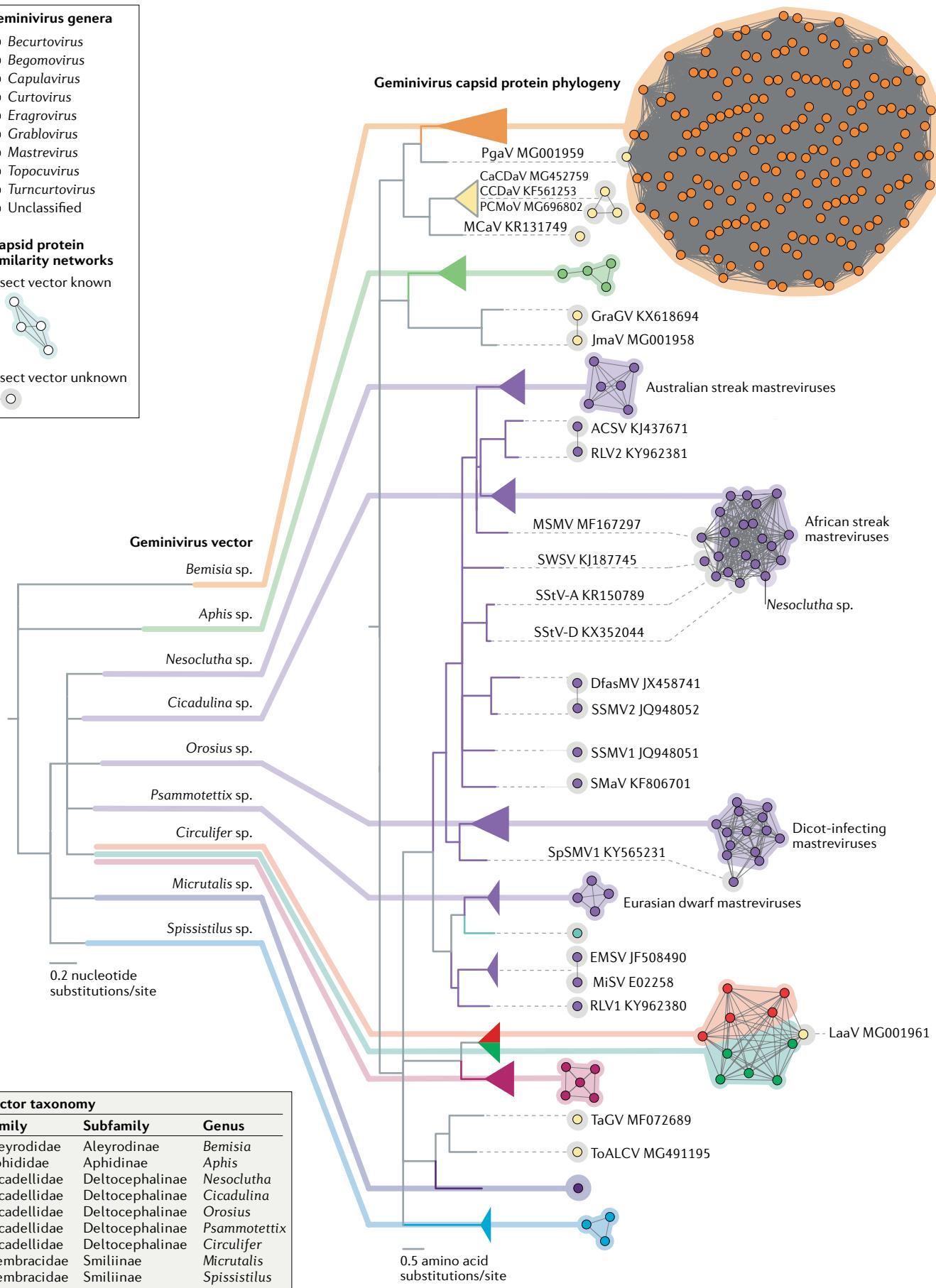
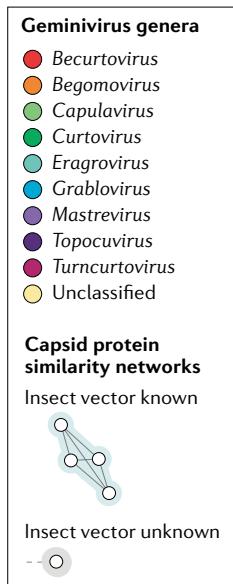
In other experimental evolution studies, viruses were passaged through differentially virus-resistant

Aphids

These small sap-sucking insects are members of the superfamily Aphidoidea in the Hemiptera order.

