Role of the Insect Supervectors *Bemisia tabaci* and *Frankliniella occidentalis* in the Emergence and Global Spread of Plant Viruses

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Annu. Rev. Virol. 2015. 2:67–93

The *Annual Review of Virology* is online at virology.annualreviews.org

This article's doi: 10.1146/annurev-virology-031413-085410

Keywords

begomovirus, ipomovirus, torradovirus, tospovirus, ilarvirus, virus evolution

Abstract

Emergence of insect-transmitted plant viruses over the past 10–20 years has been disproportionately driven by two so-called supervectors: the whitefly, *Bemisia tabaci*, and the Western flower thrips, *Frankliniella occidentalis*. High rates of reproduction and dispersal, extreme polyphagy, and development of insecticide resistance, together with human activities, have made these insects global pests. These supervectors transmit a diversity of plant viruses by different mechanisms and mediate virus emergence through local evolution, host shifts, mixed infections, and global spread. Associated virus evolution involves reassortment, recombination, and component capture. Emergence of *B. tabaci*–transmitted geminiviruses (begomoviruses), ipomoviruses, and torradoviruses has led to global disease outbreaks as well as multiple paradigm shifts. Similarly, *F. occidentalis* has mediated tospovirus host shifts and global dissemination and the emergence of pollen-transmitted ilarviruses. The plant virus–supervector interaction offers exciting opportunities for basic research and global implementation of generalized disease management strategies to reduce economic and environmental impacts.

INTRODUCTION

Viruses likely emerged well before eukaryotic organisms (1). Thus, ancestral plant viruses have coevolved with hosts over long periods of time to develop successful strategies for replication, gene expression, and cell-to-cell and long-distance movement. Plant-to-plant movement was an additional challenge because plants are stationary. Plant-feeding insects provided an ideal solution. Indeed, roughly 75% of plant viruses are transmitted by insects. The majority of these are hemipterans, including aphids, leafhoppers, and whiteflies (2). Evolution of piercing-sucking mouthparts allowed for acquisition of nutrition from the phloem, the primary conduit for long-distance movement of viruses, making these insects effective viral vectors. Transmission of plant viruses by insect vectors represents a complex interaction between plant, virus, and vector. Depending on the nature of the interaction, at least four mechanisms of insect transmission are recognized: nonpersistent (most aphid-transmitted viruses), semipersistent (beetle- and some whitefly-transmitted viruses), persistent circulative (some aphid- and most leafhopper- and whitefly-transmitted viruses), and persistent propagative (thrips- and some leafhopper- and aphid-transmitted viruses). Recent reviews have addressed various aspects of insect-transmitted viruses, including mechanisms of transmission (2–7).

Over the past 10–20 years, changes in the predominance of insect-transmitted viruses in agricultural ecosystems have occurred due to virus emergence and evolution. We define an emerging virus as a previously unknown virus or a known virus that has expanded its geographical range, incidence, and importance because of an arthropod vector. The reasons for virus emergence include evolution of new viruses or new strains of existing viruses; changes in the geographical range, properties, and populations of insect vectors; changes in agricultural practices; and climate change (8–11). One of the most notable trends has been the emergence of two insect vectors, *Bemisia tabaci* (sweet potato, tobacco, or silverleaf whitefly) and *Frankliniella occidentalis* (Western flower thrips), as driving forces in the emergence of plant viruses in agricultural ecosystems (**Figure 1**). These two vectors alone transmit >300 viruses representing 10 genera (**Table 1**) (12–14). These include DNA and RNA viruses and viruses with geminate, spherical, rod-shaped, and enveloped virions. Therefore, *B. tabaci* and *F. occidentalis* are considered to be supervectors of plant viruses, although we recognize that other vectors may share some of these properties [e.g., the green peach aphid (*Myzus persicae*)] or be more important in certain geographical regions. In this review, we describe the properties of the *B. tabaci* and *F. occidentalis* supervectors, provide examples of plant viruses that have emerged because of these vectors, and discuss aspects of how these vectors have mediated virus emergence.

What Properties Make *Bemisia tabaci* and *Frankliniella occidentalis* Supervectors of Plant Viruses?

Although native to different parts of the world, *B. tabaci* (B biotype or Middle East–Asia Minor 1 species, unless otherwise noted), from India or the Middle East, and *F. occidentalis*, from western North America, have attained global pest status (15–17). Characteristics of these supervectors include a high rate of reproduction (fecundity), development of large populations on plants (**Figure 1**), dispersal ability (i.e., short distances by flying and long distances via wind currents or in association with plants), extreme polyphagy, and coping with or altering plant defense mechanisms. They have piercing-sucking mouthparts and feed on a large number of cultivated and noncultivated plants: 500 species in 74 families for *B. tabaci* (15) and over 500 species in 60 families for *F. occidentalis* (18). Both insects effectively metabolize, detoxify, or alter pesticides and toxic plant compounds. Their exposure to a wide range of insecticides has led to emergence of

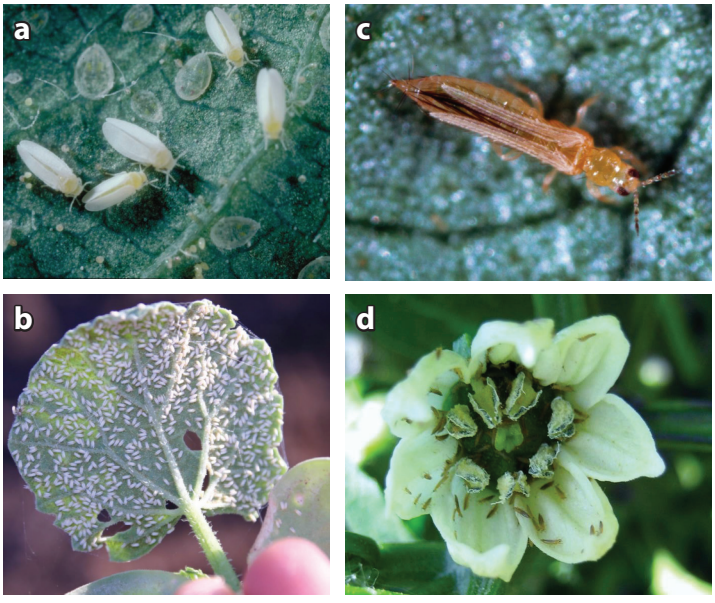


Figure 1

Supervectors driving the emergence of insect-transmitted viruses. (*a*,*b*) *Bemisia tabaci*. (*a*) Close-up of *B. tabaci* adults, pupae, and nymphs on the undersurface of a leaf. Photograph by Eric Natwick, University of California Cooperative Extension. (*b*) A large population of *B. tabaci* adults feeding on the undersurface of a melon leaf. Photograph by Eric Natwick, University of California Cooperative Extension. (*c*,*d*) *Frankliniella occidentalis*. (*c*) Close-up of an adult thrips. Photograph by Jack Kelly Clark, University of California Statewide Integrated Pest Management Program. (*d*) Adult thrips in a pepper flower. Photograph by Ozgur Batuman, University of California, Davis.

insecticide-resistant populations. Small size and thigmotactic nature (thrips), relatively short life cycles, long life spans of females, and pest status on ornamentals have facilitated global spread. Together, these insects transmit a diversity of plant viruses via multiple transmission mechanisms (**Table 1**). In some cases, this involves intricate persistent interactions, whereas in others the interaction is less specific and semipersistent. Interestingly, these two supervectors transmit distinct taxonomic groups of plant viruses, which may relate to differences in feeding behavior, receptors, or other factors. For example, *B. tabaci* is a phloem-feeding insect that typically does not probe extensively before feeding, making it an effective vector of phloem-limited and phloem-associated viruses (14–16); in contrast, *F. occidentalis* feeds on epidermal and mesophyll cells and transmits non-phloem-limited viruses (17). Finally, these properties have allowed these supervectors to drive emergence and evolution of plant viruses, leading to paradigm shifts.

