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Deep Roots and Splendid Boughs of the Global Plant Virome

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Abstract

Land plants host a vast and diverse virome that is dominated by RNA viruses, with major additional contributions from reverse-transcribing and single-stranded (ss) DNA viruses. Here, we introduce the recently adopted comprehensive taxonomy of viruses based on phylogenomic analyses, as applied to the plant virome. We further trace the evolutionary ancestry of distinct plant virus lineages to primordial genetic mobile elements. We discuss the growing evidence of the pivotal role of horizontal virus transfer from invertebrates to plants during the terrestrialization of these organisms, which was enabled by the evolution of close ecological associations between these diverse organisms. It is our hope that the emerging big picture of the formation and global architecture of the plant virome will be of broad interest to plant biologists and virologists alike and will stimulate ever deeper inquiry into the fascinating field of virus–plant coevolution.

INTRODUCTION: OVERVIEW OF VIRUS ORIGINS, HOST RANGES, AND MEGATAXONOMY

During the past decade, we have witnessed dramatic inflation in the number and diversity of known viruses, thanks to the reduction of nucleotide sequencing costs and the rapid rise of metagenomics, metatranscriptomics, and metaviromics. The preceding stasis in the study of the global virome was marked with a heavy bias toward medically or economically important virus diseases. Although the remarkable diversity of virus genome replication and expression cycles was well appreciated, the virus world looked rather fragmented at the time. The exponential growth in new virus discovery revealed numerous connections between virus lineages, enabled the development of unifying concepts (12, 20, 21, 55, 70, 100, 149, 194, 200), and helped to draft the first coarse-grained chart of the entire virus world (88). This new virus world emerged as a gene–genome network of high internal connectivity and perpetual dynamic exchange with the worlds of other mobile genetic elements (MGEs) and cellular host organisms (76, 86, 89).

Unlike cellular organisms that share ~100 homologous genes inherited from the last universal cellular ancestor (LUCA), there is not a single gene shared by all viruses. Therefore, as a whole, viruses are undoubtedly polyphyletic, originating on several or even numerous occasions from distinct gene sets (97). However, there are two functional types of virus genes that define the virus lifestyle: those enabling semiautonomous genome replication (replication modules) and those responsible for virion formation (morphogenetic modules). Some viruses encode no proteins directly involved in replication (69), whereas others have lost the morphogenetic module (e.g., capsidless viruses) (86). However, the vast majority of viruses carry both replication and morphogenetic modules, and disentangling their evolutionary histories is key to understanding virus origins and evolution in general.

The semiautonomous, semiparasitic genome replication model of viruses is shared with an enormous variety of selfish replicons or MGEs that do not form virions but encode at least some genes involved in their propagation. The most common MGEs are DNA plasmids and self-propagating transposons (e.g., eukaryotic retrotransposons). Replication of the diverse MGEs involves protein-primed DNA polymerase B (PolB), rolling-circle replication endonucleases (RCREs), superfamily 3 helicase (S3H), or reverse transcriptase (RT). Strikingly, all these enzymes are also typical of viruses and are either rare or completely absent in cells (97). In addition, RNA viruses, but not MGEs or cells, encode RNA-dependent RNA polymerases (RdRPs) homologous to the RTs. It remains an open question whether the extant virus RdRPs are direct descendants of primordial RdRPs that might have been involved in the replication of RNA genomes in the hypothetical RNA-protein world but before the advent of DNA genomes (194).

The shared evolutionary histories of replication modules of MGEs and viruses can be tentatively traced to ancient replication systems predating LUCA, with the primordial RNA recognition motif (RRM) being at the root of the replicative enzymes (97). Therefore, virus/MGE replication modules appear to emerge at the earliest stages of evolution, possibly even within protocellular replication systems, in accord with the concept of genetic parasite inevitability (90).

What distinguishes viruses from other MGEs are proteinaceous capsids that harbor and protect virus genomes between infections and enable genome delivery to the host cell. Despite the spectacular variety of capsid morphologies, the virus morphosphere is heavily dominated by icosahedral viruses, followed by those with elongated helical capsids. Recent analyses indicate that many if not most virus morphogenetic modules have evolved from cellular ancestors at different phases of life evolution from LUCA to this day (100). Thus, the evolutionary histories of the core modules of virus genomes point to ancient MGE-like elements providing replication-related proteins to emerging viruses while snatching protocapsid proteins from cells as the prevalent scenario in virus origins (97).

Given the apparent primordial origins of MGEs, a timeframe for the origin of bona fide, encapsidated viruses can be approximated from the evolutionary history of the respective virus hosts. A paradigm of the early origins, perhaps at the LUCA stage, is provided by viruses with single jelly roll (SJR) and double jelly roll (DJR) capsid proteins (CPs). These virion proteins were proposed to emerge on several occasions via repurposing of a wide variety of cellular carbohydrate-binding homologs sharing an SJR fold (100). Because the dsDNA viruses possessing icosahedral capsids formed by DJR-CP infect bacteria, archaea, and diverse eukaryotes, it seems likely that the DJR-CP evolved in the pre-LUCA virosphere, and viruses encoding this CP diversified, adapting to newly evolving host organisms. Although SJR-CPs are particularly common in eukaryotic RNA and ssDNA viruses, there are some DNA bacteriophages and archaeal viruses utilizing this archetypal CP fold. However, it appears that these groups of viruses have recruited cellular SJR proteins on several independent occasions.

In sharp contrast to ancient and widespread SJR-CPs and DJR-CPs, the zinc finger domain virion matrix Z protein is utilized by only one family of vertebrate −RNA viruses, *Arenaviridae* (100). The Z-protein fold is closely similar to that of eukaryotic E3 ubiquitin ligases, implying relatively recent recruitment by ancestral arenavirus, apparently within a timeframe of vertebrate evolution. Therefore, emergence of viruses with novel combinations of replication and morphogenetic modules covers the entire history of life, from LUCA to vertebrates, and likely extends to this day (see the section titled ssDNA Viruses).

In general, viruses with distinct forms of encapsidated genomes (Baltimore classes) are differentially represented among evolutionary lineages of cellular host organisms (88). The archaea host the most restricted set of viruses, namely, dsDNA and ssDNA viruses only. In contrast, animals are the only group of organisms known to host viruses of all seven Baltimore classes, including RNA, reverse-transcribing, and DNA viruses. The virome of the land plants has a distinct composition that is heavily dominated by diverse +RNA viruses, with a more limited representation of dsRNA, −RNA, reverse-transcribing, and ssDNA viruses, to the exclusion of bona fide dsDNA viruses (34). In contrast, the virome of green algae is rich in large dsDNA viruses of the family *Phycodnaviridae* (22, 129, 185). The virome of fungi has a similar composition, lacking dsDNA viruses as well, but exhibits a bias toward dsRNA viruses (35, 48).

Although the Baltimore classes provide a useful framework for comparing virome compositions, the most recently developed and International Committee on Taxonomy of Viruses (ICTV)-approved classification of viruses is not based on Baltimore classes or virion morphology (88). Rather, this megataxonomy is underpinned by virus phylogenomics complemented by bipartite (gene–genome) network analysis and comparison of the virion and CP structures. This evolutionary classification includes four virus realms, each subdivided into kingdoms, phyla, classes, orders, families, genera, and species (88). The virome of land plants fits in two realms, *Riboviria* (RNA and reverse-transcribing viruses) and *Monodnaviria* (ssDNA viruses).

Below, we discuss the composition and large-scale evolution of the plant virome from the vantage point of phylogenomics. Because of the sparse sampling of lower plants (green algae, bryophytes, lycophytes, and ferns) as well as gymnosperms, the analysis of the plant virome is mostly limited to flowering land plants (angiosperms). However, viruses of lower plants are briefly covered in the context of the plant virome evolution. Our principal conclusion is that the plant virome was largely shaped by horizontal virus transfer (HVT), often between extremely divergent hosts. The HVT events appear to occur through tight ecological associations between diverse organisms, including predation and parasitism as well as commensalism and symbiosis. The shorter-term evolution of viruses via mutations (46, 125) is beyond the scope of this article.

COMPOSITION OF THE ANGIOSPERM VIROME

There are at least three metrics that are useful for classifying plant virome components: (*a*) evolutionary, i.e., classified via phylogenomic and taxonomic diversity; (*b*) ecological, i.e., classified via the virus host range and infection frequency within plant populations; and (*c*) economical, i.e., classified via virus disease impacts on crop, bioenergy, or ornamental plants. Here, we focus on the evolutionary approach but also mention virus ecology and disease impacts where these are most relevant.

By and large, the replication and morphogenetic modules of plant viruses are shared with other viruses of eukaryotes, and animal viruses in particular (34). What distinguishes plant viruses from their kin are processes defined by the specifics of plant biology: plant-to-plant virus transmission followed by two-phase systemic infection that involves local cell-to-cell movement and systemic transport through the plant vasculature (134).

The active intercellular virus spread occurs through plasmodesmata (14) and typically requires specialized, virus-encoded movement proteins (MPs) (64). The evolutionarily diverse MPs represent the most prominent signature of plant viruses (131). Typically, systemic transport relies on additional functionalities of virus proteins such as MPs, CPs, or counter-defense proteins (41). However, in some viruses with larger genomes, this function involves dedicated long-distance transport proteins.

The plant-to-plant transmission of viruses requires vectors such as plant-feeding arthropods, nematodes, plant-parasitic fungi, and plasmodiophorids (protists of the phylum *Cercozoa*) (9, 42, 140, 159). The process of transmission is virus and vector specific and often involves genetic determinants associated with virions and additional transmission factors known as helper components.

Plants possess potent RNA-based defense systems against both RNA and DNA viruses, including RNA interference (RNAi), also known as RNA silencing (6, 62, 111). To facilitate infection, many plant viruses rely on either specialized RNAi suppressor proteins or suppression activity of proteins with other functionalities (e.g., MPs, CPs, or transmission factors) (23).

Thus, a genome of an archetypal plant virus contains replication, morphogenesis, transport, transmission, and RNAi suppression modules. Many virus genes, particularly in viruses with small genomes, contribute to more than one of these activities. There are, however, plant viruses with reduced, minimal genomes that have either lost or never acquired transport, transmission, or counter-defense functions. Such viruses lead a persistent lifestyle characterized by vertical transmission through seeds and/or pollen and a lack of pathogenicity and infectivity (ability to infect new hosts de novo via plant-to-plant transmission) (142, 160). In this section, we describe the taxonomic structure of the global plant virome based primarily on the evolutionary provenance of the virus replication and morphogenetic modules.

RNA Viruses: Realm *Riboviria*, Kingdom *Orthornavirae*

As mentioned above, most of the plant virome diversity fits into the realm *Riboviria*. Within this realm, the kingdom *Orthornavirae* harbors the bona fide RNA viruses with no DNA stage in their replication cycles (88). The replication modules of RNA viruses are organized around the RdRP, the only gene that is conserved in all viruses of this kingdom, to the exclusion of the rest of the global virome. Therefore, the phylogenetic tree of the RdRPs is used as a scaffold to reconstruct the RNA virus evolution and develop the corresponding taxonomy (194). According to this tree, *Orthornavirae* splits into five branches at the phylum rank (**Figure 1**).

Phylum *Lenarviricota*. The deepest branching phylum *Lenarviricota* harbors +RNA bacteriophages that are believed to be the ancestors of eukaryotic virus families *Mitoviridae*, *Narnaviridae*,

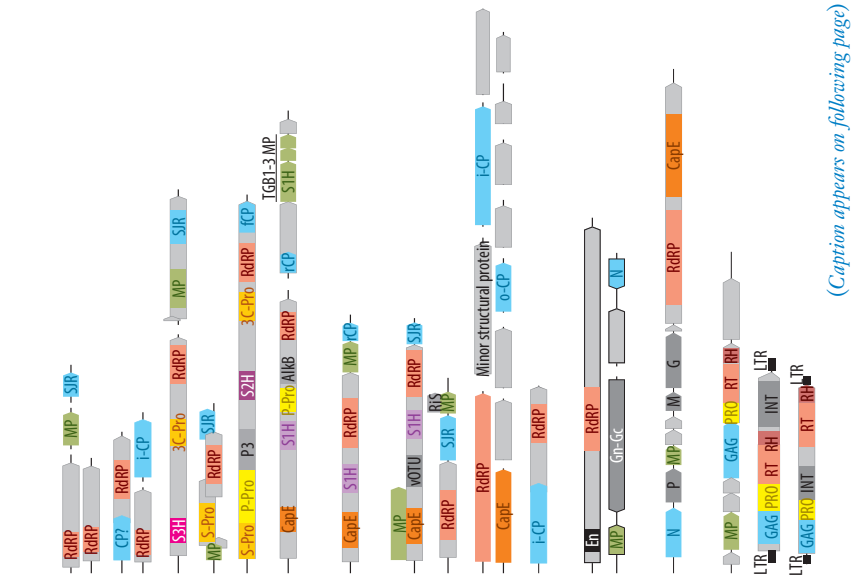
REALM
KINGDOM
PHYLUM
CLASS
ORDER
FAMILY
GENOME
Riboviria
Orthornavirae
Pararnavirae
Kitrinoviricota
Duplornaviricota
Negarnaviricota
Pisuviricota
Artverviricota
Alsuviricetes
Tolucaviricetes
Duplopiviricetes
Pisoniviricetes
Stelpaviricetes
Resentoviricetes
Chrymoviricetes
Ellioviricetes
Milneviricetes
Monjiviricetes
Retroviricetes
Levivirales
Durnavirales
Picornavirales
Sobelivirales
Patatavirales
Hepelivirales
Martellivirales
Tymovirales
Tolivirales
Reovirales
Ghabrivirales
Bunyavirales
Serpentovirales
Mononegavirales
Ortervirales
Botourmiaviridae, Mitoviridae*
Amalgaviridae, Partitiviridae
Secoviridae
Solemoviridae
Potyviridae
Benyviridae
Bromoviridae, Closterovirida, Endornaviridae, *Kitaviridae, Virgaviridae
Alphaflexiviridae, Betaflexiviridae, Tymoviridae
Luteoviridae, Tombusviridae
Reoviridae
Totiviridae
Fimoviridae, Phenuiviridae, Tospoviridae
Aspiviridae
Rhabdoviridae
Caulimoviridae
Metaviridae
Pseudoviridae
OuMV
CpMV1
STV
BCV1
CPMV
SBMV
TEV
BdMoV
TMV
TYMV
TBSV
RDV
L-A
TSWV
BYSMV
CaMV
Athila
Hopscotch
RdRP
MP
SIR
CP7
I-CP
S3H
3C-Pro
S-Pro
P-Pro
P3
S2H
CapE
S1H
RdRP
AlkB
rCP
vOTU
RNS
Minor structural protein
o-CP
Gn-Gc
N
NI
G
GAG
PRO
RT
RH
INT
LTR
TGB1-3 MP
fCP
S1H
(Caption appears on following page)

Figure 1 (*Figure appears on preceding page*)