Phloem

The phloem is the vascular system in plants within which soluble organic compounds that are produced during photosynthesis are transported.



◀ Fig. 3 | Co-divergence of a viral capsid protein and transmission vector sequences.

The evolutionary relationships between capsid proteins of viruses in the *Geminiviridae* family are broadly congruent with the evolutionary relationships of the different insect species that transmit geminiviruses (in this case determined using cytochrome oxidase I gene nucleotide sequences). This pattern strongly supports the hypothesis that the capsid protein sequences of geminiviruses are co-evolving with their vectors. Capsid protein sequences were extracted from all available geminivirus full genome sequences ($n = 6,760$) before clustering them using CD-HIT¹⁶⁵. Only one exemplar sequence was retained from each cluster of sequences sharing >90% identity to leave only a subset of sequences representative of the entire known breadth of geminivirus capsid protein sequence diversity ($n = 276$). These sequences were aligned using MAFFT¹⁶⁶ with the satellite tobacco necrosis virus capsid protein sequence as an outgroup and used to construct a maximum likelihood phylogenetic tree with PhyML 3.0 (REF.¹⁶⁷) with the LG+I+G amino acid substitution model. The cytochrome oxidase I gene nucleotide sequences of known geminivirus vector species were obtained from the GenBank¹⁶⁸ and BOLD¹⁶⁹ databases and aligned with Nemouridae sequences (in the order Plecoptera) as outgroups using MAFFT¹⁶⁶. This alignment was used to infer a maximum-likelihood tree using PhyML 3.0 (REF.¹⁶⁷). Branches with less than 0.8 approximate likelihood ratio test support were collapsed in both the geminivirus capsid protein and the cytochrome oxidase I gene phylogenetic trees. The geminivirus capsid sequence similarity networks for different groups of geminiviruses were generated from the representative 276 sequence dataset using the Enzyme Function Initiative-Enzyme Similarity Tool¹⁷⁰ with an alignment score threshold of 60 and minimum E -value of 1×10^{-5} . Note that one of the geminivirus genera, *Mastrevirus*, has viruses with capsid proteins so diverse that they fall into seven distinct clusters and three singletons, whereas capsid protein sequences from the other genera all fall within individual clusters. ACSV, Axonopus compressus streak virus; CaCDaV, Camellia chlorotic dwarf-associated virus; CCDAV, Camellia citrus chlorotic dwarf-associated virus; DfASMV, dragonfly-associated mastrevirus; EMSV, Eragrostis minor streak virus; GraGV, grapevine geminivirus; JmaV, Juncus maritimus-associated virus; LaaV, Limeum africanum-associated virus; MCaV, mulberry crinkle-associated virus; MiSV, Miscanthus streak virus; MSMV, maize streak Reunion virus; PCMoV, passion fruit chlorotic mottle virus; PgaV, *Polygala* *garcinii*-associated virus; RLV1, rice latent virus 1; RLV2, rice latent virus 2; SmaV, switchgrass mosaic-associated virus; SpSMV1, sweetpotato symptomless mastrevirus 1; SSMV1, *Sporobolus* striate mosaic virus 1; SSMV2, *Sporobolus* striate mosaic virus 2; SStV-A, sugarcane striate virus A; SStV-D, sugarcane striate virus D; SWSV, sugarcane white streak virus; TaGV, tomato-associated geminivirus; ToALCV, tomato apical leaf curl virus.

genotypes of a single host species that differed in their level of virus resistance⁸⁴. Some reports have evaluated natural variations in the resistance of host genotypes to virus infection^{98,99}, whereas others have focused on artificially engineered virus resistance genes^{100,101}. In both cases, fitness trade-offs apparently depend on the particular host genotypes in which they evolve, with more-resistant host genotypes selecting highly virulent generalists and more-permissive host genotypes selecting low-virulence specialists. These differences could be partially explained by the differential effects that each type of viruses has on the transcriptional regulation of host genotypes¹⁰². Whereas generalists influence the same set of transcripts in all host genotypes, resulting in a relatively homogeneous response, specialists influence different sets of transcripts in each host genotype, resulting in heterogeneous responses.

