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The Hidden Diversity of *Benyviridae* and their *Polymyxa* Vectors: A Comparative Analysis

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Abstract

The *Benyviridae* family encompasses multipartite soil-borne phytoviruses characterized by rod-shaped virions and positive single-stranded RNA genomes. The family is mostly known for its type species, the beet necrotic yellow vein virus, the causal agent of rhizomania on sugar beet. However, the recent description of candidate *Benyviridae* species and "beny-like" sequences suggests a far greater diversity than previously recognized. Also, their increasing relevance in agriculture has drawn attention to this family of viruses. In this review, we provide a comparative analysis of *Benyviridae* viruses, including newly identified emerging relatives. We highlight recent advances in understanding their diversity, pathogenicity, and interaction with plant hosts and their plasmodiophorid vectors, *Polymyxa* spp., that remain poorly characterized. Finally, we identify critical knowledge gaps and exciting opportunities—particularly in vector biology, host interactions, and the ecological dynamics of viral spread—that will shape the research ahead.

1. INTRODUCTION

For decades, the beet necrotic yellow vein virus (BNYVV), a member of the species *Benyvirus necrobetae*, has served as a model for studying soilborne viruses. Its interaction with its vector, *Polymyxa betae*, and sugar beet exemplifies a coevolved tripartite system where the virus manipulates both its vector's and its host's physiology. BNYVV significantly modifies the root architecture of sugar beets (22), such as the proliferation of lateral roots and stunted taproots (129), to better accommodate *P. betae* (35). This results in yield losses up to 95% in highly susceptible cultivars (85). This three-way interaction goes beyond simple transmission. It indicates a coevolutionary dynamic between the virus, its vector, and the plant host. This certainly explains why benyviruses have been chosen as model viruses and how their study has helped to study fundamental plant virology questions regarding cell-to-cell movement and soil-borne plasmodiophorid transmission.

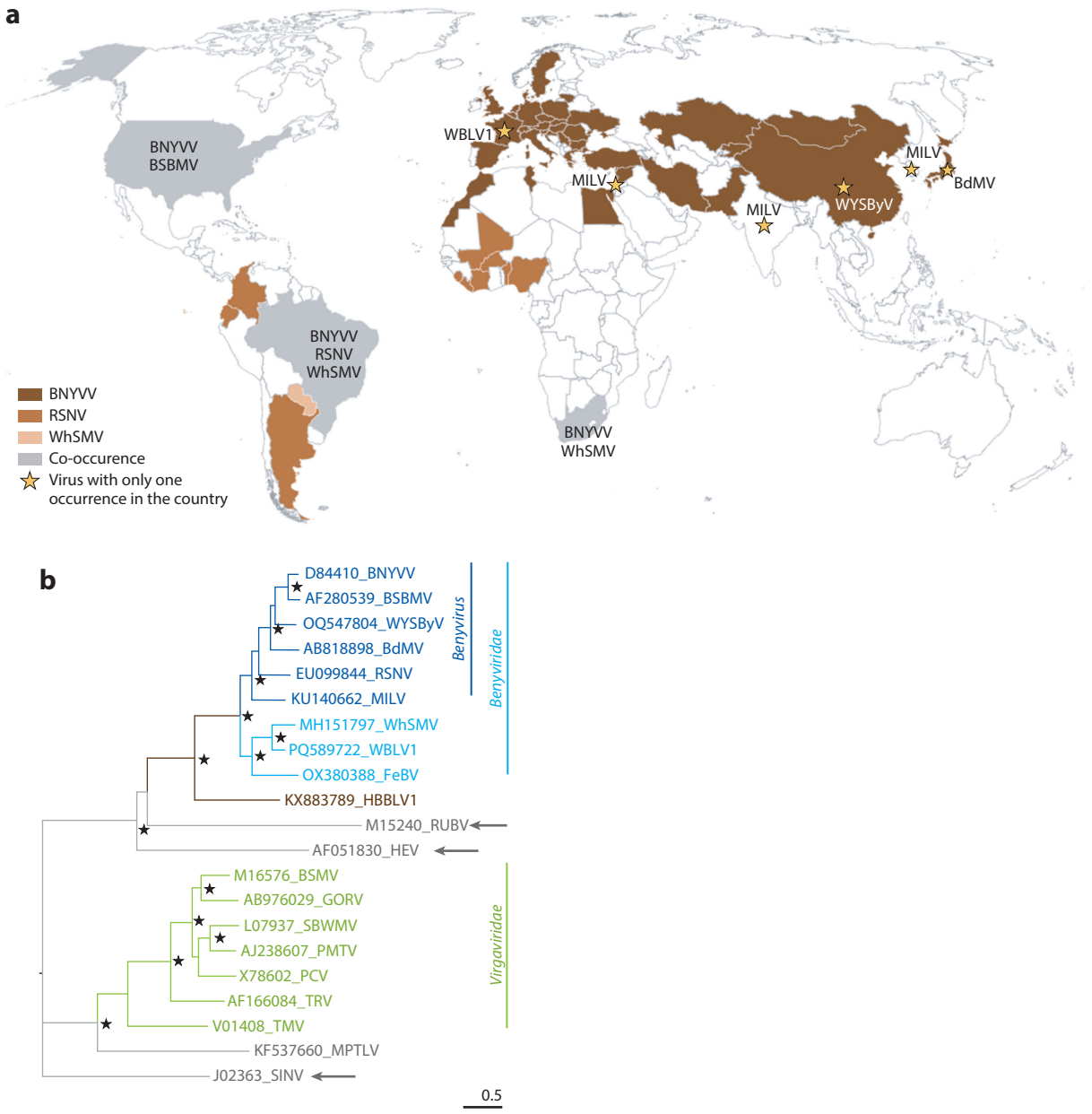
Beyond BNYVV, three other *Benyvirus* species are recognized by the International Committee on Taxonomy of Viruses (ICTV) (51) (**Table 1**). The beet soil-borne mosaic virus (BSBMV), a member of the species *Benyvirus solibetae*, also infects sugar beet, primarily causing leaf symptoms such as light-yellow vein banding, mottling, mosaic patterns, and growth disorders (63). Although roots often appear asymptomatic, rhizomania-like symptoms can occur in some infected plants (19). The burdock mottle virus (BdMV), a member of the *Benyvirus arctii* species, induces chlorosis and mottling on leaves of burdock, a root vegetable cultivated in East Asia (65, 72). The rice stripe necrosis virus (RSNV), a member of the *Benyvirus oryzae* species, leads to yellow stripes and deformation of leaves, stunted growth, reduced tillering, and abnormal panicle formation leading to lower seed production on rice plants. Field incidence ranges locally from 37% to 80% (44, 88, 95).

Benyvirids have long stood as a clearly defined family of single-stranded positive-sense RNA multipartite viruses transmitted by vectors belonging to the *Polymyxa* genus (*Plasmodiophoridae*, Rhizaria), except for BdMV, for which no vector has been identified (51). The *Polymyxa* genus includes two species, *P. betae* and *P. graminis*, which are both asymptomatic root endoparasites and distinguished based on their hosts and virus ranges. Although the first genomes of *P. betae* have been recently sequenced (34), providing insight into host–*Polymyxa*–virus interactions in sugar beet, the entire genome of *P. graminis* is not yet known, limiting our understanding of vector diversity and transmission determinants across the family. Given the long-term survival of benyviruses within *Polymyxa* resting spores, which renders crop rotation inefficient, understanding the biology and diversity of vectors is crucial for assessing the epidemiological risks of *Benyviridae*.

Table 1 List of *Benyvirus* species

Species	Virus name	Virus acronym	Host	Genomic organization	RefSeq numbers	Reference
Benyvirus necrobetae	Beet necrotic yellow vein virus	BNYVV	Sugar beet	4–5 RNAs	NC_003514 NC_003515 NC_003516 NC_003517 NC_003513	126
Benyvirus solibetae	Beet soil-borne mosaic virus	BSBMV	Sugar beet	4 RNAs	NC_039226 NC_039225 NC_039224 NC_039227	79
Benyvirus arctii	Burdock mottle virus	BdMV	Burdock	2 RNAs	NC_021735 NC_021736	72
Benyvirus oryzae	Rice stripe necrosis virus	RSNV	Rice	2 RNAs	NC_038775 NC_038774	89

Over recent years, the *Benyviridae* family has drawn growing attention due to its increasing agricultural relevance and expanding host range (**Figure 1*a***). Recent discoveries have revealed the increasing impact of RSNV on rice, particularly in Latin America, and its ability to infect other major monocots (9, 103). Another bipartite virus related to *Benyviridae*, currently under review at ICTV, the wheat stripe mosaic virus (WhSMV), a member of the proposed species *Benyvirus tritici*, was recently discovered on wheat in Brazil (136). This virus causes yellow-green chlorotic streaks on leaves and stems, stunting, excessive tillering, and rosette formation, leading to yield losses