Supervectors Mediate Global Spread of Economically Important Plant Viruses

The global spread of a number of economically important plant viruses has been facilitated by these supervectors. A key factor in this phenomenon has been increased, unregulated global trade of plants, especially ornamentals. This has led to the global dissemination of *B. tabaci* and *F. occidentalis*. Because of their polyphagous nature and their capacity to transmit a diversity of viruses, these vectors can acquire and inoculate viruses from many host species, thereby increasing the

Table 1 Properties of plant virus genera transmitted by the supervectors *Bemisia tabaci* and *Frankliniella occidentalis*

Supervector	Genus	Properties		Mode of transmission	Number of species[a]
		Virion shape and size	Genetic material		
Bemisia tabaci (sweet potato/tobacco/silverleaf whitefly)	*Begomovirus*	Geminate, 18 × 30 nm	ssDNA	Persistent circulative	288
	Ipomovirus	Flexuous, filamentous, 12–15 × 800–950 nm	ssRNA	Semipersistent	6
	Crinivirus	Flexuous, filamentous, 10–13 × 650–900 nm	ssRNA	Semipersistent	13
	Carlavirus	Flexuous, filamentous, 12–13 × 470–1,000 nm	ssRNA	Semipersistent	2 (of 52)[b]
	Torradovirus	Spherical, 28–30 nm	ssRNA	Semipersistent	2 (3)[c]
Frankliniella occidentalis (Western flower thrips)	*Tospovirus*	Enveloped, pleomorphic, spherical, 80–120 nm	Ambisense ssRNA	Persistent propagative	9 (14)[c]
	Ilarvirus	Quasi-spherical, 29 nm	ssRNA	Thrips-mediated (pollen)	19 (3)[c]
	Machlomovirus	Spherical, 28–34 nm	ssRNA	Semipersistent	1
	Carmovirus	Spherical, 28–34 nm	ssRNA	Thrips-mediated (pollen)	1 (of 19)[b]
	Sobemovirus	Spherical, 25–33 nm	ssRNA	Thrips-mediated (pollen)	1 (of 14)[b]
				Total number of species	362

[a]Official number of species listed by the International Committee on Taxonomy of Viruses.
[b]Number (out of the total) of species in the genus reported to be transmitted by these supervectors.
[c]Number in parentheses indicates additional proposed/accepted species.

number and types of virus-infected plants transported over long distances. Such transport is further facilitated by the fact that some of these plants have symptomless infections, either because they are true symptomless hosts or because they have not yet developed symptoms, allowing for unknowing transport. Once such plants are transported, the global distribution of these polyphagous vectors allows for virus acquisition, including from low-titer symptomless hosts, and the subsequent establishment, amplification, and spread of an exotic virus in a new geographical location. The persistent association of some viruses with these vectors (e.g., begomoviruses with *B. tabaci* and tospoviruses with *F. occidentalis*) also allows for global transport in juvenile, pupal, or adult stages. However, this is not a prerequisite, as some semipersistently transmitted viruses have also spread globally, such as the crinivirus *Cucurbit yellow stunting disorder virus* in association with *B. tabaci* (8).

EMERGENT VIRUSES DRIVEN BY THE SUPERVECTOR *BEMISIA TABACI*

Geminiviruses

Geminiviruses have emerged as the largest and one of the most diverse plant virus families. These viruses have small (~2.6–5.2-kb) circular ssDNA genomes encapsidated in twinned quasi-icosahedral virions (19–22). Geminiviruses are closely associated with the phloem of plants, are not seed-transmitted, and utilize *B. tabaci* and various leafhoppers for plant-to-plant spread. Plant diseases caused by geminiviruses are among the most damaging and economically important in the world (8–10, 23).

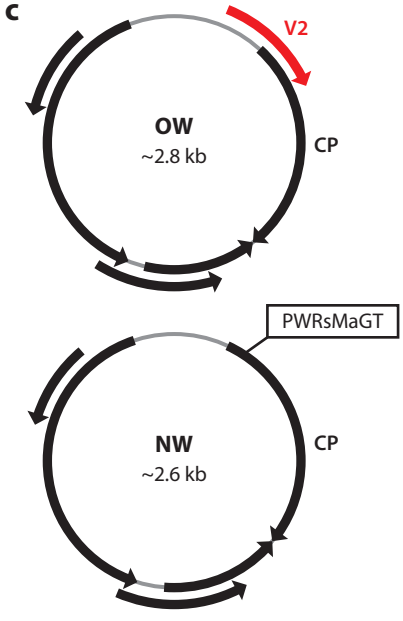
The family Geminiviridae is divided into seven genera based on genome size and structure, phylogenetic relationships, type of insect vector, and host range (20–22). The genus *Begomovirus* contains viruses transmitted by *B. tabaci* and has the largest number of species (288 of 325) (24). Viruses in the genera *Curtovirus* and *Mastrevirus* are transmitted by leafhoppers, whereas the single species in the genus *Topocuvirus* is transmitted by a treehopper (20, 21). Application of rolling-circle amplification and other new tools (25–27) has resulted in the identification of new geminiviruses and geminivirus-like viruses, some of which have been accommodated in three newly created genera: *Becurtovirus*, *Eragrovirus*, and *Turncurtovirus* (28). These genera have a small number of species (one or two), infect dicot or monocot hosts, and are either known or suspected to be vectored by leafhoppers.

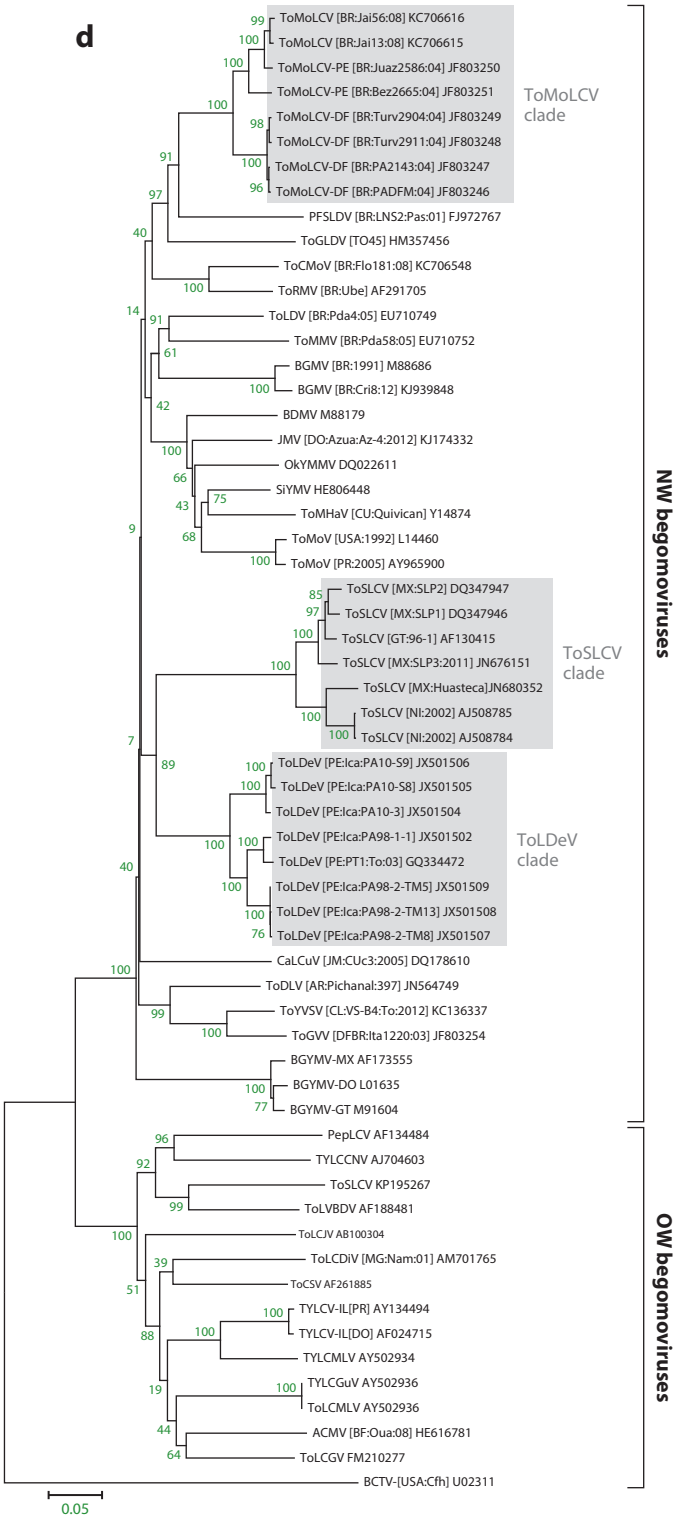
Begomoviruses: a genus of viruses that has emerged through the activities of the *Bemisia tabaci* supervector. The remarkable emergence of begomoviruses has been driven by the *B. tabaci* supervector. The prevalence and economic importance of begomovirus diseases in tropical and subtropical regions closely mirror the distribution and polyphagous feeding habits of *B. tabaci*. Its key role is evident when considering that, although geminiviruses are ancient viruses (20–22), emergence of the majority of begomovirus species has occurred over the past 20 years and has followed the global spread of *B. tabaci*. Begomoviruses are geographically separated into New World (NW) and Old World (OW) viruses, likely a consequence of continental drift. NW begomoviruses have bipartite genomes (two ~2.6-kb ssDNA components referred to as DNA-A and DNA-B), whereas most OW viruses (~85%) have monopartite genomes (one ~2.8-kb genomic DNA) and associated ssDNA satellites (20–22). Prior to the global spread of *B. tabaci*, begomoviruses in the NW and OW were widely distributed in noncultivated plants (e.g., weeds), presumably via indigenous whiteflies. Furthermore, in many regions of the world, beans, cucurbits, and tomatoes were cultivated for hundreds of years without major begomovirus disease outbreaks. As the *B. tabaci* supervector spread globally, crop-infecting begomoviruses subsequently emerged, evolving from local viruses and reflecting ancient lineages. This local evolution resulted in a diversity of begomovirus species causing similar disease symptoms in susceptible crop plants in distinct geographical regions (8–10, 14, 29–35).