Hierarchical taxonomy of the plant RNA and reverse-transcribing viruses. Only taxa containing plant-infecting viruses are shown. Genome maps of selected viruses are shown on the right. Functionally equivalent domains or genes are indicated with the same color. Asterisks denote families including capsidless viruses. Viruses: OuMV, Ourmia melon virus (NC_011068, NC_011069, NC_011070); CpMV1, Cryphonectria parasitica mitovirus 1 (NC_004046); BCV1, Beet cryptic virus 1 (NC_011556, NC_011557); STV, Southern tomato virus (NC_011591); CPMV, Cowpea mosaic virus (NC_003549, NC_003550); SBMV, Southern bean mosaic virus (NC_004060); TEV, Tobacco etch virus (NC_001555); BdMoV, Burdock mottle virus (NC_021735, NC_021736); TMV, Tobacco mosaic virus (NC_001367); TYMV, Turnip yellow mosaic virus (NC_004063); TBSV, Tomato bushy stunt virus (NC_001554); RDV, Rice dwarf virus (NC_003760, NC_003761, NC_003762, NC_003763, NC_003764, NC_003765, NC_003766, NC_003767, NC_003768, NC_003772, NC_003773, NC_003774); L-A, Saccharomyces cerevisiae virus L-A (NC_003745); TSWV, Tomato spotted wilt virus (NC_002052, NC_002050, NC_002051); BYSMV, Barley yellow striate mosaic cytorhabdovirus (NC_028244); CaMV, Cauliflower mosaic virus (NC_001497); Athila, Arabidopsis thaliana Athila virus (X81801); Hopscotch, Zea mays Hopscotch virus (ZMU12626). Abbreviations: RdRP, RNA-dependent RNA polymerase; MP, movement protein; SJR, single jelly roll capsid protein; i/o/f/r-CP, inner/outer/filamentous/rod-shaped capsid protein; S1/2/3H, superfamily 1/2/3 helicase; 3C-Pro, 3C protease; S/P-Pro, serine/cysteine protease; vOTU, viral homolog of the ovarian tumor protease; CapE, capping enzyme; En, cap-snatching endonuclease; AlkB, alpha-ketoglutarate-dependent dioxygenase; RiS, RNA interference suppressor; G/Gn-Gc, receptor-binding and membrane fusion glycoprotein; G, glycoprotein; N, nucleocapsid; M, matrix protein; P, phosphoprotein; GAG, group-specific antigen; INT, integrase; RT, reverse transcriptase; RH, RNase H; LTR, long terminal repeat.

and *Botourmiaviridae* (35, 194). The mitoviruses are capsidless RNA replicons that encode only the RdRP and replicate within the mitochondria. Most of the known mitoviruses have been identified in fungi, but recently members of this family have been detected in plants as well (66, 143). Technically, capsidless, noninfectious mitoviruses are mobile RNA elements, their claim to virusness being solely the RdRP. The family *Botourmiaviridae* is also populated by fungal viruses but contains a small genus *Ourmiavirus* that includes encapsidated, MP-encoding plant viruses (122, 155). In addition, a rich diversity of related yet unclassified viruses has been described in invertebrates (173). Thus, plant ourmiaviruses represent but a twig within *Lenarviricota*, a phylum that is expected to spawn several new virus taxa.

Phylum *Pisuviricota*. This second phylum of RNA viruses corresponds to a massive lineage previously described as the picornavirus supergroup (85, 91) and now splits into three classes (**Figure 1**) (88). The class *Duplopiricetes* consists of simple icosahedral dsRNA viruses that typically encode only the RdRP and a distinct type of CP. In this class, there are two families that contain plant viruses, *Partitiviridae* and *Amalgaviridae*. *Partitiviridae* is a vast family that includes a variety of fungal and unclassified invertebrate viruses (142, 173). The currently recognized plant partitivirids are corralled into genera *Alphapartitivirus* and *Betapartitivirus*, both shared with their fungal and unclassified invertebrate kin. This striking host range diversity among closely related viruses is suggestive of their exceptional propensity to HVT. A broad plant metavirome screening has shown that partitivirids are a prevalent component of the plant virome present in a variety of the wild plant species (161). The reason why the apparent ecological dominance of partitivirids was overlooked is that they lead a nonpathogenic lifestyle. The dsRNA amalgavirids of plants and, again, fungi encode RdRPs related to those of partitivirids and a protein distantly related to nucleocapsid proteins (NCs) of −RNA plant viruses in the *Phenuiviridae* family (see below) (96, 153). Similar to partitivirids, plant amalgavirids apparently resigned to a noninfectious, vertically transmitted lifestyle (168). It is not clear whether amalgaviruses form virions, but they provide an apparent case of a dsRNA virus that, instead of transcribing its genome in virio, which is typical of dsRNA viruses, adopted the replication mechanism involving NCs that is characteristic of −RNA viruses.

In contrast to the dsRNA *Duplopiricetes* included in *Pisuviricota* solely by virtue of the phylogenetic affinity of the RdRPs, *Pisoniviricetes* and *Stelpaviricetes* share a chymotrypsin-like protease

responsible for the polyprotein processing, the SJR-CP and protein (VPg)-primed mechanism of RNA synthesis (**Figure 1**). The class *Pisoniviricetes* includes two orders, *Picornavirales* and *Sobelivirales*, each harboring a family of icosahedral plant viruses, *Secoviridae* and *Solemoviridae*, respectively. The secovirids are rank-and-file picornaviruses with two-component genomes that share S3H with other members of *Picornavirales* (**Figure 1**). Most of the family members are transmitted by insect (e.g., aphids, beetles, whiteflies) or nematode vectors (169). By contrast, solemovirids have much smaller, densely packed genomes (**Figure 1**) and are transmitted by beetles and a variety of other insects (179).

Finally, the class *Stelpaviricetes* includes the order *Patatavirales*, which has a single, expansive, economically important plant virus family, *Potyviridae* (49, 156). The potyvirids are a highly derived family of picorna-like viruses that encode a superfamily 2 helicase (S2H) not found in other picorna-like viruses as well as two additional proteases with multiple functions in virus replication, RNAi suppression, and vector transmission. Unlike most of the viruses in this phylum that have icosahedral virions made of SJR-CPs, potyviruses possess a distinct type of CP that forms flexuous filamentous virions of diverse plant +RNA viruses (fCP) (33, 198). Strikingly, structural analysis has shown that fCP is also homologous to phenuivirid (−RNA viruses) NCs, indicating yet another evolutionary connection between RNA viruses from different phyla (2). Most potivirids share the nonpropagative, nonpersistent transmission mode that involves virion attachment to receptors within the arthropod stylet or foregut mediated by the virus helper component (192). Interestingly, potyvirus genera evolved affinity to distinct vectors, including aphids, whiteflies, and mites, whereas the genus *Bymovirus* exploits plasmodiophorid protists for virus transmission (159).

Phylum *Kitrinoviricota*. Unlike other *Riboviria* phyla, where plant viruses are in the minority, the phylum *Kitrinoviricota* includes a large fraction of plant viruses that heavily dominate the class *Alsuviricetes* (88). Viruses in this class (formerly known as the Alphavirus-like supergroup) share a universal signature of genome architecture that includes the capping enzyme (CapE), superfamily 1 helicase (S1H), and RdRP (**Figure 1**) (85, 87). Aside from this replication module, these viruses show a remarkable diversity of genome organization and virion structure. On the minimalist end of the complexity spectrum is the family *Virgaviridae*, which includes the archetypal *Tobacco mosaic virus* (TMV) with a 6.4-Kb genome that encodes only CapE–S1H–RdRP RNA replicase, MP, and a single CP forming rigid rod-shaped particles (rCP). TMV employs an atypical, vectorless mode of transmission via mechanical damage of host plants, be it caused by wind, passing animals, or agricultural activities (170).

On the more baroque side is the family *Closteroviridae*, where the prototype member Beet yellows virus (BYV) has a ∼15.5-Kb genome encoding 10 proteins, of which 5 form the morphogenetic module and assemble into complex filamentous virions (32). Three of these CPs are homologous to the fCP of potyvirids as well as to those of alpha-, beta-, and gammaflexivirids, also members of *Alsuviricetes* (33, 132). The six-component transport module of closteroviruses includes a dedicated MP and the entire morphogenetic module, suggesting that a complex virion architecture evolved to facilitate virus movement (36). In addition, closteroviruses encode potent RNAi suppressors (19) and, altogether, present one of the most spectacular examples of genome complexification among RNA viruses (25, 36, 41). Analogous to potivirids, closterovirids from distinct genera are transmitted in a nonpropagative, semipersistent manner by different insect vectors, e.g., aphids, whiteflies, and mealybugs (75).

Taxonomically, *Alsuviricetes* split into three orders, of which *Hepelivirales* harbors a single plant virus family, *Benyviridae* (51). The rCP of benyvirids is homologous to that of virgavirids (33) and forms the rod-shaped virions transmitted by plasmodiophorid vectors (159). The much larger order *Martellivirales* includes *Bromoviridae*, *Kitaviridae*, and *Endornaviridae* in addition to *Virgaviridae*

and *Closteroviridae* discussed above. The bromovirids have small tripartite genomes and icosahedral virions that are typically transmitted by various insects in a nonpersistent manner. The most notorious of the bromovirids, *Cucumber mosaic virus*, infects no less than 1,000 plant species and is transmitted by aphids (171). The kitavirids are the only plant viruses in this phylum with enveloped virions transmitted by mites (107, 150, 154) and are related to insect viruses in the provisional genus *Negevirus* (186). The endornavirids are a peculiar group of viruses that, in addition to the replication module typical of *Alsuvericetes*, encode various enzymatic domains but have lost the morphogenetic module altogether. These capsidless viruses are found in fungi and oomycetes but are extremely widespread in plants, where they cause symptomless, persistent, vertically transmitted infections that have been almost completely overlooked in the premetaviromics era (43, 163).

The third *Alsuvericetes* order, *Tymovirales*, consists of five families, including three families of filamentous viruses that share fCP, *Alphaflexiviridae*, *Betaflexiviridae*, and *Gammaflexiviridae* (formerly combined in one family *Flexiviridae*); spherical *Tymoviridae* encoding SJR-CPs; and capsidless *Deltaflexiviridae* (121). Among these, *Alphaflexiviridae*, *Betaflexiviridae*, and *Tymoviridae* infect plants, whereas *Gammaflexiviridae* and *Deltaflexiviridae* infect plant-pathogenic fungi. In addition to highly conserved RdRPs, CapE, and S1H, many *Tymovirales* possess a papain-like protease. However, these plant virus families have unrelated transport modules: triple-gene block MPs in *Alphaflexiviridae*, 30K-like MP in *Betaflexiviridae*, and a unique MP in *Tymoviridae* (131, 187). The alphaflexivirids and betaflexivirids are transmitted by a variety of arthropods, including aphids, mites, and mealybugs, although viruses in the genus *Potexvirus* and some other genera appear to lack vectors and are transmitted mechanically (121). This latter property is apparently shared by some tymovirids, whereas others are transmitted by beetles in a nonpropagative manner. Strikingly, it has been reported that tymovirids in the genus *Marafivirus* are transmitted by leafhoppers in a propagative fashion, that is, they replicate within the insect (67), a feature that is so far unique among the nonenveloped +RNA plant viruses.

The second class in the phylum *Kitrinoviricota*, *Tolucaviricetes*, contains a single order, *Tolivirales*, with two families of icosahedral plant viruses, *Tombusviridae* (191) and *Luteoviridae* (182). This class is linked to *Alsuvericetes* chiefly through the RdRP phylogeny; other genes encoded in the small, densely packed tombusvirus and luteovirus genomes encode only SJR-CPs (distantly related to those in tymoviruses) and unique types of MPs and RNAi suppressors (**Figure 1**). Many of the tombusvirids are transmitted by fungi, whereas some (e.g., members of the genus *Tombusvirus*) could be soil-transmitted without vectors (159). The luteovirids are transmitted by aphids in a distinct, persistent, circulative, and nonpropagative manner whereby viruses travel from the insect's gut to its salivary gland without replicating and are deposited through saliva when the aphid feeds on a next plant's phloem (9, 54).

The third class of *Kitrinoviricota*, *Flasuvericetes* (Flavivirus supergroup), currently includes the order *Amarillovirales* with a single family of exclusively animal, enveloped viruses, *Flaviviridae*. However, a single flavi-like virus, Gentian Kobu-sho-associated virus (GKaV), has been identified in alpine ornamentals cultivated in Japan (4, 82). At ~23 Kb, this virus possesses the largest among all known plant virus monopartite genomes. Because the two viruses most closely related to GKaV infect plant-feeding invertebrates, [*Macrosiphum euphorbiae* virus 1 identified in a potato aphid (183) and soybean cyst nematode virus 5 (7)], it seems likely that this virus was relatively recently transferred from invertebrates to plants.

Phylum *Duplornaviricota*. This phylum of dsRNA viruses encompasses a limited diversity of plant viruses that belong to extremely dissimilar families, *Totiviridae* and *Reoviridae*. Totivirids are among the simplest RNA viruses, encoding just a CP and the RdRP (**Figure 1**), the same gene

complement as in partitiviruses. Furthermore, although the RdRPs of these dsRNA virus families are widely separated in the phylogenetic tree, both form similar icosahedral virions in which 60 CP homodimers are organized on a pseudo T = 2 lattice (123). This capsid architecture typical of diverse dsRNA viruses is not seen among other viruses. The genome organizations of partitiviruses and totiviruses are, however, distinct: Whereas the former possess bipartite genomes, totiviruses typically express CP and RdRP from a single genome-size mRNA via translational frameshift.

Plant-infecting totiviruses have been discovered only recently in ecogenomics studies. The persistent, apparently nonpathogenic lifestyle of these viruses appears to be similar to that of plant partitiviruses, although much is to be learned about the biology and ecology of plant totiviruses (M. Roossinck, personal communication). So far, *Totiviridae* has been known to include a variety of viruses that infect fungi, parasitic protists, and invertebrates (48, 60, 173).

In contrast, plant reoviruses are well-studied, pathogenic, insect-transmitted viruses (128, 189) that form three genera within the family that is otherwise heavily dominated by animal viruses and also includes a few fungal viruses and a virus from a green picoplankton alga (5, 173). Unlike the +RNA viruses discussed above, which are transmitted by arthropod or nematode vectors without replicating (nonpropagative transmission), plant reoviruses replicate in their insect (leaf- or planthopper) vectors (propagative transmission). Such dual host range involving extremely divergent organisms provides a striking example of virus adaptability as well as potential clues to the routes of reovirus evolution (see the section titled Origins and Diversification of the Plant Virome: Horizontal Virus Transfer and Virus–Vector Associations).

Similar to animal reoviruses, their plant kin possess large segmented genomes that consist of 10 or 12 unique dsRNA molecules (**Figure 1**) encapsidated in a peculiar double-shelled, concentric icosahedral virion. These genomes produce up to seven virion proteins, including outer and inner CPs as well as RdRP and CapE, which are coencapsidated with the genome. These virion proteins function in virion assembly, vector transmission (outer CP), and RNA replication within infected cells. The nonstructural proteins are involved in the formation of the viroplasm (where genome replication and virion assembly apparently take place), in RNAi suppression, and in virus cell-to-cell movement in plants and insects (128).

Phylum *Negarnaviricota*. Similar to dsRNA viruses, −RNA viruses in this phylum encapsidate their RdRPs and additional replication proteins. Their virions, however, adopt a highly distinct architecture, typically with a condensed, helical, filamentous nucleocapsid and a membrane envelope adorned with virus-encoded glycoproteins (47, 92).

