ANNUAL REVIEWS

Annual Review of Virology

Life on the Edge: Geminiviruses at the Interface Between Crops and Wild Plant Hosts

Fernando García-Arenal[1]
and Francisco Murilo Zerbini[2]

[1]Centro de Biotecnología y Genómica de Plantas UPM-INIA and Escuela Técnica Superior de Ingeniería Agronómica, Alimentaria y de Biosistemas, Universidad Politécnica de Madrid, 28223 Pozuelo de Alarcón, Madrid, Spain; email: fernando.garciaarenal@upm.es

[2]Departamento de Fitopatologia, Instituto de Biotecnologia Aplicada à Agropecuária (BIOAGRO), and National Research Institute for Plant-Pest Interactions, Universidade Federal de Viçosa, Viçosa, Minas Gerais 36570-900, Brazil; email: zerbini@ufv.br

Annu. Rev. Virol. 2019. 6:411–33

First published as a Review in Advance on June 10, 2019

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

https://doi.org/10.1146/annurev-virology-092818-015536

Keywords

begomovirus, ecosystem anthropization, emerging virus, genetic variability, mastrevirus, wild hosts

Abstract

Viruses constitute the largest group of emerging pathogens, and geminiviruses (plant viruses with circular, single-stranded DNA genomes) are the major group of emerging plant viruses. With their high potential for genetic variation due to mutation and recombination, their efficient spread by vectors, and their wide host range as a group, including both wild and cultivated hosts, geminiviruses are attractive models for the study of the evolutionary and ecological factors driving virus emergence. Studies on the epidemiological features of geminivirus diseases have traditionally focused primarily on crop plants. Nevertheless, knowledge of geminivirus infection in wild plants, and especially at the interface between wild and cultivated plants, is necessary to provide a complete view of their ecology, evolution, and emergence. In this review, we address the most relevant aspects of geminivirus variability and evolution in wild and crop plants and geminiviruses' potential to emerge in crops.

INTRODUCTION

Despite recent evidence that plant viruses are often neutral or beneficial to their hosts and that the outcome of plant-virus interactions varies along the antagonism-mutualism continuum (1, 2), the study of plant viruses has long been prompted by the relevance of the diseases they cause in crops. Viruses are second only to fungi in the number and socioeconomic impact of crop diseases, with reported losses in yield often above 60% (3), and the efficacy of virus disease control is lower than for other plant pathogens (4). Perhaps more significantly, viruses are the major class of emerging pathogens, defined as "the causative agents of infectious diseases whose incidence is increasing following its appearance in a new host population or whose incidence is increasing in an existing host population as a result of long-term changes in its underlying epidemiology" (5, p. S3). About half of the plant diseases that emerged in the last few decades are caused by viruses, which is also the case for diseases of humans, domestic animals, and wildlife (6–8), and emergence is often associated with increased virulence (2, 6, 9). Interest in virus emergence has boosted the study of emergence's ecological and evolutionary drivers, including the identification of wild host reservoirs, factors favoring spillover from reservoir to crop, and evolutionary processes resulting in virus adaptation to the new host. Deep sequencing techniques have allowed the analysis of the patterns of virus infection in wild plant communities (10–12), but studies that explicitly analyze the role of wild hosts in the ecology and evolution of viruses are not yet abundant (13, 14). Still, a good fraction of such studies has focused on geminiviruses, which arguably constitute the major group of emerging plant viruses in crops (15–17).

Viruses classified in the family *Geminiviridae* include economically important plant pathogens in tropical and subtropical regions of the world, and geminivirus prevalence is increasing in temperate regions, which are often associated with the invasion of new areas by vectors (18, 19). Geminiviruses have genomes composed of one or two molecules of circular, single-stranded DNA packaged into twinned, quasi-icosahedral particles and are transmitted in nature by different types of insect vectors (20). Geminiviruses have provided models for basic studies on cell cycle regulation, pathogenesis, viral replication and movement, interaction with insect vectors, and viral evolution (21). Not surprisingly, many studies have focused on species whose members cause economically important diseases in crops such as bean golden mosaic virus (BGMV), cotton leaf curl virus (CLCuV), east African cassava mosaic virus (EACMV), maize streak virus (MSV), and tomato yellow leaf curl virus (TYLCV) (19).

Geminiviruses have a high potential for genetic variation both by accumulating mutations and by recombination, which is an identified factor in virus adaptation and emergence into new hosts (9). The study of evolutionary factors in geminivirus emergence has been prioritized, and emergence has often been linked to the large genetic changes associated with recombination (22). As analyses of geminivirus emergence have put emphasis on their genetic variation, we review first what is known about the generation and maintenance of genetic diversity in their populations. Research on the ecology of emergence is less abundant. Specifically, research on geminivirus infection in wild plant communities, or in wild plants growing in anthropic ecosystems, is still in its infancy, focusing on a few pathosystems and a few geographic regions. Still, data indicate that wild plants can be the original hosts from which spillover to crops has occurred as a first step to emergence but also that spill back from crops to wild plant hosts occurs, resulting in the appearance of new inoculum sources for epidemic outbreaks in crops. **Figure 1** summarizes the factors behind spillover and spill back between wild hosts and crops. Understanding the ecology of geminiviruses at the interface between crops/agroecosystems and wild plant hosts/wild ecosystems is essential to understand their evolution and emergence. We review here what is known about this exciting topic, discuss trends, and suggest new avenues for future research.

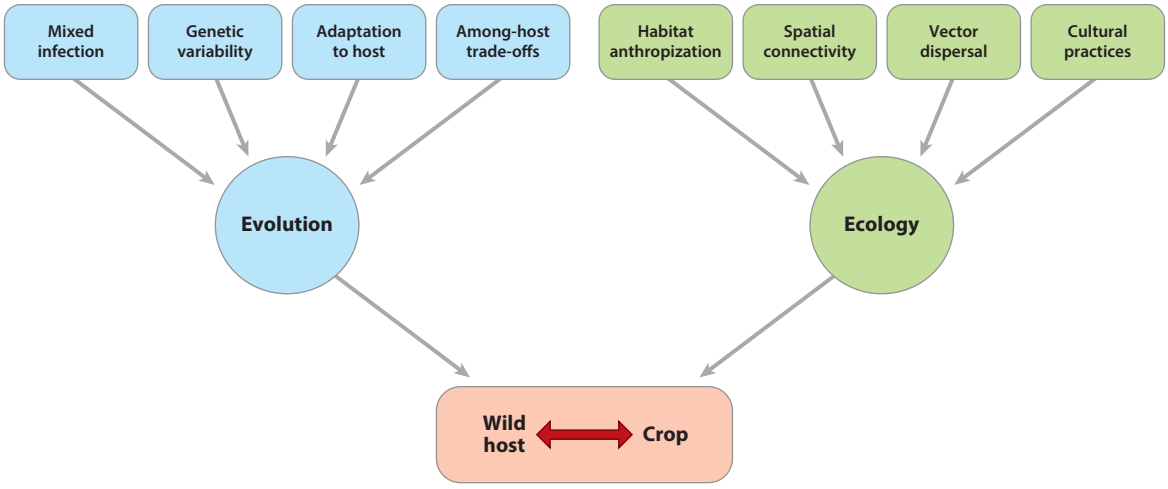


Figure 1

Factors that modulate geminivirus infection at the interface between wild and cultivated hosts. A virus can acquire a new host by spillover from a wild to a cultivated host or by spill back from a cultivated host to a wild one, represented by the double-headed red arrow in the red rectangle. The success of the virus in the new host depends on its evolutionary potential (*blue*), and the probability of spillover or spill back depends on virus ecology (*green*). Factors that determine virus evolution and ecology discussed in this review are indicated in boxes.

EVOLUTIONARY CAUSES OF EMERGENCE: HIGH GENETIC VARIABILITY

Spillover from an original (wild) host to a new (crop) host most often will result in dead-end infections that do not propagate virus to new host plants. For emergence to occur the virus needs to become adapted to the new host and/or vectors so that efficient transmission within the new host population is assured (9). Adaptation to a new environment, such as a new host, requires that enough viral genetic variation is available for selection to operate (**Figure 1**).

A primary source of genetic variation is mutation. Mutation rates have not been estimated for any geminivirus, but estimates for viruses with small ssDNA genomes (bacteriophages M13 and phiX174) indicate that they are at the lower end of the range of mutation rate values reported for RNA viruses (23). Geminivirus mutation rates may also be in that range despite the dependence on host DNA polymerases for DNA chain elongation during geminiviruses' genome replication (21), which need not result in high replication fidelity. High mutation rates can be due to processes acting specifically on ssDNA and/or reducing its accessibility to the DNA proofreading enzymes (24–26).

The first indication that geminivirus mutation rates are in the order of those of RNA viruses was derived from analyses of the genetic structure and diversity of field populations of the begomoviruses tobacco leaf curl virus (TLCV) and CLCuV, estimating high nucleotide diversities at synonymous positions, of ~0.2–0.5 for different genes (24, 27). Mutation rates similar to those of RNA viruses are also supported by high short-term nucleotide substitution rates, which for different viruses in the genera *Begomovirus* and *Mastrevirus* are of 10^{-3}–10^{-4} substitutions/site/year (28–32), similar to those reported for RNA viruses (33).

Another major source of genetic variation is recombination, which has been reviewed recently (22). Recombination plays an important role in geminivirus evolution, having contributed to the diversification of the species and genera of the family (34, 35). Frequent recombination in

geminiviruses has been explained by the coexistence of two mechanisms of genome replication, a rolling-circle replication and a recombination-dependent replication, which will recycle incomplete DNA molecules to produce full-size genomes (36). Recombinants recovered from experiments or from natural populations show recombination hot and cold spots within viral genomes and demonstrate that recombinant fitness is dependent on the preservation of interaction networks among both viral proteins and domains within proteins, with network disruption resulting in fitness penalties (37–40). Numerous studies have reported that inter- and intraspecies recombinants occur at high frequency in geminivirus populations (41–48). Interspecies recombination was invoked as a cause of the emergence of cassava mosaic disease (CMD) in Uganda and of cotton leaf curl disease in Pakistan in the late 1990s (41, 43).

In viruses with bipartite genomes, the frequency of recombinants is either similar for the DNA-A (which encodes structural and replication-associated proteins) and DNA-B (which encodes movement-associated proteins) (47) or higher for the DNA-B (49). Interestingly, the selection acting on recombinants may differ among virus species, as judged by recombinant frequency. Thus, recombinants were significantly more frequent in the population of pepper golden mosaic virus (PepGMV) than in that of pepper huasteco yellow vein virus (PHYVV) infecting the same host in the same region (47).

In bipartite begomoviruses, reassortment of DNA-A and DNA-B components from coinfecting virus genotypes or species (also called pseudorecombination by plant virologists) results in genetic exchange with similar evolutionary consequences as recombination (50). It has been proposed that reassortment has been particularly relevant in the evolution of old-world begomoviruses (35). Reassortment may have phenotypic effects on infectivity, virulence, and host range (51, 52). Reassortants have been shown to occur frequently in field populations between African cassava mosaic virus (ACMV) and EACMV (51) and between PepGMV and PHYVV (47).