A new direction in begomovirus evolution: emergence of indigenous New World monopartite begomoviruses. The recent emergence of indigenous NW monopartite begomoviruses is an example of a paradigm shift mediated by the *B. tabaci* supervector. The established paradigm was that all NW begomoviruses had bipartite genomes (22). In the mid-1990s, a new leaf curl disease of tomato appeared in Peru following outbreaks of *B. tabaci* (**Figure 2*a***), and a similar disease appeared in tomatoes in northwestern Brazil following the introduction of this supervector (**Figure 2*b***). Although these symptoms were suggestive of a begomovirus disease, they were not typical of NW bipartite begomoviruses (36, 37). A begomovirus DNA-A


a


b


c
V2
OW
~2.8 kb
CP
PWRsMaGT
NW
~2.6 kb
CP


d
ToMoLCV [BR:Jai56:08] KC706616
ToMoLCV [BR:Jai13:08] KC706615
ToMoLCV-PE [BR:Juaz2586:04] JF803250
ToMoLCV-PE [BR:Bez2665:04] JF803251
ToMoLCV-DF [BR:Turv2904:04] JF803249
ToMoLCV-DF [BR:Turv2911:04] JF803248
ToMoLCV-DF [BR:PA2143:04] JF803247
ToMoLCV-DF [BR:PADFM:04] JF803246
ToMoLCV clade
PFSLDV [BR:LNS2:Pas:01] FJ972767
ToGLDV [TO45] HM357456
ToCMoV [BR:Flo181:08] KC706548
ToRMV [BR:Ube] AF291705
ToLDV [BR:Pda4:05] EU710749
ToMMV [BR:Pda58:05] EU710752
BGMV [BR:1991] M88686
BGMV [BR:Cri8:12] KJ939848
BDMV M88179
JMV [DO:Azua:Az-4:2012] KJ174332
OkYMMV DQ022611
SiYMV HE806448
ToMHaV [CU:Quivican] Y14874
ToMoV [USA:1992] L14460
ToMoV [PR:2005] AY965900
ToSLCV [MX:SLP2] DQ347947
ToSLCV [MX:SLP1] DQ347946
ToSLCV [GT:96-1] AF130415
ToSLCV [MX:SLP3:2011] JN676151
ToSLCV [MX:Huasteca]JN680352
ToSLCV [NI:2002] AJ508785
ToSLCV [NI:2002] AJ508784
ToSLCV clade
ToLDeV [PE:Ica:PA10-S9] JX501506
ToLDeV [PE:Ica:PA10-S8] JX501505
ToLDeV [PE:Ica:PA10-3] JX501504
ToLDeV [PE:Ica:PA98-1-1] JX501502
ToLDeV [PE:PT1:To:03] GQ334472
ToLDeV [PE:Ica:PA98-2-TM5] JX501509
ToLDeV [PE:Ica:PA98-2-TM13] JX501508
ToLDeV [PE:Ica:PA98-2-TM8] JX501507
ToLDeV clade
CaLCuV [JM:CUc3:2005] DQ178610
ToDLV [AR:Pichanal:397] JN564749
ToYVSV [CL:VS-B4:To:2012] KC136337
ToGVV [DFBR:Ita1220:03] JF803254
BGYMV-MX AF173555
BGYMV-DO L01635
BGYMV-GT M91604
NW begomoviruses
PepLCV AF134484
TYLCCNV AJ704603
ToSLCV KP195267
ToLVBDV AF188481
ToLCJV AB100304
ToLCDiV [MG:Nam:01] AM701765
ToCSV AF261885
TYLCV-IL[PR] AY134494
TYLCV-IL[DO] AF024715
TYLCMLV AY502934
TYLCGuV AY502936
ToLCMLV AY502936
ACMV [BF:Oua:08] HE616781
ToLCGV FM210277
BCTV-[USA:Cfh] U02311
OW begomoviruses
0.05

component, but not a DNA-B component, was detected in plants with these symptoms from Peru, and molecular characterization indicated it was the DNA-A component of a new species of NW bipartite begomovirus (**Figure 2*c*,*d***). This new virus was named *Tomato leaf deformation virus* (ToLDeV) (36). Definitive evidence that ToLDeV was a bona fide NW monopartite begomovirus came from the fulfillment of Koch's postulates, in which tomato plants agroinoculated with the cloned ToLDeV DNA-A component developed leaf curl symptoms indistinguishable from those observed in the field (37, 38). Similar results have been obtained for *Tomato mottle leaf curl virus* (ToMoLCV), from northeastern Brazil (**Figure 2*b*,*d***), and *Tomato severe leaf curl virus* (ToSLCV), from Central America and Mexico (**Figure 2*d***). Thus, the *B. tabaci* supervector is driving the emergence of indigenous NW monopartite begomovirus species, a new direction in geminivirus evolution, in distinct geographical regions.

Begomovirus satellites: hijacked components of ssDNA viruses. Most OW monopartite begomoviruses are associated with ssDNA satellites that play a role in pathogenicity. There are two major types: alphasatellites and betasatellites, both of which are ~1.4 kb, half the size of an OW monopartite geminivirus genome. Alphasatellites are dependent on a helper begomovirus for spread but not replication, they do not play a major role in symptoms, and they were captured from nanoviruses (39, 40). Betasatellites are dependent on a helper begomovirus for replication and spread, they do play an important role in symptoms, and their origin is unknown (39). A comprehensive review of begomovirus satellites was recently published by Zhou (39), so only a few points are mentioned here. First, the association of betasatellites with begomoviruses is host specific. They are either required for begomovirus symptom development (malvaceous hosts), required in the case of only some virus-host combinations (solanaceous hosts), or not required for begomovirus symptom development (many legume and cucurbit hosts) (39, 41). Second, betasatellites provide a benefit to the helper virus and *B. tabaci* by suppressing gene silencing and reducing expression of insect defense response genes, respectively (39, 42). Third, betasatellites are promiscuous and can be replicated by a wide range of helper begomoviruses, even a monocot-infecting mastrevirus (43), and in a wide range of host plants. Finally, the betasatellite must be encapsidated in the helper virus capsid protein for plant-to-plant spread by *B. tabaci*. The acquisition of begomovirus satellites by component capture from other ssDNA viruses was facilitated by the polyphagous feeding activities of the *B. tabaci* supervector and has resulted in the emergence and spread of new disease complexes (39).

Emergence of new geminivirus-satellite disease complexes mediated by *Bemisia tabaci*. In Mali, West Africa, a leaf curl disease of okra is caused by the monopartite begomovirus *Cotton*

←

Figure 2

A new direction in begomovirus evolution mediated by the supervector *Bemisia tabaci*: emergence of indigenous New World (NW) monopartite begomoviruses. (*a*) Stunting and leaf curl symptoms in tomato plants in Peru induced by the indigenous NW monopartite begomovirus *Tomato leaf deformation virus* (ToLDeV). (*b*) Leaf curling, distortion, and vein swelling and purpling symptoms in a tomato plant in northeastern Brazil (Piaui state) induced by the indigenous NW monopartite begomovirus *Tomato mottle leaf curl virus* (ToMoLCV). (*c*) Comparison of the properties of the genomic DNAs of Old World (OW) and NW monopartite begomoviruses: The typical genomic DNA of OW viruses is ~2.8 kb, possesses the V2 gene (*red arrow*), and lacks the NW amino acid motif (PWRsMaGT) in the capsid protein (CP), whereas the genomic DNA of NW viruses is ~2.6 kb, lacks the V2 gene, and possesses the NW amino acid motif in the CP. (*d*) Phylogenetic tree showing that the sequences of the indigenous NW monopartite begomoviruses ToLDeV, ToMoLCV, and *Tomato severe leaf curl virus* (ToSLCV) cluster with DNA-A components of NW bipartite begomoviruses and emerged independently. Virus abbreviations are as described in Reference 24.