The host range of *Negarnaviricota* is dominated by invertebrate viruses followed by vertebrate viruses (110, 172). Several −RNA viruses were recently discovered in protists (59) and fungi (112, 122). The plant viruses in this phylum are notably less diverse than their animal cousins, forming three families (*Tospoviridae*, *Fimoviridae*, and *Aspiviridae*) and several genera within two large families of mostly animal viruses (*Phenuiviridae* and *Rhabdoviridae*). Again, similar to plant reovirids, most of these viruses are dual-host parasites that reproduce in both plants and arthropod vectors. Thus, plant virus members of *Tospoviridae* possess three-component ambisense genomes encapsidated into enveloped virions that also infect their minute insect transmission vectors, thrips (17). Perhaps the most notorious of the plant −RNA viruses, Tomato spotted wilt virus, which is endowed with extremely broad host range and infects a variety of crops, is the prototype species in this family (1). The *Fimoviridae* are characterized by enveloped virions that harbor four- to eight-component genomes; fimovirids are transmitted by mites, which are tiny arachnid arthropods (38). Finally, most plant rhabdovirids possess monopartite genomes and are transmitted by the hemipteran insects, including leafhoppers, planthoppers, aphids (*Cytorhabdovirus* and *Nucleorhabdovirus*), and arachnid mites (*Dichorhavirus*) (29, 193).

The −RNA plant viruses are closely related to their animal relatives in virion and genome architectures except for encoding MPs and RNAi suppressors that are required for systemic infectivity in plants (92). There are, however, three taxa of plant −RNA viruses that depart from this paradigm to different degrees. The genus *Tenuivirus* (*Phenuiviridae*) is distinguished by segmented RNA genomes that are the largest among all known plant viruses (up to 8 segments totaling up to ∼25 Kb) (108, 184). Apparently, the tenuiviruses have lost the ancestral membrane envelopes and switched to using their filamentous nucleocapsids as virions (39). However, tenuiviruses retained nonstructural glycoproteins as helper components mediating the typical propagative mode of transmission by planthoppers (115, 118). Likewise, the envelopeless, rod-shaped virions of *Varicosavirus* members (*Rhabdoviridae*) are formed by nucleocapsid-like CPs. The varicosaviruses exhibit a further notable departure from the majority of arthropod-associated −RNA plant viruses in being transmitted by zoospores of soil fungi (193). In a similar manner, the aspivirids (previously classified as *Ophioviridae*) have also shed their envelopes and use nucleocapsids, albeit of unclear provenance, as fungus-transmitted virions (92, 159). Given the dominance of nonenveloped plant viruses, the evolution of these three virus taxa clearly reflects the adaptation of −RNA viruses to a plant-specific lifestyle.

Reverse-Transcribing Viruses

The second kingdom within the realm *Riboviria*, *Pararnavira*, consists of reverse-transcribing viruses encoding a RT, which is homologous to the RdRPs of RNA viruses. Hence, RNA viruses and reverse-transcribing viruses are assumed to have evolved from a common ancestor, warranting their classification within the same highest-level taxon (88). Among six officially recognized families within *Pararnavira*, plants host a share of *Metaviridae* and *Pseudoviridae* that encapsidate +RNA and the family *Caulimoviridae* (informally referred to as pararetroviruses) that encapsidate dsDNA. The metavirids, pseudovirids, and caulimovirids are classified into the phylum *Artverviricota*, class *Retraviricetes*, and order *Ortervirales* (**Figure 1**) (88, 95). All orterviruses share the replication module that consists of the RT and RNase H, a morphogenetic module (Gag polyprotein containing the characteristic α-helical CP and zinc-knuckle NC domains) and the polyprotein-processing aspartic protease (95, 99).

Similarly to vertebrate *Retroviridae*, metavirids and pseudovirids encode integrases and abundantly colonize genomes of diverse eukaryotes, plants being no exception (116, 117). However, unlike infectious retrovirids, for most metavirids and pseudovirids, infectivity and intercellular spread have not been described, despite the conservation of the gene encoding structural Gag polyprotein and occasional presence (including in plant viruses) of genes encoding putative envelope proteins responsible for virus entry (105, 120, 196). Thus, metavirids and pseudovirids have been historically considered transposable elements and are also known as long terminal repeat (LTR) retrotransposons of the Ty3/Gypsy and Ty1/Copia families, respectively (116). Accordingly, the majority of the identified LTR retrotransposons have not been included in the ICTV framework, rendering the genus-level classification of these viruses incomplete (139).

Recent analysis of 80 plant genomes resulted in the identification of nearly 14,000 metavirids and pseudovirids (139). Both families are represented in all major groups of green plants, including the basal *Chlorophyta*, suggesting that both were present in the *Viridiplantae* genomes since their origin approximately 700–1,500 million years ago (27, 117, 139, 148). Neither plant metavirids nor pseudovirids encode recognizable MPs (139), suggesting that these viruses do not move between cells. Despite the lack of detectable infectious particles, widespread and frequent horizontal transfer of metavirids/pseudovirids in plants has been reported (37). Although the involved mechanisms remain unknown, high similarity between some fungal and plant metavirids suggests that plant-pathogenic fungi might participate in their horizontal dissemination (147).

The caulimovirids share the replication and morphogenetic modules with metavirids and pseudovirids (**Figure 1**) but lead a radically different lifestyle: They encapsidate circular dsDNA genomes and form infectious, isometric, or bacilliform nonenveloped virions (68). Phylogenetic analysis of the RT suggests that caulimovirids share the most recent common ancestor with metavirids (95). Similar to metavirids and pseudovirids, the basal caulimovirids, such as Petunia vein clearing virus, express all proteins as a single polyprotein, which is subsequently processed by the virus-encoded protease (158). Unlike the other orterviruses, replication of caulimovirids does not depend on integration into the host chromosome. Even though most caulimovirids do not encode an integrase, caulimovirus-derived endogenous virus elements (EVEs) are widespread in plant genomes and are thought to be integrated through nonhomologous end-joining during DNA repair (16, 45). Although most of these EVEs are inactive, some are infectious upon reactivation by various stress factors (44, 157).

Similar to many other plant viruses, caulimovirids encode a movement protein of the 30K superfamily (130) and are insect transmissible in a genus-specific manner by aphids, mealybugs, or leafhoppers. The mechanism of transmission involves two virus helper factors that bridge virions to the specific receptor at the tip of an insect's stylet (10, 192). Notably, the reactivation-competent endogenous petuniavirus has no known insect vectors, further suggesting that it is a living intermediate between the metavirids and more complex caulimovirids. Thus, evolution of caulimovirids from a metavirus-like ancestor likely involved the loss of the integrase gene and acquisition of the MP and vector transmission factors.

ssDNA Viruses

Viruses with ssDNA genomes encoding rolling-circle replication endonucleases (RCRE) of the HUH superfamily are classified into the realm *Monodnaviria*, which currently encompasses six phyla (88). The phylum *Cressdnaviricota* includes all eukaryotic viruses with circular ssDNA genomes that encode homologous replicases (Reps) containing the N-terminal HUH endonuclease and C-terminal S3H domains (94, 102, 202). This phylum unifies seven virus families and a vast number of viruses discovered by metagenomics that are affiliated tentatively (clades CRESSV1–6) (77). All plant viruses of this realm fall into families *Geminiviridae* and *Nanoviridae* within the phylum *Cressdnaviricota* (**Figure 2**).

Most members of *Cressdnaviricota* have extremely small virus genomes (~2 Kb) encoding only two proteins, Rep and CP, whereas plant viruses in this phylum encode MP and RNAi suppressor in addition to Rep and CP. The virions of geminivirids have unique morphology of twinned (geminate) icosahedra encapsidating mono- or bipartite genomes (199). These virions are transmitted by insects (whiteflies, aphids, leafhoppers) in a genus-specific manner, via a circulative, nonpropagative mechanism similar to that of +RNA luteovirids (192, 199).

By contrast, the genomes of nanovirids are partitioned into 6–8 circular DNA molecules of ~1 Kb, each encoding a single protein and separately encapsidated into simple icosahedral virions (58). Remarkably, different genomic segments of nanovirids rarely co-occur in the same plant cell; instead, they individually accumulate in distinct cells so that virus reproduction is achieved by complementation, whereby the gene products are shuttled between the cells (175). Conversely, many nanovirus particles enter every susceptible aphid vector cell so that distinct genome segments always remain together (30). Furthermore, during circulative, nonpropagative aphid transmission, the frequencies of genome fragments change, implying more intimate relationships with the insect than simply passing from gut to salivary glands (176). Unlike geminivirids, in addition to virions, nanovirid transmission requires a helper component (57).

Both geminivirids and nanovirids are associated with diverse satellite nucleic acids, including alphasatellites (*Alphasatellitidae*), which encode their own Reps but not the CPs and thus depend on

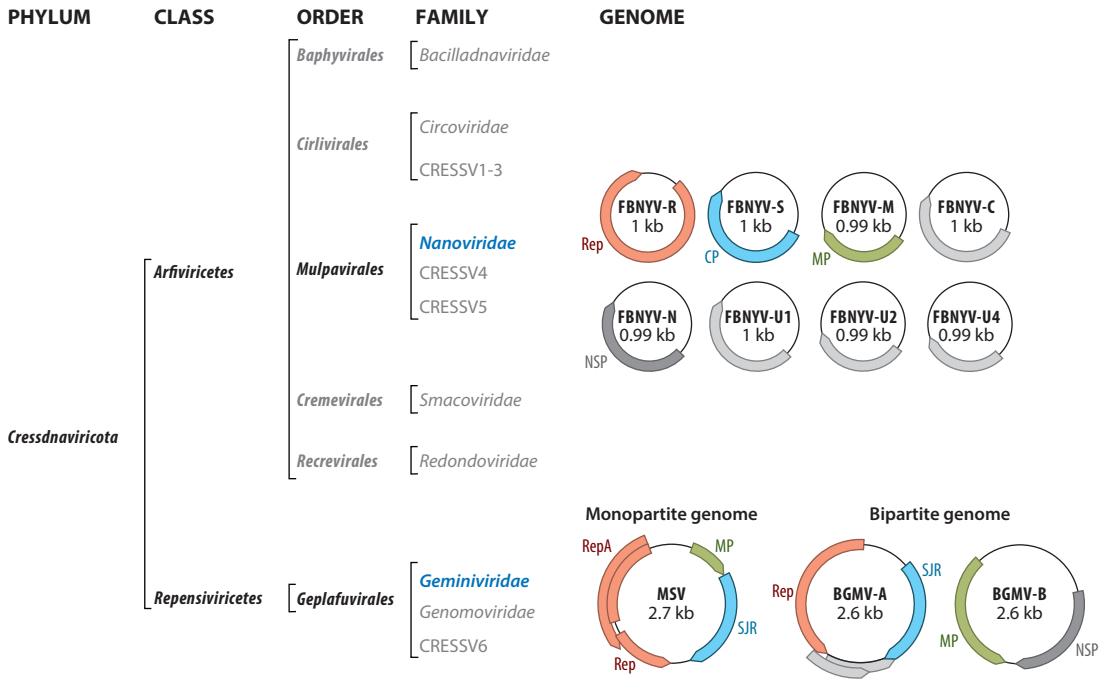

Figure 2

Hierarchical taxonomic structure of the phylum *Cressdnaviricota* of the ssDNA viruses. Taxa that do not contain plant viruses are indicated in gray font. Genome maps of selected viruses are shown on the right: FBNYV, Faba bean necrotic yellows virus (NC_003560, NC_003563, NC_003562, NC_003559, NC_003566, NC_003561, NC_003564, NC_024457); MSV, Maize streak virus (NC_001346); BGMV, Bean golden mosaic virus (NC_004042, NC_004043). Abbreviations: MP, movement protein; NSP, nuclear shuttle protein; Rep/RepA, rolling circle replication initiation protein; SJR, single jelly roll capsid protein.

the helper viruses for transmission (13). The alphasatellites evolved from the Rep-encoding components of the nanovirus genomes, whereas the second genomic component of bipartite geminiviruses (DNA-B) could have originated from a satellite nucleic acid of unknown provenance (135). Furthermore, the ultimate origin of eukaryotic *Cressdnaviricota* appears to be rooted in bacterial rolling-circle plasmids, and the diversity of these viruses apparently has been seeded on at least two independent occasions (76). Phylogenetic analysis indicates that the two classes in the phylum *Cressdnaviricota*, *Repensiviricetes* and *Arfiviricetes* (**Figure 2**), evolved from two subgroups of related bacterial plasmids (76), implying that nanovirids and geminivirids are not monophyletic. The transformation of a plasmid into a virus necessitated the acquisition of a CP-encoding gene. Comparison of the CP structures has shown that CPs of different taxa within *Cressdnaviricota* have closer homologs among +RNA viruses than among themselves. For instance, the CP of geminivirids is most closely related to that of Satellite tobacco necrosis virus (65, 101), whereas CPs of cruciviruses are most similar to CPs of tombusvirids (28, 167). Thus, eukaryotic ssDNA viruses appear to have evolved from plasmids through the acquisition of reverse-transcribed CP genes (potentially aided by RTs of endogenous reverse-transcribing viruses) from different groups of RNA viruses (76). In the case of plant ssDNA viruses, the CPs are at the forefront of virus interaction with the insect vectors. Accordingly, phylogenies of the geminivirid CPs mirror those of their vector species far more closely than those of the host species (109).

Remarkably, transformation of a plasmid into a virus is not a one-way street: Geminivirids have apparently given rise to plasmids of phytoplasma (phloem-parasitic bacteria) by losing the CP gene (76). Evolution of the ssDNA viruses is thus one of the most compelling manifestations of tight evolutionary connections between viruses and capsidless MGEs, which appears to be a general trend in virus evolution (97).

PLANT EVOLUTION SHAPES THE VIROME

The evolution of the green branch of the eukaryotic tree of life (Archeoplastida) included successive emergence of the rhodophytes (red algae), glaucophytes, and chlorophytes and streptophytes (green algae), lineages that split ~1 billion years ago, and finally, a monophyletic lineage of the land plants (Embryophytes) that appeared approximately 400 Mya (26, 27, 148). Apparently, the land plants evolved from the Zygnematophyceae branch of freshwater green algae, which acquired resistance to desiccation and shared wet terrestrial habitats with the earliest embryophytes, the bryophytes (hornworts, liverworts, and mosses) (18). The evolutionary sequence within the land plant branch that follows bryophytes includes lycophytes, ferns, gymnosperms (conifers), and, finally, angiosperms (flowering plants with two major lineages, monocots and eudicots). The angiosperms diversified greatly in the Early Cretaceous, 140–100 Mya, and flourished to dominate the terrestrial phytosphere as grasses, herbs, shrubs, and trees (178). Virtually all agricultural output consists of the flowering plants, from rice to potatoes to apples to oranges, whereas timber production is based on both gymnosperm and angiosperm trees, and all of the flowering plants host viruses.

What are the key evolutionary transitions in plant biology that are relevant to the formation of the contemporary virome of the land plants? One obvious consequence of terrestrialization is the switch from the aquatic to the soil/aerial lifestyle. From the virus perspective, this lifestyle change means losing the benefits of the aquatic environment, which protects viruses outside the infected host from desiccation and UV damage as well as promotes virus dissemination via diffusion, convection, and currents. By contrast, even in the wet soil environment, passive transmission of viruses between root systems of the host plants is very inefficient.

The land plant anatomy and cell architecture pose another set of limitations for plant-to-plant transmission of viruses, be it leaving the infected plant or entering a new one. The first barrier to virus penetration is the epidermal cuticle, a layer of insoluble lipid polymers, such as polyester impregnated with hydrophobic waxes (144). There is simply no way for a virus to penetrate the undamaged cuticle except through open stomata formed by guard cells and functioning in gas exchange (53). Even if a virus manages to sneak through stomata into the leaf parenchyma, it faces the thick and rigid cell walls made of crystalline cellulose and matrix polysaccharides (e.g., hemicelluloses and pectins). These cell walls are an ancient feature shared by green plants: Their composition is nearly identical in a lineage of charophycean algae and land plants (152).

