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World Management of Geminiviruses

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Abstract

Management of geminiviruses is a worldwide challenge because of the widespread distribution of economically important diseases caused by these viruses. Regardless of the type of agriculture, management is most effective with an integrated pest management (IPM) approach that involves measures before, during, and after the growing season. This includes starting with resistant cultivars and virus- and vector-free transplants and propagative plants. For high value vegetables, protected culture (e.g., greenhouses and screenhouses) allows for effective management but is limited owing to high cost. Protection of young plants in open fields is provided by row covers, but other measures are typically required. Measures that are used for crops in open fields include roguing infected plants and insect vector management. Application of insecticide to manage vectors (whiteflies and leafhoppers) is the most widely used measure but can cause undesirable environmental and human health issues. For annual crops, these measures can be more effective when combined with host-free periods of two to three months. Finally, given the great diversity of the viruses, their insect vectors, and the crops affected, IPM approaches need to be based on the biology and ecology of the virus and vector and the crop production system. Here, we present the general measures that can be used in an IPM program for geminivirus diseases, specific case studies, and future challenges.

INTRODUCTION

Geminiviruses (family *Geminiviridae*) are a large group of plant viruses that possess small circular single-stranded (ss) DNA genomes that are encapsidated in twinned or geminate virions (194). These viruses cause economically important diseases of food, feed, and fiber crops worldwide (59, 84, 182). These diseases have resulted in substantial losses to agricultural production, especially in tropical and subtropical regions, and a heavy reliance on insecticides for management (i.e., the pesticide treadmill). Thus, effective management of these diseases can have substantial economic, environmental, and human health benefits.

Geminivirus Emergence, Evolution, and Properties

Modern day geminiviruses evolved from ancient prokaryotic ssDNA plasmids to infect and cause a wide diversity of diseases in many dicotyledonous plants (68, 90, 149). This involved the acquisition of additional genes necessary to infect plants and the interaction with phloem-feeding insects that would become vectors for plant-to-plant spread of geminiviruses (149). These insect vectors, especially the polyphagous supervector *Bemisia tabaci*, have disseminated geminiviruses extensively and introduced them to a diversity of potential host plants (56). Viral genetic mechanisms such as mutation, recombination, and pseudorecombination facilitated the emergence of new species (99, 106, 141, 149). In some crops, such as tomato and common bean, these events have occurred multiple times in different geographical regions, giving rise to multiple geminivirus strains and species that cause similar symptoms in the same host (local evolution) (57, 197). These mechanisms have led to an explosion of geminivirus species. There are currently 441 geminivirus species

recognized by the International Committee on Taxonomy of Viruses, making them the largest family of viruses as well as one of the most diverse (194).

The genome of geminiviruses is monopartite [a single genomic DNA of ~2,600–3,000 nucleotides (nt)] or bipartite [two ~2,600 nt DNA components (termed DNA-A and DNA-B) for a genome size of ~5,200 nt] (67, 149). The monopartite genomic DNA and each of the DNA components of the bipartite genome are individually encapsidated into the twinned quasi-icosahedral virions (194). All geminivirus genomes encode a replication-associated protein (Rep) and have a noncoding intergenic region that contains the common region of the bipartite genome, with the conserved nonanucleotide sequence TAATATTAC contained within a stem-loop structure that is part of the origin of replication (67). The Rep protein introduces a nick in the highly conserved nonanucleotide sequence to initiate rolling circle replication of the viral genome. Genome structure varies depending on the type of geminivirus and the genome encodes six to eight proteins with essential functions (194). The nature of the genomes and their gene functions have been addressed elsewhere (67, 144, 149).

Genera of Geminiviruses

Nine genera are currently recognized in the family *Geminiviridae* (1, 194). Genera are established based on the type of insect vector, host range, genome structure and organization, and phylogeny (149, 194). These genera are *Becurtovirus*, *Begomovirus*, *Capulavirus*, *Curtovirus*, *Eragrovirus*, *Grablovirus*, *Mastrevirus*, *Topocuvirus*, and *Turncurtovirus* (183, 184, 194). The genus *Begomovirus* has the greatest number of species (388), and members have monopartite or bipartite genomes (149, 194). Members of the other genera have monopartite genomes. In nature, geminiviruses are transmitted by various hemipterous insects. *B. tabaci*, a whitefly species complex, transmits members of the genus *Begomovirus*; various species of leafhoppers transmit members of the genera *Becurtovirus*, *Curtovirus*, *Mastrevirus*, and *Turncurtovirus*; treehoppers transmit the single members of the genera *Grablovirus* and *Topocuvirus*; and an aphid transmits one of the members of the genus *Capulavirus*.

Members of the genera *Begomovirus*, *Mastrevirus*, *Curtovirus*, and *Grablovirus* cause the majority of economically important diseases and are the focus of this review. The biological properties of members of these four key genera are further discussed in the following sections, with an emphasis on aspects that pertain to management.

Begomoviruses. These viruses have monopartite or bipartite genomes and show a phylogeographical distribution, with most bipartite begomoviruses occurring in the New World (NW) and most monopartite begomoviruses occurring in the Old World (OW) (30, 149). This reflects the ancient origin of these viruses, i.e., they were clearly present prior to continental drift (68, 149). Collectively, begomoviruses infect a wide range of dicotyledonous crops, noncultivated plants (e.g., weeds), and ornamental plants. However, individually, begomoviruses have relatively narrow host ranges. Plants affected include crops such as common bean, cotton, cucurbits, okra, papaya, peppers, and tomatoes; weeds such as *Sida* spp., *Macroptilium* spp., *Malva* spp., and *Jatropha* spp.; and ornamentals such as *Abutilon* spp. and *Lonicera* spp. (honeysuckle). Furthermore, local evolution has resulted in multiple begomovirus species that infect a given crop or weed species and cause similar symptoms; e.g., there are more than 90 species that infect tomato and more than 30 species that infect *Sida* spp. Thus, the precise identification of the begomovirus involved is important for the selection of management measures.

Whiteflies of the *B. tabaci* cryptic species complex (43) transmit begomoviruses. The mode of transmission is generally believed to be circulative (persistent) and nonpropagative (38). However, results of some studies have raised the question of whether all geminiviruses are truly nonpropagative in their vectors. This evidence mostly comes from studies of the invasive monopartite

begomovirus *Tomato yellow leaf curl virus* (TYLCV), in which limited viral replication and transovarial transmission in *B. tabaci* have been reported (38, 54, 60, 135, 188). However, to date, no other geminivirus has been shown to have such an intricate association with their insect vector. Furthermore, in terms of disease management, there are several lines of evidence that suggest replication and transovarial transmission of TYLCV may not be important in the epidemiology of tomato yellow leaf curl disease. For example, the success of host-free periods in managing TYLCV and other begomoviruses (i.e., the capacity to clean the virus from the whitefly vector following multiple generations in the absence of infected tomato plants) argues against extensive replication and transovarial transmission (152, 179). However, these properties may help explain why TYLCV has been spread worldwide (98). Clearly, this is an area that warrants further research.

The cryptic species of *B. tabaci* are morphologically indistinguishable but have different biological and molecular characteristics (43). Thus, biotypes A, B, and Q are now classified as NW, Middle East Asia Minor 1, and Mediterranean species, respectively (43). All three species transmit begomoviruses, with variable efficiencies, depending on the virus-host combination (54, 128). *B. tabaci* is highly polyphagous and populations can survive year-round in tropical and subtropical regions, which can lead to serious outbreaks of begomovirus diseases in overlapping crops. Indeed, this pest is considered a supervector because it is difficult to manage and is distributed worldwide (56).

Monopartite begomoviruses in the OW are associated with circular ssDNAs of ~1,400 nt, which are referred to as alphasatellites and betasatellites (27, 195). Alphasatellites share an origin of replication with members of the family *Nanoviridae* and encode a nanovirus-like Rep protein, which allows for independent replication. By contrast, betasatellites cannot self-replicate, but instead encode a major pathogenicity and symptom determinant, the βC1 protein, which is a suppressor of gene silencing. Betasatellites depend completely on their helper begomovirus for replication, encapsidation, and vector transmission (195). Recently, the families *Alphasatellitidae* and *Tolecusatellitidae* were established for these ssDNA satellites (1).