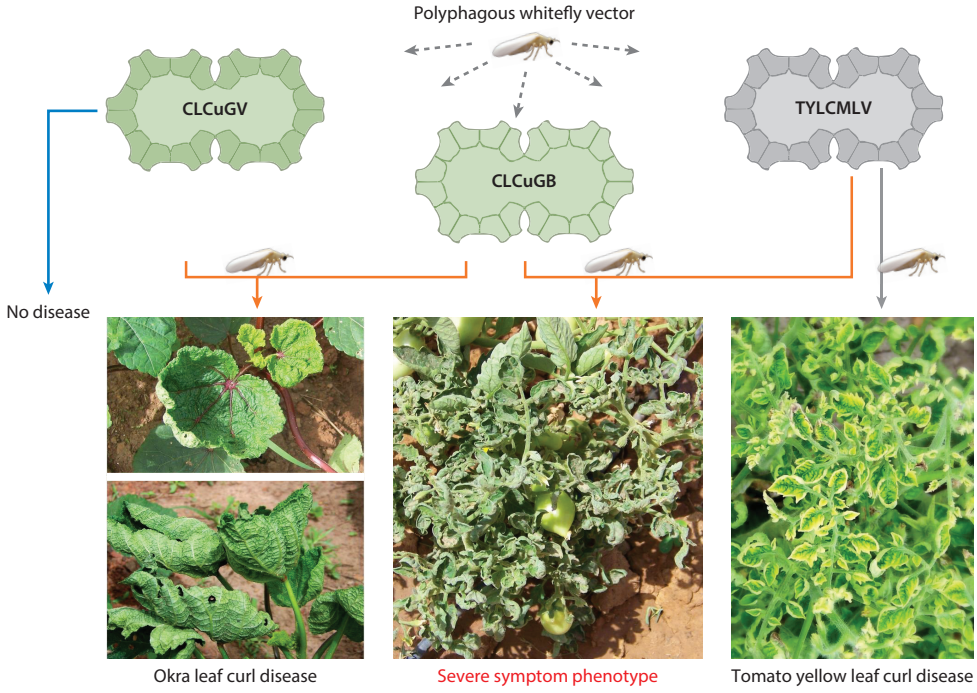


Figure 3

The supervector *Bemisia tabaci* has driven the emergence of a new tomato disease with a severe symptom phenotype in Mali, West Africa, caused by a reassortant of the tomato-infecting begomovirus *Tomato yellow leaf curl Mali virus* (TYLCMLV) and the malvaceous betasatellite Cotton leaf curl Gezira betasatellite (CLCuGB). TYLCMLV alone induces tomato yellow leaf curl disease (*gray arrow*), whereas the severe symptom phenotype develops in tomatoes coinfected with TYLCMLV and CLCuGB (*orange bracket and arrow on right*). *Cotton leaf curl Gezira virus* (CLCuGV) is the cognate helper virus for CLCuGB; okra plants coinfected with CLCuGV and CLCuGB develop okra leaf curl disease (*orange bracket and arrow on left*), whereas okra plants infected with CLCuGV alone develop no symptoms (*blue arrow*; plants not shown).

leaf curl Gezira virus (CLCuGV) and cognate Cotton leaf curl Gezira betasatellite (CLCuGB) (44), whereas a yellow leaf curl disease of tomato is caused by *Tomato yellow leaf curl Mali virus* (TYLCMLV) (45); both diseases are transmitted by *B. tabaci* and cause economic losses (**Figure 3**). In addition, a new disease of tomato appeared, characterized by extremely stunted and distorted plants, and was referred to as the severe symptom phenotype. Tomatoes with this new disease were coinfected with TYLCMLV and CLCuGB (45) (**Figure 3**). Thus, TYLCMLV was a helper virus for this malvaceous betasatellite, which was functional (pathogenic) in a heterologous host (tomato). The polyphagous *B. tabaci* supervector therefore mediated the emergence of a new disease caused by a begomovirus/betasatellite reassortant (45) (**Figure 3**).

In India, the leafhopper-transmitted monocot-infecting mastrevirus *Wheat dwarf India virus* (WDIV) is sympatric with *B. tabaci*–transmitted begomoviruses and satellites. Reassortants of WDIV and begomovirus satellites were detected, and the satellites increased disease severity through suppression of gene silencing (43). This is the first report of geminivirus satellites occurring outside of the genus *Begomovirus*, which likely involved mixed infections mediated by both leafhopper and *B. tabaci* vectors. This further shows the genetic flexibility of satellites and how *B. tabaci* can drive the emergence of new combinations of geminiviruses and satellites in mixed

infections. This also may be an example of global climate change bringing together viruses and vectors from tropical and temperate regions.

Differences in *Bemisia tabaci* biology and vectoring capacity can drive the emergence of begomovirus diseases. The species *B. tabaci* is composed of morphologically indistinguishable biotypes or species that can be differentiated on the basis of molecular and biological characteristics (16). The B biotype is the major driver of begomovirus emergence, but recent evidence suggests a more complicated situation, with biotypes differing in transmission efficiency, competing with and displacing each other, and having mutualistic interactions with begomoviruses.

For example, *Tomato leaf curl Taiwan virus* (ToLCTWV) was recently displaced as the predominant tomato-infecting begomovirus in Taiwan due to the introduction of the invasive *B. tabaci* B biotype and *Tomato yellow leaf curl Thailand virus* (TYLCTHV) (46). The displacement of ToLCTWV was attributed to a higher rate of transmission of TYLCTHV by the invasive *B. tabaci*. A more complex situation in China involves a mutualistic interaction between *B. tabaci* biotypes and begomoviruses (47). The invasive *B. tabaci* B biotype appeared in China in the mid-1990s and displaced the indigenous biotype (ZHJ2) (48). This was followed by the emergence of begomoviruses, such as *Tomato yellow leaf curl China virus* and a betasatellite. The B biotype was subsequently displaced by the insecticide-resistant Q biotype, introduced in 2003 (49–51). In 2006, *Tomato yellow leaf curl virus* (TYLCV) was introduced, and by 2012, it had become predominant (32). Emergence and spread of TYLCV was specifically mediated by the Q biotype due to indirect (vector preference for TYLCV-infected plants) and direct (altered feeding behavior) vector manipulation by the virus (47). Furthermore, feeding on TYLCV-infected plants increased fecundity and survival of the Q biotype but was deleterious to the B biotype, and the Q biotype had higher rates of horizontal (sexual) passage of TYLCV (51). Thus, this mutualistic interaction favored both the virus and the vector. It will be of interest to determine whether mutualism occurs in other *B. tabaci*–begomovirus interactions and the impact of such mutualism on begomovirus diversity and prevalence. It will also be of interest to identify the factor or factors in TYLCV-infected plants that are favorable to one biotype but detrimental to another.

***Bemisia tabaci* is driving the global spread and emergence of New World and Old World begomoviruses.** *B. tabaci* has also played a key role in the global spread of begomoviruses, which are not seed-transmitted. The most well documented example is the OW monopartite TYLCV, which was introduced into the NW in the early 1990s (52, 53) and has spread extensively, following the path of the invasive *B. tabaci* (54). Interestingly, of the ~60 known tomato-infecting begomoviruses, only TYLCV has spread globally. This suggests that specific factors in the TYLCV–*B. tabaci* interaction have facilitated its global spread, such as the above-mentioned mutualism and the capacity for horizontal (sexual) and vertical (transovarial) transmission. These and other findings have been the basis for suggesting that the mechanism of transmission of TYLCV by *B. tabaci* is in between persistent circulative and persistent propagative, possibly involving viral replication in cells of the insect or in the endosymbionts (5, 51).

Global spread is not unique to TYLCV, because there are recent examples for other begomoviruses and other types of whitefly-transmitted viruses (8–10, 14). In 2002, the NW bipartite begomovirus *Squash leaf curl virus* (SLCV) was detected in Israel, and it subsequently spread throughout the Middle East. The resulting sympatry of SLCV and the indigenous OW bipartite begomovirus *Watermelon chlorotic stunt virus* led to the emergence of a new disease of melon caused by mixed infection with these viruses (55). Other examples of recent intra- and intercontinental spread include introductions of the NW bipartite begomovirus *Cucurbit leaf crumple virus* (56) from the southwestern United States into Florida (57, 58) and *Tomato leaf curl New Delhi virus*

from India to southern Spain (59). Finally, CLCuGV, which together with CLCuGB causes cotton leaf curl disease in Africa, was recently detected infecting cotton in Pakistan (60). The cognate CLCuGB was not detected, but CLCuGV interacted with Asian betasatellites, revealing another example of *B. tabaci* mediating a functional reassortant between a begomovirus and a noncognate betasatellite.

A begomovirus without a supervector means no disease epidemic. An introduced begomovirus or begomovirus-betasatellite complex may not emerge due to the absence of *B. tabaci*. When TYLCV was introduced into southern California in 2007 (61), there was concern it could cause substantial losses to the approximately $1 billion/year processing tomato industry. However, most of this production is in central and northern counties that have cold winter temperatures not tolerated by *B. tabaci*. The winter also provides a natural 3–4-month tomato-free period. This has limited the spread and establishment of TYLCV in California.