(*Caption for **Figure 1** appears on following page*)

Figure 1 (*Figure appears on preceding page*)

Phylogeny and geographical distribution of *Benyviridae* species. (*a*) Geographical distribution of *Benyviridae* species. Countries where viral species have been reported several times are colored, single occurrences were indicated as yellow stars, and co-occurrences are indicated in gray. (*b*) Phylogenetic trees based on the RNA-dependent RNA polymerase. The nucleotide sequences were downloaded from the NCBI database, trimmed to be compared to the reference dataset (51), and aligned using MUSCLE in Seaview version 5.0.5 software (54). The phylogenetic tree based on the codon-aligned nucleotide sequences was built using the maximum likelihood method based on the General Time Reversible model with gamma-distributed rate variation and invariant sites (GTR+I+G) model and 100 bootstrap replicates. A black star represents bootstraps greater than 75%. Gray arrows show reference viruses from other families: rubella virus (RUBV; *Matonaviridae*), sindbis virus (SINV; *Togaviridae*), and hepatitis E virus (HEV; *Hepeviridae*). Viral species related to the genus *Benyvirus* and the family *Benyviridae* are colored in dark and light blue, respectively. Viruses of the *Virgaviridae* family are in green. Abbreviations: BdMV, burdock mottle virus; BNYVV, beet necrotic yellow vein virus; BSBMV, beet soilborne mosaic virus; BSMV, barley stripe mosaic virus; FeBV, fern benyvirus; GORV, gentian ovary ringspot virus; HBBLV1, Hubei beny-like virus 1; MILV, mangifera indica latent virus; MPTLV, macrophomina phaseolina tobamo-like virus; PCV, peanut clump virus; PMTV, potato mop top virus; RSNV, rice stripe necrosis virus; SBWMV, soil-borne wheat mosaic virus; TMV, tobacco mosaic virus; TRV, tobacco rattle virus; WBLV1, wheat beny-like virus 1; WhSMV, wheat stripe mosaic virus; WYSByV, wheat yellow stripes associated with beny-like virus.

exceeding 50% in susceptible wheat cultivars (106, 136). Moreover, two bipartite "beny-like" RNA sequences have been identified on wheat: the wheat yellow stripe associated virus (WYSAV) in China (57) and the wheat beny-like virus 1 (WBLV1) in France (15). Owing to the previous existence of two viruses related to *Polerovirus* and *Betaflexiviridae*, also tentatively named Wheat yellow stripe associated virus (WYSaV) and Wheat yellow stunt-associated betaflexivirus (WYSaBV), respectively, we propose changing the name of the benyvirus WYSAV to wheat yellow stripe associated with beny-like virus (WYSByV) to avoid confusion. WYSByV was found in coinfection with wheat yellow mosaic virus, a potyvirid also transmitted by *P. graminis*, and WBLV1 with both barley yellow dwarf virus strains, PAV and PAS, transmitted by aphids. These coinfections make it unclear if they cause symptoms in wheat. Additional bipartite beny-like RNA sequences were uncovered on different hosts: the mangifera indica latent virus (MILV), the first virus reported in the transcriptome of mango trees (118), and the fern benyvirus (FeBV) found in two ferns, *Asplenium nidus* and *Lonchitis hirsutae* (94). These viruses suggest a much broader host range for beny-like viruses.

Furthermore, the identification of numerous monopartite beny-like RNA sequences (**Table 2**, **Supplemental Table 1**) in a wide range of plant and nonplant organisms challenges the traditional view that *Benyviridae* is restricted to plant-infecting viruses (98). These discoveries raise questions about virus evolution and, consequently, classification criteria and taxonomy.

This review offers a critical perspective on the current state of research on *Benyviridae*, moving beyond known facts to explore what makes this virus family both scientifically intriguing and agriculturally significant. Although BNYVV remains the best-characterized member, the expanding diversity of other benyviruses and recently identified beny-like species signals that we are only beginning to grasp the true scope of this group. We highlight not just the established stages of the virus life cycle but also the surprising complexity of its transmission through *Polymyxa* vectors—organisms whose biology remains an underexplored frontier. Recent advances in molecular and ecological research are reshaping our understanding of how these viruses emerge, adapt, and persist. Critical gaps and exciting opportunities that will shape future research can be identified from this evolving picture, especially regarding vector biology, host–virus interactions, and the dynamics of virus evolution.

2. GENETIC DIVERSITY OF *BENYVIRIDAE*

2.1. Taxonomy of *Benyviridae*

The *Benyviridae* is a family of multipartite plant viruses possessing two to five single-stranded, positive-sense RNA segments, with 5' m7G caps and polyadenylated tails, each encapsidated

Table 2 List of viruses related to *Benyviridae* and beny-like viruses found on plants

Status	Virus name	Virus acronym	Genomic organization	Accession number	Origin of sequence	Sampling location	Reference(s)
Tentative *Benyvirus*	Mangifera indica latent virus	MILV[a]	2 RNAs	KU140662 KU140663	*Mangifera indica*	India, Israel, South Korea	118
	Wheat yellow stripe associated virus	WYSByV[b]	2 RNAs	OQ547804 OQ547805	*Triticum aestivum*	China	57
Tentative *Benyviridae*	Wheat stripe mosaic virus	WhSMV	2 RNAs	NC_040760 NC_040761	*Triticum aestivum*	South Africa, Brazil, Paraguay	134, 136
	Wheat beny-like virus 1	WBLV1	2 RNAs	PQ589722 PQ589723	*Triticum aestivum*	France	15
	fern benyvirus	FeBV	2 RNAs	OX380388 OX380448	*Asplenium nidus*	NA	94
Unclassified	Abrau grapevine-associated virus	AGaV	1 RNA	PQ606058	*Vitis vinifera*	NA	13
	Arceuthobium sichuanense-associated virus 3	ASaV3	1 RNA	BK059270	*Arceuthobium sichuanense*	China	120
	Carrot associated RNA virus 1	CaRNAV1	1 RNA	OP889248	*Daucus carota*	France	116
	Chara australis virus	CAV	1 RNA	JF824737	*Chara australis*	Australia	48
	Dactylorhiza hatagirea beny-like virus	DhBLV	1 RNA	BK013327	*Dactylorhiza hatagirea*	India	107, 121
	Epipactis helleborine associated beny-like virus	EhaBLV1	1 RNA	NA	*Epipactis helleborine*	NA	107
	Goji berry chlorosis virus	GBCV	1 RNA	MH791331	*Lycium chinense* Miller	South Korea	75
	Gymnadenia rhellicani associated beny-like virus	GraBLV1	1 RNA	NA	*Gymnadenia rhellicani*	NA	107
	Hemipilia forrestii associated beny-like virus	HfaBLV1	1 RNA	NA	*Hemipilia forrestii*	NA	107
	Orchis hatagirea associated beny-like virus	OhaBLV1	1 RNA	NA	*Orchis hatagirea*	NA	107
	Orchis militaris associated beny-like virus	OmaBLV1	1 RNA	NA	*Orchis militaris*	NA	107
	Ophrys fusca associated beny-like virus	OfaBLV1	1 RNA	NA	*Ophrys fusca*	NA	107
	Red clover RNA virus 1	RCRV1	1 RNA	MG596242	*Trifolium pratense*	Czech Republic	124

[a]Related to Benyviridae species by ITCV (51).
[b]WYSAV is written WYSByV.