Mastreviruses. The genus *Mastrevirus* is the second largest in the family, with 34 species. These viruses have a monopartite genome of ~2,700 nt, and most occur in the OW. The majority of *Mastrevirus* species infect monocotyledonous plants of the family Poaceae, e.g., barley (*Hordeum vulgare*), maize (*Zea mays*), sugarcane (*Saccharum officinarum*), and wheat (*Triticum aestivum*). Several mastreviruses infect dicotyledonous plants, mainly those in the family Fabaceae. Mastreviruses are transmitted by leafhoppers (family Cicadellidae) in a circulative (persistent) and nonpropagative manner (69, 146). The transmission mechanism of the *Mastrevirus* type species *Maize streak virus* (MSV) by *Cicadulina mbila* has been most extensively studied (92, 161). Each *Mastrevirus* species is transmitted efficiently by a certain set of leafhopper species, often of the same genus; e.g., *Cicadulina* spp. for MSV and *Psammotettix* spp. for *Wheat dwarf virus* (WDV). In the case of the dicot-infecting viruses, transmission is by the common brown leafhopper *Orosius orientalis*. Because maize is a staple crop, often grown by subsistence farmers, disease management can be a challenge.

Curtoviruses. Members of the genus *Curtovirus* have a monopartite genome of ~2,900 nt and are associated with curly top disease (CTD) in a wide range of dicotyledonous crops and noncultivated plants (23, 37). CTD is characterized by stunting and curling and distortion of leaves (23). The classification of *Curtovirus* species was recently reevaluated and the number of species was reduced to three (183). *Beet curly top virus* (BCTV) is the type species and contains most of the previous strains and species associated with CTD. Thus, BCTV is the major species in terms of economic importance and knowledge of curtovirus biology (37). BCTV has a recombinant genome, with the complementary-sense genes of a begomovirus and virion-sense genes of a mastrevirus (37).

The beet leafhopper (BLH) (*Circulifer tenellus*) and other *Circulifer* spp. transmit BCTV (and presumably the other two *Curtovirus* species). BLHs acquire the phloem-limited BCTV during feeding in the phloem. The mode of BCTV transmission by the BLH is circulative (persistent) and nonpropagative (37). BCTV is a good example of the long-distance spread and subsequent establishment of a virus because of human activities. The virus likely originated in the OW and was introduced (along with the BLH) into the NW in the early 1900s in association with sugar beet (*Beta vulgaris*) propagative material (23, 37). BCTV and the BLH became well-established in the western United States, where most of the reported economic damage by CTD occurs. In this region, the BLH overwinters in the foothills and migrates down into agricultural valleys in the spring, where it transmits BCTV. In the fall, female BLHs migrate back to the foothills. The complexity of the BCTV-BLH interaction makes CTD a challenge to manage.

Grablovirus. The surreptitious identification of a circular ssDNA associated with red blotch symptoms in grapevines led to the identification of a geminivirus-like agent that causes these symptoms (9, 169). This virus was named *Grapevine red blotch virus* (GRBV), and it is considered a highly divergent type of geminivirus, mostly because of having (limited) sequence identity with known members of the family. GRBV has a monopartite genome of ~3,400 nt that possesses some features of geminiviruses, including a Rep protein and an origin of replication with the conserved nonanucleotide sequence within a stem-loop structure (169). However, it has not been clearly established that this relatively large genomic DNA is encapsidated into geminate virions. To accommodate this putative new type of geminivirus, the genus *Grablovirus* was established (184). For now, this genus contains GRBV as the only member, although a putative second species has been recently reported (138). GRBV has been reported only in North America; however, given the extensive (global) exchange of grapevine propagative material, it is expected that the virus will be detected in other grape-growing regions of the world (10). For the isolates characterized to date (~15), two groups or phylogenetic clades have been identified (169). GRBV appears to have a narrow host range, limited to cultivated and wild grapevines. Recent studies have established that GRBV is transmitted by the three-cornered alfalfa treehopper (*Spissistilus festinus*) (15). However, the role of this vector in the epidemiology of the disease remains to be determined. Management of GRBV presents unique challenges, as grapevine is a perennial host.

Long-Distance Spread of Geminiviruses

International trade of seed, seedlings, propagation materials, and agricultural commodities has opened new routes for agricultural products to move between countries and worldwide. This also has allowed for a dramatic increase in the long-distance spread of geminiviruses and their insect vectors (56; see section titled Begomoviruses of Pepper and Tomato). Additional factors that contribute to their spread and establishment include (*a*) relaxation of quarantine regulations, (*b*) increased intensification and diversification of agriculture together with changes in cropping practices, and (*c*) production of crops in new areas. Climate change is yet another factor contributing to the successful spread of geminiviruses into areas that were previously unfavorable for the virus or vector. The combination of all these circumstances is enhancing long-distance spread and establishment of geminiviruses on a global scale.

Geminivirus Diagnosis

Before developing a management program, it is necessary to confirm that a given disease is, in fact, caused by a geminivirus. Certain types of foliar symptoms, such as golden and yellow mosaic,

leaf curling and distortion, enations, and vein yellowing, swelling, and purpling, are associated with geminivirus diseases. In monocots infected with mastreviruses, streaks and striates are typical symptoms. The presence of potential vectors, such as leafhoppers and whiteflies, may also suggest a geminivirus etiology. However, these symptoms may be caused by other viruses, and it is often important to identify the specific virus involved. Thus, diagnosis based on symptoms alone is often not sufficient and diagnostic tests must be used to confirm geminivirus infections. For some geminiviruses, such as TYLCV, *African cassava mosaic virus* (ACMV), and BCTV, antibodies and serological tests, e.g., enzyme-linked immunosorbent assay, were developed, but these are not commonly used. Recombinant DNA technology allowed for the development of rapid and specific tests for detection and characterization of geminivirus DNA. Initially, dot- and squash-blot hybridization tests with cloned geminivirus DNAs as probes were used (58). However, it was the application of polymerase chain reaction (PCR) technology, often together with DNA sequencing, that revolutionized the detection and characterization of geminiviruses (148). PCR is currently the method of choice for geminivirus detection, as it is rapid, sensitive, and precise. Furthermore, it can be tailored for detection of a specific virus or all members of a genus (148). Sequencing of the PCR-amplified geminivirus DNA fragments allows for precise identification of the virus. Rolling circle amplification (RCA), which enriches for circular DNA molecules in a nonspecific manner, has proved to be a valuable tool for the detection and cloning of uncharacterized and novel geminiviruses (74). Indeed, this was the method that led to the identification of GRBV (169). When combined with restriction enzyme digestion, RCA can also be used to identify mixed infections of geminiviruses (62). More recently, high-throughput (or next-generation) sequencing has become popular, as it allows for detection of uncharacterized geminiviruses and mixed infections. Methods based upon isothermal amplification have also been developed for geminivirus detection, including loop-mediated isothermal amplification tests for *Squash leaf curl virus* (SLCuV) (91) and TYLCV (52) and recombinase polymerase amplification (111). Once the geminivirus involved in a disease is identified, it is then possible to determine the current knowledge of the biological properties of the virus and to select appropriate management strategies.

GENERAL MANAGEMENT MEASURES FOR GEMINIVIRUSES

Numerous factors affect the occurrence, incidence, and economic losses caused by geminivirus diseases. In the development of an integrated pest management (IPM) approach, many of these factors are considered in order to select the measures that are used to limit economic loss and epidemics. Here, general strategies for geminivirus management are described in terms of three phases of the growing season: before, during, and after.

Before the Growing Season

These are measures that can be taken prior to the establishment of a field or vineyard. Perhaps most important is the selection of the cultivar, particularly if resistance is available. If resistant cultivars are not available, then efforts need to be made to obtain virus- and vector-free planting materials, e.g., transplants and propagative material. Other measures include time of planting and field placement.

Cultivars with conventional resistance. Selection of the cultivar to be grown and the source of the seed or propagative materials are critical factors before the growing season. In terms of geminivirus management, efforts should be made to determine the availability of horticulturally desirable cultivars with conventional resistance to the geminivirus(es) affecting the crop plant being

planted. Here, this refers to cultivars generated with resistance genes introgressed via conventional breeding methods. Geminivirus resistance genes can be dominant, semidominant, or recessive, and sources of these genes are often wild species or landraces. However, it can be challenging to introgress resistance genes from these species or landraces into commercially acceptable cultivars. When commercially available, the planting of cultivars resistant to geminivirus or, in some cases, to the insect vector is strongly recommended as this can provide complete protection from geminivirus diseases. Examples of crop plants having effective geminivirus resistance include tomato (*Solanum lycopersicum*), cotton (*Gossypium hirsutum*), and cassava (*Manihot esculenta*).