The yellow vein symptoms in Gold Net or Yellow Net cultivars of Japanese honeysuckle (*Lonicera japonica*) are due to infection with OW monopartite begomoviruses and a betasatellite (62). These aesthetically pleasing (but invasive) ornamental plants, which are native to Japan, have been spread globally. The honeysuckle begomoviruses also cause a yellow stunting disease of tomato (62, 63), although the capacity of these viruses to be whitefly-transmitted is not clear. Thus, these ornamental honeysuckle plants could serve as a Trojan horse for global spread of tomato-infecting begomoviruses and betasatellites. Indeed, a honeysuckle plant with yellow vein symptoms on the University of California, Davis, campus was infected with *Honeysuckle yellow vein virus* (HYVV); a betasatellite, Honeysuckle yellow vein betasatellite (HYVVB); and a recombinant betasatellite (T. Kon & R.L. Gilbertson, unpublished observations). Tomato and tobacco plants agroinoculated with this HYVV isolate developed stunting and leaf curling symptoms, and HYVVB increased symptom severity. Therefore, this honeysuckle plant contained a potential tomato-infecting begomovirus and betasatellite and represents one of the first detections of a betasatellite in the NW. The probability of this complex jumping into tomato in California is unlikely because (*a*) the *B. tabaci* vector is not indigenous in northern California, (*b*) HYVV isolates may not be whitefly-transmissible (i.e., honeysuckle is a dead-end host), and (*c*) these viruses inefficiently infect tomato, even in Japan and Korea.

Ipomoviruses

The capacity of *B. tabaci* to transmit diverse types of plant viruses and to do so by different mechanisms is exemplified by vectoring viruses in the genus *Ipomovirus* (family Potyviridae). Transmission by *B. tabaci* sets ipomoviruses apart from other genera of this family, and the mechanism of transmission is semipersistent. Like many other potyviruses, ipomoviruses have flexuous rod-shaped particles 800 to 950 nm in length and monopartite positive-sense ssRNA genomes of 9.0 to 10.8 kb (64).

The emergence of potyviruses transmitted by *Bemisia tabaci*. Ipomoviruses have relatively recently emerged on a global scale (65). Six species are currently recognized: *Cassava brown streak virus* (CBSV), *Cucumber vein yellowing virus* (CVYV), *Squash vein yellowing virus* (SqVYV), *Sweet potato mild mottle virus* (SPMMV), *Tomato mild mottle virus* (TomMMoV), and *Ugandan cassava brown streak virus* (UCBSV). As with other potyviruses, the ipomovirus genome has a single open reading frame that encodes 11 mature proteins (by polyprotein processing and an additional overlapping open reading frame) (66). However, three different types of ipomovirus genome organization are currently known and have been recently reviewed (65, 67, 68).

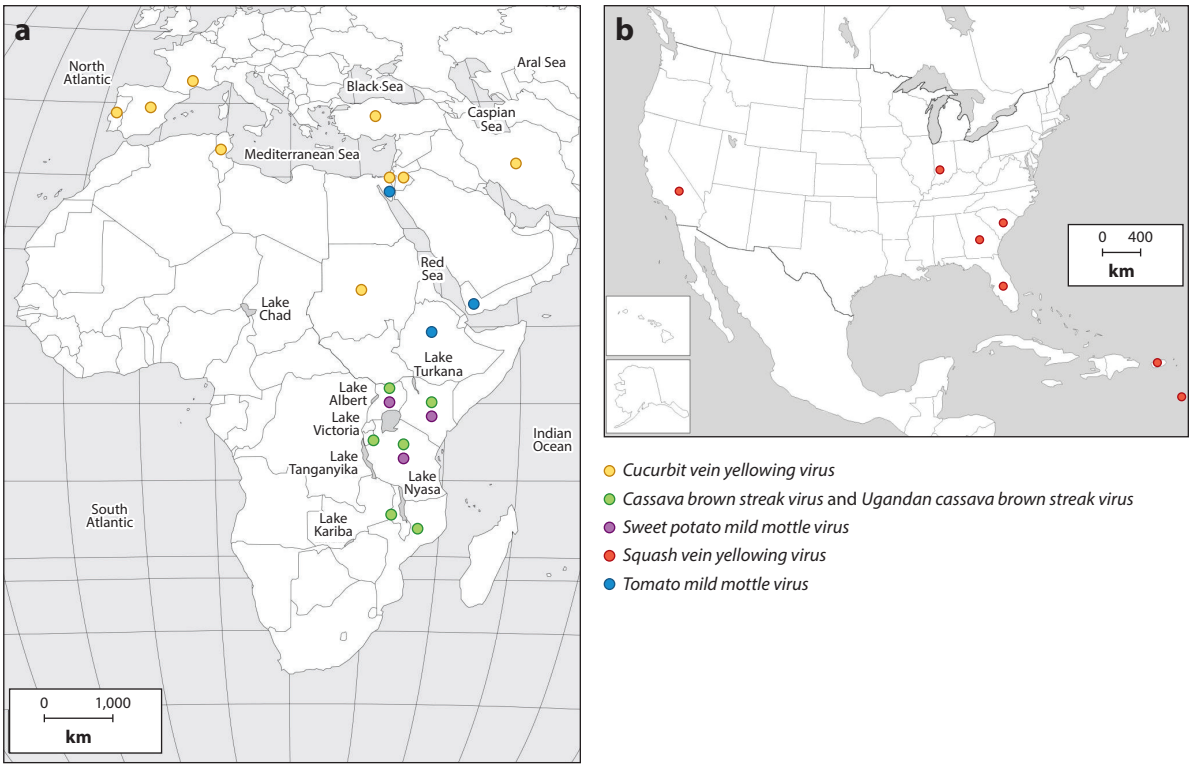


Figure 4

Worldwide distribution of *Cucurbit vein yellowing virus* (*yellow*); cassava brown streak disease caused by *Cassava brown streak virus* and *Ugandan cassava brown streak virus* (*green*); *Sweet potato mild mottle virus* (*purple*); *Squash vein yellowing virus* (*red*); and *Tomato mild mottle virus* (*blue*). (*a*) Locations in Africa, the Middle East, and the Mediterranean Basin. (*b*) Locations in the United States and the Caribbean Basin.

Ipomoviruses: a diversity of symptom types and host plants. Cassava brown streak disease, reported in East Africa in the early 1900s (69), was the first known ipomovirus disease; it is characterized by leaf vein yellowing and necrosis of stems and tuberous roots (70–72). However, only recently were these symptoms attributed to CBSV and UCBSV, which show genetic and geographic separation, although both occur in East Africa (**Figure 4*a***) (73–75). CVYV was reported as the cause of a severe yellowing disease of cucumber in the 1960s in Israel (76) and has subsequently spread throughout the Mediterranean Basin, the Middle East, and Africa (**Figure 4*a***) (65). SqVYV was first reported in the United States, in Florida, and subsequently spread to the states of Indiana, Georgia, South Carolina, and California as well as to Puerto Rico and Guadeloupe (**Figure 4*b***) (77–80). In watermelon, SqVYV induces necrosis of petioles and stems, leading to collapse of leaves and vines as fruits approach harvest (77, 81). This disease is known as watermelon vine decline and causes economic losses due to the internal rind necrosis that renders fruits nonmarketable. Interestingly, in melon and squash, SqVYV induces a yellow vein symptom, indicating substantial differences in host response. SPMMV is the type member of the genus and was described from sweet potato plants in Africa with a foliar mottle (**Figure 4*a***) (82). TomMMoV was first reported in the 1990s from solanaceous plants in Africa with stunting and leaf mottle but only recently was classified as an ipomovirus (**Figure 4*a***) (83, 84). It can cause economic loss in

eggplant due to fruit deformation (84). Thus, ipomoviruses infect a wide range of host plants from different families across a wide geographical area. *B. tabaci* has played a major role in the emergence of these viruses. In addition, vegetative propagation and movement of infected planting material, especially for cassava and sweet potato, are important factors in long-distance spread (85).

Ipomoviruses are inefficiently transmitted by *Bemisia tabaci* in a semipersistent manner. The establishment of the genus *Ipomovirus*, and confirmation of transmission by *B. tabaci*, has been a long and curious journey. CVYV was the first ipomovirus for which convincing evidence of whitefly transmission was shown, and transmission required relatively large numbers of whiteflies (76). At the time, CVYV was considered unusual because it was also mechanically (sap) transmissible (76). This is yet another paradigm shift, because multiple types of mechanically transmitted viruses are now known to be vectored by the *B. tabaci* supervector (e.g., carlaviruses, ipomoviruses, torradoviruses, and some begomoviruses).

Difficulty establishing unequivocal evidence of whitefly transmission has been a common feature of these viruses, although the reasons for this difficulty differ. CBSV and UCBSV were long thought to be whitefly-transmitted, but this was only recently established (86). SqVYV took longer to be described as a new ipomovirus species, because whitefly transmission was not originally explored due to the ease of mechanical transmission (77). The initial report of whitefly transmission of SPMMV has been difficult to repeat (65, 82). TomMMoV was originally thought to be aphid-transmitted, but recent studies have definitively demonstrated whitefly transmission (83, 84).

The semipersistent transmission of ipomoviruses is relatively inefficient, which reflects aspects of the plant-virus-vector interaction. For example, the phloem-feeding *B. tabaci* may not efficiently acquire ipomovirus virions from nonphloem tissues. Thus, many questions remain to be answered regarding ipomovirus transmission by *B. tabaci* (65).