The multicellularity and complex vascular anatomy, which originated in land plants independently of other organisms (26, 145), pose additional severe challenges to viruses. For successful infection followed by plant-to-plant transmission, a virus must be able to move from cell to cell and/or through the vascular tissue, a route lying through plasmodesmata interconnecting plant cells and tissues. The Zygnematophyceae ancestors of land plants are unicellular or simple filamentous algae that lack plasmodesmata. Therefore, these essential organelles evolved de novo to mediate intercellular communications in land plants (14). Because the plasmodesmatal channels are narrow and highly structured, they serve as checkpoints for smaller macromolecular complexes and do not allow free passage of virions.

Finally, at both the cellular and organismal levels, flowering plants possess potent innate immune responses to pathogens, including viruses (15, 72, 111). The most powerful antiviral acquired immune response in land plants is RNAi (6, 62). In brief, RNAi is based on the recognition of abnormal (highly structured and/or overexpressed) virus RNA, generation of small interfering RNAs (siRNAs) homologous to the virus genome, and inactivation of this genome by the siRNA-guided Argonaute effector complex (40, 164). Importantly, induction of RNAi in a single virus-infected plant cell triggers the amplification and systemic spread of the RNAi that follows or precedes the virus spread (126).

Although it is difficult to assess the exact contributions of the cuticle, cell wall, and plasmodesmata and soil–aerial lifestyle and immunity to limitations in plant virome composition, one outcome of these defenses is a strictly reinforced taboo: No bona fide dsDNA viruses are allowed in land plants. This is in contrast to Chlorophyte algae where phycodnaviridids with large dsDNA flourish (see below). Furthermore, integrated leftovers of distant phycodnavirid relatives are present in moss, implying that these fossilized viruses have infected algal ancestors of moss (124). The reason for the banishment of phycodnaviridids in land plants could be that plasmodesmata are impenetrable for the large virions or dsDNA. Unlike phycodnaviridids that break the cell walls via enzymatic digestion (177), none of the known viruses of land plants has this capacity.

However, what about small dsDNA viruses such as animal *Papillomaviridae* and *Polyomaviridae*? Both papillomavirids and plant reverse-transcribing caulimovirids encapsidate ~8-Kb circular dsDNA genomes into 40–55 nm icosahedral virions. The caulimovirids move cell to cell through tubules formed by virus MPs, implying that small, papillomavirid-like dsDNA viruses could have evolved a similar strategy. However, the host range of papillomavirids is limited to vertebrates, where they are highly host-species specific and tissue restricted. Thus, a potential explanation of their absence from plants could be that a virus transfer route from vertebrates to plants is lacking.

The obvious follow-up question is how the extant, non-dsDNA viruses managed to prosper in land plants using their limited genomic resources. One fundamental solution is to surrender infectivity, i.e., virus transmission between cells and plants, altogether. This solution is employed by cryptic viruses that cause no disease and survive by means of vertical transmission through seed and pollen. This low-profile lifestyle is characteristic of minimalist persistent viruses (mitovirids, partitivirids, and totivirids) (142, 143, 160). The persistent endornaviruses have larger genomes and appear to represent a transition from the infectious lifestyle of their ancestors in the *Alsuvericetes* lineage to persistence (43).

However, the majority of the known groups of plant viruses evolved a more radical strategy, using invertebrates, fungi or protists, for vector-assisted penetration into and transmission between plant cells and tissues. In particular, piercing–sucking insects deliver viruses by perforating the leaf cuticle and epidermal or phloem cell walls with their stylets (10, 140). Likewise, soil-dwelling ectoparasitic nematodes deliver stylet-borne viruses into root cells (42). The virus transfer by fungal or protist vectors into the root cells is achieved via encystment of zoospores that either absorb the virus on their surface or internalize it (159).

As mentioned above, some viruses, for example, TMV, eschew vector transmission and rely on stochastic mechanical transmission facilitated by the extreme environmental endurance of their virions. Another, relatively small subset of plant viruses takes advantage of both horizontal (vectors or mechanical) and vertical (seed or pollen) transmission, ensuring their long-term survival. This dual strategy is particularly important for viruses infecting annual hosts (63).

In the next section, we discuss the interplay between host and virus evolution that shaped the compositions of plant virus genomes and a dynamic contemporary plant virome, as well as underlying evolutionary scenarios for major plant virus lineages.

ORIGINS AND DIVERSIFICATION OF THE PLANT VIROME: HORIZONTAL VIRUS TRANSFER AND VIRUS–VECTOR ASSOCIATIONS

Phylogenomics is the foundation on which the virome evolution concepts and scenarios rest. For *Viridiplantae*, our ability to reconstruct the path of viromes across time and hosts requires adequate sampling. We know little about the viruses represented in most of the plant lineages (22, 129), with the exception of angiosperms because of their importance to agriculture. Hopefully, the availability of hundreds of transcriptomes covering the entire plant kingdom (Archaeplastida) will resolve these big questions (148).

Taking stock of the known viruses of alga is essential. In Rhodophytes, the presence of dsRNA totivirid-like entities was reported for two red macroalgal holobionts (103, 166); however, it is not clear whether these viruses reproduce in algae or associated fungi. Our literature search for viruses of glaucophytes yielded no hits.

The current insight into the virome of *Chlorophyta* is more advanced, and, so far, large dsDNA viruses of the family *Phycodnaviridae* are found in a variety of marine picoplankton and freshwater algae (185, 190). In addition, chlorophytes host small ssDNA viruses (8), a dsRNA reovirus (5), dsRNA partitivirus-like, capsidless replicons (83, 84), and, potentially, a few other dsRNA viruses (129).

We know discouragingly little about the virome of *Streptophytae* (*Charophyta*) algae that include zygnematophycean ancestors of land plants. Two very similar +RNA viruses related to benyvirids of the flowering plants have been identified in *Chara australis* and freshwater metaviromes in Canada (50, 188). In addition, three RdRPs apparently belonging to dsRNA viruses have been detected in algal transcriptomes (129). Although deeper sampling of the zygnematophycean virome is needed, it seems extremely unlikely that its diversity will ever approach that of the land plants. Indeed, the species richness, a key determinant of the virome diversity, is ~100 times lower in *Zygnematophycea* than in the vascular plants (61, 80). Therefore, it seems safe to assume that the algal ancestors of vascular plants could not harbor the seeds of all virus diversity represented in the extant land plants and rather served as a bottleneck in the virome evolution. Put another way, it appears likely that a substantial part, if not most, of the land plant virome was not inherited from the algal ancestors but was rather acquired via HVT from plant-associated organisms such as invertebrates, fungi, and protists (35).

As radical as this claim might seem, it finds strong support in the phylogenomic analysis of the rapidly growing data on the global virome and, in particular, the RNA viruses that are the dominant component of the plant virome (88). One of the early realizations that plant viruses might originate from viruses of arthropods was concerned with −RNA viruses (34, 92). Most of these viruses have a dual host range, i.e., they reproduce in both plants and vectors. Furthermore, the diversity of plant viruses is a subset of the arthropod virus diversity: In RdRp phylogenetic trees, plant-specific branches reside within a broader radiation of arthropod and arthropod/vertebrate viruses (110, 194). This same trend is prominent for both +RNA and dsRNA viruses in the phyla *Lenarviricota*, *Pisuviricota*, *Kitrinoviricota*, and *Duplornaviricota* (173, 174, 194).

In addition to RdRP phylogeny, the invertebrate-to-plant HVT scenario for RNA viruses is supported by general evolutionary considerations. Despite a considerable margin of uncertainty, the current consensus is that plants started to colonize land somewhat earlier than invertebrates, but together they proceeded to form a terrestrial ecosystem after ~500 Mya (79, 127, 165). At that time, invertebrates had already diversified greatly in the aquatic environments before and during the Cambrian explosion (195). Accordingly, most of the currently known large-scale RNA virus diversity was likely present in aquatic invertebrates such as mollusks and crustaceans (173) and

followed invertebrates to land. In contrast, vascular plants just started to emerge after ∼400 Mya, going through their own Early Cretacean explosion when angiosperms flourished merely 140–100 Mya (178). Therefore, compared to flowering plants, invertebrates had a few hundred million years' head start to evolve their vast RNA virome, bring it to land, and share it with evolving land plants.

The above scenario is also supported by partial analysis of the primitive plant transcriptomes, which showed that the diversity of +RNA virus RdRPs grew along with land plant evolution from mosses to angiosperms (129). Furthermore, virus MP gene transcripts were detected in lycophytes but not in the more ancient mosses, pointing to gradual virus adaptation to the growing plant complexity.

The composition of the extant biosphere is also in agreement with the evolutionary dominance of the invertebrate RNA virome, the diversity of which is roughly proportional to the hosts' species richness. The terrestrial arthropods alone account for ∼7,000,000 species, far exceeding all other land-dwelling eukaryotes combined (181), let alone vascular plants, which have only ∼300,000 species (80). Thus, plants are exposed to an enormous pool of invertebrate viruses that continuously sample the entire ecological space associated with the lifestyles of their hosts.

The dominant plant virus vectors are insects, particularly Hemiptera (aphids, whiteflies, mealybugs, leafhoppers, planthoppers), Thysanoptera (thrips), and Coleoptera (beetles), which transmit +RNA, dsRNA, −RNA, ssDNA, and pararetroviruses (192). In addition, mites of the *Arachnida* class of arthropods vector some of the +RNA and −RNA viruses (29, 47). Among the nonarthropod invertebrates, nematodes transmit +RNA secovirids (42).

As discussed above, the evolutionary scenario for the plant −RNA viruses seems to be the simplest: These viruses emerged from viruses of plant-feeding arthropods that acquired MPs and assorted RNAi suppressors via, presumably, recombination with preexisting plant viruses (**Figure 3**). Among these, plant tospovirids, fimovirids, and rhabdovirids (*Cytorhabdovirus* and *Nucleorhabdovirus* genera) appear to be the least plant specialized, possessing envelopes atypical for plant virome (17, 29, 38, 193). The tenuiviruses (*Phenuiviridae*) made a step toward plantness by losing envelopes and repurposing a membrane glycoprotein as an insect transmission factor (39, 118). Other envelopeless −RNA plant viruses, varicosaviruses (*Rhabdoviridae*) and aspivirids, are transmitted by soil-dwelling fungi, raising the possibility that their origin is via *trans*-kingdom HVT between fungi and plants (92).

The plant dsRNA reovirids also fit an insect-to-plant HVT scenario: They closely resemble their animal cousins (except for having acquired MPs and RNAi suppressors), reproduce in their hemipteran vectors, and reside within clades of insect reovirids (128, 189). Likewise, the +RNA kitavirids appear to descend from arthropod negeviruses to which they are phylogenetically closer than to any plant viruses of the class *Alsuviricetes*. Furthermore, kitavirids reproduce in mite vectors and are enveloped, similar to negeviruses, but not to other plant viruses in this class (107, 186).

In contrast, most of the remaining plant +RNA viruses appear to be much more host specialized, departing from their animal kin to different degrees. The case in point is *Alsuviricetes*, a class of the +RNA viruses that accounts for a large share of plant virome (**Figure 1**). Indeed, 9 of the 15 officially recognized families in this class are plant specific. Of these nine families, five harbor viruses with rod-shaped and filamentous virions that are formed by rCP and fCP, respectively, the two CP types historically believed to be plant-virus specific (33). That belief, however, predates the recent discovery of many viruses of this class in insects, chelicerates, myriapods, crustaceans, mollusks, and nematodes. Phylogenetic analysis of RdRPs of *Alsuviricetes* placed plant-specific virus families, such as *Bromoviridae*, *Closteroviridae*, and *Virgaviridae*, deep within the radiation of related invertebrate viruses, implying an ancestral relationship (173). Furthermore, the genomes of three invertebrate viruses (Behai charybdis crab virus 1 and insect Hubei virga-like viruses 2

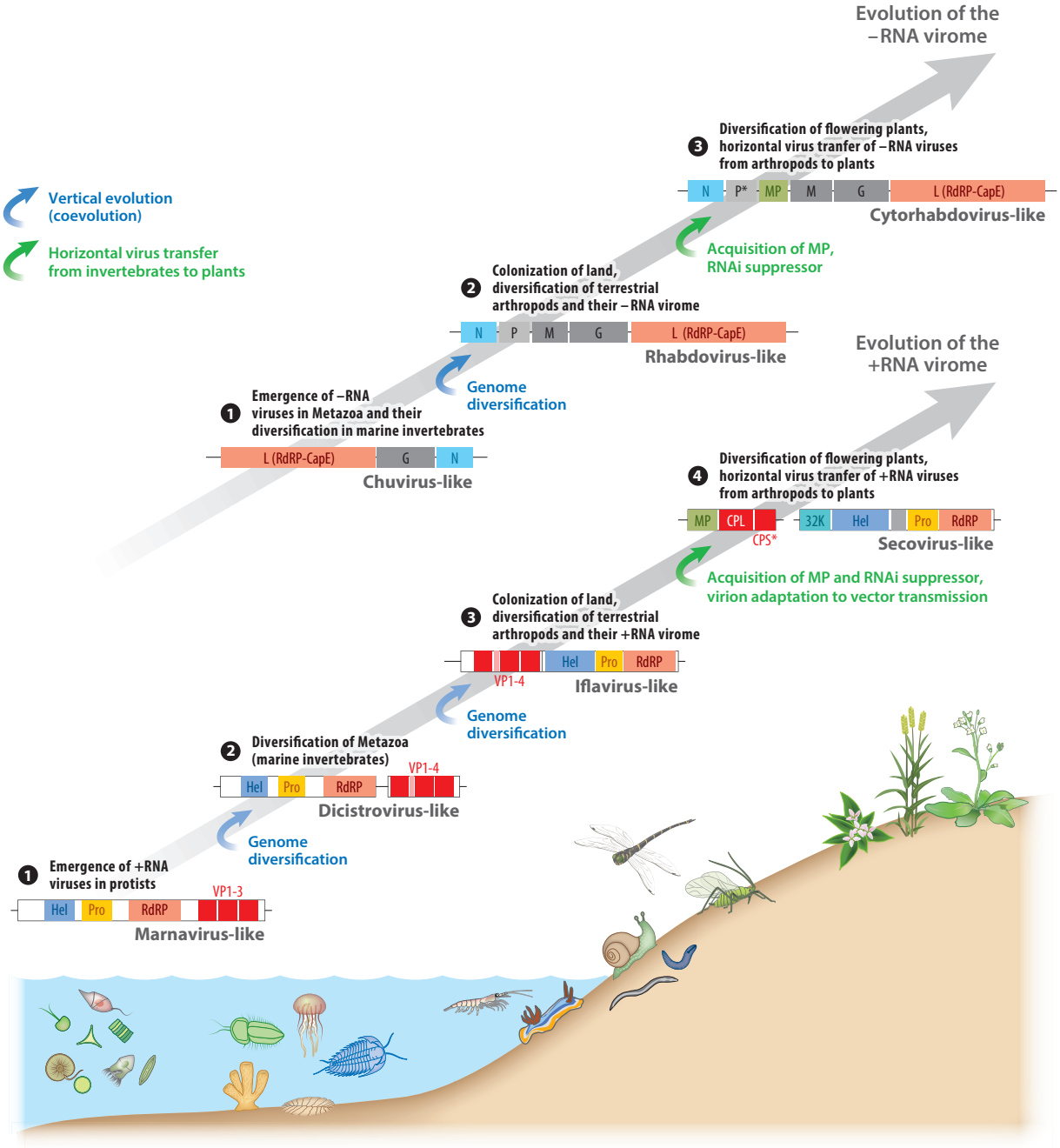

Figure 3

Evolutionary scenarios for the origin of two lineages of plant RNA viruses, cytorhabdovirids (*top*) and secovirids (*bottom*). Genome maps are not drawn to scale. Major evolutionary changes for each step are explained above the corresponding genome maps. Asterisks indicate gain of additional function—RNA interference suppression—by preexisting proteins. Abbreviations: 32K, 32-kDa protein; CapE, capping enzyme; CPL, large capsid proteins; CPS, small capsid proteins; G, glycoprotein; Hel, helicase; M, matrix protein; MP, movement protein; N, nucleocapsid; P, phosphoprotein; Pro, protease; RdRP, RNA-dependent RNA polymerase; RNAi, RNA interference; VP, capsid proteins.

and 9) contained the rCP-encoding genes (but no MP genes), supporting rCP origin within the invertebrate virome (173). The integrated copies of rCP genes were also excavated from several fly genomes, implying the long-term presence of rCP-coding viruses in insects (81). Along the same lines, the evolutionary origins of the fCP were traced to the −RNA viruses (2).