Virus- and vector-free planting materials. When resistant cultivars are not available, the planting of virus-free transplants and propagation materials is very important to reduce primary inoculum and delay geminivirus disease development. In vegetable crops such as cucurbits, peppers (*Capsicum annuum*), and tomato, transplants should be produced in high-quality greenhouses or screenhouses instead of in seedbeds in open fields (**Figure 1*a***). At a minimum, when grown in areas where vectors are present, seedbeds in open fields should be protected with netting to prevent vectors from transmitting geminiviruses to transplants. In crops propagated from cuttings (e.g., cassava), virus-free mother plants selected based on a lack of symptoms and, ideally, molecular tests (e.g., PCR) should be used as the source of these cuttings. In the case of grapevines (*Vitis vinifera*), which are propagated from rooted or grafted cuttings, virus-free mother plants are often maintained by dedicated facilities that utilize modern technologies for virus testing (e.g., PCR, quantitative PCR, and next-generation sequencing) to provide clean material for increase and establishment of commercial vineyards.

Selection of planting dates and field locations. Having selected the cultivar and source of seed/propagative materials, the next step in the process is to select when and where fields or vineyards will be established. When possible, fields of annual crops should be established when the population of the insect vector and sources of inoculum are at the lowest possible levels. This delays infection and provides protection to young plants, which are typically highly susceptible and, when infected, sustain the greatest yield losses. This is best accomplished following host-free periods or, at least, in areas distant from established fields with infected plants (one of the most important sources of inoculum). Later plantings should also be located upwind of earlier plantings. It is also recommended that barrier crops, such as maize, be planted between fields established at different times to reduce vector movement between plantings.

Protected culture. Protected culture allows for effective management of geminivirus diseases and insect vectors through exclusion and enhanced efficiency of other measures. In the most extreme cases, the crop is protected during the entire growing season, e.g., vegetable crops grown in greenhouses and screenhouses (**Figure 1*b***). In these structures, it is important to have a double-door airlock system that prevents insect vectors from entering. The use of UV-absorbing components in plastic or net covers increases exclusion of insect vectors by disrupting vector orientation and landing (13). Protected culture has greatly reduced losses due to geminivirus (begomovirus) diseases in high-value vegetable crops in southern Spain, the Baja Peninsula of Mexico, and Guatemala. In cases of vegetable crops that are grown in open fields, protection for the entire growing season is not feasible. Here, row covers (e.g., Agribon or Agril) can be used to protect young plants for ~30–45 days after transplanting (DAT) (**Figure 1*c***). Because the greatest yield losses due to geminivirus disease occur when plants are infected at a young age, the use of row covers protects young plants from early infection. Furthermore, when combined with other measures following

removal of the row covers, economically acceptable yields can be achieved in areas with high disease pressure.

Enhanced plant and soil health. Growing healthy plants, with adequate nutrition and water, is important not only for obtaining high yields but also for enhancing the capacity of virus-infected plants to produce a crop (tolerance). This is especially the case when plants are infected at later

(Caption appears on following page)

Figure 1 (*Figure appears on preceding page*)

Examples of integrated pest management (IPM) measures for geminivirus diseases. (*a*) Production of virus- and vector-free tomato transplants in a greenhouse. (*b*) Protected culture in screenhouses allows for effective management of geminivirus diseases of vegetable crops (e.g., cucurbits, peppers, and tomatoes), especially when combined with other measures (e.g., roguing infected plants and vector management with insecticides or biological control). (*c*) Row covers provide a barrier that protects young plants of susceptible crops from infection by insect-transmitted geminiviruses (mostly whitefly-transmitted begomoviruses). (*d*) Reflective mulching disrupts landing of insect vectors on vegetables. (*e*) Yellow sticky cards can be used to monitor insect vector populations (e.g., leafhoppers and whiteflies). (*f*) Insecticides are applied to manage insect vector populations, ideally only after populations reach established thresholds. (*g*) Roguing of geminivirus-infected plants early in the growing season reduces inoculum sources and slows the spread of disease. (*h*) Implementation of a mandatory three-month whitefly host-free period in the Dominican Republic has reduced vector populations and delays the appearance of *Tomato yellow leaf curl virus* in the following tomato crop.

stages of growth. Plant health can be enhanced in various ways, and some of these contribute to a reduction in geminivirus disease incidence. Mulch is a soil surface cover that helps conserve soil moisture, improves soil fertility and health, and reduces the number of weeds. There are several types of mulch. Synthetic mulch, typically some type of plastic, is more expensive and is commonly used in intensive agricultural systems (**Figure 1*d***). Organic mulch includes a range of materials, e.g., leaves, bark chips, field hay, straw, and living plants and is less expensive and more common in subsistence agriculture. Reflective plastic mulches can prevent insect vectors from recognizing crop plants via disorientation and reduced landing rates mediated by reflected UV and visible light (97). The most appropriate type of mulch depends on the economic value of the crop, the size of the field, and the type of farming involved.

During the Growing Season

These are the measures that can be used for management of geminivirus diseases after the crop has been established. The most commonly used measure is vector management with insecticides. This measure is most effective and safe when used as a component of an IPM program that includes monitoring vector populations and using thresholds to trigger insecticide applications. Furthermore, insecticides should be used in combination with the other measures that can reduce geminivirus disease development during the growing season.

Vector management with insecticides. The use of insecticides to reduce vector populations is the most commonly used method for management of geminivirus diseases. It is common for seeds or transplants in greenhouses or seedbeds to be treated with systemic insecticides such as neonicotinoids (e.g., acetamiprid, dinotefuran, imidacloprid, and thiamethoxam) or the more recently available cyazypyr to manage vector populations, especially whiteflies. Yellow sticky cards can be used to monitor vector populations (e.g., adult leafhoppers and whiteflies) in protected culture and in open fields (**Figure 1*e***). This information is used to make decisions about when to apply insecticides (**Figure 1*f***). Whitefly populations can also be estimated from the abaxial side of leaves using the leaf-turn method (59). Leafhoppers and treehoppers can be monitored with yellow sticky cards or collected with sweep nets. In IPM, insecticides should be applied only when vector populations reach a threshold, which must be determined for each region (**Figure 1*f***). The threshold values and type of insecticide required depend on the crop, the disease pressure in the area, and other factors. Options include continued use of systemic insecticides such as the neonicotinoids and cyazypyr] and application of contact insecticides (e.g., bifenthrin, fenpropathrin, and lambda-cyhalothrin) or insect growth regulators (e.g., buprofezin, pyriproxyfen, and spiromesifen). An environmentally friendly alternative is the use of botanical insecticides, such as neem oil. These materials are applied to foliage or roots, e.g., via drip irrigation. Furthermore, to most

effectively reduce the spread of geminivirus diseases, insecticides must act rapidly, otherwise the virus is transmitted before the insect vector is killed. Indeed, most insecticides require hours to act, whereas virus transmission may require 5–15 minutes. Thus, for most effective management, an insecticide must act quickly and be applied when vector populations are relatively low.

Another problem is that insect vector populations with resistance to various insecticides have emerged (126). This has been largely studied and documented for the supervector *B. tabaci* (70, 71). Repeated application of the same active ingredient has led to the selection of individuals with resistance to many of the most frequently used insecticides (71). Populations with insecticide resistance tend to be selected in regions where the intensive use of insecticides is practiced. These resistant populations may displace indigenous or other established invasive populations. A good example of this is the insecticide-resistant Mediterranean species (Q biotype) in China (56). Therefore, it is important to rotate insecticides with different modes of action and to apply only the recommended number of applications during a growing season.

Roguing. Roguing is the removal of virus-infected plants during the growing season (**Figure 1*g***). This practice reduces sources of inoculum that contribute to the secondary spread of the virus in protected culture and open fields. It is especially effective when implemented at the beginning of the season (up to 30–45 DAT) and in protected culture and open fields with relatively low disease incidences. To avoid releasing viruliferous insect vectors, rogued plants should be immediately placed in plastic bags and disposed of. Regular inspections (weekly or biweekly) and roguing of plants with symptoms are recommended for protected culture and open fields.

Biological control. Biological control can be used to manage insect vectors and geminivirus diseases, especially for *B. tabaci* and begomovirus diseases in protected culture. Here, released biocontrol agents are contained and can achieve high rates of parasitism and predation. Three agents have been effectively used for biocontrol of *B. tabaci*: predators, parasitoids, and fungi. These agents may be efficiently used in an IPM program, e.g., in the reduction of whitefly populations and the incidence of TYLCV in protected cultures in Spain (165).

After the Growing Season

During the period following harvest and before the planting of the next crop, multiple measures should be used to reduce viral inoculum sources in a given geographical region. The harvested crop plants should be promptly removed and destroyed or deep plowed, as they represent an important inoculum source. In open fields, this often involves plowing, whereas in protected culture, plants are removed and destroyed. Host-free periods can be effective in reducing viral inoculum sources and insect vector populations, but they can be a challenge to implement and enforce. Thus, this time period is critical for reducing viral inoculum sources for the next crop.