Diversity of ipomoviruses. Genetic diversity has been observed in Africa and/or the Middle East for CBSV and UCBSV isolates from cassava (87), SPMMV isolates from sweet potato (82, 88), and TomMMoV isolates from tomato and eggplant (83, 84). In contrast, less genetic diversity has been reported for CVYV in Spain (89) and SqVYV in the United States (68), possibly reflecting recent emergence of these viruses in these locations. A divergent SqVYV strain found in Florida since 2005 is similar to recently reported California SqVYV isolates (68, 80) and has revealed greater diversity in SqVYV. Nonetheless, the higher levels of genetic diversity observed in CBSV, UCBSV, SPMMV, and TomMMoV in Africa and/or the Middle East may indicate proximity to their center of origin and coevolution with wild plants over a long period of time (e.g., 90, 91).

Emergence of ipomoviruses: a series of first encounters? With the exception of SqVYV in the United States and the Caribbean Basin, ipomovirus species occur in Africa and/or the Middle East (**Figure 4**) (68, 69, 73, 76–80, 83, 84). Ipomoviruses from these regions, except CVYV, have the highest levels of genetic diversity. This suggests that ipomoviruses emerged in Africa and/or the Middle East, most likely from native plants. These progenitor viruses were acquired by *B. tabaci*, were introduced into cultivated plants, and evolved to infect cassava (CBSV and UCBSV), sweet potato (SPMMV), and tomato and eggplant (TomMMoV). SPMMV probably existed in East Africa in native convolvulaceous plants (88) before the introduction of sweet potato from the NW ~300 years ago. CBSV and UCBSV likely emerged after the introduction of cassava from Brazil in the sixteenth century, although neither virus has been detected in plants native to Africa. Following the emergence of ipomoviruses, intercontinental movement resulted in global spread. For example, CVYV has spread around the Mediterranean Basin to Spain, and SqVYV has spread

to the continental United States and the Caribbean Basin. Presumably, genetic diversity of CVYV and SqVYV is greater at the origin of these species.

Intercontinental movement of plant materials and the global distribution of *B. tabaci* likely resulted in a series of first encounters that led to the emergence of crop-infecting ipomoviruses in Africa and the Middle East. The interaction of CBSV and UCBSV with the NW crop cassava has been proposed as such a situation (9, 72). The other cultivated crops in Africa and the Middle East affected by currently known ipomoviruses were also introduced from the NW by Europeans, indicating a similar pattern of emergence. Consistent with the recent ipomovirus-host interaction is the limited or lack of resistance to ipomovirus infection in cultivated varieties of cassava, cucumber (92), and watermelon (77). Interestingly, the moderate resistance to SqVYV identified in wild relatives of watermelon from Africa (Zimbabwe, Botswana, South Africa, and Kenya) and the Middle East (Iran) may indicate a previous interaction (93). Therefore, these geographic regions should be searched for reservoirs of SqVYV and other ipomoviruses, as well as for additional sources of resistance.

Torradoviruses

The torradoviruses provide another paradigm shift in whitefly-transmitted viruses. These viruses represent an entirely new type of RNA virus, transmitted by *B. tabaci* and two other whitefly species.

First example of a whitefly-transmitted spherical virus. Torradoviruses were first recognized in the early to mid-2000s, when a new disease of tomatoes appeared in Spain, Mexico, and Guatemala (14, 94–97). Affected plants show extensive necrosis or burning of leaves, petioles, and stems, and the disease was named *torrado* (roasted) in Spain, *marchitez* (wilted) in Mexico, and *mancha de chocolate* (chocolate spot) in Guatemala. Depending on the geographical location, the disease was associated with *B. tabaci* or the greenhouse whitefly (*Trialeurodes vaporariorum*) (94). The causal agent was a previously unknown mechanically transmissible virus with spherical virions (~30 nm in diameter) and a bipartite positive-sense ssRNA genome (**Table 1**) (95–97). The genome possessed features similar to plant-infecting picorna-like viruses in the family Secoviridae but was sufficiently distinct to be placed into a new genus, *Torradovirus*, named after the type member *Tomato torrado virus* (ToTV) (14, 94, 98). ToTV was subsequently identified infecting tomatoes in other countries of Europe as well as in Australia and the NW (Panama) (14); isolates from Guatemala and Mexico were distinct species (95–97). More recently, torradoviruses infecting hosts other than tomato were identified in the Netherlands (99) and Korea (100). These new torradoviruses are highly divergent from those infecting tomato, their origin and vector are unknown, and their identification involved next-generation (deep) sequencing. Thus, torradoviruses are an emerging genus of viruses, with additional members likely to be identified in the future.

Torradoviruses are transmitted in a semipersistent manner by three whitefly species. Transmission experiments established that three whitefly species can vector torradoviruses: *B. tabaci*, *T. vaporariorum*, and *Trialeurodes abutilonea* (101). Torradoviruses are the first example of a spherical virus transmitted by whiteflies, and they are one of the few examples of a virus transmitted by multiple species of whiteflies [criniviruses are the other (14, 101)]. The mechanism of transmission is semipersistent: Acquisition and inoculation take ~2 h and retention is <8 h. Interestingly, the virus was localized to the stylet, rather than the foregut like many other viruses transmitted in this manner (101). It will be interesting to identify viral factors involved in this mechanism of transmission—for example, whether a helper component is involved in virion

binding to the stylet, and from what cell and tissue types torradoviruses are acquired, as they are not phloem-limited (102). The development of infectious clones and an agroinoculation system for ToTV will help answer these and other questions about torradovirus gene function (103).

It appears that torradoviruses have emerged independently in multiple geographic regions by jumping out of noncultivated host plants into tomato (or other species) via the activity of polyphagous whitefly vectors. This notion is consistent with the detection of torradoviruses infecting weeds (14, 100) and with the nearly simultaneous emergence of genetically distinct torradoviruses in different geographical regions (94). In the case of ToTV, this was followed by global spread to the NW and Australia, presumably via movement of infected plants as there is no evidence that torradoviruses are seed-transmitted (104).

EMERGENT VIRUSES DRIVEN BY THE *FRANKLINIELLA OCCIDENTALIS* SUPERVECTOR

Tospoviruses

F. occidentalis is the other supervector, and it has played a key role in the global emergence of viruses in the genus *Tospovirus* (family Bunyaviridae). The tospoviruses are the only plant-infecting members of this family, but they also have the capacity to infect cells of their thrips vectors; thus, tospoviruses infect and replicate in plants and animals.

***Frankliniella occidentalis* transmits multiple tospoviruses and does so by a persistent propagative mechanism.** *F. occidentalis* transmits multiple tospovirus species worldwide (North and South America, Europe, Africa, the Middle East, and Australia), including most that occur in the NW (105, 106). Because *Thrips palmi* plays a similar role in some regions, it has been referred to as the *F. occidentalis* of tropical and subtropical Asia (105). *F. occidentalis* was recently introduced into India (107), and it will be of interest to see whether it displaces *T. palmi* or mediates emergence of new tospoviruses. Thus, although we consider *F. occidentalis* to be a thrips supervector, other thrips species may be important vectors of a particular tospovirus species or in particular crops or geographic regions (106, 108, 109).

Tospoviruses have pleomorphic spherical particles 80 to 120 nm in diameter and tripartite ambisense ssRNA genomes of ~16 kb (109, 110). The three genomic RNAs are designated large (L), medium (M), and small (S) (109). The type member is *Tomato spotted wilt virus* (TSWV), which has a worldwide distribution and an extremely wide host range (infects ~800 plant species) (105). There are 11 recognized tospovirus species (110) and 14 proposed species. In general, these species can be divided into NW (Americas) and OW (Asia) viruses, though a few have a worldwide distribution, including TSWV, *Impatiens necrotic spot virus* (INSV), and *Iris yellow spot virus* (105). *F. occidentalis* transmits five of these: *Groundnut ringspot virus* (GRSV), INSV, *Tomato chlorotic spot virus* (TCSV), TSWV, and Chrysanthemum stem necrosis virus.

Of the thousands of recognized thrips species, only about a dozen transmit tospoviruses, indicating a highly specific relationship between the virus and vector (2, 111). Adult thrips transmit tospoviruses only if the virus has been acquired during feeding of larval stages on infected plants, and the virus is not transovarially transmitted (109). Tospoviruses replicate in the thrips vectors (112, 113), although the full consequences of infection of the thrips is still being determined. It has been shown that TSWV infection of *F. occidentalis* results in a 3-fold increase in feeding as well as altered feeding behavior of male thrips, thereby increasing the probability of virus transmission (114). Determination of the mechanisms underlying these complex virus-vector interactions is important but is challenged by the lack of infectious clones.