Because genetic exchange is considered a main driver of geminivirus genetic variation, few studies have determined the relative contribution of recombination and mutation to genetic diversity (34, 42, 51–53). Lima et al. (44, 54) performed this analysis for 17 begomoviruses, and in every case mutation played a larger role than recombination in shaping genetic diversity. However, the relative contribution of recombination and mutation to genetic variation was not a function of their relative frequencies, as even low recombination rates could provide a significant amount of genetic variation (47, 54).

The diversity of begomovirus populations has been shown to vary largely, in the range of 0.003–0.130 nucleotide substitutions per site (24, 31, 44, 45, 47, 55, 56). This variation is not due to differences in the size of the sampled area or in the number of individuals making up the population. Rather, the degree of diversity seems to be an intrinsic property of each begomovirus. Other significant trends are that diversity is higher for DNA-B than for DNA-A in viruses with two-component genomes and that for all analyzed viruses, viral proteins are consistently under strong purifying selection (35).

ECOLOGICAL CAUSES OF EMERGENCE: ECOSYSTEM SIMPLIFICATION

Emergence requires new virus-host encounters for the virus to spill over from an original or reservoir host to the new one (9). Consequently, ecological changes favoring virus-host encounters are frequently identified as drivers of virus emergence (57, 58). The environmental effects of human activity resulting in land-use change, agriculture, and, ultimately, biodiversity loss and ecosystem simplification (**Figure 1**) have been identified as factors in the emergence of pathogens of plants and animals (59, 60). Interestingly, plant pathologists have invoked for a long time the loss of

species diversity in agroecosystems, as compared with wild ecosystems, and the loss of genetic diversity of crops, as compared with their wild relatives, as major causes of the high impact of diseases in crops (61, 62). The relationship between biodiversity and disease risk has been proposed to be either negative (dilution effect) or positive (amplification effect) according to different traits, including the pathogen's host range (63). However, it is difficult to predict disease risk only from biodiversity. Community-level studies at different scales, up to the landscape, are necessary to encompass the environmental heterogeneity of ecosystems in which patches of crops and wild plant communities are interspersed. These landscapes would provide ample opportunities for an interface between wild and crop plants to arise. Note that such interface requires connectivity between plant species and/or communities but not necessarily physical adjacency between crops and wild plants (64, 65). Such analyses are still few for plant viruses.

Deep sequence analyses did not provide evidence of geminiviruses infecting plant species in two different wild ecosystems, the Area de Conservación Guanacaste (Costa Rica) and the Tall Grass Prairie (Oklahoma, United States) (12, 66, 67). While this could have been expected at the first site, as analyses were based on double-stranded RNA preparations from plants, in the second site nucleic acids associated with virus-like particles (VLP) were analyzed. Deep sequencing of VLP-associated nucleic acids was also used to study virus diversity and prevalence in two Mediterranean climate regions, the Rhone delta in France and the Western Cape in South Africa. At each region, virus infection was analyzed in plants growing in areas with different levels of human disturbance, from wild to crops. Virus prevalence was higher in agricultural than in wild areas and also higher in cultivated than in wild plant species (10). Interestingly, and at odds with several families of RNA viruses, in neither site did geminiviruses show significant associations with agriculture, being most prevalent in noncultivated, anthropic habitats such as old, abandoned fields. Another interesting result of this study was that new, highly divergent geminiviruses were found in wild plants, four in South Africa and one in France, suggesting that wild ecosystems harbor a large fraction of still-undescribed geminivirus diversity (68).

A similar deep sequencing–based study examined infection by geminiviruses in 115 wild plant species growing in wild or anthropic habitats in central Spain. Geminivirus infection was not detected in any species growing in the highly biodiverse wild habitats but concentrated on 25 species growing in simplified anthropic habitats next to crops and in two weed species within crops (M.J. McLeish & F. García-Arenal, unpublished results). Although still few, these studies suggest that ecosystem simplification due to anthropization favors geminivirus incidence and that anthropic habitats near crops house potential reservoirs for geminivirus emergence.

The relationship between landscape heterogeneity and virus infection was also studied in populations of the wild pepper or chiltepin (*Capsicum annuum* var. *glabriusculum*) from native wild woods, anthropic habitats (live edges, pasturelands), or intentionally cultivated small traditional fields in Mexico. Chiltepin plants were infected by two begomoviruses, PepGMV and PHYVV, whose incidence increased with the level of human management of the habitat. Habitat biodiversity, host genetic diversity, and host plant density were major predictors of incidence (69). However, the level of human management of the habitat was not a factor in the genetic structure of PepGMV or PHYVV populations (31). There was a correspondence of the spatial structure of the viruses and host populations, which was interpreted as due to similar spatial restriction in the dispersion of viruses and chiltepin rather than to plant-virus codivergence (31). The level of human management of the habitat was also associated with the frequency of mutation and recombination of PepGMV and PHYVV, but the effects were virus specific (47). At odds with numerous reports of virus infection being asymptomatic in wild plants (2), infection by PepGMV and PHYVV induced symptoms in chiltepin and negatively impacted its fitness by reducing both plant fecundity and survival (70). Interestingly, PepGMV and PHYVV virulence to chiltepin, estimated from the

number of infected plants that developed disease symptoms, was higher in cultivated than in wild populations, which should be attributed to ecological factors, as these two populations did not differ genetically (70). These studies illustrate that ecosystem simplification may affect the infection dynamics, the virulence, and the evolution of geminiviruses, all factors involved in virus emergence.

TRYING TO UNDERSTAND GEMINIVIRUS EMERGENCE IN CROPS: THREE CASE STUDIES

The last four decades have seen the regional or worldwide emergence of important phytopathological problems in crops associated to geminiviruses. The causes of the emergence and expansion of some of these viruses have been analyzed extensively, particularly for genetic factors related to virus evolvability. Begomovirus emergence has also been associated with the worldwide dispersion of the vector *Bemisia tabaci* Middle-East-Asia Minor 1 (MEAM1; previously *B. tabaci* biotype B) (71–73). Other ecological factors, such as the occurrence of suitable alternative hosts or changes in the cultural practices of crops, have also been invoked as major determinants of emergence. We present here studies on geminiviruses causing three important diseases that illustrate the role of some of these factors in emergence. However, two common features of these viruses are that the original host has not been identified and that hypotheses on wild host reservoirs have not been supported beyond verbal arguments.

Cassava Mosaic Disease–Associated Begomoviruses: A Highly Diverse Virus Complex Infecting an Introduced Crop

In Sub-Saharan Africa (SSA) cassava (*Manihot esculenta*), a major staple crop, is severely affected by CMD, which is caused by a complex of seven begomoviruses with bipartite genomes (74). Since cassava is a South American domesticate introduced into Africa by Portuguese traders in the sixteenth century (75), and CMD begomoviruses are not present in South America, a reasonable assumption is that they must have originated from indigenous viruses infecting local hosts (76). One significant feature of CMD is the frequent occurrence of mixed infections and synergistic interactions, mostly between ACMV and EACMV (77, 78). Frequent mixed infections facilitate genetic exchange by either recombination or reassortment, which have played an important role in the emergence of CMD begomoviruses (42, 77). Thus, east African cassava mosaic virus-Uganda (EACMV-UG), a recombinant between EACMV and ACMV that was responsible for the severe epidemics of CMD in Uganda during the 1990s, has a higher virulence than its parentals (42, 51). EACMV-UG has expanded to other east African countries such as Kenya and Tanzania, where it has displaced previously existent CMD begomoviruses (79–81), indicating that an epidemiological advantage added to its increased virulence.

Early epidemiological studies indicated that cultivated cassava is the most important reservoir for CMD and that temporal and geographical differences of incidence and severity of CMD could be attributed to variations in the incidence and/or presence of distinct genotypes of the whitefly vector (82, 83). Possibly as a result of these conclusions, the vast majority of the studies on the genetic and pathogenic diversity of CMD begomoviruses in different SSA countries have focused exclusively on isolates from cassava (79, 81, 84, 85). These studies indicated that genetic variation differs among the various CMD begomoviruses. ACMV appears to be the least variable species, with >95% DNA-A sequence identity among isolates from Cameroon, Ivory Coast, Kenya, Nigeria, Tanzania, and Uganda and no evidence of intraspecific recombination events (79). EACMV isolates are more diverse. Thus, a countrywide study in Kenya indicated the presence of three

EACMV-like viruses with a clear geographical segregation (81). Isolates of EACMV-UG and east African cassava mosaic Kenya virus (EACMKV) were found in the highlands and central areas, while EACMV and east African cassava mosaic Zanzibar virus were found in the coastal and central areas. Interestingly, and against the most common trend in begomoviruses, the DNA-B was much less diverse than the DNA-A. East African cassava mosaic Cameroon virus (EACMCV) has the highest diversity among CMD begomoviruses. Isolates from east and west Africa cluster into two groups, suggesting a long divergence time (79), and recombination among isolates from each group has been detected in both the DNA-A and DNA-B (84). Higher genetic diversity and the occurrence of unique recombination events in isolates from east Africa have led researchers to propose east Africa as the origin of EACMCV. In fact, east Africa was suggested as the center of origin of all EACMV-like begomoviruses (79), so this region should be prioritized for studies aimed at identifying their original, indigenous hosts that could act as reservoirs.

One of the most comprehensive studies of CMD begomovirus diversity was carried out in Madagascar (85). Four viruses were prevalent: ACMV, EACMCV, EACMKV, and South African cassava mosaic virus (SACMV). ACMV and SACMV populations had a low degree of genetic variability (mean identities of 97% and 98.5%, respectively), while EACMKV and EACMCV populations were relatively more diverse (mean identities of 94.4% and 96.3%, respectively) and were geographically structured. Phylogeographic analyses indicated that all four viruses were introduced from mainland Africa. For the two most prevalent viruses (ACMV and SACMV), introduction was estimated to be very recent (approximately 20 years ago), which could account for their low genetic diversity. Interestingly, movement of EACMCV from Madagascar to Angola was also revealed. Overall, the study indicated that the different begomoviruses have their own evolutionary dynamics despite sharing the same (cultivated) host and being transmitted by the same vector.

Even considering the widely accepted view that noncultivated hosts are unimportant in the epidemiology of CMD, the lack of studies on the role of wild hosts in the origin of the related begomoviruses, as virus reservoirs and as sources of CMD begomoviruses' genetic diversity, is striking. The fact that such hosts have not been identified is a serious limitation in understanding the evolutionary dynamics and emergence of these viruses.

Tomato Yellow Leaf Curl Virus: Global Spread of a Virus with a Narrow Host Range

Although, as a group, geminiviruses can be found worldwide, there is only one truly global geminivirus, the monopartite begomovirus TYLCV. Other begomoviruses cause tomato yellow leaf curl disease in restricted regions of the world, such as tomato yellow leaf curl China virus and tomato yellow leaf curl Sardinia virus (TYLCSV).