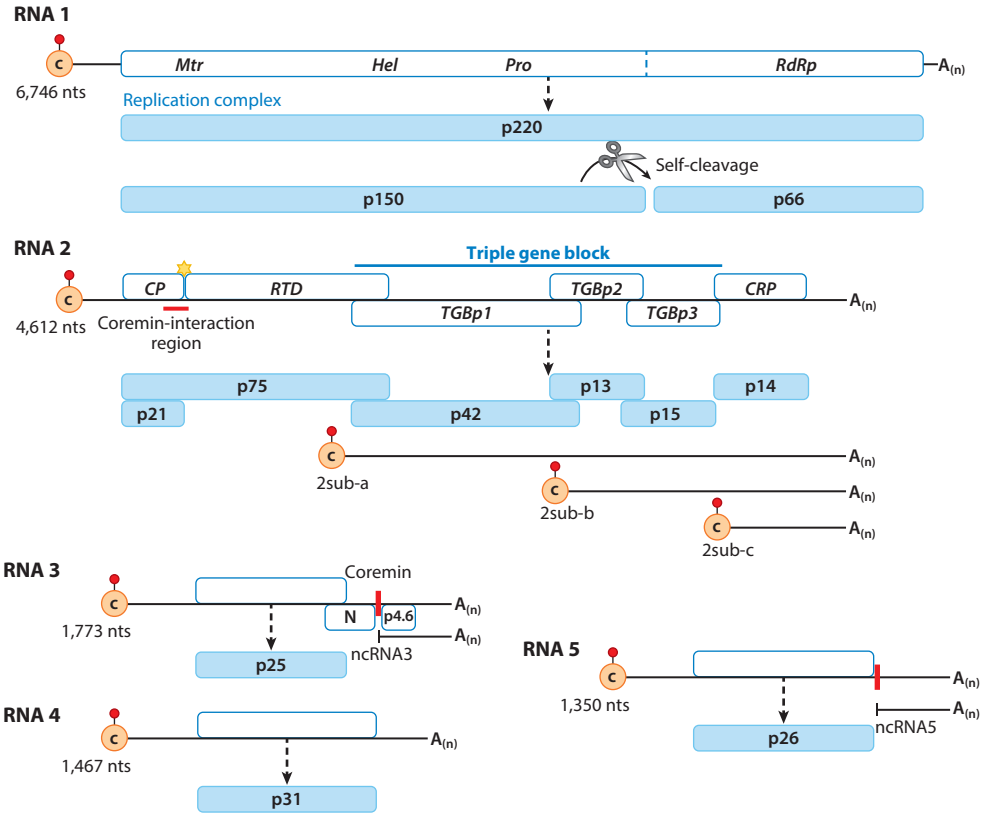


Figure 2

Genome organization and translation strategies of beet necrotic yellow vein virus, a typical member of the *Benyviridae* species. The scheme indicates the open reading frames (*white rectangles with blue borders*), the encoded proteins (*light blue rectangles*), the self-cleavage of the replicase protein (*black arrow and scissor*), a suppressible UAG stop codon (*yellow star*), m^7Gppp (*c in orange circle*), the 3′ poly(A)-tails [A(n)], a coremin sequence (*vertical red line*), and the coremin binding site (*horizontal red line*). Abbreviations: CP, coat protein; CRP, cysteine-rich protein; Hel, helicase; Mtr, methyltransferase; ncRNA, noncoding RNA; nts, nucleotides; Pol, RNA-dependent RNA polymerase; Pro, protease; RdRp, RNA-dependent RNA polymerase; RTD, readthrough domain; sub, subgenomic RNAs; TGBp1–3, triple gene block protein 1–3.

independently (**Figure 2**). The virions are rod-shaped and nonenveloped with a diameter of 20 nm and a length of up to 390 nm (51). The *Benyviridae* contains only a single genus (*Benyvirus*) initially assigned to the family *Virgaviridae*, due to morphological, genomic, and transmission mode similarities.

Since the adoption of the novel taxonomy rules by ICTV, the classification of RNA viruses is first guided by the phylogeny of their RNA-dependent RNA polymerase (RdRp) (122). To determine the status of the bipartite and monopartite beny-like viruses, we reconstructed the phylogeny of their polymerase sequences following the method and the reference dataset (51) (**Figure 1*b***). As the rubella virus now belong to *Matonaviridae* (91), it is noteworthy that the sindbis virus was added to the initial dataset to represent *Togaviridae* and visualized its relatedness with *Virgaviridae* in the order Martellivirales and with *Benyviridae* and *Hepeviridae* in Hepelivirales (73). Based on this analysis and as described by their authors, WYSByV and MILV belong to the genus *Benyvirus*,

whereas WhSMV, WBLV1, and FeBV belong to a distinct lineage, likely a new genus, in the family *Benyviridae*.

The available genomic sequences of the 59 monopartite beny-like viruses were also examined, in addition to the bipartite ones (**Table 2**, **Supplemental Table 1**). The complete polymerase domain was available for only 40 beny-like viruses (**Table 2**, **Supplemental Table 1**). As the order Hepelivirales also contains two other families infecting nonplant hosts, type member species of the *Alphatetraviridae* and *Mycoalphaviridae* were added to the dataset (**Figure 3**). On the one hand, based on the genetic distance to the known *Benyviridae*, the five monopartite viruses described in plants, including chara australis virus (48) and goji berry chlorosis virus (75), would form two new genera in this family or in a new (sub)family. On the other hand, our phylogenetic reconstruction showed that none of the beny-like sequences found from nonhost plants belong to the genus *Benyvirus* or the family *Benyviridae*. Hubei beny-like virus 1, found in mosquitos and previously described as an unclassified virus related to *Benyviridae* (51), appears very distant from them.

Based on these analyses and without complementary data on other RNA segments, we focused this review on bipartite beny-like viruses, all found in plants and referred to as *Benyviridae* below.

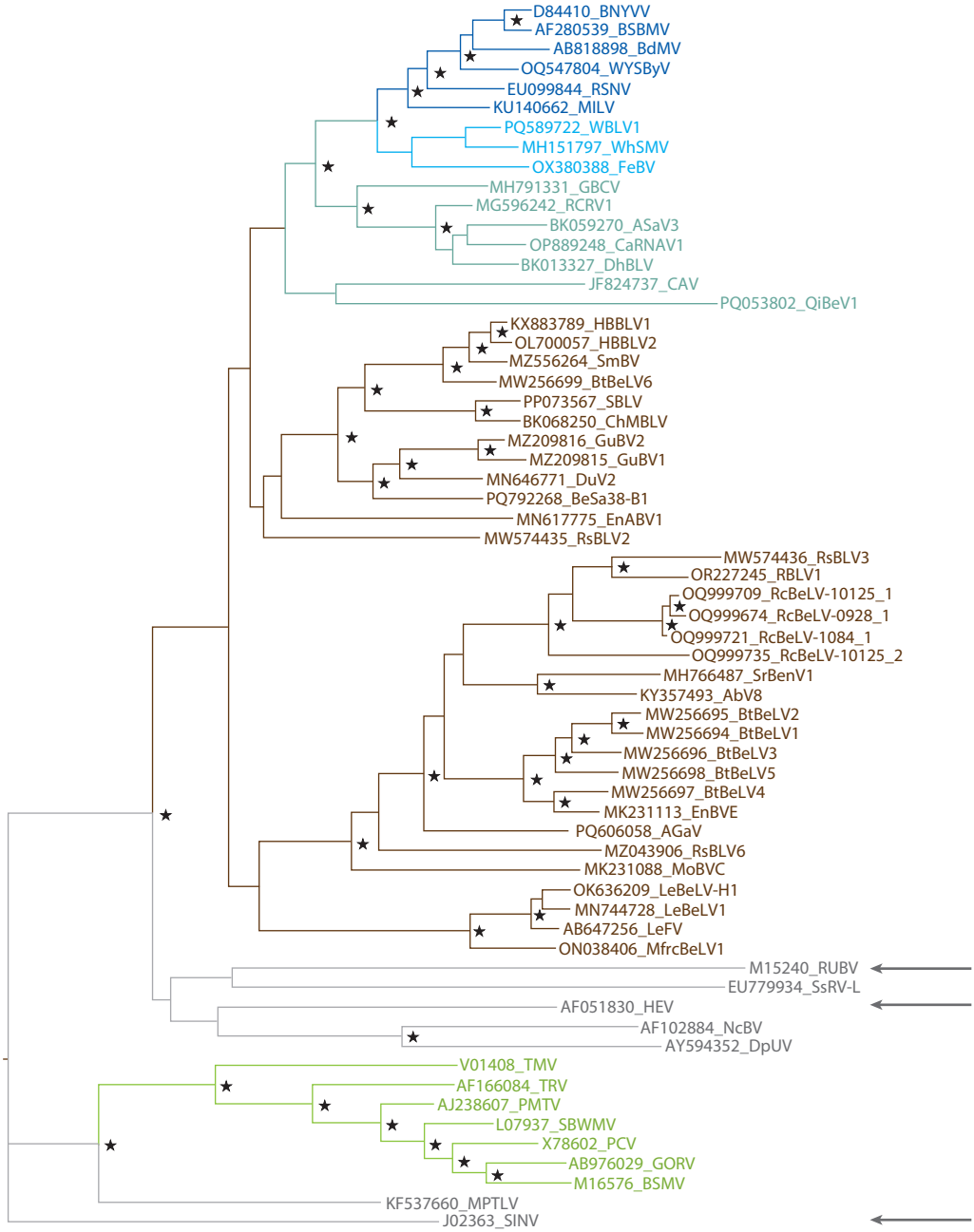
2.2. Genomic Organization of *Benyviridae* Species

Benyviridae species have at least two positive-sense RNA segments (approximately 7 and 4.6 kb in size), with up to three additional RNA segments (each between 1.3 and 1.8 kb) (51) (**Figure 2**). RNA1 contains a single large open reading frame (ORF) encoding a replication complex polyprotein that harbors motifs for methyltransferase (Mtr), helicase (Hel), papain-like protease (Pro), and RdRp (9, 17, 51, 57, 136). RNA2 contains six overlapping ORFs encoding the coat protein (CP), a readthrough domain (RTD) of CP, three proteins clustered in a triple gene block (TGBp1–3), and a cysteine-rich protein (CRP) (51). Except for WYSByV, all RNA2 segments of the new *Benyviridae* are smaller, suggesting either recombinant events or in silico assembly problems. The ORF6 is shorter in WhSMV or absent in WBLV1, FeBV, and MILV sequences. In addition, RTD and TGBp3 are lacking in MILV. However, the phylogenetic trees based on CP and TGBp1 sequences remain congruent for these viruses (**Supplemental Figure 1**).