Furthermore, within the large order *Tymovirales*, plant and insect viruses intermix in the *Maculavirus* genus (173). Even more damning is a tymovirid genus of plant marafiviruses, which can reproduce in their insect vectors (67), likely an atavism going back to their arthropod-infecting ancestors. Collectively, phylogenomic analysis of the *Alsuviricetes* points to the evolutionary primacy of invertebrate viruses over their plant-infecting offspring.

This same trend of plant virus taxa nesting within virus RdRP phylogenetic trees of animal/invertebrate viruses is apparent in the classes *Alassoviricetes* (plant *Ourmiavirus*), *Pisoniviricetes* (plant *Secoviridae* and *Solemoviridae*) (**Figure 3**), and *Tolucaviricetes* (plant *Luteoviridae* and *Tombusviridae*) (173, 194).

A pertinent question concerning the nonpropagatively transmitted viruses discussed above is how they acquire vector transmissibility upon switching to plant-only reproduction mode. In many of these viruses, the virion is the only essential transmission determinant that is apparently responsible for the receptor binding in the vector stylet or midgut and/or for guiding virus from the arthropod gut to salivary glands in the case of circulative transmission (9, 54, 140). Given the high evolvability of the virus CPs, adaptation to the receptor binding appears to be a relatively low evolutionary barrier for viruses to cross. In addition, many plant viruses employ helper components/vector transmission factors, proteins that specifically function in bridging virions to vector receptors (10).

Although an invertebrate-to-plant HVT scenario seems to be prevalent in plant virome formation, distinct scenarios were also likely in action for several plant virus lineages. Thus, plant partitivirids in the *Duplopviricetes* class share the family with fungal viruses, implying possible HVT from fungi (162). The ancestry of the class *Stelpaviricetes*, which includes a single plant virus family, *Potyviridae*, is rather enigmatic, having no close relatives among animal viruses except the phylogenetic affinity with the astrovirid RdRP. Apart from the RdRP, potyvirids and astrovirids share only homologous trypsin-like proteases that do not appear to form a clade. In addition, the potyvirids encode fCP likely borrowed from other plant viruses, S2H related to those of flavivirids, and several other proteins with unclear evolutionary provenance attesting to a highly mosaic origin of the potyvirid genomes (49). It seems likely that the ancestors of potyvirids are lurking somewhere waiting to be found, a possibility supported by recent discovery of plant "plastroviruses," apparent evolutionary intermediates between astrovirids and potyvirids (106).

An intriguing nuance relevant to the enigma of potyvirid ancestry is offered by the potyvirids of the genus *Bymovirus*, which, along with benyvirids (*Alsuviricetes*), use plasmodiophorid protists as vectors (159). Because these viruses and their proteins were found inside spores, they are likely capable of reproducing within the vector cells (74, 119). Remarkably, plasmodiophorids and related phagomyxids are cosmopolitan eukaryotic parasites of diatoms, oomycetes, brown algae, and land plants and are prone to cross-kingdom shifts between these diverse hosts (138). Such unusual ecological mobility makes plasmodiophorids plausible vehicles of HVT from diverse aquatic protists to land plants, thus short-circuiting the need for invertebrate vectors. Indeed, diatoms and other protists are known to host a relatively diverse RNA virome that is considered ancestral to the vast RNA virome of invertebrates (35, 56).

Other striking departures from the invertebrate-to-plant HVT leitmotif are the evolutionary scenarios for ssDNA geminivirids and nanovirids, starting with two related but distinct bacterial plasmids and gradually evolving into plant-specific viruses (76). Their evolutionary paths included acquisition of distinct SJR-CPs from +RNA viruses followed by the virion adaptation for

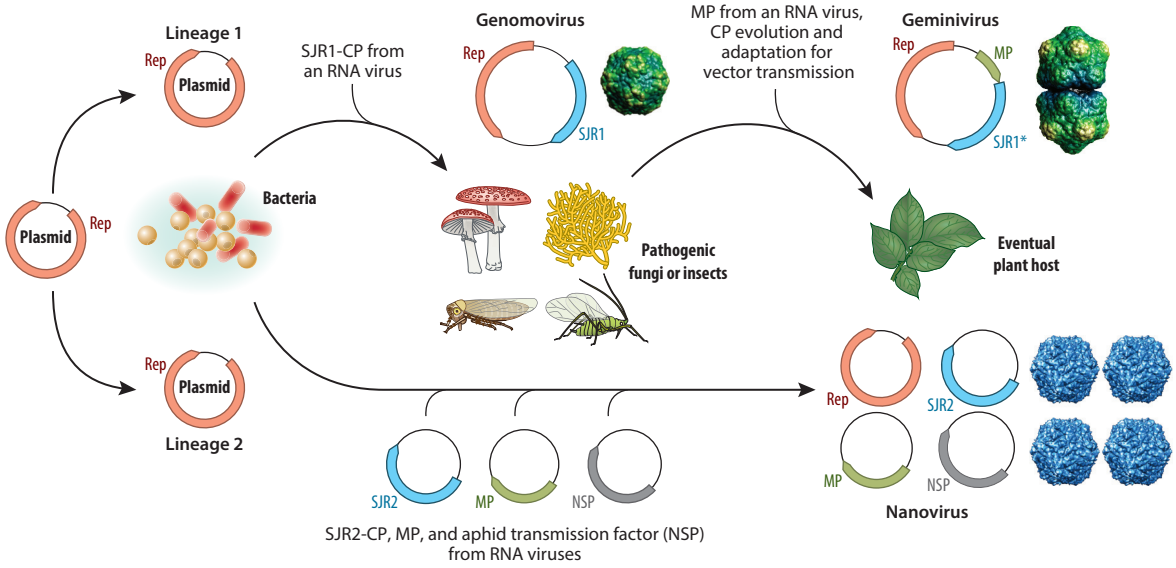

Figure 4

Evolutionary scenario for the origin of plant ssDNA viruses. Geminivirids (*top*) and nanovirids (*bottom*) have evolved from two lineages of related bacterial plasmids through acquisition of the genes encoding capsid proteins (SJR1-CP and SJR2-CP) from RNA viruses on two independent occasions. The evolution of geminivirids has likely proceeded through a genomovirid-like ancestor infecting plant-pathogenic fungi or insects. Abbreviations: CP, capsid protein; MP, movement protein; NSP, nuclear shuttle protein; Rep, rolling circle replication initiation protein; SJR, single jelly roll capsid protein.

circulative, nonpropagative insect transmission, capture of the MPs from pre-existing plant viruses, and, in the case of nanovirids, capture of the helper component from an unknown source (**Figure 4**).

Yet other evolutionary scenarios have been in action for the plant *Pararnavira*. In particular, the *Metaviridae* and *Pseudoviridae* are widely represented in diverse protists, fungi, and plants (95, 116). The wide spread of mostly noninfectious metavirids and pseudovirids (better known as LTR retrotransposons) across eukaryotes implies their presence in the last eukaryotic common ancestor (LECA), but their subsequent history involved extensive horizontal transfer, including between plants and fungi (37, 146). By contrast, *Caulimoviridae* are limited to plants and *Retroviridae* to animals, implying independent emergence from LTR retrotransposons (99). Thus, the evolution of the infectious lifestyle of caulimovirids in plants has involved acquisition of MP and helper components (10, 68) but no interkingdom HVT.

In conclusion, we need to consider the question of the ultimate origins of the genes defining the plant virus lifestyle, including MPs, vector transmission factors, and RNAi suppressors. Although there are examples of clear appropriation of the host proteins for virus transport (e.g., Hsp70 homolog of closterovirids) as well as some cases of likely duplication of virus genes (e.g., the S1H of the triple gene block movement module) (36, 187), the majority of the diverse MPs, including the ubiquitous TMV 300-kDa-like MPs, have no detectable homologs outside plant viruses (131). Likewise, no direct ancestors were identified for the virus transmission factors. It seems likely that these proteins evolved by exaptation of other host or virus proteins followed by rapid divergence erasing all traces of the ancestry. In the case of the MPs, the starting material could be the host genes encoding proteins that possess cell-to-cell trafficking and RNA-binding capacities, which are involved in plant development and antiparasite defense (114). Similar to the

extremely diverse MPs and transmission factors, virus RNAi suppressors are typically virus-family specific (23). Many of these proteins function by binding siRNAs and thus can be recruited from some of the numerous RNA-binding proteins available from host and virus genomes (19, 104). Alternatively, de novo evolution, which uses the recoding of preexisting genes or chimeric genes arising through recombination, is also a distinct possibility (31).

EVOLUTION OF THE OVERLAPPING RNA VIROMES OF PLANTS, FUNGI, AND ANIMALS

In this section, we present a brief overview of the relationships among the viromes of flowering plants, animals, and fungi. The discovery of related RNA viruses in plants and animals was a veritable sensation in the early days of virus genomics (3, 52, 73). Since then, it has become clear that such relationships are a recurrent pattern in virus evolution that can be explained by HVT, independent capture of homologous genes from hosts or other viruses, or long-term coevolution with the hosts. The latter scenario implies a highly diverse virome in the LECA, given that plants and opisthokonts (animals and fungi) share common ancestry only at a very early stage of eukaryotic evolution (78). Distinguishing between these alternatives with confidence is difficult because the inadequate virome sampling complicates assessment of the virus spread across the host taxa. Nevertheless, for some groups of viruses, by combining the information on the depth of mixing in phylogenetic trees, evolutionary scenarios, and the biology of the host relationships, the most plausible route of evolution becomes apparent.

As emphasized in the previous section, a pervasive phylogenetic blending of animal and plant viruses is observed among *Orthornavirae* (**Figure 1**), where the majority of the plant RNA virome has likely evolved through HVT from a much more diverse invertebrate virome (**Figure 5**).

A contrasting RNA virome evolution paradigm is apparent between invertebrates (all prechordate metazoa) and vertebrates. All major lineages of metazoa diversified in the Ediacaran era ~600 Mya (151). Furthermore, the jawed vertebrates that make up more than 99% of modern vertebrates started to diversify at least 420 Mya (11). Therefore, most of the animal lineages were in place before animal terrestrialization or diversification of the flowering plants. Importantly, all this animal diversity shared the marine habitat conducive to HVT where the early vertebrates were continuously sampled by invertebrate viruses, forming the emerging vertebrate virome, and vice versa.

Although the invertebrate virome appears to be much larger than that of vertebrates, all major *Orthornavirae* lineages (except for *Lenarviricota*) are present in both invertebrates and early aquatic jawed vertebrates (172, 173). The viromes of the successive lineages of the terrestrial vertebrates, amphibians, reptiles, birds, and mammals show strong signals of coevolution with their respective hosts reflected in monophyletic, host-specific clades in the RdRP phylogenetic trees (201). Thus, following plausible multidirectional HVT during diversification of animals in the marine environment, as well as more recent HVT events (e.g., from blood-sucking arthropods transmitting arboviruses) (9, 201), the evolutionary scenario for vertebrates has a major virus–host coevolution aspect (**Figure 5**).

The split between likely aquatic unicellular fungi and animals within the opisthokont supergroup occurred very early in eukaryote evolution (78). However, unlike marine animals that diversified greatly before coming to land, now-ubiquitous mycelial fungi flourished upon terrestrialization, reminiscent of the flowering plants (133, 180). According to the green scenario, land colonization involved association of fungi with the algal ancestors of land plants, accompanied by evolution of fungi capable of decomposing organic matter and cycling nutrients, resulting in a tightly knitted phytomycobiome. Therefore, the evolutionary success of fungi and plants on land

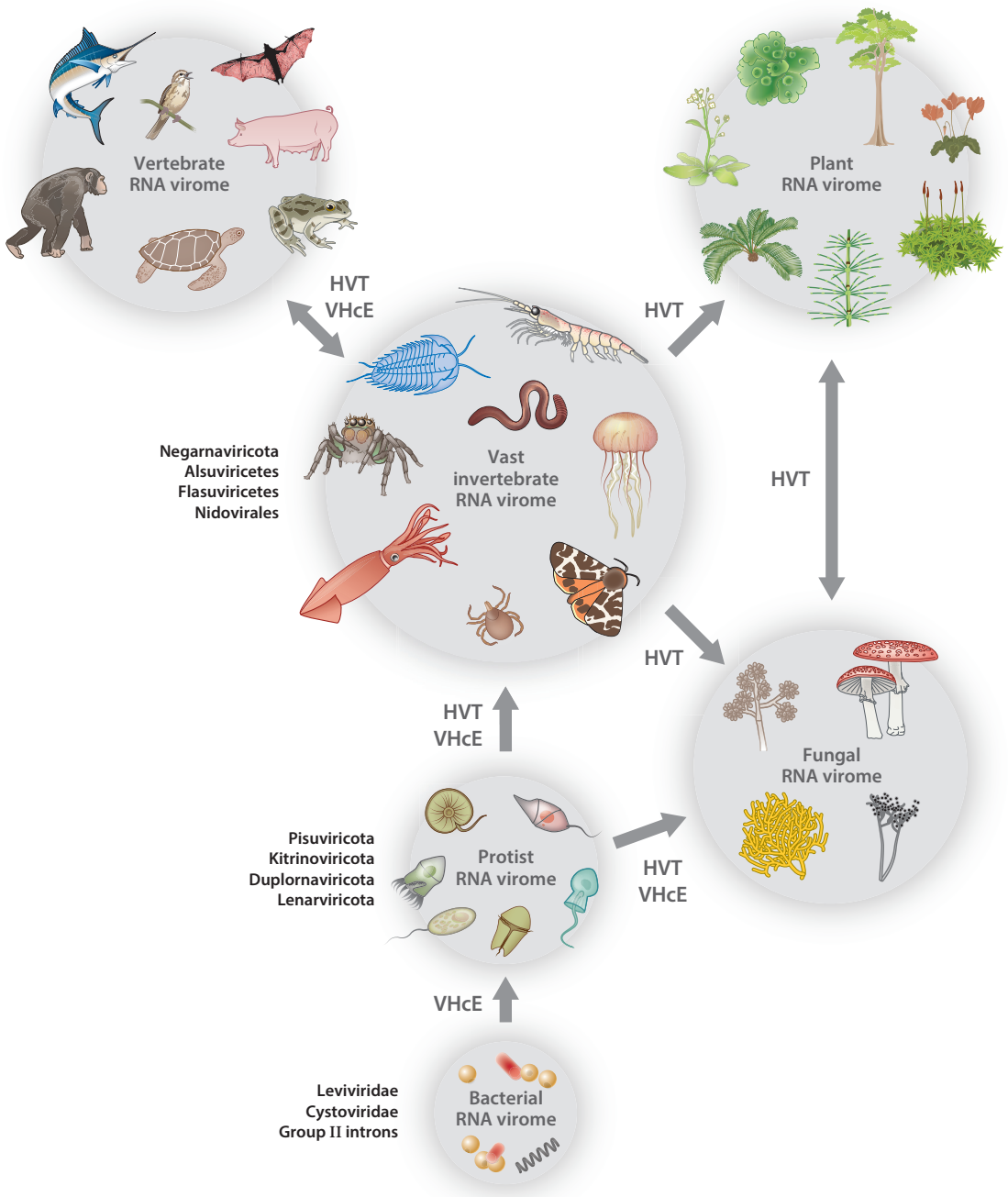

Figure 5

Hypothetical evolutionary pathways for the origin of the protist, fungal, invertebrate, vertebrate, and plant RNA viromes. Dominant mechanisms of the virus lineage macroevolution, including virus–host coevolution (VHcE) and cross-species horizontal virus transmission (HVT), are depicted by arrows. Major virus taxa that emerged in each type of the organisms are listed at the left. Potential HVT pathways from protists to vertebrates and plants are not shown for the sake of clarity.

was mutually assured. During the course of terrestrial evolution, fungi diversified their lifestyles from being plant parasites to being critical plant symbionts and saprophytes. In parallel, fungi evolved associations with other organisms, including metazoa (133).