Sanitation. Sanitation consists of all measures aimed at eliminating or reducing geminivirus inoculum sources in and around fields. After harvest, it is important to remove and destroy crop plants, as these can serve as sources of inoculum. This is relatively easy with annual crops such as cucurbits, peppers, and tomatoes, whereas it can be more difficult for other crops, such as cassava and grapevines. Additionally, sanitation efforts should include weed management in and around fields, particularly if a weed host of a crop-infecting virus is known. Volunteer crop plants also should be eliminated. Thus, the time period between harvest and the next growing season is critical for the implementation of sanitation measures.

Host-free period. Host-free periods provide a break in the continuous cropping of annual crops in a defined geographical region. For some begomovirus diseases, host-free periods of two to three months allow multiple generations of the whitefly vector and a cleansing of the geminiviruses, most of which are nonpropagated and not transovarially transmitted. This can result in the elimination or reduction of virus inoculum and, in some cases, vector populations. Ideally, the host-free period provides a window of time (e.g., 4–8 weeks) during which newly established crops are not subjected to high levels of virus pressure. Host-free periods can be very effective for management of some begomovirus diseases because (*a*) the host range of these viruses is often narrow, (*b*) the crop plant is the most important inoculum source, and (*c*) most begomoviruses are not transovarially transmitted. In temperate regions, the winter season provides a natural host-free period. However, in tropical and subtropical regions, where crops can be grown continuously, host-free periods should be implemented on a regional basis. Host-free periods can be voluntarily established by growers or legally enforced (**Figure 1*b***). The properties of a particular host-free period, e.g., time of year, length, crop(s) involved, and geographical region, depend on the crop, cropping system, and host-virus-vector interactions.

Promising New Technologies

Numerous transgenic strategies have been evaluated for generating geminivirus-resistant crops. Some of these have been very effective, particularly those utilizing RNA silencing (interference). However, for various reasons, geminivirus-resistant transgenic plants have not reached the level of commercialization. Transgenic beans with resistance to *Bean golden mosaic virus* (BGMV) have been approved for commercial production, but, to date, this has not happened. A major issue influencing commercialization is public acceptance of genetically modified organisms (GMOs), which continue to be viewed as controversial.

Transgenic plants. In some cases, transgenic plants engineered to trigger transcriptional gene silencing upon infection with geminiviruses have shown high levels of resistance. In this strategy, a sequence with part of the viral or insect vector genome is transformed into the plant to induce a double-stranded RNA structure. This induces the RNA silencing response of the plant to specifically degrade or methylate the genome of the target geminivirus/insect vector. Although geminiviruses are ssDNA viruses, this approach has been effective in producing highly resistant transgenic plants through methylation of promoter and other sequences, resulting in reduced viral replication (63). For example, the transgenic common bean (*Phaseolus vulgaris*) cultivar Embrapa 5.1 was developed to use RNA silencing via the expression of an intron-containing hairpin RNA corresponding to a portion of the *Rep* (AC1) gene of BGMV (26). Field experiments under natural infection have shown high levels of resistance to BGMV in this cultivar (14). However, major challenges remain for the commercialization of geminivirus-resistant transgenic plants, including standardization of this strategy and the complex regulatory structure involved in implementing and managing these new technologies (175, 176).

CRISPR-Cas. The clustered regularly interspaced short palindromic repeat (CRISPR)–CRISPR-associated protein (Cas) system is an adaptive immune system used by bacteria and archaea against viruses and mobile genetic elements, and it has been adapted for genome editing in eukaryotes (154). Engineered resistance against geminiviruses can be achieved by (*a*) editing the viral genome, (*b*) directing a catalysis-deficient Cas9–single-guide RNA (sgRNA) to interfere with binding of viral and/or host factors required for replication, and (*c*) editing genes encoding host factors required for the virus life cycle. CRISPR-Cas9 has been used to edit the genome of

Bean yellow dwarf virus, BCTV, TYLCV, *Cotton leaf curl Kokhran virus* (CLCuKoV), and *Merremia mosaic virus* (7, 8, 17, 76). In these studies, sgRNAs were designed to target coding and noncoding regions of the viral genome; however, these approaches involved GMO strategies, making them subject to the same regulations as transgenic plants. As expected, some sgRNAs were more efficient than others (17). To date, there are no published examples of a catalysis-deficient Cas9 or an edited host factor that interferes with geminivirus replication/transcription and confers geminivirus resistance.

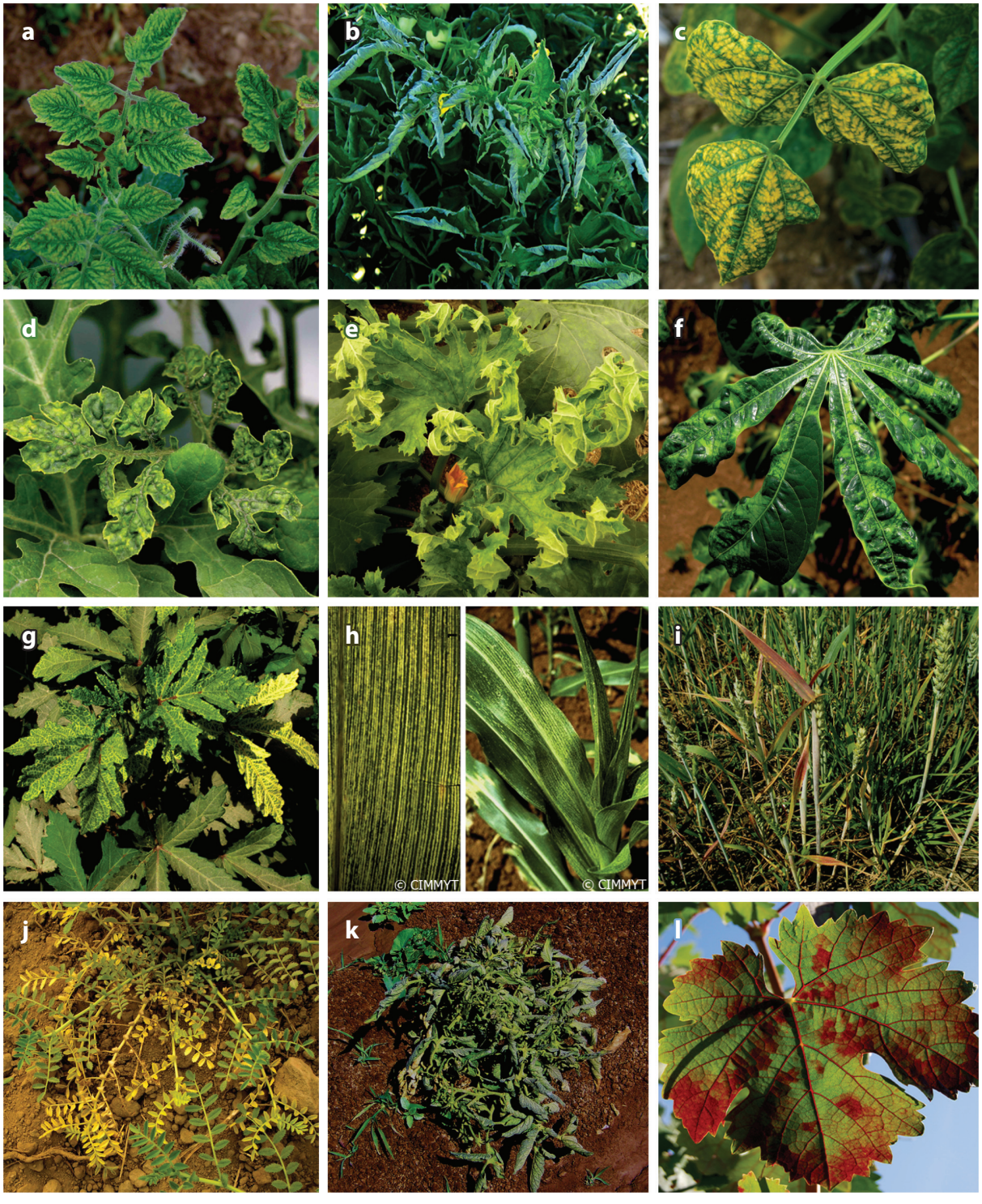
CASE STUDIES: BEGOMOVIRUS

Begomoviruses of Pepper and Tomato

Pepper-infecting begomoviruses occur in India, Indonesia, Africa, Mexico, Central America, and the southern United States. Begomoviruses that infect tomato are more widely distributed and seriously impact production in tropical and subtropical regions worldwide. Monopartite begomoviruses infect peppers in Asia (e.g., *Chili leaf curl virus* and *Pepper leaf curl Bangladesh virus*) and Africa (e.g., *Pepper yellow vein Mali virus*), whereas the bipartite *Pepper yellow leaf curl Indonesia virus* occurs in Indonesia (84, 103, 151, 163, 173, 177, 197). In the southwestern United States, Mexico, and Central America, the bipartite begomoviruses *Pepper huasteco yellow vein virus* and *Pepper golden mosaic virus* cause economically important diseases of pepper (19, 178). The importance and diversity of begomoviruses infecting *Capsicum* species have increased over the past 5–10 years (84). On the Indian subcontinent in particular, leaf curl disease caused by a complex of begomoviruses and betasatellites is considered the major constraint on pepper production (186).