RNA1 and RNA2 segments are sufficient for infection, viral replication, virus movement, and suppression of gene silencing in hosts (9, 57, 72, 89, 136). However, BNYVV and BSBMV possess two additional RNAs, and experimental inoculation without the RNA3 and RNA4 segments drastically reduces symptoms. RNA3 encodes a single protein (p25 and p29 for BNYVV and BSBMV, respectively) and RNA4 codes for one protein (p31) or two overlapping proteins (p32 and p13) (127) (**Figure 2**). They influence symptom expression, transmission, long-distance movement, viral multiplication in roots, and resistance breakdown (9, 70, 76, 104, 132). Some BNYVV isolates also possess an additional RNA segment (RNA5) that modulates symptom severity and encodes a single protein p26 (130).

2.3. Interspecies Genetic Distance and Reassortment

Devoted to the replication function, an RNA1 segment is more conserved than RNA2 regardless of the *Benyviridae* species. Using the reference sequences for each viral species and as observed on the polymerase gene, BSBMV and BNYVV show the highest levels of pairwise sequence identity compared to other species for RNA1 and RNA2 (77% and 67%) (79) (**Supplemental Figure 2**). As they share the same host range and *Polymyxa* vector, experimental reassortments of BNYVV and BSBMV RNA1 and RNA2 were performed using infectious clones. They are viable and produce symptoms sometimes more severe than wild-type viruses (78). BNYVV and BSBMV show lower sequence identity for RNA3 and RNA4 segments (56% and 58%) (79), which could



(*Caption for* ***Figure 3*** *appears on following page*)

Figure 3 (*Figure appears on preceding page*)

Phylogenetic analysis of *Benyviridae* species and "beny-like" sequences. The nucleotide sequences of the RNA-dependent RNA polymerases were downloaded from the NCBI database and trimmed and aligned using MUSCLE. A phylogenetic tree based on the codon-aligned nucleotide sequences was built using the maximum likelihood method based on the General Time Reversible (GTR) model and 100 bootstrap replicates. A black star represents bootstraps greater than 75%. Grey arrows show reference viruses from other families: RUBV (*Matonaviridae*), SINV (*Togaviridae*), and HEV (*Hepeviridae*). Viral species related to the genus *Benyvirus* and the family *Benyviridae* are colored in dark and light blue, respectively. Beny-like viruses from plants are colored aquamarine, and other beny-like viruses from fungi, insects, and algae are colored black. Viruses of the *Virgaviridae* family are colored in green. Other viruses are colored in gray: SsRV-L (sclerotinia sclerotiorum RNA virus L), NcBV (nudaurelia capensis beta virus), DpUV (dendrolimus punctatus virus), and MPTLV (macrophomina phaseolina tobamo-like virus).

explain the differences in symptomatology caused by the two viruses on common hosts. Reports of experimental reassortments and hetero-encapsidation of RNA3 and RNA4 (30, 112) show that complementation is still possible between the two viruses. Although coinfections have been found in fields, natural recombination and reassortment events between BNYVV and BSBMV have never been found. The two viruses remain spatially separated even when they coinfect common hosts; a suppression and/or an exclusion phenomenon has been suggested (77). WhSMV and WBLV1 show 66% and 58% of pairwise sequence identity in RNA1 and RNA2, respectively. All the other *Benyviridae* species are more distant (<55%) regardless of their host type (dicotyledons or monocotyledons) or *Polymyxa* vector species (89) (**Supplemental Figure 2**). Although WYSByV and WBLV1 have been found in coinfection with viruses from other families, neither coinfection nor reassortment events have been reported or tested among other benyviruses and benyvirids.

2.4. Intra-Species Genetic Diversity and Geographical Distribution

The number of complete genomes and partial sequences available in public databases is very contrasted depending on the *Benyviridae* species (**Supplemental Table 3**, **Supplemental Table 4**). Although the hundreds of RNA2-CP and RNA3-p25 sequences of BNYVV represent an exception (see above), genetic information remains limited to one to four complete genomes for most other viruses.

The genomic diversity of only BNYVV, RSNV, and WhSMV has been studied so far. Although the geographical distribution area of the former is more extensive (**Figure 1*a***), the genetic diversity of its RNA1 and RNA2 segments, assessed on 12 representative isolates, is lower or similar to the latter (**Supplemental Table 3**). For BNYVV, the genetic diversity is higher in RNA2, 4, and 5 segments (94%, 92%, and 93%, respectively) compared to its other segments, but it does not show any clear geographic structure (25). By considering RNA2-CP, RNA3-p25, and RNA4-p31 gene sequences and the presence of RNA5, several lineages of BNYVV have been described: types A and B and, within type A, three subtypes, A-I, A-II, and A-III (25, 146). Nucleotide differences for CP, p25, p31, and p26 are relatively low, meaning that BNYVV does not evolve rapidly or have recently diverged (25).

The genetic diversity of RSNV was assessed on 18 genomes (9). Phylogenetic analyses of RSNV sequences have identified two main lineages: an African lineage with isolates from Mali and Burkina Faso, and a South American lineage with isolates from Argentina and Brazil (9, 31, 32). Additionally, some RSNV isolates exhibit higher divergence from the two major lineages, suggesting the existence of additional basal lineages, recombination, and reassortment events (9). Up to now, we consider that the origin of RSNV is still unknown, in contrast to de Oliveira Scherer et al. (31). Phylogenetic analyses of WhSMV based on 30 genomes also revealed two lineages based on geographical origin (South African and Brazilian isolates) (125). Among these lineages, genetic diversity remains low.

3. PLANT HOST–*BENYVIRIDAE* MOLECULAR INTERACTIONS

Our understanding of the molecular interactions between benyvirids and their host plant mostly relies on the results obtained on the BNYVV model and comprehensively reviewed by Gilmer et al. (50). Here, we provide a summary of key points and focus instead on recent findings and comparisons with other *Benyviridae* species.

xrRNA: exoribonuclease-resistant RNA

chRNA: CP-encoding chimeric RNA derived from BSBMV RNA2 and xrRNA

coremin: conserved 20-nt motif involved in a stem-loop structure that stalls 5′-to-3′ exoribonuclease activity

CP-RTD: coat protein fused with the read-through domain

3.1. Viral Replication and Virion Assembly

The replication of BNYVV is dependent on the p237 polyprotein, encoded by RNA1. This polyprotein undergoes post-translation auto-cleavage to yield two products: p150, which contains the Mtr, Pro, and Hel domains, and p66, which harbors the RdRp (**Figure 2**) (62). The N-terminal domain of p150 interacts with both itself and the internal domain of p66-RdRp, thus participating in the formation of a replication complex that localizes to the endoplasmic reticulum membrane (101). The conservation of the main amino acid motifs in other benyviruses suggests a similar processing and mode of action (**Supplemental Table 2**, **Supplemental Figure 3**). However, the cleavage site xGG/K is absent in other benyvirids, and alignment problems were identified for the catalytic triad of the protease in FeBV.

Conserved secondary structures of the 3′- and 5′-untranslated regions (UTRs) drive the specific replication of genomic RNAs and subgenomic RNAs. All subgenomic RNAs derived from RNA2 are required for the expression of internal ORFs (112).

In addition to genomic and subgenomic RNA, two distinct RNA species have been identified during infection by BNYVV and BSBMV: noncoding exoribonuclease-resistant RNAs (xrRNAs) and chimeric RNAs (chRNAs). BNYVV xrRNA3 corresponds to the 3′ end of RNA3 and begins with a highly conserved 20-nucleotide sequence, known as the coremin motif, that promotes the formation of a hairpin structure. xrRNA3 is generated by degradation of RNA3 by a 5′-3′ plant exoribonuclease, which is halted upon reaching this structured region, resulting in its stable accumulation (37, 47). Notably, coremin motifs have also been found in RNA3 and RNA4 of BSBMV, RNA3 and RNA5 of BNYVV but not RNA4, and several cucumoviruses. Sequence alignment suggested their presence in RNA1 of WhSMV. Interestingly, similar elements have recently been identified in other virus families such as *Betaflexiviridae*, *Virgaviridae*, *Potyviridae*, and *Secoviridae* (37).