Bayesian phylogeographic inference from 82 full-length TYLCV sequences indicated that the virus arose in tomato crops in the Middle East (between the Jordan Valley and Iran), most likely between 1930 and 1950, and spread globally during the 1980s and 1990s (86). A more recent study with a larger data set indicated that this global spread is still ongoing (87). Although TYLCV has been reported to infect several hosts in addition to tomato (88–90), in most regions its detection in alternate hosts is only sporadic (90), and viral populations are primarily associated with tomato. The global spread of TYLCV and its occurrence mostly in tomato crops suggests that the virus has evolved to become highly adapted to tomato and poorly adapted to other hosts, although nongenetic factors of an epidemiological or ecological nature could also explain the observed dispersion and host range patterns. The occurrence of genotypes able to efficiently infect different hosts in Spain (see the next paragraph) indicates that adaptation to tomato was not at the cost of infecting other hosts and underscores the relevance of ecological factors. Nonetheless, within

the TYLCV complex, some species or strains appear to be better adapted to tomato and/or local ecological conditions, as shown by the displacement of the local viruses by TYLCV after its introduction to a new area (87, 91–93). Identified causes for that displacement include more efficient transmission by different *B. tabaci* species (92), the capacity to infect other crops (92), and increased multiplication in tomato cultivars with the resistance gene *Ty-1* (94), which demonstrate the joint role of genetic and ecological factors in geminivirus emergence.

Different estimates of the nucleotide substitution rate of TYLCV give similar values of about 10^{-4} substitutions/site/year (28, 95, 96), the same as for other begomoviruses. Although a recombinant origin has been proposed for TYLCV (86), the role of recombination in TYLCV evolution differs in different regions. Recombinant genomes have been detected in the Middle East, Spain, Italy, and Morocco (53, 86, 97–99), but they may incur fitness costs relative to the parentals. Thus, in Sicily (Italy), recombinants between TYLCV and TYLCSV were frequently detected in the field but always in mixed infection with the parental viruses, and their replication was approximately tenfold lower than that of the parentals, indicating lower within-host fitness (100). A different scenario was reported in Spain, where recombinants with a host range larger than the parentals', and with higher virulence, have become established (48, 53, 101). The crop rotation of tomato and common bean (*Phaseolus vulgaris*), both hosts of TYLCV, and the common occurrence of the wild host *Solanum nigrum*, host of TYLCSV, may provide the conditions for the selection and increased prevalence of TYLCV/TYLCSV recombinants able to infect all three hosts (48). The diversification of the virus through mutations could be also affected by the host (102). The absence of relevant hosts other than tomato in Sicily may have hindered the selection and spread of recombinants there. In Morocco a recombinant TYLCV has also replaced the parental viruses (103), which has been linked to the deployment of tomato cultivars containing the TYLCV resistance gene *Ty-1* (98), as the recombinant multiplies in *Ty-1* tomato genotypes more efficiently than the parentals (103). High infectivity of recombinants in *Ty-1* cultivars was also reported in Spain (53). These studies show how changes in cultural practices—in this case, the deployment of resistant cultivars—can favor geminivirus emergence. The fact that recombinants have not become prevalent in other Mediterranean countries despite the widespread use of *Ty-1* cultivars further points to the relevance of poorly understood ecological factors in geminivirus emergence.

As for CMD begomoviruses, no local, wild or cultivated, reservoir has been identified from which emergence in tomato would have started somewhere in the Near East. Interestingly, spill back from tomato or other crops (bean) to wild hosts such as *S. nigrum* in Spain may create a secondary wild reservoir with a role in generating genetic diversity (48) and as inoculum sources for epidemic outbreaks in the crops.

Tomato-Infecting Begomoviruses in the Americas: Emergence of Multiple Indigenous Viruses in a Crop Host

In the Americas, begomoviruses have significantly impacted tomato production since the 1980s, following the introduction and dispersion at the continental scale of *B. tabaci* MEAM1 (71–73). A large number of tomato-infecting, mostly bipartite begomoviruses have been identified and characterized (72, 73, 104–111). Most of these viruses have never been detected in other continents, and they are considered indigenous to the Americas (56). Multiple introductions and the subsequent spread of TYLCV since the mid-1990s (112, 113) have led to the displacement of some of these local viruses.

In southeastern Brazil, nine different begomoviruses were found infecting tomato, with four of them—tomato common mosaic virus, tomato chlorotic mottle virus (ToCMoV), tomato

severe rugose virus (ToSRV), and tomato yellow vein streak virus—accounting for 90% of infections (56). The distribution of the incidence of these four viruses showed a clear spatial structure, which was interpreted as indicative of differential adaptation to tomato and/or the environment or differential transmission by local populations of the insect vector. In northeastern Brazil, other begomoviruses were mostly found infecting tomato crops, with tomato mottle leaf curl virus being most prevalent (104), again indicating strong spatial structure of infection patterns. ToCMoV, ToSRV, and other less-prevalent begomoviruses are also found infecting wild species growing in anthropic habitats near crops or as weeds within the crop, such as *Datura stramonium*, *Nicandra physaloides*, or *S. nigrum* (56, 105, 114). Other viruses infecting tomato with lower incidence, such as tomato mild mosaic virus and tomato yellow spot virus, are present in wild hosts with higher incidence (115, 116) and phylogenetically are closer to viruses infecting wild hosts than to viruses primarily infecting tomato (56, 117). Collectively, these data are compatible with the hypothesis that the diverse tomato-infecting begomoviruses in Brazil have their origin in local wild hosts from which different viruses have emerged in tomato in different geographical areas, reaching different levels of adaptation to the host and/or the environment.

ToSRV is particularly successful among these viruses, being the most common tomato begomovirus in central Brazil and highly prevalent in the southeastern and southern regions, where it also infects pepper crops (118). As with other major begomoviruses in crops, its persistence in tomato seems to be the main factor associated with epidemics (119, 120). Viral populations have a low degree of genetic diversity, with little evidence that intraspecific recombination has contributed significantly to their evolution (44). Using coalescence analysis based on variable sites from a data set composed of 33 isolates, researchers mapped 71 mutation sites along the ToSRV genome (**Figure 2**). The oldest mutations along the DNA-A were located in the *Rep* gene, followed by the *CP* gene. All mutations observed in isolates obtained from pepper (but not from wild hosts) were shared with isolates from tomato and had the same relative age. Altogether, these results are consistent with the hypothesis that ToSRV is well adapted to tomato and pepper and that it is occasionally transferred to noncultivated hosts by the insect vector *B. tabaci* (O.F.L. Sande & F.M. Zerbini, unpublished results).

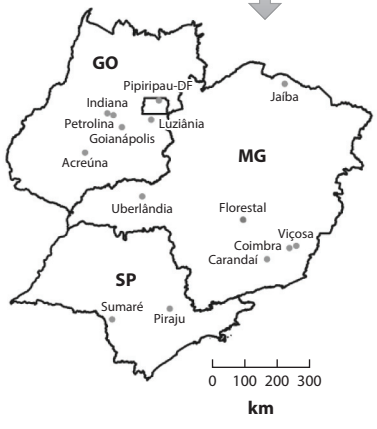
THE SEARCH FOR WILD HOST RESERVOIRS AS ORIGINS FOR GEMINIVIRUS EMERGENCE

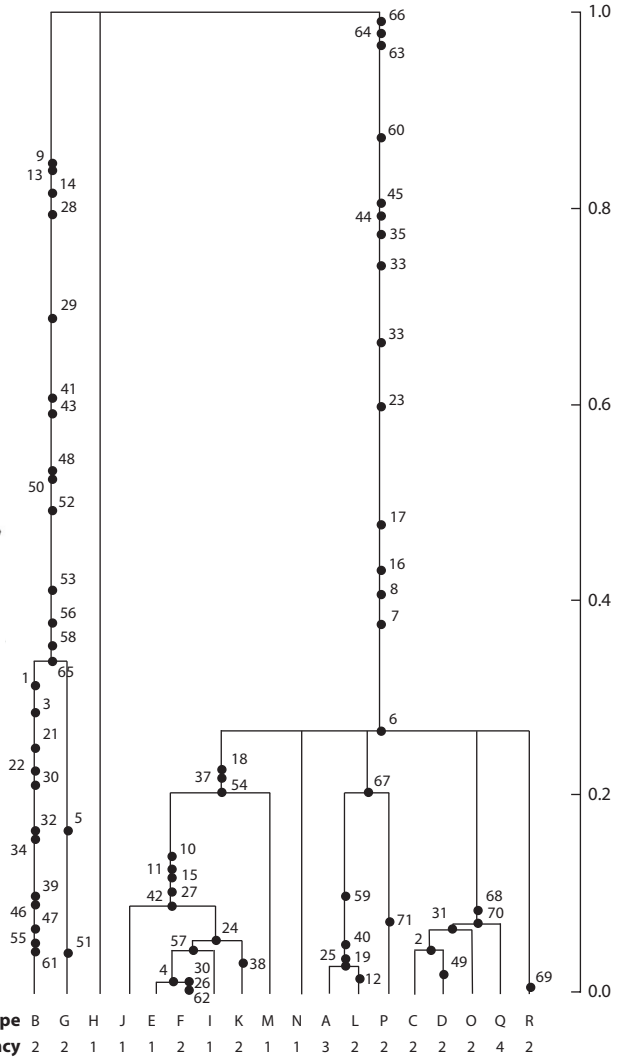
The role of wild hosts as sources of viral diversity and reservoirs for emergence has been investigated for viruses other than geminiviruses. The studies performed on the role of wild hosts in the evolution and emergence of potyviruses, and on the spill back of potyviruses and their impact on the native flora of Australia, are particularly relevant (121–123). To our knowledge these are the only studies comparable in detail to those performed with geminiviruses, as stated in the introduction of this review.

Patterns of Geminivirus Infection in Wild Hosts as Related to Reservoir Potential

The search for wild reservoirs of geminiviruses has been primarily directed to plant species growing in anthropic habitats near crops, or as weeds within the crop, as they would have the highest potential connectivity with crops. Although current studies have a restricted geographical range, mostly referring to Brazil, they provide valuable information from which patterns in virus infection and diversity can be derived.