Induction of similar symptoms by multiple tospovirus species complicates identification. Symptoms induced by many tospovirus species in ornamental and vegetable crops often include necrotic or chlorotic ringspots and necrosis (105). Tomato plants infected with TSWV develop symptoms of yellowing, necrosis, and wilting, hence the names spotted wilt or bronze wilt given to the disease when it was first observed in the early 1900s (115, 116). The identification and characterization of additional tospovirus species in the NW and OW in the 1990s revealed the global emergence of these viruses, mediated by activities and population increases of *F. occidentalis* and other thrips vectors. This also led to the establishment of the genus *Tospovirus* (109, 110).

It also became apparent that many of these new tospovirus species, regardless of their origin, induced similar symptoms in crop hosts (e.g., lettuce, pepper, tobacco, and tomato). For example, the NW tospoviruses TSWV, GRSV, and TCSV cause similar symptoms in tobacco, tomato, and pepper, whereas TSWV, TCSV, and INSV cause similar symptoms in lettuce. In Australia, the Asian tospovirus Capsicum chlorosis virus (CaCV) induces spotted wilt symptoms in pepper and tomato and has displaced TSWV in some areas, suggesting a competitive advantage (117, 118). TSWV, GRSV, and *Peanut bud necrosis virus* induce similar symptoms in peanut in the southeastern United States, South America, and Africa and Asia, respectively; these infections cause significant losses (105, 108, 119). Therefore, precise identification of tospoviruses often requires molecular tests, such as reverse transcription–polymerase chain reaction (RT-PCR) and sequencing (e.g., 120). Precise identification is important for developing disease management strategies, including deployment of resistance genes.

Global spread of *Frankliniella occidentalis* and *Tomato spotted wilt virus*. The emergence of TSWV as a global pathogen was preceded by the worldwide dispersal of *F. occidentalis* (121). This occurred for many of the same reasons previously described for begomoviruses and *B. tabaci*, including the polyphagous and thigmotactic properties of *F. occidentalis*, especially on ornamental plants, and the propensity of thrips to develop resistance to insecticides, which makes them difficult to detect and eliminate. In contrast to most individual begomoviruses, which have narrow host ranges, TSWV (and to a lesser extent some other tospoviruses) has a very wide host range. Together, these properties have resulted in *F. occidentalis* and TSWV emerging as global constraints on the production of ornamentals, peanuts, and vegetables (122).

Evolution and emergence of tospoviruses: migration and host expansion of *Groundnut ringspot virus* and *Tomato chlorotic spot virus*. The recent migration of GRSV and TCSV to the United States and the Caribbean Basin is an example of global spread of tospoviruses mediated by *F. occidentalis* (**Figure 5**). GRSV was first detected in Florida in 2009 and more recently appeared in South Carolina and New York (118). In 2012, TCSV was detected in Florida, and it was subsequently found in Puerto Rico, the Dominican Republic, Haiti, and Ohio (**Figure 5**) (120, 123–127; O. Batuman & R.L. Gilbertson, unpublished observations). Currently, TSWV, GRSV, and TCSV are sympatric in southern Florida, similar to the situation in tomato reported in Argentina (128). The establishment and spread of GRSV and TCSV in the United States and the Caribbean Basin have been remarkably rapid and have been driven by *F. occidentalis* with help from locally important thrips species, such as *Frankliniella schultzei*, which is a particularly efficient vector of GRSV and TCSV (126, 129).

In Florida and the Caribbean Basin, *F. occidentalis* and *F. schultzei* have also facilitated host range expansion of GRSV and TCSV, which is fully consistent with the polyphagous nature of these insects. New hosts reported for GRSV in Florida include tomatillo, eggplant, American black nightshade (*Solanum americanum*), and cutleaf groundcherry (*Physalis angulata*) (120, 129). New hosts reported for TCSV in Puerto Rico and Florida include jimsonweed (*Datura stramonium*),

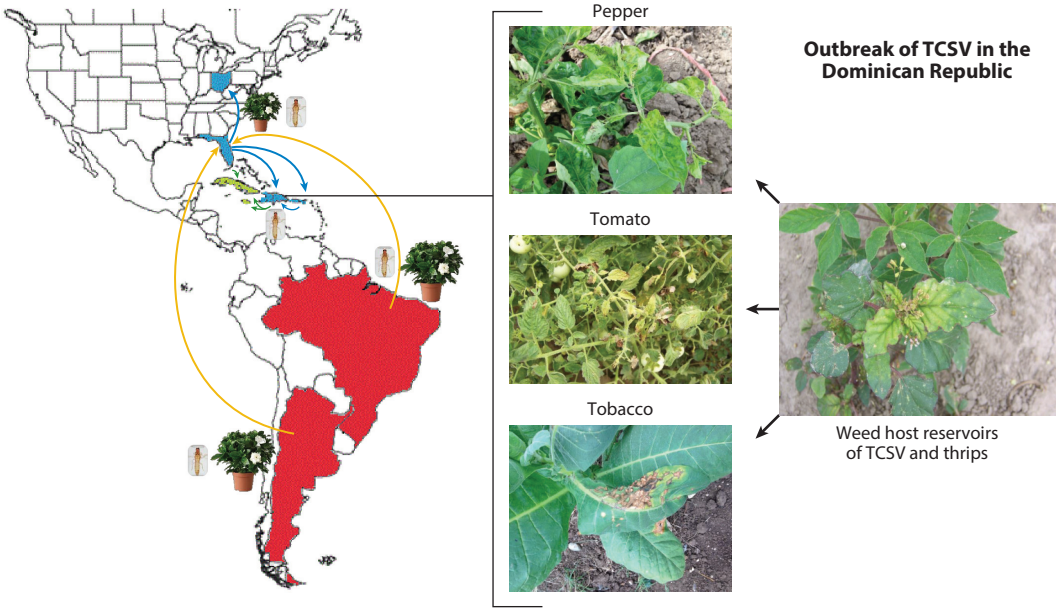


Figure 5

Intercontinental spread of an invasive tospovirus mediated by the supervector *Frankliniella occidentalis*. The tospovirus *Tomato chlorotic spot virus* (TCSV) is indigenous to Argentina and Brazil, shown in red. Possible routes for the intercontinental spread and introduction of TCSV into Florida, most likely via infected plants or viruliferous thrips carried on plant materials, are indicated with yellow arrows. Subsequent spread in the Caribbean Basin and the United States is indicated with blue arrows, and confirmed detections of TCSV in Puerto Rico, the Dominican Republic, Haiti, and Ohio are shown in blue. Likely future spread to Cuba and Jamaica is indicated with green arrows. The current situation with the TCSV outbreak in the Dominican Republic is shown on the right. Infected weeds serve as sources of thrips and TCSV for pepper, tobacco, and tomato crops.

lettuce, waxflower (*Hoya wayetii*), false Christmas cactus (*Schlumbergera truncata*), and annual vinca (*Catharanthus roseus*) (124, 125, 130, 131). In the Dominican Republic, two new nonsolanaceous weed hosts of TCSV, *Cleome viscosa* and *Boerhavia erecta*, play an important role in disease epidemiology (126).

Investigations of the outbreaks of GRSV and TCSV in Florida and the Caribbean Basin have revealed several important lessons. First is the difficulty of identifying tospoviruses that cause similar disease symptoms. As mentioned earlier, symptoms caused by TSWV, GRSV, and TCSV are indistinguishable in most crop hosts; thus, GRSV or TCSV may have been present earlier (with disease symptoms attributed to TSWV), although the low level of genetic diversity in GRSV and TCSV isolates from these locations suggests recent introductions. A lateral flow device developed for TSWV (e.g., ImmunoStrip tests; Agdia, Inc.) allows for the rapid detection of the virus and confirmation of the disease. However, this test also detects GRSV and TCSV. This can be advantageous, as the diseases caused by these viruses are similar in terms of symptoms, ecology, and epidemiology, but specific molecular tests (RT-PCR and sequencing) are required for species identification (120, 126). For example, in the Dominican Republic, tospovirus symptoms in processing tomatoes were initially attributed to TSWV until molecular tests revealed the virus was TCSV (126). Second, the rapid spread of GRSV and TCSV clearly demonstrated that the necessary components were present for disease spread and establishment, including the polyphagous *F. occidentalis* (and other thrips vectors, such as *F. schultzei*) and a wide range of susceptible

cultivated and noncultivated plants. Finally, integrated pest management programs developed for management of thrips and TSWV can be applied to manage these new invasive tospoviruses. The major TSWV resistance gene in tomato, *Sw-5*, also provides resistance to GRSV and TCSV, although care must be used in deployment because, as with TSWV, this gene can be overcome. Indeed, evidence is emerging that the gene may not confer the same level of resistance to these viruses, as TCSV appears to more readily overcome or break *Sw-5*-based resistance. In the Dominican Republic, an integrated pest management program that has helped manage TCSV on processing tomatoes involves (*a*) planting resistant varieties, (*b*) roguing symptomatic plants early in the season, (*c*) monitoring and management of thrips, (*d*) management of weeds, and (*e*) sanitation.