Tomatoes are infected by more begomovirus species (~90) than any other plant species. This can be attributed to the worldwide cultivation of this vegetable crop, the innate susceptibility to geminivirus infection and local evolution mediated by the supervector *B. tabaci* (56). Furthermore, the worldwide dissemination of the invasive monopartite begomovirus TYLCV (**Figure 2*a***) has resulted in economic losses in regions of the NW and OW. Originally described from the Middle East around 1940, TYLCV was introduced into the NW in the early 1990s, and it has now been reported in the southern United States (e.g., states of Arizona, California, Florida, and Texas), northern Mexico, Central America, the Caribbean Basin, and Venezuela (40, 45, 98, 152). It has also spread into China, Japan, and the Korean Peninsula, where it causes economic losses in open fields and protected culture (84). The spread of TYLCV has been associated with the global dissemination of the supervector *B. tabaci* (biotype B, or the Middle East Asia Minor 1 species) (56, 98) (**Figure 3*a*,*b***). However, this also may be a consequence of the more intricate association of TYLCV with *B. tabaci* and the possible transmission in tomato seed (54, 86).

There are many other economically important monopartite and bipartite tomato-infecting begomoviruses (56, 84, 103). In Asia, new begomoviruses are emerging and some previously known viruses are spreading and displacing indigenous species (84). *Tomato leaf curl New Delhi virus* is spreading and displacing less-aggressive species in India, and it has been recently reported from North Africa and Southern Europe (124). In China, multiple monopartite tomato-infecting begomoviruses have been described, including the indigenous *Tomato yellow leaf curl China virus* and *Tomato leaf curl China virus*, which require *Tomato yellow leaf curl betasatellite* and *Tomato leaf curl China betasatellite*, respectively, to induce typical symptoms (84, 192). *Tobacco curly shoot virus* and *Tobacco leaf curl Yunnan virus* are monopartite begomoviruses that cause severe symptoms in tomato without the need for a betasatellite (105). Many of these tomato-infecting begomoviruses from China have recombinant genomes (191), revealing the importance of this genetic mechanism in the emergence of new strains and species. The invasive TYLCV has now displaced the indigenous *Tomato leaf curl China virus* in many areas (56, 84).



(*Caption appears on following page*)

Figure 2 (*Figure appears on preceding page*)

Symptoms of geminivirus infection. (*a*) *Tomato yellow leaf curl virus* (tomato). (*b*) *Tomato mottle leaf curl virus* (tomato). (*c*) *Bean golden mosaic virus* (common bean). Photograph by Roberto Ramos-Sobrinho. (*d*) *Cucurbit leaf crumple virus* (watermelon). (*e*) *Tomato leaf curl New Delhi virus* (zucchini). (*f*) Mixed infection of *African cassava mosaic virus* and *East African cassava mosaic virus* (cassava). (*g*) *Bhendi yellow vein mosaic virus* and *Bhendi yellow vein betasatellite* (okra). (*h*) *Maize streak virus* (maize). Photographs by the New South Wales Department of Primary Industries courtesy of CIMMYT. (*i*) *Wheat dwarf virus* (wheat). (*j*) *Chickpea chlorotic dwarf virus* (chickpea). (*k*) *Beet curly top virus* (tomato). (*l*) *Grapevine red blotch virus* (grapevine).

In the NW, the bipartite begomovirus *Tomato severe rugose virus* is important in central Brazil, whereas the NW monopartite begomovirus *Tomato mottle leaf curl virus* (**Figure 2*b***) is prevalent in northern production areas (56, 75, 113). In Central America, mixed infections of begomoviruses are common, e.g., *Tomato mosaic Havana virus* and *Tomato severe leaf curl virus* in Guatemala, and *Tomato yellow mottle virus*, *Tomato leaf curl Sinaloa virus*, and TYLCV in Costa Rica (19). Finally, since 2010, NW monopartite begomoviruses continue to be discovered infecting tomatoes in Brazil, Ecuador, and Peru (56, 112, 118). Together, these events have added new complexity to the geographic distribution and genetic diversity of tomato-infecting begomoviruses worldwide and have led to a blurring of the distinction between NW (bipartite) and OW (monopartite) begomoviruses.

Symptoms and epidemiology. Begomovirus symptoms in pepper range from symptomless to different degrees of stunting and curling, distortion, mosaic, mottling, and vein yellowing of leaves. In some cases, infected pepper plants show premature leaf or flower drop. In tomato, many begomoviruses induce similar symptoms, such as stunting and distorted and upright growth of plants; leaves with chlorosis; upward or downward curling mosaic, mottling, and purple veins; flower abortion; and small and unmarketable fruits (**Figure 2*a*,*b***). Mixed infections of begomoviruses (and other viruses) are common and can challenge diagnosis. In addition, synergistic interactions can lead to increased symptom severity (112, 120). In most cases, diagnosis of the causal virus(es) based on symptoms alone is difficult and molecular tests are required.

Pepper and tomato begomoviruses have narrow host ranges, mostly infecting crops and noncultivated plants in the family Solanaceae. A few tomato-infecting begomoviruses also infect plants of other families, e.g., Fabaceae [common bean and soybean (*Glycine max*)] and Cucurbitaceae (78, 113). Thus, overlapping crops of susceptible peppers and tomatoes should be avoided because infected plants in established fields are important inoculum sources for newly planted fields. Solanaceous weeds and volunteers can also serve as inoculum sources.

It should be noted that a recent report has presented evidence that TYLCV is seed-transmitted in tomato (86). In this study, an intricate association of the virus with seed tissues was demonstrated by PCR tests, but it was not established that the seed-associated virus induces tomato yellow leaf curl disease in plants established from contaminated seed. It is possible that there is a longer latent period in plants infected from seedborne inoculum, but this remains to be demonstrated. Furthermore, if TYLCV is seed-transmitted to any great extent, there should be more instances of infection of transplants grown in protected culture or in fields in regions where the virus does not occur, especially for hybrid seed produced in areas where the virus occurs (e.g., China and Thailand). However, seed transmission could have helped in the worldwide spread of TYLCV (98). To date, seed transmission of geminiviruses does not appear to be widespread in the family or epidemiologically important. Thus, management measures (e.g., seed testing) are not warranted at the present time. However, this is clearly a phenomenon that needs to be investigated further.

Management strategies. Effective management of begomovirus diseases of pepper and tomato can be achieved with protected culture (e.g., greenhouses and screenhouses), whereas this is