So far, the virome of the presumed ancient lineages of marine fungi remains largely unexplored, whereas in the terrestrial fungi, most of the available data deal with plant-associated fungi (48, 122, 137). Despite these limitations, significant aspects of mycovirus diversity are apparent. One striking feature of the fungal virome shared with the plant virome is the complete absence of the dsDNA mycoviruses. The possible factors explaining this similarity include the fungal chitinous cell walls that block virus entry and the apparent lack of the virus-vectoring organisms that would surmount this barrier (35). Accordingly, the majority of the mycoviruses possess no extracellular infectivity and are transmitted vertically or through anastomosis (48). Another claim to originality is the unusual richness of the fungal virome in mycovirus-dominated dsRNA virus families in the classes *Chrymoviricetes* (totividrids, chrysovirids, quadrividrids, megabirnavirids) and *Duploviricetes* (partitivirids, amalgavirids). In addition, there is a propensity of fungi to hosting the capsidless viruses (mitovirids, narnavirids, amalgavirids, hypovirids, deltaflexivirids) (48, 86).

Despite the unexpected recent discoveries of the fungal −RNA mymonavirids (71) and unusual, extracellularly transmissible ssDNA genomovirid (98, 197), the rest of the fungal virome appears to be borrowed from fungus-associated organisms, often plants. Indeed, some mycovirus families are derived from plant viruses losing or repurposing their MPs and CPs in adaptation to fungal hosts (162). Thus, fungal *Deltaflexiviridae* and *Gammaflexiviridae* are derivatives of plant alpha- and betaflexivirids acquired via interkingdom HVT, whereas the presumed hypovirid ancestor is related to potyvirids (24, 48).

The plant-to-fungus HVT is, however, bidirectional, with plant-specific mitovirids and partitivirids likely resulting from fungus-to-plant HVT (**Figure 5**) (141, 142, 162). In the cases of botourmiavirids and endornavirids, the HVT direction is uncertain with present sampling. Many fungi are also associated with diverse animals (133), suggesting a strong potential for HVT between these organisms (**Figure 5**) (93, 113, 136).

A hypothetical coarse-grained network of some major HVT and vertical RNA virus–host co-evolution pathways is presented in **Figure 5**. This network starts with bacteria that possess two RNA virus families, *Leviviridae* and *Cystoviridae*, which spawned *Lenarviricota* and, potentially, *Duplornaviricota* lineages of eukaryotic viruses, respectively. It has also been suggested that RTs from bacterial group II introns might have evolved into RdRPs of RNA viruses (194). The next step in eukaryotic RNA virus diversification occurred in ancient protists, contemporary progenitors of which host a relatively diverse RNA virome that, in turn, could have seeded explosive RNA virus diversification in the invertebrates (35). In addition to expanding then existing *Riboviria* lineages, the new lineages of *Negarnaviricota*, *Alsuviricetes*, *Flasuviricetes*, and *Nidovirales* were apparently conceived at that time (**Figure 5**).

Following its inflation, the invertebrate RNA virome served as a vast pool from which viromes of land plants, fungi, and vertebrates have drawn generously (35, 162, 201). However preliminary, this network provides a cornerstone to a future complete picture of eukaryotic virus evolution achievable with comprehensive sampling of eukaryotes for viruses.

CONCLUDING REMARKS

The plant virome covers two of the four realms of viruses, with the dramatic exception of the two dsDNA virus realms. Most likely, the exclusion of the dsDNA viruses that dominate the algal virome during the early evolution of plants is due to physical constraints, i.e., the inability of large and even moderate-sized dsDNA to pass through the plasmodesmata. The lack of dsDNA viruses

in plants is compensated for by the enormous diversification of +RNA viruses. Phylogenomic analysis of plant viruses demonstrates extensive phylogenetic mixing between viruses within numerous groups of RNA viruses as well as reverse-transcribing viruses. The plant virome appears to have been shaped by the interplay of four major evolutionary processes: (*a*) inheritance of a relatively small set of RNA and reverse-transcribing viruses from the algal ancestor, (*b*) acquisition of diverse viruses via HVT from invertebrates and fungi, (*c*) de novo emergence of ssDNA viruses (geminivirids and nanovirids) via recombination between plasmids and pre-existing RNA viruses, and (*d*) within-plant diversification and adaptation of viruses acquired via each of the above three routes.

Although plant viruses have been studied since the late nineteenth century, until the past decade the investigation of the plant virome was almost entirely limited to viruses that cause diseases in model and economically important plants. The advances of metaviromics have changed this situation dramatically by expanding the virome and revealing the abundance of noninfectious, symptomless viruses, such as mitovirids, totivirids, partitivirids, and endornavirids. This rapid progress in the characterization of the viromes of plants and other eukaryotes stimulated in-depth phylogenomic studies that revealed the evolutionary trends outlined above and led to the creation of the all-encompassing megataxonomy of viruses. Despite these major developments, our current understanding of the viromes of plants and other groups of organisms is still based on sampling a small minority of the host diversity. However, representative sampling of the entire earth virome could be achievable within one to two decades, showing whether or not our current evolutionary reconstructions and megataxonomic schemes represent an accurate outline of the organization of the virus world.

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LITERATURE CITED

1. Adkins S. 2000. Tomato spotted wilt virus: positive steps towards negative success. *Mol. Plant Pathol.* 1:151–57
2. Agirrezabala X, Mendez-Lopez E, Lasso G, Sanchez-Pina MA, Aranda M, Valle M. 2015. The near-atomic cryoEM structure of a flexible filamentous plant virus shows homology of its coat protein with nucleoproteins of animal viruses. *eLife* 4:e11795
3. Ahlquist P, Strauss EG, Rice CM, Strauss JH, Haseloff J, Zimmern D. 1985. Sindbis virus proteins nsP1 and nsP2 contain homology to nonstructural proteins from several RNA plant viruses. *J. Virol.* 53:536–42
4. Atsumi G, Tomita R, Kobayashi K, Sekine KT. 2013. Prevalence and genetic diversity of an unusual virus associated with Kobu-sho disease of gentian in Japan. *J. Gen. Virol.* 94:2360–65
5. Attoui H, Jaafar FM, Belhouchet M, de Micco P, de Lamballerie X, Brussaard CP. 2006. *Micromonas pusilla* reovirus: a new member of the family Reoviridae assigned to a novel proposed genus (*Mimoreovirus*). *J. Gen. Virol.* 87:1375–83
6. Baulcombe D. 2004. RNA silencing in plants. *Nature* 431:356–63
7. Bekal S, Domier LL, Niblack TL, Lambert KN. 2011. Discovery and initial analysis of novel viral genomes in the soybean cyst nematode. *J. Gen. Virol.* 92:1870–79
8. Benites LF, Poulton N, Labadie K, Sieracki ME, Grimsley N, Piganeau G. 2019. Single cell ecogenomics reveals mating types of individual cells and ssDNA viral infections in the smallest photosynthetic eukaryotes. *Philos. Trans. R. Soc. Lond. B* 374:20190089
9. Blanc S, Gutierrez S. 2015. The specifics of vector transmission of arboviruses of vertebrates and plants. *Curr. Opin. Virol.* 15:27–33

10. Blanc S, Uzest M, Drucker M. 2011. New research horizons in vector-transmission of plant viruses. *Curr. Opin. Microbiol.* 14:483–91
11. Brazeau MD, Friedman M. 2015. The origin and early phylogenetic history of jawed vertebrates. *Nature* 520:490–97
12. Breitbart M, Bonnain C, Malki K, Sawaya NA. 2018. Phage puppet masters of the marine microbial realm. *Nat. Microbiol.* 3:754–66
13. Briddon RW, Martin DP, Roumagnac P, Navas-Castillo J, Fiallo-Olive E, et al. 2018. Alphasatellitidae: a new family with two subfamilies for the classification of geminivirus- and nanovirus-associated alphasatellites. *Arch. Virol.* 163:2587–600
14. Brunkard JO, Zambryski PC. 2016. Plasmodesmata enable multicellularity: new insights into their evolution, biogenesis, and functions in development and immunity. *Curr. Opin. Plant Biol.* 35:76–83
15. Carr JP, Murphy AM, Tungadi T, Yoon JY. 2019. Plant defense signals: players and pawns in plant-virus-vector interactions. *Plant Sci.* 279:87–95
16. Chabannes M, Iskra-Caruana ML. 2013. Endogenous pararetroviruses: a reservoir of virus infection in plants. *Curr. Opin. Virol.* 3:615–20
17. Chen Y, Dessau M, Rotenberg D, Rasmussen DA, Whitfield AE. 2019. Entry of bunyaviruses into plants and vectors. *Adv. Virus Res.* 104:65–96
18. Cheng S, Xian W, Fu Y, Marin B, Keller J, et al. 2019. Genomes of subaerial Zygnematophyceae provide insights into land plant evolution. *Cell* 179:1057–67.e14
19. Chiba M, Reed JC, Prokhnevsky AI, Chapman EJ, Mawassi M, et al. 2006. Diverse suppressors of RNA silencing enhance agroinfection by a viral replicon. *Virology* 346:7–14
20. Chow CE, Suttle CA. 2015. Biogeography of viruses in the sea. *Annu. Rev. Virol.* 2:41–66
21. Cobián Güemes AG, Youle M, Cantu VA, Felts B, Nulton J, Rohwer F. 2016. Viruses as winners in the game of life. *Annu. Rev. Virol.* 3:197–214
22. Coy SR, Gann ER, Pound HL, Short SM, Wilhelm SW. 2018. Viruses of eukaryotic algae: diversity, methods for detection, and future directions. *Viruses* 10(9):487
23. Csorba T, Kontra L, Burgyan J. 2015. Viral silencing suppressors: tools forged to fine-tune host-pathogen coexistence. *Virology* 479–480:85–103
24. Dawe AL, Nuss DL. 2013. Hypovirus molecular biology: from Koch's postulates to host self-recognition genes that restrict virus transmission. *Adv. Virus Res.* 86:109–47
25. Dawson WO, Bar-Joseph M, Garnsey SM, Moreno P. 2015. *Citrus tristeza virus*: making an ally from an enemy. *Annu. Rev. Phytopathol.* 53:137–55
26. Delwiche CF, Cooper ED. 2015. The evolutionary origin of a terrestrial flora. *Curr. Biol.* 25:R899–910
27. de Vries J, Archibald JM. 2018. Plant evolution: landmarks on the path to terrestrial life. *New Phytol.* 217:1428–34
28. Diemer GS, Stedman KM. 2012. A novel virus genome discovered in an extreme environment suggests recombination between unrelated groups of RNA and DNA viruses. *Biol. Direct* 7:13
29. Dietzgen RG, Freitas-Astua J, Chabi-Jesus C, Ramos-Gonzalez PL, Goodin MM, et al. 2018. Dichorhaviruses in their host plants and mite vectors. *Adv. Virus Res.* 102:119–48
30. Di Mattia J, Vernerey MS, Yvon M, Pirolles E, Villegas M, et al. 2020. Route of a multipartite nanovirus across the body of its aphid vector. *J Virol.* 94:e01998-19
31. Dinman JD. 2012. Control of gene expression by translational recoding. *Adv. Protein Chem. Struct. Biol.* 86:129–49
32. Dolja VV. 2003. Beet yellows virus: the importance of being different. *Mol. Plant Pathol.* 4:91–98
33. Dolja VV, Boyko VP, Agranovsky AA, Koonin EV. 1991. Phylogeny of capsid proteins of rod-shaped and filamentous RNA plant viruses: two families with distinct patterns of sequence and probably structure conservation. *Virology* 184:79–86
34. Dolja VV, Koonin EV. 2011. Common origins and host-dependent diversity of plant and animal viromes. *Curr. Opin. Virol.* 1:322–31
35. Dolja VV, Koonin EV. 2018. Metagenomics reshapes the concepts of RNA virus evolution by revealing extensive horizontal virus transfer. *Virus Res.* 244:36–52
36. Dolja VV, Kreuze JF, Valkonen JP. 2006. Comparative and functional genomics of closteroviruses. *Virus Res.* 117:38–51