Different types of chRNAs have been described for BSBMV and BNYVV. D'Alonzo et al. (30) reported chRNA corresponding to the 5′ end of RNA4 and 3′ end of RNA3 of BNYVV following serial inoculations. More recently, monocistronic CP-encoding chRNAs, which exist at the 5′ end of RNA2 and either xrRNA3 or xrRNA4, were identified during BSBMV infection (97). These chRNAs are translationally functional, move systemically, and may promote CP accumulation. They likely result from a template switching of RdRp facilitated by intermolecular base-pairing interactions between the coremin motif of xrRNA and the 3′-proximal part of the CP cistron located immediately upstream of a hairpin structure. Interestingly, stem-loop structures have also been predicted downstream of RSNV and BdMV CP cistron (97), although no coremin motif has been found in their genomes.

Once replicated, genomic RNAs are encapsidated. The 5′ UTRs of BNYVV contain *cis*-acting packaging signals that mediate the encapsidation of genomic, but not subgenomic, RNAs (53). Interestingly, similarities between the UTRs of BNYVV and BSBMV allow replication and encapsidation of BSBMV RNA3 by the BNYVV machinery (112). The BNYVV virion is mainly composed of the CP (p21). The helicoidal particles have an axial repeat of four turns, each involving 49 CP subunits. Their assembly is initiated by the CP-RTD fusion protein (p75) that arises from a translational readthrough of the CP stop codon in ∼10% of cases and is found at

one extremity of the virion particle (126). Amino acid alignment of benyvirid CP reveals probable errors at the C-terminus of FeBV and, as indicated above, the absence of the MILV RTD in the published sequences.

Like other multipartite viruses, benyvirids face challenges in assembling all genome segments necessary for infection. Regulation of segment copy numbers has recently been reported for several multipartite viruses (93, 119). It may affect virus accumulation, symptom expression, and host-specific adaptation while mitigating the costs of genome segmentation. To date, within the *Benyviridae* family, only the genomic formula of BNYVV has been investigated (29). Results indicate that RNA1 and RNA2 are consistently replicated at a lower level than RNA3 and RNA4 within the plant host, despite variations in the set point genomic formula among different plant hosts or organs.

3.2. Toward Systemic Infection: Movement and Suppression of Silencing

Like pomoviruses, pecluviruses, and hordeiviruses in the *Virgaviridae* family, benyviruses rely on the TGB proteins for cell-to-cell movement through the plasmodesmata (143). TGBp1 of BNYVV contains an NTP-dependent helicase activity and binds viral RNAs to form ribonucleic complexes. They rely on interactions with TGBp2 and TGBp3 and their transmembrane domains (TMDs) to facilitate its delivery to plasmodesmata. All three TGB proteins also accumulate in membrane-rich peripheral bodies likely derived from endoplasmic reticulum (41). Recent studies suggest a close interplay between viral replication and cell-to-cell movement of plant viruses, but little is currently known about this relationship in the context of benyviruses (50). All bipartite *Benyviridae* species encode TGB proteins and contain conserved motifs, with the exception of MILV, for which TGBp1 and TGBp3 are missing, suggesting that they employ a similar cell-to-cell movement strategy, albeit with species-specific features (143). For example, compared to BNYVV, RSNV TGBp1 contains an 81-amino-acid insertion in the N-terminal part. This insertion contains a nucleolar localization signal. Recent results highlight the role of the nucleolus and the nucleolar protein fibrillarin Fib2 in virus infection, particularly in virus movement (84, 145).

Mechanisms of long-distance movement of *Benyviridae* through the vascular vessels appear to depend on both the host and the virus. RNA1 and RNA2 of both BNYVV and BSBMV are sufficient for systemic infection of *Nicotiana benthamiana* (6, 76, 78). In this case, long-distance movement involves the CRP (24). In contrast, xrRNA are also required for systemic infection of *Beta* spp. (76, 78, 104). Both CRP and xrRNA3 are also involved in the suppression of RNA silencing, pointing out a close relationship between long-distance movement and the ability to counteract host silencing mechanisms: No systemic movement occurs when the virus is not able to counteract RNAi-based antiviral immunity. The role of CRP as a viral suppressor of RNA silencing (VSR) has been demonstrated for BNYVV, BSBMV, and BdMV (24, 56). BNYVV CRP VSR activity requires a functional zinc-finger domain and inhibits the RdRp 6 (RDR6) pathway, thus operating on the secondary small interfering RNA production or systemic movement (24, 47). Although BNYVV CRP localizes to both the cytoplasm and the nucleolus, its VSR function is independent of nucleolar localization. In contrast, BdMV CRP is restricted to the nucleus, highlighting functional and localization differences among CRP proteins within the *Benyviridae* family. These differences likely reflect the high sequence divergence of CRP, which represents the most variable ORF among *Benyviridae* members. However, sequence alignment showed the presence of the conserved zinc-finger motif in RSNV and WYSByV CRPs. In addition to CRP, although less efficient, xrRNA3 shows a VSR activity that can partially complement non-fully functional CRP (47).

3.3. Pathogenicity and Host Plant Deregulations

Infections caused by members of the *Benyviridae* family are associated with a wide range of symptoms that vary in both manifestation and severity. This variability is influenced by several factors, including the virus species, the host plant, the method of inoculation, and, notably, the composition of the viral genome, which help in identifying specific viral RNAs or proteins that function as pathogenicity factors. All these elements are also associated with distinct patterns of host gene deregulation, which may be associated with the observed symptoms. Transcriptomic studies have been conducted on sugar beet, wild beet (*Beta macrocarpa*), and *N. benthamiana* infected by BNYVV or BSBMV (42, 43, 46). In sugar beet, BNYVV infection leads to approximately twice the number of deregulated genes compared to BSBMV (46). In *N. benthamiana*, the presence of RNA4-encoded p31 correlates with severe symptoms, such as leaf curling and dwarfing, and significantly amplifies the host response, resulting in a 12-fold increase in the number of deregulated genes (42).

As expected, genes involved in response to stress and defense are deregulated by BNYVV and BSBMV infections. For example, several classes of pathogenesis-related (PR) proteins are upregulated and can account for 10% of upregulated defense-associated transcripts (43). Additionally, the expression of defense-associated transcription factors (AP2, bHLH, bZIP, ERF, MYB, NAC, and WRKY), as well as genes involved in the metabolism of reactive oxygen species or phenylpropanoid biosynthesis and the RNA silencing pathway, is markedly altered (42, 43, 46). On *N. benthamiana*, induction of PR10 by BNYVV requires the presence of p31, encoded by RNA4 and coinciding with the appearance of severe symptoms (142). Genes of the ubiquitin–proteasome pathway are also deregulated, potentially involving the BNYVV p25, which has been shown to interact with several components of this pathway (135). However, although these genes and pathways are major determinants of antiviral defense, the alteration of their expression may be related to other aspects of cellular disturbance.

Several genes and pathways related to development are also strongly impacted by BSBMV and BNYVV infections. Notably, cell wall–associated genes—such as cellulose synthases, endoglucanases, and pectinesterases—as well as the gibberellin pathway are affected in BNYVV-infected *N. benthamiana*, especially in the presence of BNYVV RNA4 (42). This may explain symptoms such as stunted growth. The auxin pathway is also highly affected in BNYVV-infected sugar beet. Its deregulation is associated with the lateral root proliferation (LRP), the most well-described symptom of rhizomania, which notably appears to benefit the vector *P. betae* (105). LRP is initiated by the conversion of root cells into meristematic cells and also involves the regulation of genes associated with cell wall organization (35, 45, 46). According to Fernando Gil et al. (46), two different pathways of auxin biosynthesis are activated in BNYVV-infected plants, whereas auxin inactivation is inhibited, resulting in lateral organ boundary domain and expansin expression, which directly impacts root proliferation. LRP is mediated by BNYVV-p25 (105), which interacts with the aux/IAA proteins BvIAA2, BvIAA6, and BvIAA28, whose expression inhibits root development in *N. benthamiana* (45, 96). These findings suggest that p25 may contribute to LRP by sequestering negative regulators. By contrast with BNYVV, BSBMV infection has a lower effect on the auxin pathway, and, accordingly, root-associated symptoms are rarely reported on BSBMV-infected sugar beets (46).

4. TRANSMISSION OF *BENYVIRIDAE* BY *POLYMYXA* SPP.

Polymyxa spp. are asymptomatic obligate root endoparasites classified in the Plasmodiophorida order (Rhizaria supergroup) (20). *Polymyxa* spp. transmit 22 viruses, of which 18 are transmitted by *P. graminis* and four by *P. betae* (128).

4.1. Diversity and Taxonomy of *Polymyxa* spp.

The separation of *P. betae* and *P. graminis* into two species is based on their distinct host ranges (138). This separation into two species was then confirmed using phylogenetic analyses based on ITS1–5.8S-ITS2, despite a low bootstrap value (138). However, a greater diversity of host ranges, ecotypes, and viruses, as well as a larger genetic diversity in the nuclear rDNA regions (internal transcribed spacer between 5.8S and 18S), are reported for *P. graminis* (rDNA variations >20%) than *P. betae* (rDNA variations <2%) (123), raising questions about the current taxonomy (**Supplemental Figure 4**).