Pioneering studies of whitefly-transmitted viruses causing either a mosaic disease in *Euphorbia prunifolia* (syn. *E. heterophylla*) or infectious chlorosis in species of the Malvaceae were conducted





Haplotype	B	G	H	J	E	F	I	K	M	N	A	L	P	C	D	O	Q	R
Frequency	2	2	1	1	1	2	1	2	1	1	3	2	2	2	2	2	4	2

Locality	Host																	
Florestal-MG	*Solanum lycopersicum*	2	2	.	1	1	2	1	2	.	.	.	.	.	.	.	.	1	.
Coimbra-MG	*Solanum lycopersicum*	.	.	.	.	.	.	.	.	.	.	1	.	.	.	.	.	.	.
Viçosa-MG	*Solanum lycopersicum*	.	.	.	.	.	.	.	.	.	.	.	2	.	.	.	.	.	.
Carandaí-MG	*Solanum lycopersicum*	.	.	.	.	.	.	.	.	.	.	.	.	1	.	.	.	.	.
	***Sida* sp.**	.	.	.	.	.	.	.	.	.	.	.	.	1	.	.	.	.	.
Jaíba-MG	*Solanum lycopersicum*	.	.	.	.	.	.	.	.	.	.	2	.	.	.	.	.	.	.
Uberlândia-MG	*Solanum lycopersicum*	.	.	1	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
Acreúna-GO	*Solanum lycopersicum*	.	.	.	.	.	.	.	.	.	.	.	.	.	2	.	.	.	.
Luziânia-GO	*Solanum lycopersicum*	.	.	.	.	.	.	.	.	1	.	.	.	.	.	.	.	.	.
	Crotalaria juncea	.	.	.	.	.	.	.	.	.	.	.	.	.	.	1	.	.	.
Goianápolis-GO	*Solanum lycopersicum*	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	1	.	.
	Nicandra physaloides	.	.	.	.	.	.	.	.	.	.	.	.	.	.	1	1	.	.
Petrolina-GO	***Capsicum annuum***	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	1
Indiana-GO	*Solanum lycopersicum*	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	1	.
Piraju-SP	***Capsicum annuum***	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	1	.
Sumaré-SP	***Nicandra physaloides***	.	.	.	.	.	.	.	.	.	1	.	.	.	.	.	.	.	.
Pipiripau-DF	*Solanum lycopersicum*	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	1	1

(*Caption appears on following page*)

Figure 2 (*Figure appears on preceding page*)

Coalescence analysis based on variable sites showing the distribution of 71 mutations in the tomato severe rugose virus genome. The data set is composed of 33 isolates obtained from samples from multiple hosts collected in different localities of Brazil (*left*). The analysis was inferred using the program GENETREE (150), considering 10 independent runs of 1,000,000 Markov chain Monte Carlo replications with a burn in of 100,000 runs. The coalescence timescale (*right*) is presented in units of effective population size. Filled numbered circles represent individual mutations mapped in the viral genome. Names in boldface indicate isolates obtained from hosts other than tomato. Abbreviations: DF, Federal District; GO, state of Goiás; MG, state of Minas Gerais; SP, state of São Paulo.

during the 1940s–1960s by Costa & Bennett (124) and Orlando & Silberschmidt (125). It was demonstrated that the two syndromes were caused by different viruses that shared transmission properties but differed in host range (126). The virus in *E. heterophylla*, now named *Euphorbia* yellow mosaic virus (EuYMV), is the only begomovirus reported to infect this species naturally in Brazil (49). The virus causing the infectious chlorosis was initially considered to be an isolate of *Abutilon* mosaic virus (AbMV) (126). Much later it was shown that the mosaic in *Sida micrantha* (a species in the Malvaceae) was caused by a virus distinct from AbMV that was named *Sida micrantha* mosaic virus (SimMV) (127), and it is now well established that infectious chlorosis of malvaceous hosts in Brazil is associated with a complex of several begomoviruses (56, 128). In fact, throughout the Americas it has been shown that *Sida* spp. host literally dozens of begomoviruses, often in mixed infections (129–134). That similar viral-disease syndromes in different wild hosts can be caused by a single begomovirus or by many is also illustrated by the yellow spot disease of *Blainvillea rhomboidea* (Asteraceae), which is associated exclusively with *Blainvillea* yellow spot virus (BlYSV) (56), and the yellow mosaic of *Macroptilium* spp. (in the Fabaceae), which can be caused by a complex of several legume-infecting begomoviruses (46, 135, 136). It is interesting to underscore that despite mixed infections being a prerequisite for recombination or reassortment of genome segments, the genetic diversity of these viruses is unrelated to their occurrence in single or mixed-infected hosts: BlYSV displays the highest diversity among all begomoviruses studied in Brazil (56), while the diversity of EuYMV is much lower (49). Thus, the level of genetic diversity seems to be an intrinsic property of the virus, regardless of host range or coexistence in mixing infection with other viruses.

The above studies show two different patterns of plant-begomovirus interactions in wild hosts that would play different roles in emergence (**Figure 3**). *E. heterophylla* and *B. rhomboidea* exemplify sealed container hosts, in which a highly adapted virus would exclude multiple infections with other less adapted viruses and would rarely infect other less susceptible or competent hosts (EuYMV has been sporadically detected in hosts other than *E. heterophylla*, but BlYSV has been detected only in *B. rhomboidea*). *Sida* and *Macroptilium* spp., on the other hand, are mixing vessels that allow efficient infection by several begomoviruses, possibly similarly adapted to the host, which results in a high frequency of mixed infections. *E. heterophylla* and *B. rhomboidea* are annuals, while *Sida* and *Macroptilium* are perennials, suggesting a relationship between life span, the opportunity for virus infection, and reservoir potential for crops—a good topic for future studies. The mixing vessel hosts will be hubs for transmissions and for virus diversification, acting as efficient reservoirs for the emergence of begomoviruses. In agreement with this hypothesis, *Macroptilium* yellow spot virus (MaYSV) has recently emerged in bean crops, and its genetic diversity is similar in the crop and the wild hosts (137). However, so far no begomovirus has emerged in malvaceous crops (such as cotton) in Brazil, again indicating the difficulty of driving general models for virus emergence.

Geminiviruses at the Interface Between Wild and Cultivated Hosts

The question of whether wild hosts are reservoirs for geminivirus emergence in crops has been addressed by studies of geminivirus variation in wild and cultivated hosts. The first of these

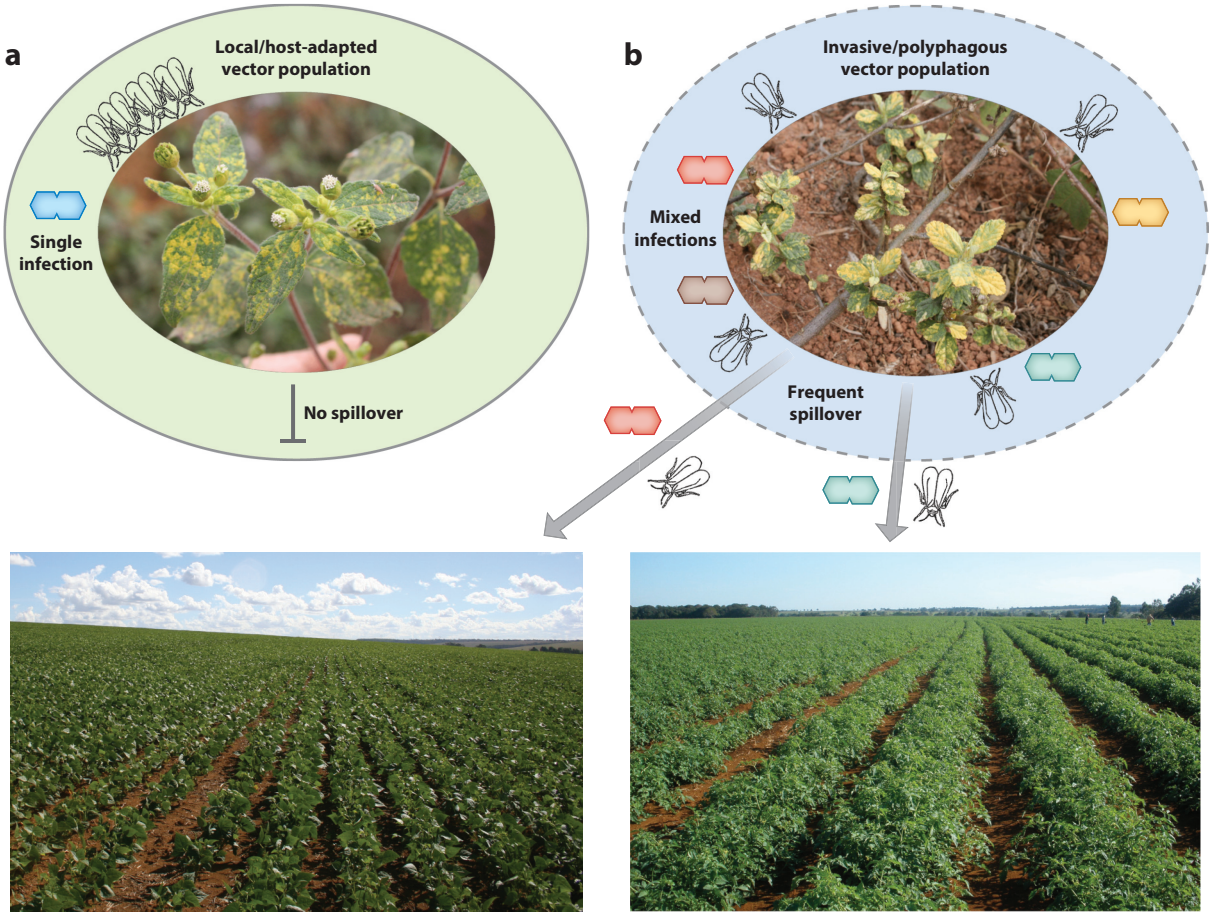


Figure 3

Two different patterns of plant-geminivirus interactions in wild hosts. (*a*) In sealed container hosts such as *Blainvillea rhomboidea*, a highly adapted geminivirus would exclude multiple infections with other less adapted viruses and would rarely infect other less susceptible or competent hosts. This high degree of adaptation, possibly coupled with colonization by local and/or host-adapted vector populations, would prevent spillover to crops. (*b*) In mixing vessel hosts such as *Sida rhombifolia*, efficient infection by several similarly adapted geminiviruses would result in a high frequency of mixed infections. Mixing vessel hosts would be hubs for virus diversification, acting as efficient reservoirs for the emergence of geminiviruses in crops mediated by polyphagous vector populations.

studies analyzed sequences of TLCV from its wild hosts *Eupatorium makinoi*, *Eupatorium glehni*, and *Lonicera japonica* from different locations in Japan (27). TLCV showed a high diversity in these wild hosts, and phylogenies indicated frequent transmission between hosts and sites. The authors argued that high diversity in *Eupatorium* spp. suggests this as a primary host for TLCV from which infection to the introduced tobacco and tomato crops would have occurred. However, this work did not analyze TLCV sequences from crops together with the sequences from wild plants.

Many of the more recent studies on the incidence and genetic variation of geminiviruses at the interface between wild and cultivated hosts have been done in Brazil, where a plethora of begomoviruses have emerged in the recent past in crops (118). These studies include the comparison of different viruses infecting wild and cultivated hosts in the same geographic area as well as of populations of a specific virus over its wild and cultivated host range.