***Frankliniella occidentalis* is driving the emergence of new tospovirus diseases.** Another example of tospovirus host expansion mediated by *F. occidentalis* is the emergence of INSV as a crop-infecting tospovirus. The established paradigm was that INSV is a pathogen of ornamental crops (105). However, a tospovirus host switch has occurred in coastal regions of California, with INSV causing a new disease in lettuce (132). Beginning in 2006, severe outbreaks of a tospovirus disease in coastal lettuce fields were caused by INSV, and these were positively correlated with large populations of *F. occidentalis*. INSV isolates from lettuce were nearly identical to those previously characterized from ornamental hosts, indicating that the virus had jumped into lettuce from ornamental hosts, due to the polyphagous *F. occidentalis*. Furthermore, numerous weeds and crop plants in this region have also been found infected with INSV, which is fully consistent with this virus emerging as a pathogen of crops (105).

***Frankliniella occidentalis* mediates mixed infections of tospoviruses and reassortants.** Due to the tripartite tospovirus genome, additional variation can be introduced by reassortment of the tospovirus genomic RNAs (reviewed in 133). Reassortment may occur between isolates of species, as has been observed with TSWV, and may result in novel phenotypes (e.g., 134–136). Interestingly, the GRSV detected in Florida is a reassortant composed of the L and S RNAs from GRSV and the M RNA from TCSV ($L_GM_TS_G$ genotype) (137); to date, all GRSV isolates characterized from the United States have this genotype (120). Either this reassortant was introduced or both GRSV and TCSV were introduced and the reassortant was generated locally. Interestingly, the TCSV isolates characterized from the United States and the Caribbean Basin are not reassortants and have the standard TCSV genotype. Thus, the GRSV in the United States is an example of a viable reassortant that is causing economic loss (137).

Because of its polyphagous nature, *F. occidentalis* will likely increase the frequency of tospovirus reassortants through the generation of mixed infections. This in turn will raise many questions about the emergence of tospoviruses: Will the frequency of viable reassortants increase as mixed infections become more common? What are the limitations for development of viable reassortants, and will these reassortants be more virulent than parental viruses or be able to infect resistant varieties?

Ilarviruses

F. occidentalis is also mediating the emergence of viruses in the genus *Ilarvirus* (isometric labile ringspot viruses) in the family Bromoviridae through thrips-mediated pollen transmission. Ilarviruses are distributed worldwide and infect a wide range of economically important plants, including fruit trees, ornamentals, and vegetables.

Emergence of ilarviruses by pollen-mediated thrips transmission. Ilarviruses have a tripartite positive-sense ssRNA genome, with the RNAs separately encapsidated within spherical particles ~28 nm in diameter (**Table 1**) (138). There are 19 recognized species and 3 proposed species, and the type member is *Tobacco streak virus* (TSV) (139). TSV infects a wide range of crops and is seed- and pollen-transmitted, like many ilarviruses. The biology and genetic diversity of ilarviruses have been recently reviewed (138). Here, we focus on how *F. occidentalis* has mediated the emergence of a new ilarvirus infecting tomatoes in California (140).

Emergence of a new tomato-infecting ilarvirus in California associated with large populations of *Frankliniella occidentalis*. In 2008, unusual disease symptoms, including necrotic spots and streaks, were observed on leaves and stems of processing tomato plants in the Central Valley of California. This new disease was associated with large populations of *F. occidentalis*. Tests for known viruses, including TSWV, were negative, but a sap- and graft-transmissible virus-like agent induced similar symptoms in tomato plants. Molecular characterization revealed a new species of ilarvirus, and it was provisionally named Tomato necrotic spot virus (ToNSV) (140). ToNSV is most closely related to *Parietaria mottle virus* (PMoV), an ilarvirus first reported in southern Europe from the weed *Parietaria officinalis* and subsequently found to infect tomato and pepper (141–144). PMoV can be economically important in Europe, especially in mixed infections with other tomato-infecting viruses (142, 144). Although ToNSV is commonly observed in California tomato fields, it has not reached high incidences (generally <1%) or caused economic loss. To date, ToNSV has been reported only from California. It is probably an indigenous virus, infecting a yet-to-be-identified perennial plant host, that emerged as a pathogen of tomato due to increased populations of *F. occidentalis*. ToNSV may have been responsible for outbreaks of similar symptoms in tomato in California reported in the 1980s and previously attributed to TSV (145), because extensive efforts to detect TSV in California tomatoes have been unsuccessful.

Evidence for pollen-mediated transmission of Tomato necrotic spot virus. The inoculum source(s) and mode of transmission of ToNSV are not known. Based on the association of the disease with *F. occidentalis*, the possibility of pollen-mediated thrips transmission was considered (146–148). In *F. occidentalis* feeding experiments, only plants that (*a*) had leaves dusted with pollen from infected plants and exposed to thrips or (*b*) were mechanically (rub) inoculated with pollen from infected plants developed symptoms (149). ToNSV also was detected in pollen from infected plants by RT-PCR. Thus, ToNSV was transmitted via pollen, and *F. occidentalis* feeding mediated this process. It was recently reported that PMoV also infects pollen and that pollen-mediated transmission occurred following feeding of *F. occidentalis* and other insects (150). In addition, the source of the pollen was important for transmission efficiency of PMoV (150), indicating that thrips transmission of ilarviruses may be more complicated than simple mechanical inoculation of virus-infected pollen.

Transmission of ilarviruses by thrips involves an opportunistic and nonspecific mechanism that is different from the highly specific and intricate interaction with tospoviruses. This type of transmission mediated by *F. occidentalis* has been reported for spherical viruses in three other genera: *Carmovirus*, *Sobemovirus*, and *Machlomovirus* (13). The machlomovirus *Maize chlorotic mottle virus* was transmitted by the feeding of *F. occidentalis* and *Frankliniella williamsi* as well as numerous leaf-feeding beetles, consistent with nonspecific mechanical-type transmission (151).

SUMMARY POINTS

1. *B. tabaci* and *F. occidentalis* are supervectors and global pests that exhibit high rates of reproduction and dispersal, extreme polyphagy, and the propensity to develop insecticide resistance and that are driving the emergence of a diversity of viruses via multiple mechanisms.
2. *B. tabaci* and *F. occidentalis* supervectors transmit different taxonomic types of viruses.
3. These insects are responsible for paradigm shifts in the transmission of viruses, including evolution of monopartite NW begomoviruses, emergence of ipomoviruses through a series of first encounters with cultivated plants, emergence of torradoviruses as the first known whitefly-transmitted spherical viruses, geographical and host range expansions of tospoviruses, and emergence of crop-infecting ilarviruses via nonspecific pollen-mediated thrips transmission.
4. Emergence of plant viruses occurs through local evolution, host shifts, mixed infections, and global spread; increased virus diversity occurs via recombination, reassortment, and component capture.
5. Application of new methods such as rolling-circle amplification and deep sequencing will likely reveal additional novel types of viruses as well as expand the geographical and host range of known viruses transmitted by these vectors (see 152 for an example with the whitefly-transmitted carlavirus *Cowpea mild mottle virus*).
6. Common biological properties of supervector-virus interactions should facilitate the development and implementation of generalized disease management strategies.

FUTURE ISSUES

1. What are the mechanisms underlying the key properties of supervectors (e.g., extreme polyphagy and propensity to develop insecticide resistance), and will these be revealed by genomics approaches?
2. What are the mechanism(s) and interactions underlying the transmission of multiple viruses by different mechanisms and the global spread of viruses mediated by these vectors, and can this information be used to develop strategies to interfere with transmission and spread?
3. Is mutualism the exception or the rule for the interaction between supervectors and viruses transmitted by persistent mechanisms, and does this provide a selective advantage for virus emergence?
4. What will be the outcome of sympatry of TCSV, GRSV, and TSWV in the United States and the thrips vectors *F. occidentalis* and *T. palmi* in Asia—coexistence or displacement?
5. Will emergence of nonspecific pollen-mediated thrips-transmitted ilarviruses lead to the appearance of new economically important diseases?

DISCLOSURE STATEMENT

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

ACKNOWLEDGMENTS

Some of the research presented in this review was supported by grants from the Integrated Pest Management Innovation Laboratory, made possible by the US Agency for International Development (USAID) and the generous support of the American people through USAID cooperative agreement number EPPA-00-04-00016-00; Transagricola SA, Dominican Republic; the California Tomato Research Institute; the US Department of Agriculture (USDA) Cooperative State Research, Education, and Extension Service Specialty Crop Research Initiative (2008-04890); the National Watermelon Association; USDA National Institute of Food and Agriculture Critical Issues (2011-37610-31178); the Florida Specialty Crop Block Grant Program (018014); the USDA Agriculture and Food Research Initiative (2012-68004-20166); the Florida Tomato Committee; the Florida Specialty Crop Foundation; and the Florida Fruit and Vegetable Association. We thank Alice Nagata and Murilo Zerbini for helpful discussions, and Li-Fang Chen and Kaitlyn Kelly for assistance with figures.

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