The low diversity among *P. betae* isolates was confirmed with the full genome sequencing of two *P. betae* isolates from Belgium (A26–41) and the United Kingdom (RES F41), which showed high similarities (~3% coding genes unique to each isolate, 99.87% average nucleotide identity in aligned portions) (34). However, genetic diversity among *P. betae* is probably underestimated, as genetic analyses have only focused on *P. betae* isolates transmitting BNYVV on sugar beets, despite *P. betae* being detected on hosts from at least eight plant families (*Amaranthaceae*, *Asteraceae*, *Brassicaceae*, *Caryophyllaceae*, *Papaveraceae*, *Poaceae*, *Portulaceae*, and *Urticaceae*) (2, 12, 81). The proposition to classify *P. betae* into three special forms (f. sp. *betae*, *amaranthi*, and *portulacae*) due to host and virus specialization (2, 12) further suggests a larger diversity, although this classification still needs to be confirmed at the genetic level.

The high genetic diversity within *P. graminis* rDNA led to its classification into different ribotypes (123, 138). Specific ecological characteristics of ribotypes have led to the distinction of five special forms (80): the two special forms from temperate areas, *temperata* [ribotype (rib) I] and *tepida* (rib II), and the three special forms from tropical areas, *tropicalis* (rib III), *subtropicalis* (rib IV), and *colombiana* (rib V) (80). This classification still needs to be confirmed with other loci. Since the initial classification, additional ribotypes (rib VI and VII) have been identified but have not yet been assigned to a special form (28, 81). Higher genetic diversity was uncovered for rib-I, rib-II, rib-III, and rib-IV, leading to the definition of subribotypes (80, 81). The large sequence differences within ITS regions between ribotypes and the particular ecological characteristics of special forms suggest that *P. graminis* could be split into several species.

Sporosori: clusters of resting spores

Zoospores: spores capable of swimming by the means of flagella

Stachel: needle-like structure for piercing cell wall during penetration of host cell

Plasmodium: life form consisting of a mass of protoplasm and containing many nuclei

Zoosporangium: asexual structure where zoospores are produced

4.2. *Benyviridae* Persistence and Transmission via *Polymyxa* spp.

Benyviruses persist inside resistant resting spores of *Polymyxa* spp., which can survive in soil for over 15 years while retaining their capacity to transmit viruses (1, 113). Yet, BNYVV is rarely observed as virions in *Polymyxa* sporosori (113), and lower quantities of BNYVV viral particles are detected in dried *P. betae* sporosori compared to fresh sporosori (58), suggesting the viruses may persist in another form. *Benyviridae* could persist and be transmitted as ribonucleoprotein complexes, similarly to *Furovirus tritici* (soil-borne wheat mosaic virus), another RNA soil-borne virus transmitted by *P. graminis* (40). This could allow the multipartite virus to maintain its genomic integrity (52).

In the presence of water and host plants, resting spores germinate into motile primary zoospores that encyst on root hairs or epidermal cells and inject their contents into the host cytoplasm via a stachel (**Figure 4**). The resulting plasmodium can initiate either a sporangial phase (producing a zoosporangium that differentiates in secondary zoospores) or a sporogenic phase (producing sporosori), depending on environmental conditions (68, 128). Virus acquisition and transmission likely occur during the injection of zoospore's content into host cells or in the early plasmodia infection stage, when *Polymyxa* and plant cytoplasm are only separated by a thin membrane (21), possibly via pinocytosis (90, 113).

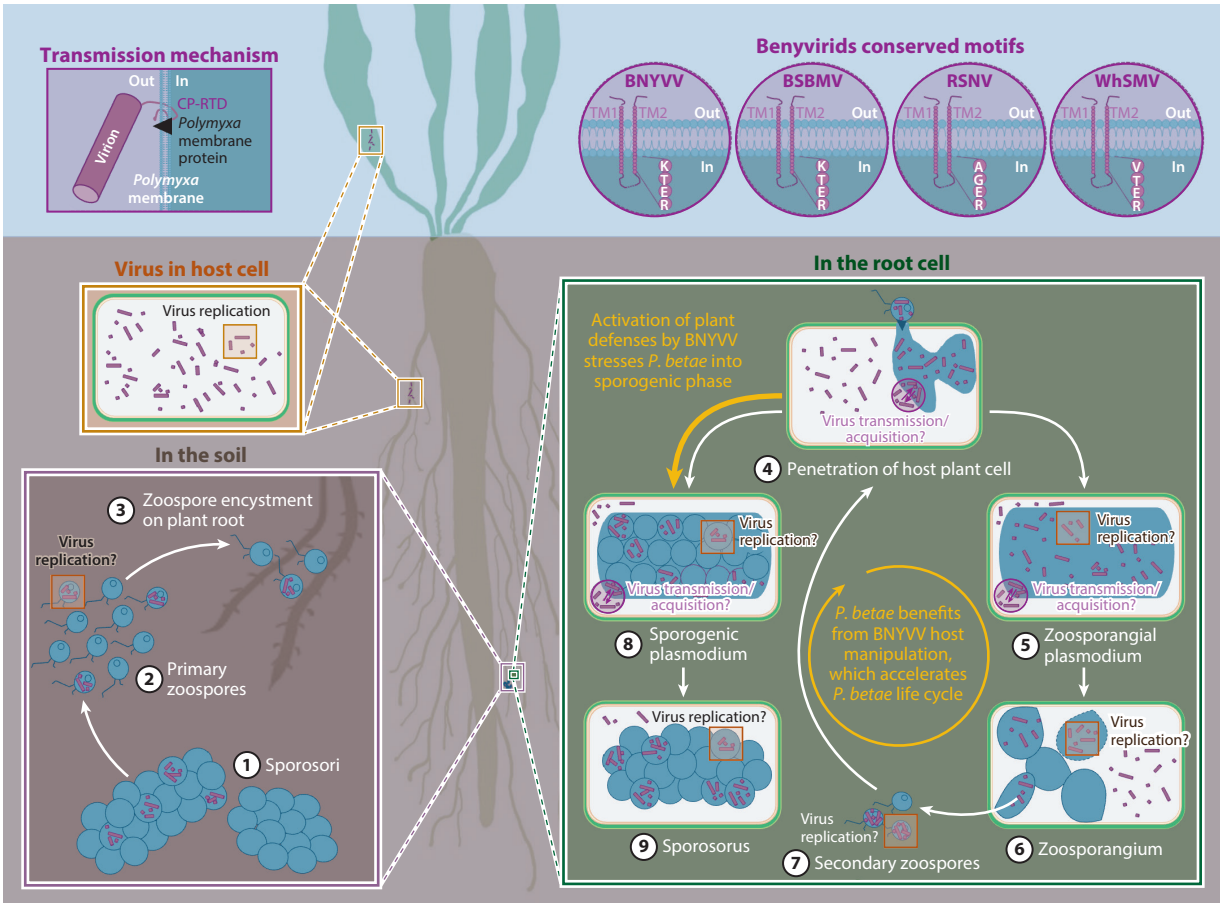


Figure 4

Lifecycle of *Polymyxa betae* in the sugar beet and its role in benyvirid, persistence, potential replication in the vector and mechanisms of acquisition and transmission. *Polymyxa* spp. (*in blue*) life cycle is represented starting from ① dormant viruliferous and aviruliferous sporosori in the soil germinating into ② primary zoospores once the environmental conditions are favorable and the plant is present. ③ Primary zoospores encyst the host cell wall and ④ inject its content into the host cytoplasm with a needle-like structure. Once the content of *Polymyxa* zoospore is inside the host cell, it forms a ⑤ sporangial plasmodium that leads to the formation of a ⑥ zoosporangium producing ⑦ secondary zoospores that can infect other root cells. Alternatively, it forms a ⑧ sporogenic plasmodium that leads to the formation of ⑨ sporosori. Transmission and acquisition of viruses by *Polymyxa* are represented in purple. Transmission is an active mechanism that involves conserved motifs in transmembrane domains in the coat protein readthrough domain of benyvirids: KTER motif for BNYVV (beet necrotic yellow vein virus) and BSBMV (beet soilborne mosaic virus), AGER motif for RSNV (rice stripe necrosis virus), and VTER motif for WhSMV (wheat stripe mosaic virus). Potential replication of benyvirids in *Polymyxa* spp. (*in orange*) is highlighted in different *Polymyxa* life forms. Impacts of BNYVV on sugar beet and subsequent acceleration of *P. betae* life cycle are represented in yellow. Abbreviation: CP-RTD, coat protein readthrough domain.