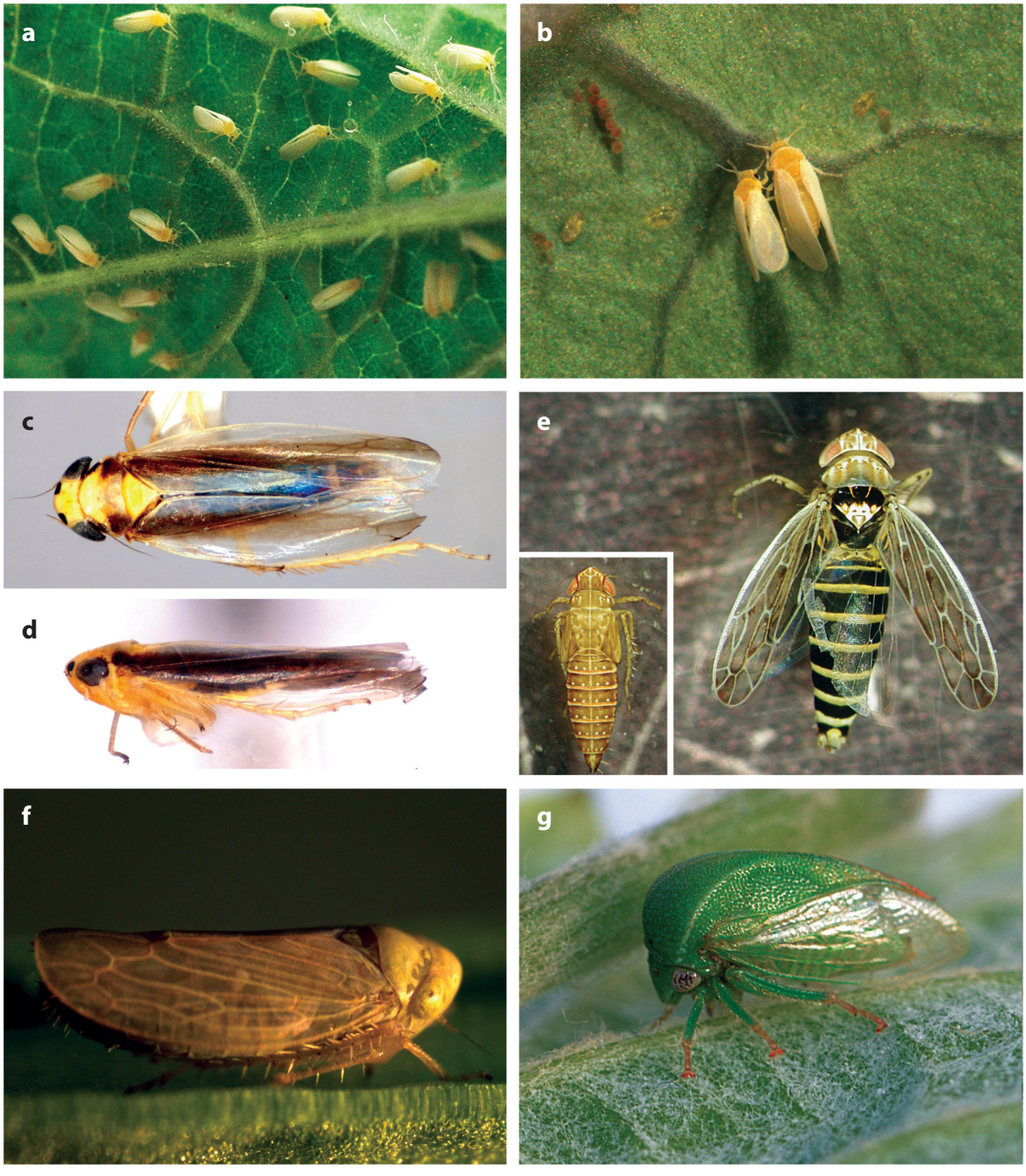


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Figure 3 (*Figure appears on preceding page*)

Insect vectors of geminiviruses. (*a*,*b*) Adult whiteflies (*Bemisia tabaci*), the vector of begomoviruses. Photograph by Rafael Fernandez-Muñoz and Alvin M. Simmons. (*c*,*d*) Adult maize leafhopper (*Cicadulina mbila*), vector of *Maize streak virus.* Photograph by the New South Wales Department of Primary Industries. (*e*) Adult and nymph (inset) of the leafhopper *Psammotettix alienus*, vector of *Wheat dwarf virus*. Photograph by Louis Vimarlund. (*f*) Adult beet leafhopper (*Circulifer tenellus*), vector of *Beet curly top virus*. (*g*) Adult three-cornered alfalfa treehopper (*Spissistilus festinus*), a vector of *Grapevine red blotch virus*. Photograph by Kathy Keatley Garvey.

considerably more difficult in open fields. This is especially true in less-developed countries in tropical and subtropical regions, where overlapping crops are common and access to resistant cultivars is limited.

Before the growing season. For pepper, resistant cultivars are not commercially available; however, pepper germplasm with promising levels of resistance to NW begomoviruses has been identified and is being used in breeding programs (53). In contrast, there are many tomato cultivars with begomovirus resistance and most of these have been generated by introgressing genes from wild tomato species (e.g., the *Ty-1* to *Ty-6* genes) with conventional breeding methods (34, 59, 84, 96, 97). Some of these cultivars have high levels of resistance to begomoviruses, including TYLCV and *Tomato severe rugose virus* and *Tomato mottle leaf curl virus* from Brazil (59). However, these genes do not provide immunity or resistance to all begomoviruses (especially bipartite species). Thus, it is important to know the specific begomovirus(es) infecting tomato in a region before selecting a resistant cultivar.

In addition to planting resistant cultivars (tomato), other IPM measures that can be used before the growing season include the selection of planting dates to avoid hot and dry periods that favor high whitefly populations. It is also important to establish fields with virus- and whitefly-free transplants. In locations where virus pressure is constantly high, protected culture or row covers may be required for all or part of the growing season. Finally, it is recommended that systemic insecticides (e.g., neonicotinoids or cyazypyr) be applied before transplanting into protected culture or open fields.

During the growing season. In protected culture and open fields, it is critical to rogue diseased plants early in the season (e.g., up to 30–45 DAT), as this eliminates an important source of inoculum. Row covers and reflective mulch can protect plants in open fields from the whitefly vector and delay begomovirus infection, although additional efforts may be needed after the row covers are removed (59). Whitefly populations should be monitored weekly or biweekly and insecticide applied only when established thresholds have been attained. Properly timed insecticide applications can delay begomovirus infection and minimize the number of applications. In general, management solely based on the application of insecticides is ineffective and can result in the emergence of insecticide-resistant whitefly populations (71). Spraying should include cultivated fields and, possibly, surrounding noncultivated areas (24, 114). Keeping fields and surrounding areas free of weeds and volunteer crop plants is also highly recommended.

After the growing season. Avoidance of intensive cropping systems involving the continuous presence of susceptible crops with begomovirus disease is very important because infected crop plants are one of the most important sources of virus inoculum and are also often propagative hosts for viruliferous whiteflies. Destruction of old crop plants must be done promptly after harvest. Host-free periods can delay and reduce begomovirus disease development in the next tomato crop. Host-free periods have been successfully used to manage begomovirus diseases of tomato in Israel, the Dominican Republic, Mali, and Brazil (75, 140, 152, 179). To manage TYLCV in the

Dominican Republic, the Minister of Agriculture established a three-month whitefly host-free period (59, 152) (**Figure 1*b***).

Begomoviruses of Common Bean

Begomovirus-like symptoms of legumes have been known since the 1960s (41, 125, 182). The first report of a whitefly-transmitted begomovirus-like agent infecting common bean in the Americas was for BGMV in Brazil (41) (**Figure 2*c***). Epidemics of bean golden mosaic disease were reported from Brazil, Central America and the Caribbean Basin (75). DNA sequencing subsequently revealed that isolates from South America (Argentina and Brazil) and Central America and the Caribbean Basin represent distinct begomovirus species, BGMV and *Bean golden yellow mosaic virus* (BGYMV), respectively (57, 75).

BGMV became (and remains) the most economically important bean-infecting virus in Brazil, Argentina, and Paraguay, whereas BGYMV causes golden mosaic disease in Mexico, Central America, the Caribbean Basin, and the southeastern United States (57, 58, 75, 147). BGMV was the only begomovirus infecting common bean in Brazil for almost 50 years until *Macroptilium yellow spot virus* was described in 2012 in northern Brazil (46, 106). Topographical barriers, coupled with the absence of seed transmission and regional preferences for different seed types, are likely responsible for the geographical separation of BGMV and BGYMV (46, 57). Bean-infecting begomoviruses of lesser economic importance in common bean include *Bean dwarf mosaic virus* in Argentina and Colombia, *Bean calico mosaic virus* in Mexico, *Bean chlorosis virus* in Venezuela, and *Cucurbit leaf crumple virus* (CuLCrV) in the United States (2, 32, 47, 123, 158).

In Asia, the major begomoviruses of summer legumes include the bipartite *Mungbean yellow mosaic virus* and *Mungbean yellow mosaic India virus*. Little is known about begomoviruses in legume crops across Africa. Three viruses, *Cowpea golden mosaic virus*, *Soybean chlorotic blotch virus*, and *Soybean mild mottle virus*, have been identified infecting legumes in Nigeria (4). In Spain, TYLCV has been associated with severe disease symptoms and economic losses in production of large-seeded (Andean gene pool) common bean cultivars (127).

Symptoms and epidemiology. BGMV and BGYMV cause similar symptoms in most common bean cultivars. Symptoms include varying degrees of yellow and golden mosaic of leaves, flower abortion, and reduced number and size of pods and seeds (**Figure 2*c***). Symptoms induced by other bean-infecting begomoviruses are less severe or limited to certain large-seeded cultivars of the Andean gene pool of common bean. For example, *Macroptilium yellow spot virus* causes yellow spotting, crumpling, and distortion of leaves, which are less severe than symptoms induced by BGMV (75). *Bean dwarf mosaic virus* induces severe stunting and curling, epinasty, and mosaic of leaves in susceptible large-seeded Andean cultivars but does not cause symptoms in small-seeded cultivars of the Middle American gene pool (158). The host range of bean-infecting begomoviruses is relatively narrow and mostly limited to common bean and other legumes. Because of the similar symptoms induced by these viruses in common bean, molecular tests are needed to confirm the specific virus(es) involved.