If no-cost generalists can experimentally evolve in resistant plants or following passages through different hosts, the question arises of why specialists exist in ecosystems that commonly contain both several potential host species and individual plants with natural antiviral resistance. A possible answer is that access to resistant hosts that select for generalist viruses may be restricted under natural conditions for any combination of the following reasons: resistant hosts occur infrequently; successful infections of

resistant hosts are rare; or viruses infecting resistant hosts tend to not be transmitted further. Alternatively, one might also argue that in natural settings the conditions that favour the emergence of specialists, such as serial passages through genetically homogeneous host plants without any competition from other viruses, would occur infrequently. Unfortunately, we presently have no data on the relative frequencies of specialist and no-cost generalist viruses in any ecosystems.

Nevertheless, even if the appropriate conditions are only infrequently met outside the laboratory, it is probable, given the size and diversity of environments on Earth, that both types of viruses would naturally occur. Specifically, the diversity and distribution in the time and space of hosts and viruses in a given environment will determine how viruses use the resources in that environment⁹¹. Whether specialists, no-cost generalists or intermediates between these two extremes are most common in nature will hopefully be revealed in the near future by ecosystem-scale viromics studies that are designed to quantitatively evaluate both the distributions of individual viral lineages in different host species (preferably with enough resolution to differentiate between genotypes) and the relative titres of different viral variants at host sites from which the viruses are acquired by vectors.

Ecology of plant viruses

The pervasive perception that plant viruses are primarily pathogens has meant that most plant virology research has focused on the causes and consequences of virus pathology. Within the pathology field, emerging diseases have garnered most attention because they cause the most damage to economically important food and ornamental plant species. Key examples of viruses that are responsible for well-studied emerging diseases are cassava-infecting begomoviruses¹⁰³, closteroviruses causing grapevine leafroll disease¹⁰⁴, luteoviruses such as barley yellow dwarf virus¹⁰⁵ and sobemoviruses such as rice yellow mottle virus¹⁰⁶.

After decades of inventorying, tracking and analysing plant viruses¹⁰⁷, it is now apparent that the emergence of new diseases following changes in viral host ranges is driven by adaptive viral evolution in response to novel ecological conditions¹⁰⁸. These novel conditions include the introduction of viruses¹⁰⁸ and vectors to new areas, the intensification of agriculture and urbanization, and ecological changes in response to changing climatic conditions^{109,110}. In agriculture, which accounts for almost all studies of plant virus emergence, emergence is likely facilitated by persistent, mostly dead-end ‘spillover’ transmissions of viruses from uncultivated plant species to genetically homogeneous crop populations at so-called ‘agro-ecological interfaces’ between cultivated and natural ecosystems^{109,110}. At such interfaces, the detected proportion of infected individuals (a proxy for virus prevalence) has been found to be higher for cultivated species than for uncultivated species^{8,111,112}.

Although the frequency of detected emergence events has increased concomitantly with the pace with which natural biomes have been replaced by managed croplands^{109,113,114}, the ‘emergence potential’ of viruses is a natural property of virus populations, which likely is

crucial for maintaining the stability of unmanaged ecosystems. The ecological impacts of plant–virus interactions may be similar to those of interactions between lytic viruses and their bacterial hosts, in which viruses limit the outgrowth of genetically homogeneous bacterial populations during ecological competitions¹¹⁵. In stable unmanaged ecosystems, plant virus emergence events may preserve the genetic richness of ecosystems by preventing them from being overgrown by genetically homogeneous plants and, thereby, may foster the capacity of ecosystems to endure environmental changes^{116,117}.

In the context of invading exotic plants, the role of viruses can be mixed. On the one hand, they can facilitate invasion if an invasive plant species has fewer natural pathogens than the indigenous plant species (the enemy release hypothesis¹¹⁸) or if the invasive species induces an increase of virus loads in the indigenous plant species with which it competes^{119,120}. On the other hand, viruses may help prevent invasions if indigenous plant species carry viruses that infect the invader and are virulent enough to prevent the invader from overrunning, or even surviving long term in, the ecosystem¹²¹.

When a virus emerges in a new host, the amount of harm that the virus inflicts on its new host will,

initially at least, not be at an optimal level for onward transmission. Often, an emergent virus causes severe disease symptoms owing to maladaptation to its new host¹²². Various trade-off hypotheses^{123,124} predict that the basic reproductive number of a virus (that is, how many new hosts a virus will, on average, be transmitted to) will be maximized if the virus can balance its transmission rate against the duration of infection, the virus-induced mortality and the reproductive capacities of hosts. For a virus that mainly infects artificially managed crops, the lifespans and/or reproductive capacities of infected plants might not affect the size of future host populations, simply because farmers will ensure a constant stream of susceptible plants. In such cases, the evolutionarily optimal amount of harm a virus inflicts might be much higher than if the virus mainly infected uncultivated plants in natural ecosystems.