A group of studies focused on legume-infecting viruses. BGMV was first reported infecting common bean (*P. vulgaris*) in Brazil in the 1960s (138) and quickly became one of the most important pathogens of that crop (139). BGMV also infects a number of wild hosts in the Fabaceae, in which it causes severe golden mosaic symptoms as in common bean (140). BGMV was the only begomovirus detected in common bean in Brazil until 2012, when samples with golden mosaic symptoms from the state of Alagoas were found to be infected by MaYSV, which emerged as the most frequent bean begomovirus in that region (137). Evidence for the recent emergence of MaYSV is the fact that it was not detected using pyro-sequencing in cultivated and wild legume host samples collected in 2003 and 2004 in three northeastern Brazilian states, including Alagoas (141). Thus, BGMV and MaYSV have host ranges encompassing wild plants and crops. Studies previous to the emergence of MaYSV in bean crops had indicated a low genetic variation for BGMV in common bean (139) and a high genetic variation for MaYSV in its wild host, *Macroptilium lathyroides* (44). After MaYSV emergence in bean crops, a large-scale systematic study of the genetic variability of BGMV and MaYSV populations in two crops, common bean and lima bean (*Phaseolus lunatus*), and the wild host *M. lathyroides*, was conducted (137). While the diversity of the whole population of both viruses was similar, the subpopulations in each of the three hosts were much less diverse for BGMV than for MaYSV, particularly the DNA-A (0.0018–0.0153 nucleotide substitutions per site for BGMV versus 0.0590–0.0662 for MaYSV). These results can be explained by the BGMV population being structured according to host, while the MaYSV population was not differentiated according to host (137). They also indicated that the diversity of the virus populations did not depend on the wild or cultivated nature of the host. Moreover, they strongly suggested that BGMV is adapted to its different hosts and that adaptation possibly involves across-host trade-offs (9). This is not the case for the MaYSV population, which would be under epidemic expansion over its host range. In this scenario, invasion of new hosts (in the case of MaYSV, bean) would not involve significant trade-offs, with the virus still exploring the fitness landscape.

The above study strongly indicates that *Macroptilium* spp. are the reservoir hosts for MaYSV emergence in *Phaseolus* crops. Other studies failed to provide evidence for a reservoir or for the virus having infected the crop from the wild host or the contrary. Thus, it could not be determined if wild chiltepin is a reservoir host of PepGMV or PHYVV for pepper crops or vice versa. Diversification of chiltepin populations of PepGMV or PHYVV is recent (~30 years ago) and coincidental in time with reports of PepGMV and PHYVV as important pathogens of domestic pepper crops (*C. annuum* var. *annuum*) in large areas of Mexico (31, 142, 143). Also, nucleotide sequences of isolates from pepper crops or from chiltepin are not differentiated, providing no information on transmission between both host plants (31). Similarly, the study of begomoviruses infecting tomato crops in Brazil did not indicate an ancestral relationship between viruses from noncultivated and cultivated hosts (56). Intriguingly, it could be that reservoirs were not identified because sampling intensity fails to detect infection by viruses that occur at low incidence or low titer. A study employing high-throughput sequencing of tomato and *Sida* plants growing side by side in fields in Minas Gerais (Brazil) showed that 99.5% of the reads from *Sida* mapped to SimMV but 0.4% mapped to ToSRV, while in tomato plants >99.3% of the reads mapped to ToSRV and 0.6% to SimMV (C.G. Ferro & F.M. Zerbini, unpublished results). These results are compatible with transmission occurring between tomato and *Sida* plants, but the within-host virus population would have evolved so that in each host the fittest virus (SimMV in *Sida*, ToSRV in tomato) became most prevalent. Although this study did not identify the reservoir host for ToSRV emergence, it shows that an overlooked wild host could have an important role as an inoculum source for epidemics in tomato crops.

We cannot finish this section without discussing what is perhaps the only case in which both the wild host reservoir and the genetic mechanisms leading to emergence have been determined. Maize streak disease is the major viral disease of maize in SSA, described in South Africa in 1901 after an important epidemic in 1896 (144). Etiology research led to the characterization of MSV as the first virus with geminate particles and the coining of the term geminivirus (145). Like cassava, maize was introduced in Africa by Portuguese traders in the sixteenth century (144), and neither the disease nor MSV existed in America, where maize was domesticated. The search for local reservoirs showed that MSV infects more than 140 species of grasses, where the leafhopper vectors (*Cicadullina* spp.) feed and breed (146). At least 11 strains of the virus (MSV-A to MSV-K) occur in different wild grasses in Africa (147), with MSV-A being the cause of the severe disease of maize. The MSV-A strain seems to have arisen by recombination of MSV-B and strains related to MSV-G and MSV-F, all from *Digitaria*, that replicate to low titers and cause mild symptoms in maize (147). Because interstrain recombination in MSV has been repeatedly shown to result in severe fitness penalties (37, 39, 40), the production of a recombinant successful in maize has probably been a rare event. Full genome analyses of 353 MSV-A isolates from all over its range in Africa led to the characterization of 24 different MSV-A lineages based on recombination event patterns, many of which have different geographical distributions (148). Phylogeographic reconstructions inferred that all MSV-A lineages have a common origin in southern Africa in the 1860s and spread in the 1950s to cause the current pandemic (148). The success of MSV-A has been explained by its high titers in maize as well as its extended host range as compared to other MSV strains, which allows it to efficiently infect and multiply in grasses from eight genera (147). Thus, MSV emergence in maize is one of the few instances in plant virology in which reservoir hosts (*Digitaria* and other wild grasses) and evolutionary mechanisms related to adaptation to the new host (maize) have been identified. Emergence of MSV-A may have been followed by spill back from maize to wild grasses, with significant epidemiological consequences. Interestingly, a recent study reported the infection of maize in Africa by MSV-C, previously reported only from Panicoidea wild grasses (149), which suggests the possibility of the emergence of new MSV strains.

CONCLUDING REMARKS AND FUTURE PERSPECTIVES

Studies of plant virus emergence have mostly focused on the evolutionary factors that determine host range and adaptation to new hosts, with the consideration of ecological factors driving emergence lagging. Specifically, understanding of plant virus emergence is currently hampered by scant knowledge on virus prevalence, transmission pathways, and genetic variability in wild plant hosts. The analysis of virus interactions with wild plants growing in wild or anthropic habitats is challenging, as infection is often asymptomatic and studies need to be based on the systematic sampling of a high number of individuals from many potential host species. Also, the often-lower titers viruses reach in wild plants compared to crops may render detection difficult. Deep sequencing technologies may alleviate these challenges and are increasingly being used in studies of plant virus ecology at the landscape scale with so-called geometagenomics approaches. Geminiviruses, and particularly begomoviruses, have emerged as important crop pathogens in the recent past, prompting the analysis of the factors that drove emergence. Geminiviruses have high genetic variability, and their populations are often highly diverse, so analyses of population structure can inform host adaptation and inoculum fluxes. Thus, a large fraction of the current knowledge of plant-virus interactions in wild systems derives from the study of geminiviruses. These studies have provided information on (*a*) actual host ranges and transmission pathways; (*b*) plant-virus and virus-virus interactions in multihost/multivirus systems; (*c*) virus genetic diversity over its host range, including wild and cultivated hosts; and (*d*) genetic structure of the virus population over its host range and

at different spatial scales. Although the current knowledge is far from complete, it suggests trends and provides the bases to develop hypotheses on the role of wild hosts on geminivirus emergence that require testing through the study of more pathosystems in more geographical areas.

We hope that this review has shown that the studies of plant virus ecology at the landscape scale in general and geminivirus infection at the interface of wild and cultivated hosts in particular are exciting areas of research in need of further efforts.

SUMMARY POINTS

1. Virus emergence requires that the virus becomes adapted to the new host and vector. Adaptation is facilitated by a high degree of genetic variability in the pathogen population, which is often the case with geminiviruses.
2. Ecosystem simplification/anthropization affects the infection dynamics, virulence, and evolution of geminiviruses and seems to be a key factor driving their emergence in crops.
3. Different begomoviruses have their own evolutionary dynamics, even when sharing the same hosts and being transmitted by the same vectors.
4. The global spread of TYLCV illustrates the combined roles of genetic and ecological factors in driving geminivirus emergence. These include efficient vector transmission by indigenous and invasive species of the *B. tabaci* complex, the ability to infect additional wild and crop plants, and the capacity to multiply in tomato cultivars carrying resistance genes.
5. Begomoviruses infecting tomato in the Americas originated from indigenous wild hosts, from which different viruses have emerged in different geographical areas (spillover), reaching different levels of adaptation to the host and/or environment. These crop-adapted viruses can be occasionally transferred back to wild hosts (spill back).
6. The level of genetic variability seems to be an intrinsic property of the virus, regardless of host range or coexistence in mixing infection with other viruses. Nevertheless, mixing vessel hosts could be hubs for transmission and for virus diversification, acting as efficient reservoirs for the emergence of begomoviruses.

FUTURE ISSUES

1. Studies at the landscape scale should illuminate the role of ecosystem simplification favoring geminivirus prevalence and of anthropic habitats as reservoir communities for emergence in crops.
2. The possible existence of two types of wild hosts (sealed containers and mixing vessels) needs to be investigated in greater detail, including their supposedly differential roles as virus reservoirs.
3. The relative significance of spillover and spill back between wild and cultivated hosts in providing inoculum sources for epidemics in crops needs to be better understood.
4. There is still much to be learned about the role of local and invasive vector populations on viral emergence, especially in Africa and the Americas.

5. The fitness of different viral genotypes in wild and crop hosts needs to be investigated. Approaches could be cross-infection experiments and short-term, forced evolution experiments in the original and assayed hosts.
6. One particularly underexplored topic is the possible role of genetic drift on the evolution of geminivirus populations in wild and crop hosts. Temporal studies using high-throughput sequencing will be useful in examining this aspect.

DISCLOSURE STATEMENT

The authors are not aware of any affiliations, memberships, funding, or financial holdings that might be favorably or adversely affected by this review.

ACKNOWLEDGMENTS

We apologize in advance to our colleagues whose work could not be appropriately cited due to space limitations. F.G.A. is supported in part by grants BFU2015-60418-R, Plan Estatal de I+D+i, Spain. F.M.Z. is supported by grants from CAPES (finance code 001), CNPq, and FAPEMIG. The authors wish to thank Dr. M.J. McLeish for assistance in preparing **Figure 1** and Osvaldo F.L. Sande for assistance in preparing **Figure 2**.