37. El Baidouri M, Carpentier MC, Cooke R, Gao D, Lasserre E, et al. 2014. Widespread and frequent horizontal transfers of transposable elements in plants. *Genome Res.* 24:831–38
38. Elbeaino T, Digiaro M, Mielke-Ehret N, Muehlbach HP, Martelli GP, ICTV Rep. Consort. 2018. ICTV virus taxonomy profile: Fimoviridae. *J. Gen. Virol.* 99:1478–79
39. Falk BW, Tsai JH. 1998. Biology and molecular biology of viruses in the genus *Tenuivirus*. *Annu. Rev. Phytopathol.* 36:139–63
40. Fang X, Qi Y. 2016. RNAi in plants: an argonaute-centered view. *Plant Cell* 28:272–85
41. Folimonova SY, Tilsner J. 2018. Hitchhikers, highway tolls and roadworks: the interactions of plant viruses with the phloem. *Curr. Opin. Plant Biol.* 43:82–88
42. Fuchs M, Schmitt-Keichinger C, Sanfaçon H. 2017. A renaissance in nepovirus research provides new insights into their molecular interface with hosts and vectors. *Adv. Virus Res.* 97:61–105
43. Fukuhara T. 2019. Endornaviruses: persistent dsRNA viruses with symbiotic properties in diverse eukaryotes. *Virus Genes* 55:165–73
44. Gayral P, Noa-Carrazana JC, Lescot M, Lheureux F, Lockhart BE, et al. 2008. A single Banana streak virus integration event in the banana genome as the origin of infectious endogenous pararetrovirus. *J. Virol.* 82:6697–710
45. Geering AD, Maumus F, Copetti D, Choisne N, Zwickl DJ, et al. 2014. Endogenous florendoviruses are major components of plant genomes and hallmarks of virus evolution. *Nat. Commun.* 5:5269
46. Geoghegan JL, Holmes EC. 2018. Evolutionary virology at 40. *Genetics* 210:1151–62
47. German TL, Lorenzen MD, Grubbs N, Whitefield AE. 2020. New technologies for studying negative-strand RNA viruses in plant and arthropod hosts. *Mol. Plant-Microbe Interact.* 33:382–93
48. Ghabrial SA, Caston JR, Jiang D, Nibert ML, Suzuki N. 2015. 50-plus years of fungal viruses. *Virology* 479–480:356–68
49. Gibbs AJ, Hajizadeh M, Ohshima K, Jones RAC. 2020. The potyviruses: an evolutionary synthesis is emerging. *Viruses* 12:E132
50. Gibbs AJ, Torronen M, Mackenzie AM, Wood JT 2nd, Armstrong JS, et al. 2011. The enigmatic genome of *Chara australis* virus. *J. Gen. Virol.* 92:2679–90
51. Gilmer D, Ratti C, ICTV Rep. Consort. 2017. ICTV virus taxonomy profile: Benyviridae. *J. Gen. Virol.* 98:1571–72
52. Goldbach R, Wellink J. 1988. Evolution of plus-strand RNA viruses. *Intervirology* 29:260–67
53. Granot D, Kelly G. 2019. Evolution of guard-cell theories: the story of sugars. *Trends Plant Sci.* 24:507–18
54. Gray S, Gildow FE. 2003. Luteovirus-aphid interactions. *Annu. Rev. Phytopathol.* 41:539–66
55. Gregory AC, Zayed AA, Conceicao-Neto N, Temperton B, Bolduc B, et al. 2019. Marine DNA viral macro- and microdiversity from pole to pole. *Cell* 177:1109–23.e14
56. Greninger AL. 2018. A decade of RNA virus metagenomics is (not) enough. *Virus Res.* 244:218–29
57. Grigoras I, Vetten HJ, Commandeur U, Ziebell H, Gronenborn B, Timchenko T. 2018. Nanovirus DNA-N encodes a protein mandatory for aphid transmission. *Virology* 522:281–91
58. Gronenborn B. 2004. Nanoviruses: genome organisation and protein function. *Vet. Microbiol.* 98:103–9
59. Grybchuk D, Akopyants NS, Kostygov AY, Konovalovas A, Lye LF, et al. 2018. Viral discovery and diversity in trypanosomatid protozoa with a focus on relatives of the human parasite *Leishmania*. *PNAS* 115:E506–15
60. Grybchuk D, Kostygov AY, Macedo DH, d'Avila-Levy CM, Yurchenko V. 2018. RNA viruses in trypanosomatid parasites: a historical overview. *Mem. Inst. Oswaldo Cruz* 113:e170487
61. Guiry MD. 2012. How many species of algae are there? *J. Phycol.* 48:1057–63
62. Guo Z, Li Y, Ding SW. 2019. Small RNA-based antimicrobial immunity. *Nat. Rev. Immunol.* 19:31–44
63. Hamelin FM, Allen LJ, Prendeville HR, Hajimorad MR, Jeger MJ. 2016. The evolution of plant virus transmission pathways. *J. Theor. Biol.* 396:75–89
64. Heinlein M. 2015. Plant virus replication and movement. *Virology* 479–480:657–71
65. Hesketh EL, Saunders K, Fisher C, Potze J, Stanley J, et al. 2018. The 3.3 Å structure of a plant geminivirus using cryo-EM. *Nat. Commun.* 9:2369
66. Hillman BI, Cai G. 2013. The family narnaviridae: simplest of RNA viruses. *Adv. Virus Res.* 86:149–76

67. Hogenhout SA, Ammar E-D, Whitfield AE, Redinbaugh MG. 2008. Insect vector interactions with persistently transmitted viruses. *Annu. Rev. Phytopathol.* 46:327–59
68. Hohn T, Rothnie H. 2013. Plant pararetroviruses: replication and expression. *Curr. Opin. Virol.* 3:621–28
69. Iranzo J, Koonin EV, Prangishvili D, Krupovic M. 2016. Bipartite network analysis of the archaeal virosphere: evolutionary connections between viruses and capsidless mobile elements. *J. Virol.* 90:11043–55
70. Iranzo J, Krupovic M, Koonin EV. 2016. The double-stranded DNA virosphere as a modular hierarchical network of gene sharing. *mnBio* 7:e00978-16
71. Jiang D, Ayllon MA, Marzano SL, ICTV Rep. Consort. 2019. ICTV virus taxonomy profile: Mymonaviridae. *J. Gen. Virol.* 100:1343–44
72. Jones JD, Vance RE, Dangl JL. 2016. Intracellular innate immune surveillance devices in plants and animals. *Science* 354:aaf6395
73. Kamer G, Argos P. 1984. Primary structural comparison of RNA-dependent polymerases from plant, animal and bacterial viruses. *Nucleic Acids Res.* 12:7269–82
74. Kanyuka K, Ward E, Adams MJ. 2003. *Polymyxa graminis* and the cereal viruses it transmits: a research challenge. *Mol. Plant Pathol.* 4:393–406
75. Karasev AV. 2000. Genetic diversity and evolution of closteroviruses. *Annu. Rev. Phytopathol.* 38:293–324
76. Kazlauskas D, Varsani A, Koonin EV, Krupovic M. 2019. Multiple origins of prokaryotic and eukaryotic single-stranded DNA viruses from bacterial and archaeal plasmids. *Nat. Commun.* 10:3425
77. Kazlauskas D, Varsani A, Krupovic M. 2018. Pervasive chimerism in the replication-associated proteins of uncultured single-stranded DNA viruses. *Viruses* 10:E187
78. Keeling PJ, Burki F. 2019. Progress towards the tree of eukaryotes. *Curr. Biol.* 29:R808–17
79. Kenrick P, Wellman CH, Schneider H, Edgecombe GD. 2012. A timeline for terrestrialization: consequences for the carbon cycle in the Palaeozoic. *Philos. Trans. R. Soc. Lond. B* 367:519–36
80. Kew Bot. Gard. 2017. *State of the World's Plants*, *2017*. London: SFUMATO Found. **https://stateoftheworldsplants.org/2017/report/SOTWP_2017.pdf**
81. Kirsip H, Abroi A. 2019. Protein structure-guided hidden Markov models (HMMs) as a powerful method in the detection of ancestral endogenous viral elements. *Viruses* 11:E320
82. Kobayashi K, Atsumi G, Iwadate Y, Tomita R, Chiba K, et al. 2013. Gentian Kobu-sho-associated virus: a tentative, novel double-stranded RNA virus that is relevant to gentian Kobu-sho syndrome. *J. Gen. Plant Pathol.* 79:56–63
83. Koga R, Fukuhara T, Nitta T. 1998. Molecular characterization of a single mitochondria-associated double-stranded RNA in the green alga *Bryopsis*. *Plant Mol. Biol.* 36:717–24
84. Koga R, Horiuchi H, Fukuhara T. 2003. Double-stranded RNA replicons associated with chloroplasts of a green alga, *Bryopsis cinicola*. *Plant Mol. Biol.* 51:991–99
85. Koonin EV, Dolja VV. 1993. Evolution and taxonomy of positive-strand RNA viruses: implications of comparative analysis of amino acid sequences. *Crit. Rev. Biochem. Mol. Biol.* 28:375–430
86. Koonin EV, Dolja VV. 2014. Virus world as an evolutionary network of viruses and capsidless selfish elements. *Microbiol. Mol. Biol. Rev.* 78:278–303
87. Koonin EV, Dolja VV, Krupovic M. 2015. Origins and evolution of viruses of eukaryotes: the ultimate modularity. *Virology* 479–480:2–25
88. Koonin EV, Dolja VV, Krupovic M, Varsani A, Wolf YI, et al. 2020. Global organization and proposed megataxonomy of the virus world. *Microbiol. Mol. Biol. Rev.* 84(2):e00061-19
89. Koonin EV, Krupovic M. 2018. The depths of virus exaptation. *Curr. Opin. Virol.* 31:1–8
90. Koonin EV, Wolf YI, Katsnelson MI. 2017. Inevitability of the emergence and persistence of genetic parasites caused by evolutionary instability of parasite-free states. *Biol. Direct* 12:31
91. Koonin EV, Wolf YI, Nagasaki K, Dolja VV. 2008. The Big Bang of picorna-like virus evolution antedates the radiation of eukaryotic supergroups. *Nat. Rev. Microbiol.* 6:925–39
92. Kormelink R, Garcia ML, Goodin M, Sasaya T, Haenni AL. 2011. Negative-strand RNA viruses: the plant-infecting counterparts. *Virus Res.* 162:184–202
93. Kotta-Loizou I, Coutts RH. 2017. Studies on the virome of the entomopathogenic fungus *Beauveria bassiana* reveal novel dsRNA elements and mild hypervirulence. *PLOS Pathog.* 13:e1006183
94. Krupovic M. 2013. Networks of evolutionary interactions underlying the polyphyletic origin of ssDNA viruses. *Curr. Opin. Virol.* 3:578–86

95. Krupovic M, Blomberg J, Coffin JM, Dasgupta I, Fan H, et al. 2018. *Ortervirales*: a new viral order unifying five families of reverse-transcribing viruses. *J. Virol.* 92(12):e00515-18
96. Krupovic M, Dolja VV, Koonin EV. 2015. Plant viruses of the Amalgaviridae family evolved via recombination between viruses with double-stranded and negative-strand RNA genomes. *Biol. Direct* 10:12
97. Krupovic M, Dolja VV, Koonin EV. 2019. Origin of viruses: primordial replicators recruiting capsids from hosts. *Nat. Rev. Microbiol.* 17:449–58
98. Krupovic M, Ghabrial SA, Jiang D, Varsani A. 2016. Genomoviridae: a new family of widespread single-stranded DNA viruses. *Arch. Virol.* 161:2633–43
99. Krupovic M, Koonin EV. 2017. Homologous capsid proteins testify to the common ancestry of retroviruses, caulimoviruses, pseudoviruses, and metaviruses. *J. Virol.* 91(12):e00210-17
100. Krupovic M, Koonin EV. 2017. Multiple origins of viral capsid proteins from cellular ancestors. *PNAS* 114:E2401–10
101. Krupovic M, Ravantti JJ, Bamford DH. 2009. Geminiviruses: a tale of a plasmid becoming a virus. *BMC Evol. Biol.* 9:112
102. Krupovic M, Varsani A, Kazlauskas D, Breitbart M, Delwart E, et al. 2020. *Cressdnaviricota*: a virus phylum unifying 7 families of Rep-encoding viruses with single-stranded, circular DNA genomes. *J. Virol.* **https://doi.org/10.1128/JVI.00582-20**
103. Lachnit T, Thomas T, Steinberg P. 2016. Expanding our understanding of the seaweed holobiont: RNA viruses of the red alga *Delisea pulchra*. *Front. Microbiol.* 6:1489
104. Lakatos L, Csorba T, Pantaleo V, Chapman EJ, Carrington JC, et al. 2006. Small RNA binding is a common strategy to suppress RNA silencing by several viral suppressors. *EMBO J.* 25:2768–80
105. Laten HM, Majumdar A, Gaucher EA. 1998. SIRE-1, a copia/Ty1-like retroelement from soybean, encodes a retroviral envelope-like protein. *PNAS* 95:6897–902
106. Lauber C, Seifert M, Bartenschlager R, Seitz S. 2019. Discovery of highly divergent lineages of plant-associated astro-like viruses sheds light on the emergence of potyviruses. *Virus Res.* 260:38–48
107. Leastro MO, Kitajima EW, Silva MS, Resende RO, Freitas-Astua J. 2018. Dissecting the subcellular localization, intracellular trafficking, interactions, membrane association, and topology of citrus leprosis virus C proteins. *Front. Plant Sci.* 9:1299
108. Lecoq H, Wipf-Scheibel C, Verdin E, Desbiez C. 2019. Characterization of the first tenuivirus naturally infecting dicotyledonous plants. *Arch. Virol.* 164:297–301
109. Lefeuvre P, Martin DP, Elena SF, Shepherd DN, Roumagnac P, Varsani A. 2019. Evolution and ecology of plant viruses. *Nat. Rev. Microbiol.* 17:632–44
110. Li CX, Shi M, Tian JH, Lin XD, Kang YJ, et al. 2015. Unprecedented genomic diversity of RNA viruses in arthropods reveals the ancestry of negative-sense RNA viruses. *eLife* 4:e05378
111. Li F, Wang A. 2019. RNA-targeted antiviral immunity: more than just RNA silencing. *Trends Microbiol.* 27:792–805
112. Lin YH, Fujita M, Chiba S, Hyodo K, Andika IB, et al. 2019. Two novel fungal negative-strand RNA viruses related to mymonaviruses and phenuiviruses in the shiitake mushroom (*Lentinula edodes*). *Virology* 533:125–36
113. Liu H, Fu Y, Jiang D, Li G, Xie J, et al. 2009. A novel mycovirus that is related to the human pathogen hepatitis E virus and rubi-like viruses. *J. Virol.* 83:1981–91
114. Liu L, Chen X. 2018. Intercellular and systemic trafficking of RNAs in plants. *Nat. Plants* 4:869–78
115. Liu W, Hajano JU, Wang X. 2018. New insights on the transmission mechanism of tenuiviruses by their vector insects. *Curr. Opin. Virol.* 33:13–17
116. Llorens C, Futami R, Covelli L, Dominguez-Escriba L, Viu JM, et al. 2011. The Gypsy Database (GyDB) of mobile genetic elements: release 2.0. *Nucleic Acids Res.* 39:D70–74
117. Llorens C, Munoz-Pomer A, Bernad L, Botella H, Moya A. 2009. Network dynamics of eukaryotic LTR retroelements beyond phylogenetic trees. *Biol. Direct* 4:41
118. Lu G, Li S, Zhou C, Qian X, Xiang Q, et al. 2019. Tenuivirus utilizes its glycoprotein as a helper component to overcome insect midgut barriers for its circulative and propagative transmission. *PLOS Pathog.* 15:e1007655
119. Lubicz JV, Rush CM, Payton M, Colberg T. 2007. Beet necrotic yellow vein virus accumulates inside resting spores and zoosporangia of its vector *Polymyxa betae* BNYVV infects *P. betae*. *Virol. J.* 4:37