As intuitively appealing as such trade-off hypotheses are, few reports that test these for plant viruses exist¹²⁵ and the outcomes for viruses with different host-range sizes and/or transmission modes are unclear^{126,127}. For example, viruses with broad host ranges that include both cultivated and uncultivated hosts might optimally have high virulence in their cultivated hosts and low

Towards understanding the ecology and evolution of plant viruses at different tiers

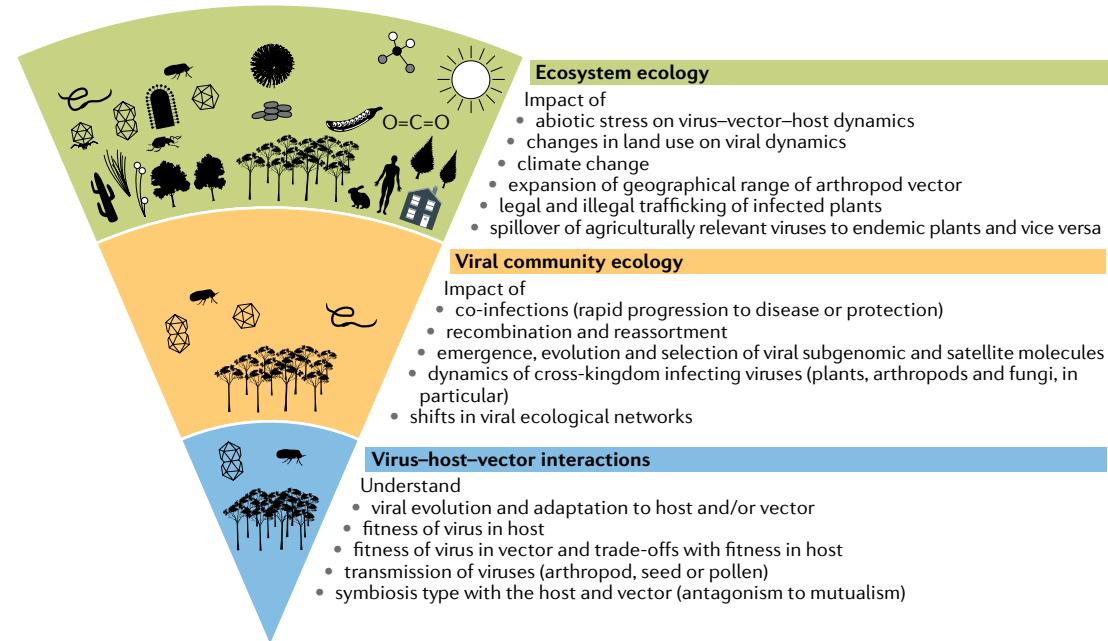


Fig. 4 | Addressing important questions in the ecology and evolution of plant viruses. High-throughput sequencing approaches, including single-molecule sequencing, coupled with traditional viral pathology methods can be used to study interactions up to the ecosystem scale. In general, these methods are becoming common for the rapid identification of plant viruses and, in certain cases, involve sequencing ‘in the field’ with portable sequencing machines such as the MinION (Oxford Nanopore Technologies). In the next decade, these methods will enable rapid mapping of viral sequence spaces and illuminate spatial and temporal virus–host–vector dynamics. Among other things, this will reveal spillover of viruses from agro-ecosystems to endemic wild flora (and vice versa), tentative expansions of virus host and/or vector ranges over time and exposure of plants in a specific region to imported viruses. On a broader scale, next-generation metagenomic studies should enable ecosystem-scale analyses of interaction networks between viruses, hosts, vectors and other microscopic and macroscopic flora and fauna in the environment. One of the big questions that such analyses could answer is whether it is possible to construct and apply synthetic microbial communities of beneficial viruses, bacteria and fungi to protect natural ecosystems from being overrun by invasive viruses, plants and insects, and to reduce the use of chemicals in modern agriculture.

virulence in their uncultivated hosts, but be constrained to have intermediate virulence across all their hosts. Alternatively, high virulence might be more beneficial for viruses that are primarily transmitted through soil or water following the death and decay of plants than it is for viruses that are primarily vertically transmitted through seeds or pollen. Therefore, in practice, the optimal degree of virulence that a virus should have in a given host will depend on the relative epidemiological importance of that host as a reservoir for onward transmission and the different transmission modes.