LITERATURE CITED

1. Roossinck MJ. 2011. The good viruses: viral mutualistic symbioses. *Nat. Rev. Microbiol.* 9:99–108
2. Fraile A, Garcia-Arenal F. 2016. Environment and evolution modulate plant virus pathogenesis. *Curr. Opin. Virol.* 17:50–56
3. Nicaise V. 2014. Crop immunity against viruses: outcomes and future challenges. *Front. Plant Sci.* 5:660
4. Sastry SK, Zitter TA. 2014. Management of virus and viroid diseases of crops in the tropics. In *Plant Virus and Viroid Diseases in the Tropics*, ed. SK Sastry, TA Zitter, pp. 149–480. Dordrecht, Neth.: Springer
5. Woolhouse ME. 2002. Population biology of emerging and re-emerging pathogens. *Trends Microbiol.* 10:S3–7
6. Anderson PK, Cunningham AA, Patel NG, Morales FJ, Epstein PR, Daszak P. 2004. Emerging infectious diseases of plants: pathogen pollution, climate change and agrotechnology drivers. *Trends Ecol. Evol.* 19:535–44
7. Dobson A, Foufopoulos J. 2001. Emerging infectious pathogens of wildlife. *Philos. Trans. R. Soc. B Biol. Sci.* 356:1001–12
8. Taylor LH, Latham SM, Woolhouse ME. 2001. Risk factors for human disease emergence. *Philos. Trans. R. Soc. B Biol. Sci.* 356:983–89
9. Elena SF, Fraile A, Garcia-Arenal F. 2014. Evolution and emergence of plant viruses. *Adv. Virus Res.* 88:161–91
10. Bernardo P, Charles-Dominique T, Barakat M, Ortet P, Fernandez E, et al. 2018. Geometagenomics illuminates the impact of agriculture on the distribution and prevalence of plant viruses at the ecosystem scale. *ISME J.* 12:173–84
11. Thapa V, McGlinn DJ, Melcher U, Palmer MW, Roossinck MJ. 2015. Determinants of taxonomic composition of plant viruses at the Nature Conservancy's Tallgrass Prairie Preserve, Oklahoma. *Virus Evol.* 1:vev007
12. Roossinck MJ. 2012. Plant virus metagenomics: biodiversity and ecology. *Annu. Rev. Gen.* 46:359–69
13. Roossinck MJ. 2003. Plant RNA virus evolution. *Curr. Opin. Microbiol.* 6:406–9

14. Alexander HM, Mauck KE, Whitfield AE, Garrett KA, Malmstrom CM. 2014. Plant-virus interactions and the agro-ecological interface. *Eur. J. Plant Pathol.* 138:529–47
15. Sattar MN, Kvarnheden A, Saeed M, Briddon RW. 2013. Cotton leaf curl disease—an emerging threat to cotton production worldwide. *J. Gen. Virol.* 94:695–710
16. Navas-Castillo J, Fiallo-Olivé E, Sánchez-Campos S. 2011. Emerging virus diseases transmitted by whiteflies. *Annu. Rev. Phytopathol.* 49:219–48
17. Moriones E, Praveen S, Chakraborty S. 2017. Tomato leaf curl New Delhi virus: an emerging virus complex threatening vegetable and fiber crops. *Viruses* 9:264
18. Gilbertson RL, Batuman O, Webster CG, Adkins S. 2015. Role of the insect supervectors *Bemisia tabaci* and *Frankliniella occidentalis* in the emergence and global spread of plant viruses. *Annu. Rev. Virol.* 2:67–93
19. Rojas MR, Macedo MA, Maliano MR, Soto-Aguilar M, Souza JO, et al. 2018. World management of geminiviruses. *Annu. Rev. Phytopathol.* 56:637–77
20. Zerbini FM, Briddon RW, Idris A, Martin DP, Moriones E, et al. 2017. ICTV virus taxonomy profile: *Geminiviridae*. *J. Gen. Virol.* 98:131–33
21. Hanley-Bowdoin L, Bejarano ER, Robertson D, Mansoor S. 2013. Geminiviruses: masters at redirecting and reprogramming plant processes. *Nat. Rev. Microbiol.* 11:777–88
22. Lefeuvre P, Moriones E. 2015. Recombination as a motor of host switches and virus emergence: geminiviruses as case studies. *Curr. Opin. Virol.* 10:14–19
23. Sanjuán R, Nebot MR, Chirico N, Mansky LM, Belshaw R. 2010. Viral mutation rates. *J. Virol.* 84:9733–48
24. Sanz AI, Fraile A, Gallego JM, Malpica JM, García-Arenal F. 1999. Genetic variability of natural populations of cotton leaf curl geminivirus, a single-stranded DNA virus. *J. Mol. Evol.* 49:672–81
25. Harkins GW, Delport W, Duffy S, Wood N, Monjane AL, et al. 2009. Experimental evidence indicating that mastreviruses probably did not co-diverge with their hosts. *Virol. J.* 6:104
26. Duffy S, Shackelton LA, Holmes EC. 2008. Rates of evolutionary change in viruses: patterns and determinants. *Nat. Rev. Genet.* 9:267–76
27. Ooi K, Ohshita S, Ishii I, Yahara T. 1997. Molecular phylogeny of geminivirus infecting wild plants in Japan. *J. Plant Res.* 110:247–57
28. Duffy S, Holmes EC. 2008. Phylogenetic evidence for rapid rates of molecular evolution in the single-stranded DNA begomovirus tomato yellow leaf curl virus. *J. Virol.* 82:957–65
29. Duffy S, Holmes EC. 2009. Validation of high rates of nucleotide substitution in geminiviruses: phylogenetic evidence from East African cassava mosaic viruses. *J. Gen. Virol.* 90:1539–47
30. Lefeuvre P, Harkins GW, Lett JM, Briddon RW, Chase MW, et al. 2011. Evolutionary time-scale of the begomoviruses: evidence from integrated sequences in the *Nicotiana* genome. *PLOS ONE* 6:e19193
31. Rodelo-Urrego M, Pagán I, González-Jara P, Betancourt M, Moreno-Letelier A, et al. 2013. Landscape heterogeneity shapes host-parasite interactions and results in apparent plant-virus codivergence. *Mol. Ecol.* 22:2325–40
32. Xu C, Sun X, Taylor A, Jiao C, Xu Y, et al. 2017. Diversity, distribution, and evolution of tomato viruses in China uncovered by small RNA sequencing. *J. Virol.* 91:e00173-17
33. Sanjuán R. 2012. From molecular genetics to phylodynamics: evolutionary relevance of mutation rates across viruses. *PLOS Path.* 8:e1002685
34. Padidam M, Sawyer S, Fauquet CM. 1999. Possible emergence of new geminiviruses by frequent recombination. *Virology* 265:218–24
35. Briddon RW, Patil BL, Bagewadi B, Nawaz-ul-Rehman MS, Fauquet CM. 2010. Distinct evolutionary histories of the DNA-A and DNA-B components of bipartite begomoviruses. *BMC Evol. Biol.* 10:97
36. Jeske H, Lutgemeier M, Preiss W. 2001. DNA forms indicate rolling circle and recombination-dependent replication of *Abutilon* mosaic virus. *EMBO J.* 20:6158–67
37. Martin DP, Lefeuvre P, Varsani A, Hoareau M, Semegni JY, et al. 2011. Complex recombination patterns arising during geminivirus coinfections preserve and demarcate biologically important intra-genome interaction networks. *PLOS Path.* 7:e1002203
38. Lefeuvre P, Lett JM, Reynaud B, Martin DP. 2007. Avoidance of protein fold disruption in natural virus recombinants. *PLOS Path.* 3:e181

39. Martin DP, van der Walt E, Posada D, Rybicki EP. 2005. The evolutionary value of recombination is constrained by genome modularity. *PLOS Genet*. 1:e51
40. Monjane AL, Martin DP, Lakay F, Muhire BM, Pande D, et al. 2014. Extensive recombination-induced disruption of genetic interactions is highly deleterious but can be partially reversed by small numbers of secondary recombination events. *J. Virol.* 88:7843–51
41. Sanz AI, Fraile A, García-Arenal F, Zhou X, Robinson DJ, et al. 2000. Multiple infection, recombination and genome relationships among begomovirus isolates found in cotton and other plants in Pakistan. *J. Gen. Virol.* 81:1839–49
42. Zhou X, Liu Y, Calvert L, Munoz C, Otim-Nape GW, et al. 1997. Evidence that DNA-A of a geminivirus associated with severe cassava mosaic disease in Uganda has arisen by interspecific recombination. *J. Gen. Virol.* 78:2101–11
43. Zhou X, Liu Y, Robinson DJ, Harrison BD. 1998. Four DNA-A variants among Pakistani isolates of cotton leaf curl virus and their affinities to DNA-A of geminivirus isolates from okra. *J. Gen. Virol.* 79:915–23
44. Lima ATM, Sobrinho RR, Gonzalez-Aguilera J, Rocha CS, Silva SJC, et al. 2013. Synonymous site variation due to recombination explains higher genetic variability in begomovirus populations infecting non-cultivated hosts. *J. Gen. Virol.* 94:418–31
45. Silva SJC, Castillo-Urquiza GP, Hora-Júnior BT, Assunção IP, Lima GSA, et al. 2011. High genetic variability and recombination in a begomovirus population infecting the ubiquitous weed *Cleome affinis* in northeastern Brazil. *Arch. Virol.* 156:2205–13
46. Silva SJC, Castillo-Urquiza GP, Hora-Junior BT, Assunção IP, Lima GSA, et al. 2012. Species diversity, phylogeny and genetic variability of begomovirus populations infecting leguminous weeds in northeastern Brazil. *Plant Pathol.* 61:457–67
47. Rodelo-Urrego M, García-Arenal F, Pagán I. 2015. The effect of ecosystem biodiversity on virus genetic diversity depends on virus species: a study of chiltepin-infecting begomoviruses in Mexico. *Virus Evol.* 1:vev004
48. García-Andrés S, Monci F, Navas-Castillo J, Moriones E. 2006. Begomovirus genetic diversity in the native plant reservoir *Solanum nigrum*: evidence for the presence of a new virus species of recombinant nature. *Virology* 350:433–42
49. Mar TB, Xavier CAD, Lima ATM, Nogueira AM, Silva JCF, et al. 2017. Genetic variability and population structure of the New World begomovirus *Euphorbia* yellow mosaic virus. *J. Gen. Virol.* 98:1537–51
50. Varsani A, Lefeuvre P, Roumagnac P, Martin D. 2018. Notes on recombination and reassortment in multipartite/segmented viruses. *Curr. Opin. Virol.* 33:156–66
51. Pita JS, Fondong VN, Sangare A, Otim-Nape GW, Ogwal S, Fauquet CM. 2001. Recombination, pseudorecombination and synergism of geminiviruses are determinant keys to the epidemic of severe cassava mosaic disease in Uganda. *J. Gen. Virol.* 82:655–65
52. Silva FN, Lima ATM, Rocha CS, Castillo-Urquiza GP, Alves-Júnior M, Zerbini FM. 2014. Recombination and pseudorecombination driving the evolution of the begomoviruses *tomato severe rugose virus* (ToSRV) and *tomato rugose mosaic virus* (ToRMV): two recombinant DNA-A components sharing the same DNA-B. *Virol. J.* 11:66
53. Monci F, Sanchez-Campos S, Navas-Castillo J, Moriones E. 2002. A natural recombinant between the geminiviruses *tomato yellow leaf curl Sardinia virus* and *tomato yellow leaf curl virus* exhibits a novel pathogenic phenotype and is becoming prevalent in Spanish populations. *Virology* 303:317–26
54. Lima ATM, Silva JCF, Silva FN, Castillo-Urquiza GP, Silva FF, et al. 2017. The diversification of begomovirus populations is predominantly driven by mutational dynamics. *Virus Evol.* 3:vex005
55. Sánchez-Campos S, Diaz JA, Monci F, Bejarano ER, Reina J, et al. 2002. High genetic stability of the begomovirus *tomato yellow leaf curl Sardinia virus* in southern Spain over an 8-year period. *Phytopathology* 92:842–49
56. Rocha CS, Castillo-Urquiza GP, Lima ATM, Silva FN, Xavier CAD, et al. 2013. Brazilian begomovirus populations are highly recombinant, rapidly evolving, and segregated based on geographical location. *J. Virol.* 87:5784–99