Management strategies. There are sources of resistance to bean-infecting begomoviruses, and these are mostly in small-seeded genotypes of the Middle American gene pool. This resistance has been introgressed into commercial cultivars, which have shown good levels of resistance in the field. However, other measures may be needed to manage BGMV/BGYMV, including planting when whitefly populations are low, managing the whitefly vector with insecticides and host-free periods.

Before the growing season. Common bean cultivars with varying degrees of resistance to BGMV and BGYMV have been bred, including those with the recessive *bgm-1* gene. In Brazil, resistant cultivars have moderate resistance and are not widely available (25). Selection of the planting date to avoid peak whitefly populations and high rates of early BGMV infection can be very important. The best planting dates will vary from region to region and will depend on various agricultural and environmental factors. In Brazil, planting common bean crops near established fields of preferred whitefly hosts, such as soybeans, should be avoided.

During and after the growing season. Despite increasing concerns about environmental contamination and selection of insecticide-resistant whitefly populations (35, 59, 70, 71), neonicotinoids and other insecticides are widely used to manage whitefly populations in an attempt to slow the spread of BGMV and BGYMV. In Brazil, center pivot irrigation (common in winter crops) helps keep whitefly populations low (107). Following harvest, the implementation of regional bean-free periods can be an effective measure that takes advantage of the narrow host range of BGMV and BGYMV. For example, implementation of a two-to-three-month bean-free period has helped manage BGYMV in the Dominican Republic, whereas a one-month bean-free period has reduced disease incidences in fields in three states of Brazil (75). In general, bean plants should be removed and destroyed following the harvest, and fields should be kept free of weeds and volunteers prior to the next crop.

Begomoviruses of Cucurbits

Most begomoviruses that infect cucurbits have a bipartite genome. These viruses can cause severe disease symptoms in a range of cucurbits and cause economic losses in the OW and NW. The first cucurbit-infecting begomovirus reported in the OW was *Watermelon chlorotic stunt virus*, which was described in Yemen in 1986 and subsequently in Sudan, Iran, Oman, and other countries in the Middle East (18, 85). More recently, a cucurbit-infecting strain of *Tomato leaf curl New Delhi virus* was introduced to Iran, Spain, and other countries in the western Mediterranean Basin, where it has caused severe disease symptoms in zucchini (*Cucurbita pepo*) (50, 78) (**Figure 2*e***).

The first cucurbit-infecting begomovirus reported in the NW was SLCuV in the southwestern United States in the early 1980s. Subsequently, SLCuV was reported infecting cucurbits (especially squash) in southern Arizona, southern California, and northern Mexico. The recombinant CuLCrV was first reported in the Imperial Valley of California in 1998 and may have evolved from an SLCuV-like virus (31, 48, 61, 64) (**Figure 2*d***). CuLCrV was identified in Florida in 2006, where it has caused economic losses in cucurbit and green bean crops (2). In the early 2000s, SLCuV was introduced into the Mediterranean Basin (95). A synergistic interaction between *Watermelon chlorotic stunt virus* and SLCuV was reported (170).

Symptoms and epidemiology. Symptoms induced by begomoviruses in cucurbits are similar and include stunting and distorted growth (often severe); leaves with crumpling, upward and downward curling, enations, light green to yellow mosaic or mottle, and vein distortion and swelling; and bumpy, deformed, and discolored fruit (**Figure 2*d,e***). Symptoms vary depending on the susceptibility of the species and the age at which plants are infected. In general, pumpkin (*C. pepo* and *Cucurbita maxima*) and squash (*C. pepo*) are more susceptible than are watermelon (*Citrullus lanatus*) and melon (*Cucumis melo*). Because of the similar symptoms induced by these viruses and the different symptoms induced by individual cucurbit-infecting begomoviruses in different cucurbit species, molecular tests are needed to precisely identify the virus(es) involved.

Most cucurbit-infecting begomoviruses have narrow host ranges and predominantly infect members of the family Cucurbitaceae, including cultivated and weed species. *Tomato leaf curl*

New Delhi virus has a wider host range, which includes cucurbits and solanaceous species (50) (**Figure 2*e***). Cucurbit bridge crops, weeds, and volunteers can serve as inoculum sources for newly planted cucurbit crops.

Management strategies. There are currently no commercially available cucurbit cultivars with resistance to begomoviruses. As mentioned, cucurbit species vary in susceptibility to these viruses, and some undergo recovery from disease symptoms (63). Therefore, selection of the type of cucurbit and cultivar to be planted in different regions will depend on the prevalent begomovirus(es) and other viruses. Regardless, in areas with high begomovirus disease pressure, other measures will need to be taken, such as protected culture, row covers, whitefly vector management, avoidance of overlapping crops, and effective sanitation.

Before the growing season. Begomovirus-resistant cucurbit cultivars are not commercially available. There are reports of resistant germplasm, and the development of agroinoculation systems for many cucurbit-infecting begomoviruses should facilitate breeding of resistant cultivars (18, 64). Some commercial cultivars of melon, watermelon, and cucumber (*Cucumis sativus*) possess an innate resistance to SLCuV and CuLCrV. With CuLCrV, this resistance was associated with the recovery phenotype, which is an antiviral defense mechanism mediated by gene silencing that results in methylation of the viral genome (63).

Management of cucurbit begomoviruses prior the growing season also involves the selection of planting dates and appropriate field locations. New fields should not be established near or downwind of existing fields with whiteflies and begomovirus-infected cucurbit plants. When possible, planting should occur during periods of low whitefly pressure. Cucurbit weeds in and around fields should be eliminated before the growing season. There is no evidence that cucurbit-infecting begomoviruses are seed-transmitted. However, it is important to establish fields with virus- and whitefly-free transplants, preferably those that are locally produced.

During and after the growing season. Once cucurbit fields are established, whitefly management is a major challenge, as cucurbits are a preferred host. Ideally, this is done using an IPM approach based on monitoring of whitefly populations rather than on regular calendar-based sprays. Yellow sticky cards can be used to monitor whiteflies to determine whether populations warrant the application of an insecticide. Insecticides with different modes of action should be rotated. In areas of high virus and whitefly pressure, cucurbits can be grown in protected culture, or row covers and reflective mulch used to protect young plants (~30–45 DAT). Fields should be frequently inspected (e.g., every 3–7 days), and symptomatic plants rogued and destroyed, especially early in the growing season (e.g., up to 30–45 DAT) or following removal of row covers. After harvest, cucurbit plants should be promptly destroyed via removal or deep plowing. Potential reservoir hosts (e.g., weeds, old cucurbit plants left in harvested fields, and volunteers) should be eliminated and fields kept clean until the next planting. A regional cucurbit-free period of two to three months can be considered.

Begomoviruses of Cassava

Cassava mosaic disease (CMD) only occurs in the OW and is a major constraint on cassava production in Africa. Multiple cassava mosaic begomovirus (CMB) species cause this disease. Approximately half of all cassava plants on the continent are infected, and annual losses have been estimated at more than US$1 billion (100, 102). CMD also occurs in Sri Lanka and southern India, and an outbreak was reported in Cambodia in 2016 (5, 156, 187). This has recently become a regional problem, as more extensive spread has been reported from six provinces of Cambodia as well as new outbreaks in two provinces of neighboring Vietnam (149a).

Symptoms and epidemiology. Symptoms of CMBs include stunting and distorted growth, leaves with striking green to yellow mosaic, and tubers that are reduced in size and number (**Figure 2*f***). Currently, the International Committee on Taxonomy of Viruses recognizes ten CMB species, all of which are bipartite (83, 102, 196). The most widely occurring species are ACMV (throughout Africa), *East African cassava mosaic virus* (EACMV) (East Africa), and *East African cassava mosaic Cameroon virus* (Central and West Africa). The recombinant Uganda strain of *East African cassava mosaic virus* (EACMV-UG) caused a severe epidemic in East and Central Africa beginning in the late 1980s (196).

During the expansion of the severe CMD pandemic in East and Central Africa in the 1990s, the EACMV-UG strain was disseminated by the whitefly vector over distances of 20–30 km/year (133). The pandemic affected 11 countries and continues to spread southward through the eastern Democratic Republic of Congo toward Zambia and westward through Cameroon toward Nigeria. Most recently, geographic information systems have been used to quantify the distance and direction of movement of the CMD pandemic in northwestern Tanzania (172).

Management strategies. Cassava is vegetatively propagated with stem cuttings. Infected cuttings can mediate the long-distance spread of CMBs and serve as primary inoculum sources in newly planted fields. Cassava is also a crop that is primarily grown by subsistence farmers in relatively small plots. Thus, developing and implementing CMD management are a major challenge.