120. Malik HS, Henikoff S, Eickbush TH. 2000. Poised for contagion: evolutionary origins of the infectious abilities of invertebrate retroviruses. *Genome Res.* 10:1307–18
121. Martelli GP, Adams MJ, Kreuze JF, Dolja VV. 2007. Family Flexiviridae: a case study in virion and genome plasticity. *Annu. Rev. Phytopathol.* 45:73–100
122. Marzano SY, Nelson BD, Ajayi-Oyetunde O, Bradley CA, Hughes TJ, et al. 2016. Identification of diverse mycoviruses through metatranscriptomics characterization of the viromes of five major fungal plant pathogens. *J. Virol.* 90:6846–63
123. Mata CP, Luque D, Gomez-Blanco J, Rodriguez JM, Gonzalez JM, et al. 2017. Acquisition of functions on the outer capsid surface during evolution of double-stranded RNA fungal viruses. *PLOS Pathog.* 13:e1006755
124. Maumus F, Epert A, Nogue F, Blanc G. 2014. Plant genomes enclose footprints of past infections by giant virus relatives. *Nat. Commun.* 5:4268
125. McLeish MJ, Fraile A, García-Arenal F. 2019. Evolution of plant-virus interactions: host range and virus emergence. *Curr. Opin. Virol.* 34:50–55
126. Melnyk CW, Molnar A, Baulcombe DC. 2011. Intercellular and systemic movement of RNA silencing signals. *EMBO J.* 30:3553–63
127. Misof B, Liu S, Meusemann K, Peters RS, Donath A, et al. 2014. Phylogenomics resolves the timing and pattern of insect evolution. *Science* 346:763–67
128. Miyazaki N, Nakagawa A, Iwasaki K. 2013. Life cycle of phytoreoviruses visualized by electron microscopy and tomography. *Front. Microbiol.* 4:306
129. Mushegian A, Shipunov A, Elena SF. 2016. Changes in the composition of the RNA virome mark evolutionary transitions in green plants. *BMC Biol.* 14:68
130. Mushegian AR, Elena SF. 2015. Evolution of plant virus movement proteins from the 30K superfamily and of their homologs integrated in plant genomes. *Virology* 476:304–15
131. Mushegian AR, Koonin EV. 1993. Cell-to-cell movement of plant viruses. Insights from amino acid sequence comparisons of movement proteins and from analogies with cellular transport systems. *Arch. Virol.* 133:239–57
132. Napuli AJ, Alzhanova DV, Doneanu CE, Barofsky DF, Koonin EV, Dolja VV. 2003. The 64-kilodalton capsid protein homolog of Beet yellows virus is required for assembly of virion tails. *J. Virol.* 77:2377–84
133. Naranjo-Ortiz MA, Gabaldon T. 2019. Fungal evolution: major ecological adaptations and evolutionary transitions. *Biol. Rev. Camb. Philos. Soc.* 94:1443–76
134. Navarro JA, Sanchez-Navarro JA, Pallas V. 2019. Key checkpoints in the movement of plant viruses through the host. *Adv. Virus Res.* 104:1–64
135. Nawaz-ul-Rehman MS, Fauquet CM. 2009. Evolution of geminiviruses and their satellites. *FEBS Lett.* 583:1825–32
136. Nerva L, Forgia M, Ciuffo M, Chitarra W, Chiapello M, et al. 2019. The mycovirome of a fungal collection from the sea cucumber *Holothuria polii*. *Virus Res.* 273:197737
137. Nerva L, Turina M, Zanzotto A, Gardiman M, Gaiotti F, et al. 2019. Isolation, molecular characterization and virome analysis of culturable wood fungal endophytes in esca symptomatic and asymptomatic grapevine plants. *Environ. Microbiol.* 21:2886–904
138. Neuhauser S, Kirchmair M, Bulman S, Bass D. 2014. Cross-kingdom host shifts of phytomyxid parasites. *BMC Evol. Biol.* 14:33
139. Neumann P, Novak P, Hostakova N, Macas J. 2019. Systematic survey of plant LTR-retrotransposons elucidates phylogenetic relationships of their polyprotein domains and provides a reference for element classification. *Mob. DNA* 10:1
140. Ng JC, Falk BW. 2006. Virus-vector interactions mediating nonpersistent and semipersistent transmission of plant viruses. *Annu. Rev. Phytopathol.* 44:183–212
141. Nibert ML, Debat HJ, Manny AR, Grigoriev IV, De Fine Licht HH. 2019. Mitovirus and mitochondrial coding sequences from basal fungus *Entomophthora muscae*. *Viruses* 11(4):E351
142. Nibert ML, Ghabrial SA, Maiss E, Lesker T, Vainio EJ, et al. 2014. Taxonomic reorganization of family Partitiviridae and other recent progress in partitivirus research. *Virus Res.* 188:128–41
143. Nibert ML, Vong M, Fugate KK, Debat HJ. 2018. Evidence for contemporary plant mitoviruses. *Virology* 518:14–24

144. Niklas KJ, Cobb ED, Matas AJ. 2017. The evolution of hydrophobic cell wall biopolymers: from algae to angiosperms. *J. Exp. Bot.* 68:5261–69
145. Niklas KJ, Newman SA. 2019. The many roads to (and from) multicellularity. *J. Exp. Bot.* **https://doi.org/10.1093/jxb/erz547**
146. Novikova O, Belfort M. 2017. Mobile group II introns as ancestral eukaryotic elements. *Trends Genet.* 33:773–83
147. Novikova O, Smyshlyaev G, Blinov A. 2010. Evolutionary genomics revealed interkingdom distribution of Tcn1-like chromodomain-containing Gypsy LTR retrotransposons among fungi and plants. *BMC Genom.* 11:231
148. OTPT Initiat. 2019. One thousand plant transcriptomes and the phylogenomics of green plants. *Nature* 574:679–85
149. Paez-Espino D, Eloe-Fadrosh EA, Pavlopoulos GA, Thomas AD, Huntemann M, et al. 2016. Uncovering Earth's virome. *Nature* 536:425–30
150. Pascon RC, Kitajima JP, Breton MC, Assumpcao L, Greggio C, et al. 2006. The complete nucleotide sequence and genomic organization of Citrus leprosis associated virus, cytoplasmatic type (CiLV-C). *Virus Genes* 32:289–98
151. Peterson KJ, Cotton JA, Gehling JG, Pisani D. 2008. The Ediacaran emergence of bilaterians: congruence between the genetic and the geological fossil records. *Philos. Trans. R. Soc. Lond. B* 363:1435–43
152. Popper ZA, Michel G, Herve C, Domozych DS, Willats WG, et al. 2011. Evolution and diversity of plant cell walls: from algae to flowering plants. *Annu. Rev. Plant Biol.* 62:567–90
153. Pyle JD, Keeling PJ, Nibert ML. 2017. Amalga-like virus infecting *Antonospora locustae*, a microsporidian pathogen of grasshoppers, plus related viruses associated with other arthropods. *Virus Res.* 233:95–104
154. Quito-Avila DF, Brannen PM, Cline WO, Harmon PF, Martin RR. 2013. Genetic characterization of Blueberry necrotic ring blotch virus, a novel RNA virus with unique genetic features. *J. Gen. Virol.* 94:1426–34
155. Rastgou M, Habibi MK, Izadpanah K, Masenga V, Milne RG, et al. 2009. Molecular characterization of the plant virus genus *Ourmiavirus* and evidence of inter-kingdom reassortment of viral genome segments as its possible route of origin. *J. Gen. Virol.* 90:2525–35
156. Revers F, Garcia JA. 2015. Molecular biology of potyviruses. *Adv. Virus Res.* 92:101–99
157. Richert-Poggeler KR, Noreen F, Schwarzacher T, Harper G, Hohn T. 2003. Induction of infectious petunia vein clearing (pararetro) virus from endogenous provirus in petunia. *EMBO J.* 22:4836–45
158. Richert-Poggeler KR, Shepherd RJ. 1997. Petunia vein-clearing virus: a plant pararetrovirus with the core sequences for an integrase function. *Virology* 236:137–46
159. Rochon D, Kakani K, Robbins M, Reade R. 2004. Molecular aspects of plant virus transmission by olpidium and plasmodiophorid vectors. *Annu. Rev. Phytopathol.* 42:211–41
160. Roossinck MJ. 2010. Lifestyles of plant viruses. *Philos. Trans. R. Soc. Lond. B* 365:1899–905
161. Roossinck MJ. 2012. Plant virus metagenomics: biodiversity and ecology. *Annu. Rev. Genet.* 46:359–69
162. Roossinck MJ. 2018. Evolutionary and ecological links between plant and fungal viruses. *New Phytol.* 221(1):86–92
163. Roossinck MJ, Sabanadzovic S, Okada R, Valverde RA. 2011. The remarkable evolutionary history of endornaviruses. *J. Gen. Virol.* 92:2674–78
164. Rosa C, Kuo YW, Wuriyanghan H, Falk BW. 2018. RNA interference mechanisms and applications in plant pathology. *Annu. Rev. Phytopathol.* 56:581–610
165. Rota-Stabelli O, Daley AC, Pisani D. 2013. Molecular timetrees reveal a Cambrian colonization of land and a new scenario for ecdysozoan evolution. *Curr. Biol.* 23:392–98
166. Rousvoal S, Bouyer B, Lopez-Cristoffanini C, Boyen C, Collen J. 2016. Mutant swarms of a totivirus-like entities are present in the red macroalga *Chondrus crispus* and have been partially transferred to the nuclear genome. *J. Phycol.* 52:493–504
167. Roux S, Enault F, Bronner G, Vaulot D, Forterre P, Krupovic M. 2013. Chimeric viruses blur the borders between the major groups of eukaryotic single-stranded DNA viruses. *Nat. Commun.* 4:2700
168. Sabanadzovic S, Valverde RA, Brown JK, Martin RR, Tzanetakis IE. 2009. Southern tomato virus: the link between the families *Totiviridae* and *Partitiviridae*. *Virus Res.* 140:130–37

169. Sanfaçon H, Wellink J, Le Gall O, Karasev A, van der Vlugt R, Wetzel T. 2009. *Secoviridae*: a proposed family of plant viruses within the order *Picornavirales* that combines the families *Sequiviridae* and *Comoviridae*, the unassigned genera *Cheravirus* and *Sadwavirus*, and the proposed genus *Torradovirus*. *Arch. Virol*. 154:899–907
170. Scholthof KB. 2004. Tobacco mosaic virus: a model system for plant biology. *Annu. Rev. Phytopathol.* 42:13–34
171. Scholthof KB, Adkins S, Czosnek H, Palukaitis P, Jacquot E, et al. 2011. Top 10 plant viruses in molecular plant pathology. *Mol. Plant Pathol.* 12:938–54
172. Shi M, Lin XD, Chen X, Tian JH, Chen LJ, et al. 2018. The evolutionary history of vertebrate RNA viruses. *Nature* 556:197–202
173. Shi M, Lin XD, Tian JH, Chen LJ, Chen X, et al. 2016. Redefining the invertebrate RNA virosphere. *Nature* 540:539–43
174. Shi M, Lin XD, Vasilakis N, Tian JH, Li CX, et al. 2016. Divergent viruses discovered in arthropods and vertebrates revise the evolutionary history of the Flaviviridae and related viruses. *J. Virol.* 90:659–69
175. Sicard A, Michalakis Y, Gutierrez S, Blanc S. 2016. The strange lifestyle of multipartite viruses. *PLOS Pathog*. 12:e1005819
176. Sicard A, Zeddam JL, Yvon M, Michalakis Y, Gutierrez S, Blanc S. 2015. Circulative nonpropagative aphid transmission of nanoviruses: an oversimplified view. *J. Virol.* 89:9719–26
177. Sobhy H. 2017. A comparative review of viral entry and attachment during large and giant dsDNA virus infections. *Arch. Virol.* 162:3567–85
178. Soltis PS, Folk RA, Soltis DE. 2019. Darwin review: angiosperm phylogeny and evolutionary radiations. *Proc. R. Soc. B* 286:20190099
179. Somera M, Sarmiento C, Truve E. 2015. Overview on sobemoviruses and a proposal for the creation of the family Sobemoviridae. *Viruses* 7:3076–115
180. Spatafora JW, Aime MC, Grigoriev IV, Martin F, Stajich JE, Blackwell M. 2017. The fungal tree of life: from molecular systematics to genome-scale phylogenies. *Microbiol. Spectr.* 5:FUNK-0053-2016
181. Stork NE. 2018. How many species of insects and other terrestrial arthropods are there on Earth? *Annu. Rev. Entomol.* 63:31–45
182. Taliansky M, Mayo MA, Barker H. 2003. Potato leafroll virus: a classic pathogen shows some new tricks. *Mol. Plant Pathol.* 4:81–89
183. Teixeira M, Sela N, Ng J, Casteel CL, Peng HC, et al. 2016. A novel virus from *Macrosiphum euphorbiae* with similarities to members of the family Flaviviridae. *J. Gen. Virol.* 97:1261–71
184. Toriyama S, Kimishima T, Takahashi M, Shimizu T, Minaka N, Akutsu K. 1998. The complete nucleotide sequence of the rice grassy stunt virus genome and genomic comparisons with viruses of the genus Tenuivirus. *J. Gen. Virol.* 79(Pt. 8):2051–58
185. Van Etten JL, Agarkova IV, Dunigan DD. 2019. Chloroviruses. *Viruses* 12:E20
186. Vasilakis N, Forrester NL, Palacios G, Nasar F, Savji N, et al. 2013. Negevirus: a proposed new taxon of insect-specific viruses with wide geographic distribution. *J. Virol.* 87:2475–88
187. Verchot-Lubicz J, Torrance L, Solovyev AG, Morozov SY, Jackson AO, Gilmer D. 2010. Varied movement strategies employed by triple gene block-encoding viruses. *Mol. Plant-Microbe Interact.* 23:1231–47
188. Vlok M, Gibbs AJ, Suttle CA. 2019. Metagenomes of a freshwater charavirus from British Columbia provide a window into ancient lineages of viruses. *Viruses* 11:E299
189. Wei T, Li Y. 2016. Rice reoviruses in insect vectors. *Annu. Rev. Phytopathol.* 54:99–120
190. Weynberg KD, Allen MJ, Wilson WH. 2017. Marine prasinoviruses and their tiny plankton hosts: a review. *Viruses* 9:E43
191. White KA, Nagy PD. 2004. Advances in the molecular biology of tombusviruses: gene expression, genome replication, and recombination. *Prog. Nucleic Acid Res. Mol. Biol.* 78:187–226
192. Whitfield AE, Falk BW, Rotenberg D. 2015. Insect vector-mediated transmission of plant viruses. *Virology* 479–480:278–89
193. Whitfield AE, Huot OB, Martin KM, Kondo H, Dietzgen RG. 2018. Plant rhabdoviruses: their origins and vector interactions. *Curr. Opin. Virol.* 33:198–207

194. Wolf YI, Kazlauskas D, Iranzo J, Lucia-Sanz A, Kuhn JH, et al. 2018. Origins and evolution of the global RNA virome. *mBio* 9:e02329-18
195. Wood R, Liu AG, Bowyer F, Wilby PR, Dunn FS, et al. 2019. Integrated records of environmental change and evolution challenge the Cambrian Explosion. *Nat. Ecol. Evol.* 3:528–38
196. Wright DA, Voytas DF. 1998. Potential retroviruses in plants: Tat1 is related to a group of *Arabidopsis thaliana* Ty3/gypsy retrotransposons that encode envelope-like proteins. *Genetics* 149:703–15
197. Yu X, Li B, Fu Y, Xie J, Cheng J, et al. 2013. Extracellular transmission of a DNA mycovirus and its use as a natural fungicide. *PNAS* 110:1452–57
198. Zamora M, Mendez-Lopez E, Agirrezabala X, Cuesta R, Lavin JL, et al. 2017. Potyvirus virion structure shows conserved protein fold and RNA binding site in ssRNA viruses. *Sci. Adv.* 3:eaao2182
199. Zerbini FM, Briddon RW, Idris A, Martin DP, Moriones E, et al. 2017. ICTV virus taxonomy profile: Geminiviridae. *J. Gen. Virol.* 98:131–33
200. Zhang YZ, Chen YM, Wang W, Qin XC, Holmes EC. 2019. Expanding the RNA virosphere by unbiased metagenomics. *Annu. Rev. Virol.* 6:119–39
201. Zhang YZ, Wu WC, Shi M, Holmes EC. 2018. The diversity, evolution and origins of vertebrate RNA viruses. *Curr. Opin. Virol.* 31:9–16
202. Zhao L, Rosario K, Breitbart M, Duffy S. 2019. Eukaryotic circular rep-encoding single-stranded DNA (CRESS DNA) viruses: ubiquitous viruses with small genomes and a diverse host range. *Adv. Virus Res.* 103:71–133