57. Jones RAC. 2009. Plant virus emergence and evolution: origins, new encounter scenarios, factors driving emergence, effects of changing world conditions, and prospects for control. *Virus Res.* 141:113–30
58. Jones RAC. 2018. Plant and insect viruses in managed and natural environments: novel and neglected transmission pathways. *Adv. Virus Res.* 101:149–87
59. Jones KE, Patel NG, Levy MA, Storeygard A, Balk D, et al. 2008. Global trends in emerging infectious diseases. *Nature* 451:990–93
60. Woolhouse MEJ, Gowtage-Sequeria S. 2005. Host range and emerging and reemerging pathogens. *Emerg. Inf. Dis.* 11:1843–47
61. Burdon JJ, Chilvers GA. 1982. Host density as a factor in plant-disease ecology. *Annu. Rev. Phytopathol.* 20:143–66
62. Stukenbrock EH, McDonald BA. 2008. The origin of plant pathogens in agro-ecosystems. *Annu. Rev. Phytopathol.* 46:75–100
63. Keesing F, Holt RD, Ostfeld RS. 2006. Effects of species diversity on disease risk. *Ecol. Lett.* 9:485–98
64. McLeish MJ, Fraile A, García-Arenal F. 2019. Evolution of plant-virus interactions: host range and virus emergence. *Curr. Opin. Virol.* 34:50–55
65. Mideo N, Alizon S, Day T. 2008. Linking within- and between-host dynamics in the evolutionary epidemiology of infectious diseases. *Trends Ecol. Evol.* 23:511–17
66. Roossinck MJ, Saha P, Wiley GB, Quan J, White JD, et al. 2010. Ecogenomics: using massively parallel pyrosequencing to understand virus ecology. *Mol. Ecol.* 19:81–88
67. Muthukumar V, Melcher U, Pierce M, Wiley GB, Roe BA, et al. 2009. Non-cultivated plants of the Tallgrass Prairie Preserve of northeastern Oklahoma frequently contain virus-like sequences in particulate fractions. *Virus Res.* 141:169–73
68. Claverie S, Bernardo P, Kraberger S, Hartnady P, Lefeuvre P, et al. 2018. From spatial metagenomics to molecular characterization of plant viruses: a geminivirus case study. *Adv. Virus Res.* 101:55–83
69. Pagán I, González-Jara P, Moreno-Letelier A, Rodelo-Urrego M, Fraile A, et al. 2012. Effect of biodiversity changes in disease risk: exploring disease emergence in a plant-virus system. *PLOS Path.* 8:e1002796
70. Fraile A, McLeish MJ, Pagán I, Gonzalez-Jara P, Piñero D, García-Arenal F. 2017. Environmental heterogeneity and the evolution of plant-virus interactions: viruses in wild pepper populations. *Virus Res.* 241:68–76
71. Morales FJ, Jones PG. 2004. The ecology and epidemiology of whitefly-transmitted viruses in Latin America. *Virus Res.* 100:57–65
72. Polston JE, Anderson PK. 1997. The emergence of whitefly-transmitted geminiviruses in tomato in the Western Hemisphere. *Plant Dis.* 81:1358–69
73. Ribeiro SG, Ambrozevicius LP, Ávila AC, Bezerra IC, Calegario RF, et al. 2003. Distribution and genetic diversity of tomato-infecting begomoviruses in Brazil. *Arch. Virol.* 148:281–95
74. Jacobson AL, Duffy S, Sseruwagi P. 2018. Whitefly-transmitted viruses threatening cassava production in Africa. *Curr. Opin. Virol.* 33:167–76
75. Carter SE, Fresco LO, Jones PG, Fairbairn JN. 1993. *Introduction and Diffusion of Cassava in Africa.* Ibadan, Niger.: Int. Inst. Trop. Agric.
76. Legg JP, Lava Kumar P, Makeshkumar T, Tripathi L, Ferguson M, et al. 2015. Cassava virus diseases: biology, epidemiology, and management. *Adv. Virus Res.* 91:85–142
77. Fondong VN, Pita JS, Rey MEC, Kochko A, Beachy RN, Fauquet CM. 2000. Evidence of synergism between African cassava mosaic virus and a new double-recombinant geminivirus infecting cassava in Cameroon. *J. Gen. Virol.* 81:287–97
78. Vanitharani R, Chellappan P, Pita JS, Fauquet CM. 2004. Differential roles of AC2 and AC4 of cassava geminiviruses in mediating synergism and suppression of posttranscriptional gene silencing. *J. Virol.* 78:9487–98
79. Ndunguru J, Legg J, Aveling T, Thompson G, Fauquet C. 2005. Molecular biodiversity of cassava begomoviruses in Tanzania: evolution of cassava geminiviruses in Africa and evidence for East Africa being a center of diversity of cassava geminiviruses. *Virol. J.* 2:21

80. Sserubombwe WS, Briddon RW, Baguma YK, Ssemakula GN, Bull SE, et al. 2008. Diversity of begomoviruses associated with mosaic disease of cultivated cassava (*Manihot esculenta* Crantz) and its wild relative (*Manihot glaziovii* Müll. Arg.) in Uganda. *J. Gen. Virol.* 89:1759–69
81. Bull SE, Briddon RW, Sserubombwe WS, Ngugi K, Markham PG, Stanley J. 2006. Genetic diversity and phylogeography of cassava mosaic viruses in Kenya. *J. Gen. Virol.* 87:3053–65
82. Fauquet C, Fargette D. 1990. African cassava mosaic virus: etiology, epidemiology and control. *Plant Dis.* 74:404–11
83. Legg JP, French R, Rogan D, Okao-Okuja G, Brown JK. 2002. A distinct *Bemisia tabaci* (Gennadius) (Hemiptera: Sternorrhyncha: Aleyrodidae) genotype cluster is associated with the epidemic of severe cassava mosaic virus disease in Uganda. *Mol. Ecol.* 11:1219–29
84. Fondong VN, Chen KG. 2011. Genetic variability of East African cassava mosaic Cameroon virus under field and controlled environment conditions. *Virology* 413:275–82
85. De Bruyn A, Harimalala M, Zinga I, Mabvakure BM, Hoareau M, et al. 2016. Divergent evolutionary and epidemiological dynamics of cassava mosaic geminiviruses in Madagascar. *BMC Evol. Biol.* 16:182
86. Lefeuvre P, Martin DP, Harkins G, Lemey P, Gray AJA, et al. 2010. The spread of tomato yellow leaf curl virus from the Middle East to the world. *PLOS Path.* 6:e1001164
87. Mabvakure B, Martin DP, Kraberger S, Cloete L, van Brunschot S, et al. 2016. Ongoing geographical spread of tomato yellow leaf curl virus. *Virology* 498:257–64
88. Papayiannis LC, Katis NI, Idris AM, Brown JK. 2011. Identification of weed hosts of tomato yellow leaf curl virus in Cyprus. *Plant Dis.* 95:120–25
89. Polston JE, Cohen L, Sherwood TA, Ben-Joseph R, Lapidot M. 2006. *Capsicum* species: symptomless hosts and reservoirs of tomato yellow leaf curl virus. *Phytopathology* 96:447–52
90. Salati R, Nahkla MK, Rojas MR, Guzman P, Jaquez J, et al. 2002. Tomato yellow leaf curl virus in the Dominican Republic: characterization of an infectious clone, virus monitoring in whiteflies, and identification of reservoir hosts. *Phytopathology* 92:487–96
91. Davino S, Napoli C, Davino M, Accotto GP. 2006. Spread of tomato yellow leaf curl virus in Sicily: partial displacement of another geminivirus originally present. *Eur. J. Plant Pathol.* 114:293–99
92. Sánchez-Campos S, Navas-Castillo J, Camero R, Soria C, Díaz JA, Moriones E. 1999. Displacement of tomato yellow leaf curl virus (TYLCV)-Sr by TYLCV-Is in tomato epidemics in Spain. *Phytopathology* 89:1038–43
93. Moriones E, Navas-Castillo J. 2008. Rapid evolution of the population of begomoviruses associated with the tomato yellow leaf curl disease after invasion of a new ecological niche. *Span. J. Agric. Res.* 6:147–59
94. García-Andrés S, Tomas DM, Navas-Castillo J, Moriones E. 2009. Resistance-driven selection of begomoviruses associated with the tomato yellow leaf curl disease. *Virus Res.* 146:66–72
95. Ge LM, Zhang JT, Zhou XP, Li HY. 2007. Genetic structure and population variability of tomato yellow leaf curl China virus. *J. Virol.* 81:5902–7
96. Yang XL, Zhou MN, Qian YJ, Xie Y, Zhou XP. 2014. Molecular variability and evolution of a natural population of tomato yellow leaf curl virus in Shanghai, China. *J. Zhejiang Univ. Sci. B* 15:133–42
97. Belabess Z, Urbino C, Granier M, Tahiri A, Blenzar A, Peterschmitt M. 2018. The typical RB76 recombination breakpoint of the invasive recombinant tomato yellow leaf curl virus of Morocco can be generated experimentally but is not positively selected in tomato. *Virus Res.* 243:44–51
98. Belabess Z, Dallot S, El-Montaser S, Granier M, Majde M, et al. 2015. Monitoring the dynamics of emergence of a non-canonical recombinant of tomato yellow leaf curl virus and displacement of its parental viruses in tomato. *Virology* 486:291–306
99. García-Andrés S, Tomas DM, Sanchez-Campos S, Navas-Castillo J, Moriones E. 2007. Frequent occurrence of recombinants in mixed infections of tomato yellow leaf curl disease-associated begomoviruses. *Virology* 365:210–19
100. Davino S, Napoli C, Dellacroce C, Miozzi L, Noris E, et al. 2009. Two new natural begomovirus recombinants associated with the tomato yellow leaf curl disease co-exist with parental viruses in tomato epidemics in Italy. *Virus Res.* 143:15–23