Transmission of *Benyviridae* by *Polymyxa* spp. is an active mechanism in which TMDs in CP-RTD regions facilitate the movement of virus particles between the host plant cytoplasm and the vector (4, 131). TMDs have been identified for all benyvirids, with the exception of FeBV, for which the CP-RTD is missing, and MILV, for which the CP-RTD is incomplete (**Table 3**), suggesting similar transmission mechanisms. Conserved KTER motifs in TMDs are required for BNYVV and BSBMV transmission by *P. betae* (131). RSNV and WhSMV have distinct motifs (AGER and VTER, respectively) (8, 136), but their roles in *P. graminis* transmission have not been

Table 3 ***Benyviridae*** **known vectors, transmembrane domains (TMD), and conserved amino acids motifs in their CP-RTD**

Virus acronym	Vector	TMD?	Motif	Reference(s)
BNYVV	*Polymyxa betae*	Yes	KTER	1, 4, 131
BSBMV	*P. betae*	Yes	KTER	79
BdMV	Unknown	Yes	Unknown	72
RSNV	*Polymyxa graminis* f. sp. *colombiana*	Yes	AGER[d]	9, 33, 89
WhSMV	*P. graminis* f. sp. *tepida*[a] *P. graminis* f. sp. *temperata*[a] *P. graminis* f. sp. *tropicalis*[a]	Yes	VTER	134, 136
WYSByV	*P. graminis* f. sp. *tepida*[a] *P. graminis* f. sp. *temperata*[a]	Yes[b]	Unknown	57
WBLV1	Unknown	Yes	Unknown	15
MILV	Unknown	Unknown[c]	Unknown	118
FeBV	Unknown	Unknown[c]	Unknown	94

[a]Potential vectors deduced here based on co-occurrences with *Polymyxa* structures in hosts and geographic distribution of *P. graminis* special forms.
[b]Transmembrane domain predicted with DeepTMHMM 1.0 (60).
[c]Complete sequence of CP-RTD not available for transmembrane domain prediction.
[d]Although a KTER motif was reported for one Colombian RSNV isolate, we only found AGER motifs for all RSNV sequences listed by the NCBI.
Abbreviations: BNYVV, beet necrotic yellow vein virus; BSBMV, beet soil-borne mosaic virus; BdMV, burdock mottle virus; CP-RTD, coat protein readthrough domain; FeBV, fern benyvirus; MILV, mangifera indica latent virus; NCBI, National Center for Biotechnology Information; RSNV, rice stripe necrosis virus; WBLV1, wheat beny-like virus 1; WhSMV, wheat stripe mosaic virus; WYSByV, wheat yellow stripe associated with beny-like virus.

confirmed with transmission assays. These differences of motifs could explain the preferential transmission of benyviruses by certain forms of *Polymyxa* (**Table 3**) and suggest a high degree of virus–vector specificity. In addition to TMD, BNYVV RNA4-p31 and BSBMV RNA4-p32 are essential for their efficient transmission (30, 110, 127) and can complement each other for virus transmission by *P. betae* (30, 78). This suggests that other viruses, vectors, or host factors may influence virus transmission by *Polymyxa* spp.

4.3. Nature of *Polymyxa–Benyviridae* Interactions

The potential role of *Polymyxa* spp. as propagative hosts for benyviruses is not clear. On one hand, the loss of virus transmission by *Polymyxa* spp. in resistant host plants (92) and the capacity of BNYVV to persist in host plants and cause severe symptoms independently of the vector's presence or abundance (38, 69) suggest that the virus cannot spread exclusively within the vector's structures and that the vector is not essential for viral replication. On the other hand, the detection of BNYVV movement and replication proteins inside *P. betae* secondary zoospores and sporosori (90) and the differential accumulation of BNYVV viral particles between sporosori and secondary zoospores (58) hint at possible virus replication inside *Polymyxa* spp. However, these viral components might originate in the plant as zoosporangia and secondary zoospores are formed in the host cell.

Regardless of viral replication within *Polymyxa* spp., the *P. betae*–BNYVV relationship could be a win-win. Faster sporosori production and increased *P. betae* DNA quantity during BNYVV infection suggest that the virus accelerates the *P. betae* life cycle (35). The induction of LRPs by BNYVV demonstrates the capacity of the virus to manipulate host root morphology to

accommodate its vector, resulting in higher *P. betae* quantities when inoculated with the virus (35). The production of energy sinks in sugar-beet roots by BNYVV (35) could boost the development of the vector, although the opposite effect could occur in case of a sugar shortage. Weakening of cell walls by BNYVV (35, 46) might also facilitate the faster colonization of the protist in its host. Finally, BNYVV-mediated RNA silencing could contribute to the acceleration of the *P. betae* life cycle. This acceleration could also be the result of higher activation of plant defenses following the infection with BNYVV. Indeed, *P. betae* seems to manipulate the plant defense response to remain undetected (35). The stress of plant defense activation when infected with BNYVV could force the protist into the sporogenic phase, leading to the production of sporosori rather than multiplication structures.

Interactions between *P. graminis* and the benyvirids it transmits are likely different from the *P. betae*–BNYVV interactions, as bipartite *Benyviridae* do not cause rhizomania-like root symptoms.

5. CONTROL OF THE DISEASE

Control measures against *Benyvirus*-associated diseases can be directed against the virus itself or against its vector to limit virus transmission. Several strategies have thus been considered to reduce the amount of *Polymyxa* spp. in the soil or its ability to infect host plants and transmit viruses. Adapted agricultural practices include long, successive crop rotations with nonhost species or delayed sowing. The effect of chemical treatments, soil solarization, and biocontrol agents has also been evaluated (18, 61, 64). In particular, experiments under controlled conditions have shown that antagonistic organisms (*Trichoderma* spp. and *Fusarium oxysporum*) and elicitors (*Bacillus* cyclic lipopeptides) are effective in reducing the infection by *P. betae* (36, 74, 99). However, these control methods are largely ineffective, environmentally unsustainable, and/or prohibitively expensive in the long-term (111). The use of genetic resistance is considered the most efficient and durable means of controlling diseases caused by *Polymyxa* spp. and, for over three decades, research has been conducted to identify such resistances (14, 66).

5.1. Genetic Resistance

Two major and dominant resistance genes against BNYVV, *Rz1* and *Rz2*, have been widely introgressed into commercial sugar-beet varieties (16). *Rz1*, identified in the 1980s, confers partial resistance, resulting in a reduction in symptom severity and viral load, although it does not fully prevent infection. *Rz2*, identified a few years later, provides a higher level of resistance. Both genes act by targeting the virus directly, rather than its vector, and are root specific (86, 140). Although both genes are located on chromosome 3, they have been confirmed to be genetically distinct (115). Given that *Rz2*, and possibly *Rz1*, originate from the wild beet *Beta vulgaris* subsp. *maritima*, this gene pool has been further explored to identify additional resistance sources (16, 141). As a result, three resistance genes—*Rz3*, *Rz4*, and *Rz5*—have been described (49, 141). However, current evidence suggests that *Rz3* likely corresponds to *Rz2*, whereas *Rz4* and *Rz5* may be allelic variants of *Rz1* (23, 55, 115).

Functional characterization of *Rz2* has revealed that it encodes a protein belonging to the nucleotide-binding leucine-rich repeat gene family—the largest class of plant resistance genes, which mediate resistance by recognizing diverse pathogen- or pest-derived effectors and triggering immune responses (23). Transient expression assays in *N. benthamiana* demonstrated that BNYVV TGBp1 acts as the avirulence determinant, whose recognition by Rz2 induces a hypersensitive response (140). Notably, under the same experimental conditions, Rz2 was also shown to recognize the TGBp1 of BSBMV and beet soil-borne virus (*Pomovirus*, *Virgaviridae*). In sugar beet, this recognition translates into effective resistance against BSBMV (140).

In addition to BNYVV-targeting genes, Asher et al. (7) identified natural resistance to colonization by *P. betae* in *B. vulgaris* subsp. *maritima*. Two quantitative trait loci (QTLs), *Pb1* and *Pb2*, have been mapped on chromosomes 4 and 9 and are likely to interact together (7). These loci represent valuable complementary resistance factors that can enhance the efficiency of resistance against rhizomania and contribute to improving its durability.

Transgenic approaches have also been developed to generate resistance to BNYVV in sugar beet. Expression of constructs carrying part of the 5′-UTR and p21 CP gene or part of the replicase gene results in a significant reduction of viral multiplication and leaf symptoms through an RNA silencing mechanism (82, 144). Besides, promising results have also been obtained with sugar beet expressing single-chain fragment antibodies directed against the BNYVV p21 protein (10).

In cereal crops, research on genetic resistance to *Benyvirus* infection is much less advanced than in beet. Nevertheless, resistance to crinkling disease, caused by RSNV, has been identified in the African cultivated rice species *Oryza glaberrima* (39, 59, 100). In certain cases, this resistance may be attributed to reduced colonization by *P. graminis* (39). Additionally, a major QTL associated with reduced disease incidence under field conditions has been mapped to chromosome 11 (59). Whether this QTL confers resistance through targeting the virus or the vector remains to be determined. In wheat, resistance to WhSMV has also been described. It may involve at least two dominant resistance genes, but the resistance mechanism is still unknown (11, 106).