Empirically, in natural ecosystems at least, the optimal degree of harm that plant viruses inflict on their hosts is probably very low^{128–130}. The hypothesis that most plant virus infections are essentially harmless to host plants, however, has remained largely untested and is still controversial¹³¹. Viruses are, after all, parasitic in their reliance on host-provided enzymes and cofactors for their replication, and the existence of evolutionarily ancient antiviral defence strategies throughout the plant kingdom¹³² implies that plants would rather remain uninfected. Accordingly, although some viruses cause fewer and/or less severe symptoms in uncultivated hosts than in cultivated hosts, viruses still generally inflict measurable harm on their uncultivated hosts^{133–136}.

Demonstrating that a virus benefits a host is difficult as the virus and the host do not exist in isolation in an ecosystem. Instead, they are part of a holobiont, encompassing the host plant and all bacteria, archaea, eukaryotes and viruses that it interacts with^{137,138}. In this regard, the benefit or harm of a virus for a host probably depends on how direct and indirect interactions between the virus and other members of the holobiont community affect the composition of the holobiont and, ultimately, the fitness of the host.

Interactions between hosts and parasites or symbionts can be divided into two broad categories: pre-infection interactions, which affect the susceptibility and/or exposure of a host to a second parasite or symbiont; and post-infection interactions, which affect the quality of within-host nutrient resources for, or host immune responses to, a second parasite or symbiont¹³⁹. For example, a virus infection might reduce the susceptibility of plants to mammal herbivory¹⁴⁰ or fungal infections¹³⁹. Although several greenhouse studies suggest that virus infection might slow plant desiccation during droughts^{141–144}, others have shown that heat, drought or salt stressors increase plant susceptibility to viral pathogens¹⁴⁵ or that virus infection has no effect on drought tolerance¹⁴⁶. Also, even when elements of plant–virus interactions are genuinely symbiotic, the degree of mutualism versus pathogenicity can depend

both on the host and virus genotypes and on the intensity of the environmental factor that the virus provides some resistance against^{91,147}.

Conclusions

Our rapidly expanding view of viral diversity is illuminating the deep evolutionary relationships of viruses^{3,148} and indicates that an appreciable fraction of many plant, fungal and arthropod viruses share common origins¹⁶. The future large-scale sampling and analysis of viral genomic sequence data from diverse environments will undoubtedly reveal additional evolutionary relationships, which may reveal the distant origins of all of the major virus groups (FIG. 1).

With proper design, these large-scale virus-sequencing projects could also yield insights into the roles of viruses in different ecological contexts. Of particular interest are the collateral impacts of human activities on these ecological roles. Although there are very few terrestrial environments that have remained uninfluenced by human activities, the degree to which ecosystems have been disturbed by humans ranges from minor for mostly pristine preserved environments through to extreme for industrial wastelands. Crucially, relatively undisturbed ecosystems frequently occur side by side with moderately disturbed agro-ecosystems. Viromics studies at these agro-ecosystem interfaces that include the collection of plant tissues, trapped or captured insects and soil over ecologically relevant areas and over multiple time points will likely prove crucial for understanding the impacts of ecological disturbances on the population and evolutionary dynamics of plant viruses (FIG. 4). Such studies will be particularly powerful and quantitative (relative frequencies of genomic sequence reads correspond to the relative frequencies of sequences within the virome), especially when virus-sequence reads are linked to specific geographical locations and the host or vector. In addition to enabling the large-scale quantitative determination of the relative epidemiological and evolutionary importance of different host and vector species for individual virus species, such studies could also expose epidemiological links between viruses associated with different plants, fungi and arthropods. We anticipate that recent and future methodological and analytical innovations will enable the design and implementation of plant viral metagenomics studies that reveal the spatial and temporal dynamics of virus infections and the effects on plant communities. These hypothesis-driven next-generation metagenomics studies will definitively determine if, and how, plant viruses either foster or harm the stability and productivity of ecosystems.

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Author contributions

P.L., D.P.M., S.F.E., D.N.S., P.R. and A.V. wrote and edited the manuscript. P.L. and A.V. undertook the analyses for the data presented in figures 1–3.

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