101. García-Andrés S, Accotto GP, Navas-Castillo J, Moriones E. 2007. Founder effect, plant host, and recombination shape the emergent population of begomoviruses that cause the tomato yellow leaf curl disease in the Mediterranean basin. *Virology* 359:302–12
102. Sánchez-Campos S, Domínguez-Huerta G, Díaz-Martínez L, Tomás DM, Navas-Castillo J, et al. 2018. Differential shape of geminivirus mutant spectra across cultivated and wild hosts with invariant viral consensus sequences. *Front. Plant Sci.* 9:932
103. Belabess Z, Peterschmitt M, Granier M, Tahiri A, Blenzar A, Urbino C. 2016. The non-canonical tomato yellow leaf curl virus recombinant that displaced its parental viruses in southern Morocco exhibits a high selective advantage in experimental conditions. *J. Gen. Virol.* 97:3433–45
104. Albuquerque LC, Varsani A, Fernandes FR, Pinheiro B, Martin DP, et al. 2012. Further characterization of tomato-infecting begomoviruses in Brazil. *Arch. Virol.* 157:747–52
105. Ribeiro SG, Martin DP, Lacorte C, Simões IC, Orlandini DRS, Inoue-Nagata AK. 2007. Molecular and biological characterization of tomato chlorotic mottle virus suggests that recombination underlies the evolution and diversity of Brazilian tomato begomoviruses. *Phytopathology* 97:702–11
106. Castillo-Urquiza GP, Beserra JEA Jr., Bruckner FP, Lima ATM, Varsani A, et al. 2008. Six novel begomoviruses infecting tomato and associated weeds in Southeastern Brazil. *Arch. Virol.* 153:1985–89
107. Paplomatas EJ, Patel VP, Hou YM, Noueiry AO, Gilbertson RL. 1994. Molecular characterization of a new sap-transmissible bipartite genome geminivirus infecting tomatoes in Mexico. *Phytopathology* 84:1215–24
108. Nahkla MK, Maxwell MD, Hidayat SH, Lange DR, Loniello AO, et al. 1994. Two geminiviruses associated with tomatoes in Central America. *Phytopathology* 84:467
109. Melgarejo TA, Kon T, Rojas MR, Paz-Carrasco L, Zerbini FM, Gilbertson RL. 2013. Characterization of a new world monopartite begomovirus causing leaf curl disease of tomato in Ecuador and Peru reveals a new direction in geminivirus evolution. *J. Virol.* 87:5397–413
110. Ramos PL, Guerra O, Peral R, Oramas P, Guevara RG, Rivera-Bustamante R. 1997. Taino tomato mottle virus, a new bipartite Geminivirus from Cuba. *Plant Dis.* 81:1095
111. Rojas A, Kvarnheden A, Valkonen JPT. 2000. Geminiviruses infecting tomato crops in Nicaragua. *Plant Dis.* 84:843–46
112. Nakhla MK, Maxwell DP, Martinez RT, Carvalho MG, Gilbertson RL. 1994. Widespread occurrence of eastern Mediterranean "strain" of tomato yellow leaf curl geminivirus in tomatoes in the Dominican Republic. *Plant Dis.* 78:926
113. Duffy S, Holmes EC. 2007. Multiple introductions of the old world begomovirus tomato yellow leaf curl virus into the new world. *Appl. Environ. Microb.* 73:7114–17
114. Barreto SS, Hallwass M, Aquino OM, Inoue-Nagata AK. 2013. A study of weeds as potential inoculum sources for a tomato-infecting begomovirus in central Brazil. *Phytopathology* 103:436–44
115. Fernandes NAN, Boiteux LS, Fonseca MEN, Gonzales-Segnana L, Kitajima EW. 2014. Report of tomato yellow spot virus infecting *Leonurus sibiricus* in Paraguay and within tomato fields in Brazil. *Plant Dis.* 98:1445
116. Rodríguez-Pardina PE, Hanada K, Laguna IG, Zerbini FM, Ducasse DA. 2011. Molecular characterisation and relative incidence of bean- and soybean-infecting begomoviruses in northwestern Argentina. *Ann. Appl. Biol.* 158:69–78
117. Andrade EC, Manhani GG, Alfenas PF, Calegario RF, Fontes EPB, Zerbini FM. 2006. Tomato yellow spot virus, a tomato-infecting begomovirus from Brazil with a closer relationship to viruses from *Sida* sp., forms pseudorecombinants with begomoviruses from tomato but not from *Sida*. *J. Gen. Virol.* 87:3687–96
118. Inoue-Nagata AK, Lima MF, Gilbertson RL. 2016. A review of geminivirus diseases in vegetables and other crops in Brazil: current status and approaches for management. *Hortic. Bras.* 34:8–18
119. Barbosa JC, Rezende JA, Amorim L, Filho AB. 2016. Temporal dynamics of *tomato severe rugose virus* and *Bemisia tabaci* in tomato fields in São Paulo, Brazil. *J. Phytopathol.* 164:1–10
120. Macedo MA, Costa TM, Barbosa JC, Pereira JL, Michereff-Filho M, et al. 2017. Temporal and spatial dynamics of begomovirus disease in tomatoes in central Brazil. *Plant Pathol.* 66:529–38

121. Gibbs A, Trueman J, Gibbs M. 2008. The bean common mosaic virus lineage of potyviruses: Where did it arise and when? *Arch. Virol.* 153:2177–87
122. Nguyen HD, Tomitaka Y, Ho SY, Duchene S, Vetten HJ, et al. 2013. Turnip mosaic potyvirus probably first spread to Eurasian brassica crops from wild orchids about 1000 years ago. *PLOS ONE* 8:e55336
123. Jones RAC, Coutts BA. 2015. Spread of introduced viruses to new plants in natural ecosystems and the threat this poses to plant biodiversity. *Mol. Plant Pathol.* 16:541–45
124. Costa AS, Bennett CW. 1950. Whitefly transmitted mosaic of *Euphorbia prunifolia*. *Phytopathology* 40:266–83
125. Orlando A, Silberschmidt K. 1946. Studies on the natural dissemination of the virus of "infectious chlorosis" of the Malvaceae and its relation with the insect vector *Bemisia tabaci* Genn. *Arq. Inst. Biol.* 17:1–36
126. Costa AS, Carvalho AMB. 1960. Comparative studies between *Abutilon* and *Euphorbia* mosaic viruses. *J. Phytopathol.* 38:129–52
127. Jovel J, Reski G, Rothenstein D, Ringel M, Frischmuth T, Jeske H. 2004. *Sida micrantha* mosaic is associated with a complex infection of begomoviruses different from *Abutilon* mosaic virus. *Arch. Virol.* 149:829–41
128. Fiallo-Olivé E, Zerbini FM, Navas-Castillo J. 2015. Complete nucleotide sequences of two new begomoviruses infecting the wild malvaceous plant *Melochia* sp. in Brazil. *Arch. Virol.* 160:3161–64
129. Wyant PS, Gotthardt D, Schafer B, Krenz B, Jeske H. 2011. The genomes of four novel begomoviruses and a new *Sida micrantha* mosaic virus strain from Bolivian weeds. *Arch. Virol.* 156:347–52
130. Frischmuth T, Engel M, Lauster S, Jeske H. 1997. Nucleotide sequence evidence for the occurrence of three distinct whitefly-transmitted, *Sida*-infecting bipartite geminiviruses in Central America. *J. Gen. Virol.* 78:2675–82
131. Roye ME, McLaughlin WA, Nakhla MK, Maxwell DP. 1997. Genetic diversity among geminiviruses associated with the weed species *Sida* spp., *Macroptilium lathyroides*, and *Wissadula amplissima* from Jamaica. *Plant Dis.* 81:1251–58
132. Ferro CG, Silva JP, Xavier CAD, Godinho MT, Lima ATM, et al. 2017. The ever increasing diversity of begomoviruses infecting non-cultivated hosts: new species from *Sida* spp. and *Leonurus sibiricus*, plus two New World alphasatellites. *Ann. Appl. Biol.* 170:204–18
133. Fiallo-Olivé E, Navas-Castillo J, Moriones E, Martinez-Zubiaur Y. 2012. Begomoviruses infecting weeds in Cuba: increased host range and a novel virus infecting *Sida rhombifolia*. *Arch. Virol.* 157:141–46
134. Fiallo-Olivé E, Martinez-Zubiaur Y, Moriones E, Navas-Castillo J. 2010. Complete nucleotide sequence of *Sida* golden mosaic Florida virus and phylogenetic relationships with other begomoviruses infecting malvaceous weeds in the Caribbean. *Arch. Virol.* 155:1535–37
135. Collins A, Rehman MM, Chowda-Reddy RV, Wang AM, Fondong V, et al. 2010. Molecular characterization and experimental host range of an isolate of *Macroptilium* golden mosaic virus that infects *Wissadula amplissima* in Jamaica. *Virus Res.* 150:148–52
136. Ramos PL, Fernandez A, Castrillo G, Diaz L, Echemendia AL, et al. 2002. *Macroptilium* yellow mosaic virus, a new begomovirus infecting *Macroptilium lathyroides* in Cuba. *Plant Dis.* 86:1049
137. Ramos-Sobrinho R, Xavier CAD, Pereira HMB, Lima GSA, Assunção IP, et al. 2014. Contrasting genetic structure between two begomoviruses infecting the same leguminous hosts. *J. Gen. Virol.* 95:2540–52
138. Costa AS. 1965. Three whitefly-transmitted virus diseases of beans in São Paulo, Brazil. *FAO Plant Prot. Bull.* 13:2–12
139. Faria JC, Maxwell DP. 1999. Variability in geminivirus isolates associated with *Phaseolus* spp. in Brazil. *Phytopathology* 89:262–68
140. Chagas CM, Barradas MM, Vicente M. 1981. Espécies hospedeiras do vírus do mosaico dourado do feijoeiro. *Arq. Inst. Biol.* 48:123–27
141. Wyant PS, Strohmeier S, Schafer B, Krenz B, Assunção IP, et al. 2012. Circular DNA genomics (circomics) exemplified for geminiviruses in bean crops and weeds of northeastern Brazil. *Virology* 427:151–57

142. Garzón-Tiznado JA, Torres-Pacheco I, Ascencio-Ibañez JT, Herrera-Estrella L, Rivera-Bustamante RF. 1993. Inoculation of peppers with infectious clones of a new geminivirus by a biolistic procedure. *Phytopathology* 83:514–21
143. Torres-Pacheco I, Garzón-Tiznado JA, Brown JK, Becerra-Flora A, Rivera-Bustamante R. 1996. Detection and distribution of geminiviruses in Mexico and the southern United States. *Phytopathology* 86:1186–92
144. Shepherd DN, Martin DP, van der Walt E, Dent K, Varsani A, Rybicki EP. 2010. Maize streak virus: an old and complex 'emerging' pathogen. *Mol. Plant Pathol.* 11:1–12
145. Bock KR, Guthrie EJ, Woods RD. 1974. Purification of maize streak virus and its relationship to viruses associated with streak diseases of sugarcane and *Panicum maximum*. *Ann. Appl. Biol.* 77:289–96
146. Konate G, Traore O. 1992. Les hôtes réservoirs du virus de la striure du maïs (MSV) en zone Soudano-Sahélienne: identification et distribution spatio-temporelle. *Phytoprotection* 73:111–17
147. Varsani A, Shepherd DN, Monjane AL, Owor BE, Erdmann JB, et al. 2008. Recombination, decreased host specificity and increased mobility may have driven the emergence of maize streak virus as an agricultural pathogen. *J. Gen. Virol.* 89:2063–74
148. Monjane AL, Harkins GW, Martin DP, Lemey P, Lefeuvre P, et al. 2011. Reconstructing the history of maize streak virus strain A dispersal to reveal diversification hot spots and its origin in southern Africa. *J. Virol.* 85:9623–36
149. Kraberger S, Saumtally S, Pande D, Khoodoo MHR, Dhayan S, et al. 2017. Molecular diversity, geographic distribution and host range of monocot-infecting mastreviruses in Africa and surrounding islands. *Virus Res.* 238:171–78
150. Bahlo M, Griffiths RC. 2000. Inference from gene trees in a subdivided population. *Theor. Popul. Biol.* 57:79–95