5.2. Resistance Breakdown Mechanisms

Twenty years after the massive introgression of *Rz*1 into commercial varieties resulted in high viral pressure selection, the A-type of BNYVV overcame the *Rz1*-mediated resistance in the United States (87). Other resistance-breaking isolates were also detected in Europe in the following years (108). The molecular basis of resistance-breaking appears to be related to two independent mechanisms. First, mutations leading to *Rz1* resistance-breaking have been identified in BNYVV RNA3-p25 (3, 26). Most of these mutations occur in a stretch of four hypervariable amino acids at positions 67–70 (114). More than 20 tetrads have been identified, but only some have confirmed resistance-breaking properties (AYPR, VLHG, VCHG, DHG/D-HG, TFPR, TYPR, VFHG, VHHG, and VHPG) (71, 85, 86). Second, RNA5, which is present in some BNYVV-J or -P isolates and encodes the p26 protein, is involved in BNYVV accumulation in the *Rz1* background in addition to increasing pathogenicity in susceptible sugar beet (85, 133). The combination of resistance-breaking tetrads on RNA3 with RNA5 resulted in higher accumulation of the BNYVV in resistant sugar-beet cultivars, suggesting that p25 and p26 have complementary effects. To date, no evidence of *Rz2* resistance-breaking has been reported.

6. FUTURE ISSUES

6.1. Diversity and Taxonomy of *Benyviridae*

Further genetic characterization of *Benyviridae* species is rapidly needed to better evaluate their diversity. Phylogenetic revision of the *Benyviridae* family is also required, considering the larger diversity uncovered through metagenomic analyses. Also, sequence quality remains crucial, particularly for new beny-like viruses, to support accurate taxonomic assignments and potentially redefine families or suborders in Hepevirales. Moreover, hosts of beny-like viruses should be confirmed experimentally by verifying the replication and genome expression of the virus in its host via detection of virus particles, complementary RNAs and CP in the host, and transmission with host species.

Emergence events and dispersion routes of *Benyviridae* were analyzed based on sequences of CP in a recent review (31) but warrant further attention. The low genetic diversity observed in

BNYVV and WhSMV may be due to recent host/vector adaptations. The highest diversity of BNYVV is in East Asia, suggesting adaptation to local hosts prior to the introduction of sugar beet (25, 31). Similarly, RSNV and WhSMV may have originated from native grasses before adapting to introduced crops in Africa and South America. Much is to be expected from expanding our knowledge of *Benyviridae* diversity with regard to the evolutive origin of species currently known or yet to be discovered.

Coinfection events also need to be investigated, especially for RSNV, WYSByV, WBLV1, and WhSMV, which can all infect wheat (15, 57, 103, 136). The appearance of reassortments among infectious clones of BNYVV and BSBMV in planta (78) highlights the potential for the evolution of novel infectious combinations. Different virus pairs could be interesting for future reassortment experiments: RSNV–WYSByV, which can both infect wheat and belong to the *Benyvirus* genus, or BdMV–MILV, which have the furthest host ranges, could help correlate genetic distance with protein function. WhSMV and WBLV1, which share hosts and form a distinct clade, are also promising candidates for reassortment experiments. Initially detected in coinfection with potyvirids or tombusvirids, the potential viral interactions and their impact on the hosts need to be investigated. Reassortments of *Benyviridae* inside the vector should also be investigated. Finally, putative satellite viruses have been discovered in rhizomania-infected sugar beets (139). Their role in disease severity and resistance should be investigated.

6.2. Functional Analysis of Viral RNA and Proteins

On the one hand, bipartite and multipartite *Benyviridae* share homologous proteins; however, their functional conservation across species has not been confirmed and could be explored using structural prediction tools. On the other hand, major differences are highlighted among *Benyviridae*, regarding specific proteins (e.g., low identity and size difference of CRP) but more notably regarding the presence or absence of additional RNAs. Although the role of RNA3–4–5 in rhizomania symptoms has been investigated, the implication of these differences for pathogenicity, movement, and transmission is not yet fully understood. Creating additional infectious clones is a key step toward functional characterization of the different RNAs and proteins.

The functional chRNAs associated with BSBMV infection constitute a recent and intriguing finding (97). Whether these chRNAs result from an RNA maturation mechanism intended to promote CP production must be confirmed. Moreover, the presence of coremin motifs and xrRNA elements—key features in chRNA biogenesis—in different viral taxa suggests that chRNAs may play a broader role in viral infection, representing an open question for future investigation within *Benyviridae* and beyond.

6.3. Diversity and Taxonomy of *Polymyxa* spp.

Polymyxa diversity is still poorly understood due to limited sampling, difficulties of multiplication in controlled conditions, and limited sequencing (34). Broader sequencing, including full genomes of different *P. graminis* special forms, and precise characterization of host ranges of *P. graminis* could lead to the description of several species within the current species *P. graminis*. Moreover, most diversity analyses are biased toward agricultural isolates. Metagenomic data mining could help uncover hidden diversity for this genus.

6.4. *Polymyxa*–Virus Interactions

Despite phylogenetic closeness to other plasmodiophorids, the capacity to transmit viruses to plants appears restricted to *Hillenburgia*, *Polymyxa*, and *Spongospora* genera (21, 67, 137). Sequencing and comparison of the first genomes of plasmodiophorids *Plasmodiophora brassicae* (117),

Spongospora subterranea (27), and *P. betae* (34) allows the identification of vector proteins possibly involved in virus transmission. Further genetic, functional, and structural analyses, especially for *P. graminis*, are needed to uncover mechanisms underlying vector–virus specificity. Cultivation of *Polymyxa* spp. in association or not with viruses in controlled conditions is the main challenge for these organisms and a key step to further understand host–*Polymyxa*–virus interactions.

Limited data for WBLV1, BdMV, MILV, and FeBV hinder the identification of vectors for these viruses. Investigating conserved motifs possibly involved in transmission and testing their role in vector specificity (e.g., CP-RTD motif swapping in BNYVV) will be key to progress in our understanding of virus transmission by *Polymyxa*. Furthermore, it remains unknown whether *Benyviridae* particles are transmitted as grouped or random units in *Polymyxa* as well as how genome integrity is maintained within hosts. These questions need further elucidation, in line with research on multipartite viruses transmitted by insects (93).

Assessing the potential replication of *Benyviridae* within *Polymyxa* is a critical point that may be achieved by detecting complementary RNAs and viral proteins in germinating primary zoospores while combining different detection methods (RT-PCR, northern blot, ELISA) or by analyzing GFP-tagged clones in *Polymyxa* zoospores.

Finally, although manipulation of root physiology by BNYVV to accommodate *P. betae* has been observed for the rhizomania pathosystem (35), the lack of rhizomania symptoms for other benyvirids suggests different *Polymyxa*–benyvirids interactions. Investigating the impact of other *Benyviridae* on *Polymyxa* is therefore essential for the understanding of these pathosystems. Manipulation of vectors by benyvirids should also be explored (e.g., impact of benyvirids on vector host range).

6.5. Control Strategies

Sustained research into resistance genes is critical for breeding resistant crops. Vector multiplication, virus acquisition, and virus transmission under controlled conditions are key steps required for the quantitative assessment and subsequent characterization of resistance genes. In addition, given the complexity of natural transmission, infectious clones represent a valuable tool to clarify resistance mechanisms. Resistance must be addressed at both the virus and vector levels, and expanding our understanding of *Polymyxa* spp. diversity and their interactions with host plants could uncover new resistance sources. Combining natural resistance genes may yield more durable protection, but advanced molecular approaches such as RNA interference, which have already been proven to be effective (83, 102), and precision genome editing should offer new opportunities for the development of broad-spectrum and sustainable resistances in the coming years.

In addition, epidemiological surveillance has to be improved through the use of rapid detection methods such as RT-LAMP (5, 109), and monitoring of new benyvirid species should be reinforced in light of potential mutations, coinfections, recombinations, and reassortments between species.

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The authors are not aware of any affiliations, memberships, funding, or financial holdings that might be perceived as affecting the objectivity of this review.

AUTHOR CONTRIBUTIONS

M.G. and E.L.J. wrote the initial version of the review under the coordination of A.L., C.B., E.H., and L.A. Each author contributed mostly according to their expertise: E.H. performed phylogenetic analysis and contributed to the viral diversity section, A.L. focused on the *Polymyxa* section, L.A. contributed to the sections on virus resistance. C.B. contributed to the introduction and

served as the corresponding author. C.B., E.H., and L.A. also contributed to the plant host molecular interaction section. M.G. and E.L.J. prepared tables and figures. All authors contributed to manuscript review, verification, and completion.

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