Before the growing season. Sources of resistance to CMBs have long been known in wild relatives (49, 65). The first source (CMD1) was multigenic and recessive, and it was introgressed into many genotypes throughout Africa (65). Later, CMD2 and CMD3 were identified, and pyramiding of these genes has resulted in highly resistant cultivars (3, 132). It is hoped that new breeding tools, such as genome-wide association mapping coupled with genomic selection, will facilitate resistance breeding (49, 189). Transgenic cassava with resistance to CMBs has been studied for more than 15 years, with the primary focus on pathogen-derived RNA interference strategies (175). Quantitative reduction to CMD infection and disease severity has been identified in transgenic lines, which have been evaluated in the field (176).

Phytosanitation methods include the use of virus-free cuttings (clean seed) and the selection of healthy stems as sources of cuttings for the establishment of new crops. Meristem-tip culture, thermotherapy, and chemotherapy allow for the production of healthy virus-free planting material (51). This material is propagated by tissue culture for the production of virus-free starter material. Formalized seed production systems are being developed in both East and West Africa (notably Nigeria, Tanzania, and Uganda) for seed certification. These methods all place a strong focus on developing systems for maintaining seed health and engaging all stakeholders from breeders to farmers.

During and after the growing season. Managing whitefly populations with insecticides can slow the spread of CMD. However, for cassava in Africa, insecticides are not a viable option in most situations because they are too expensive, not accessible, or both. Extensive surveillance of cassava fields for symptoms of CMD should be practiced. For seed production fields or commercial farms, early roguing of diseased plants can slow the spread of CMD. For both large-scale and smallholder producers, it is recommended to select disease-free stems at harvest for establishing the next season's crop. When implemented early in the growing season, this measure can slow the spread of CMD. Since the 1990s, serology and molecular tools have been used to more precisely detect and monitor CMBs (65, 101, 102, 130, 131, 134, 164, 171, 196). Currently, an artificial intelligence approach (including the use of smartphone apps) to distinguish the five major cassava diseases is being developed for symptom-based cassava virus disease diagnostics and surveillance (143). This has recently been made available through Google Play as PlantVillage Nuru for download and

use by anyone with an Android smartphone used in the field. Following the harvest, it is essential to practice thorough sanitation by uprooting all plants and destroying debris.

Begomoviruses of Cotton

Cotton is the most important fiber and cash crop worldwide. Symptoms typical of begomovirus infection in cotton, e.g., stunting, leaf curling and crumpling, and vein swelling and enations, were reported in Nigeria in 1912. Cotton leaf curl disease (CLCuD) is now endemic across North Africa. The causal agent in Africa is the monopartite begomovirus *Cotton leaf curl Gezira virus* and *Cotton leaf curl Gezira betasatellite* (72).

In Pakistan and India, begomovirus complexes cause major losses to cotton production. The begomoviruses involved are monopartite and require a betasatellite for development of typical disease symptoms. CLCuD was first observed in Pakistan near Multan in the 1960s. It was not considered a problem until 1988, when it reached epidemic levels and spread to northwestern India. The disease is caused by a complex of the monopartite begomoviruses *Cotton leaf curl Multan virus* (CLCuMuV) and CLCuKoV, and the *Cotton leaf curl Multan betasatellite* (CLCuMuB) (28, 116). Most recently, CLCuMuV and CLCuMuB have spread into southern China, where CLCuD is causing economic losses (33).

A virus-like disease of cotton was described in the southern United States and northern Mexico in the 1950s. The symptoms of this disease were stunting and leaf curling and crumpling. Not until the mid-2000s did researchers establish that the causal agent of cotton leaf crumple disease was a distinct NW bipartite begomovirus, which was named *Cotton leaf crumple virus* (73, 159). Here, the practice of ratoon cotton (ratooning is the practice of leaving the cut stems of harvested plants in the field to give rise to next year's crop) provided infected plants early in the season, which served as primary inoculum. The elimination of ratoon cotton and the relatively late appearance of the disease have made this disease no longer economically important (29, 73, 159).

Symptoms and epidemiology. The identification of cotton begomoviruses requires molecular tests because they can induce similar symptoms. CLCuD symptoms consist of vein swelling, upward or downward cupping of the leaves, and the formation of enations on the main veins on the abaxial side of leaves. Frequently, the enations develop into striking cup-shaped leaf-like structures. Cotton plants with CLCuD also appear darker green than noninfected plants, owing to the proliferation of chloroplast-containing tissues. Symptoms vary depending on the cotton cultivar and the age at which plants are infected. Plants infected soon after emergence are usually severely stunted, have tightly rolled leaves, and produce no harvestable lint. Plants infected late in development, e.g., after flowering, generally develop mild symptoms and experience little yield reduction. The symptoms induced by the NW *Cotton leaf crumple virus* are less severe and include stunting and leaf curling and crumpling. *Cotton leaf curl virus* and other cotton begomoviruses infect mostly malvaceous species, including cotton, okra (*Abelmoschus esculentus*), and hollyhock (*Alcea rosea*) as well as weeds such as *Sida* spp. and cheeseweed (*Malva parviflora*). Malvaceous hosts are the main reservoirs of cotton-infecting begomoviruses.

Management strategies. In many cotton-producing regions of the OW (e.g., Africa and Asia), CLCuD is a major constraint on production of this economically important crop, whereas in the NW cotton leaf crumple disease causes minimal losses. Development and release of resistant cultivars provided effective management of CLCuD in Pakistan until the emergence of a resistance-breaking strain. Until new resistant cultivars are bred, disease management involves selecting favorable planting dates and field locations, whitefly vector management with insecticides,

and sanitation measures. However, there is a need to use an IPM approach for management even when a cultivar with high levels of resistance is available.

Before the growing season. CLCuD-resistant cultivars, developed and deployed during the 1990s, provided high levels of resistance and the disease virtually disappeared (116). Unfortunately, these cultivars became ineffective following the appearance of the resistance-breaking CLCuKoV-Burewala complex in 2001. Resistant cotton cultivars are not currently available, although some promising sources of resistance have been identified. Thus, the present problem will likely be resolved again by conventional breeding/selection. Farmers in severely affected areas have reverted to growing *Gossypium arboreum* (native cotton), which is highly resistant, rather than the susceptible *G. hirsutum* (NW cotton). However, lint produced by *G. arboreum* is of poorer quality and brings lower prices.

Currently, the management of CLCuD involves establishing fields with virus- and whitefly-free transplants, preferably those that are locally produced. Seed treatment with systemic insecticides can protect cotton plants for up to 50–60 days. Additional insecticide treatments, even at late stages of development, can provide yield increases. Across South Asia, strict controls on planting dates are used to avoid an early buildup of whitefly populations and virus inoculum. Appropriate planting times, i.e., mid-April to mid-May, resulted in reduced disease incidence compared with delayed plantings, i.e., mid-May to June (55).

During and after the growing season. Roguing has not proven to be an effective measure for management of CLCuD. Application of insecticides to reduce vector populations and slow virus spread, particularly if done early in the growing season, is the major measure used during the growing season. However, excessive insecticide use is common, leading to resistance in the vector. Balanced plant nutrition, especially potassium, can minimize the effects of CLCuD on yield, especially with susceptible cultivars. After harvest, cotton plants should be promptly removed and destroyed or deep plowed. Volunteer cotton (self-set) and malvaceous reservoirs, as well as whiteflies, should be eliminated and fields kept clean until the next planting.

Begomoviruses of Okra

Begomovirus diseases are major constraints on okra cultivation in many tropical and subtropical regions of the OW. Monopartite begomoviruses in association with betasatellites are the main cause of these diseases, although bipartite begomoviruses have also been implicated. Begomovirus diseases of okra can be classified into two types based on symptoms: okra yellow vein disease (OYVD) and okra leaf curl disease (OLCD). In South Asia, the main virus complex causing OYVD is *Bhendi yellow vein mosaic virus* and *Bhendi yellow vein mosaic betasatellite*, whereas the cause of OLCD is *Okra enation leaf curl virus* and *Okra leaf curl betasatellite* (77, 185). In Africa, *Cotton leaf curl Gezira virus* and *Okra yellow crinkle virus* with *Cotton leaf curl Gezira betasatellite* are the main causes of OLCD (56, 89, 103, 104). Recently, OLCD was reported in China, and it was associated with the introduction of CLCuMuV and CLCuMuB (190).

Symptoms and epidemiology. Symptoms of OYVD include stunting, vein swelling and yellowing, and mild leaf curl (**Figure 2*g***). In contrast, OLCD causes stunting, strong leaf curling and distortion, vein swelling and enations on the abaxial surface of leaves but little or no chlorosis and vein yellowing. On occasion, plants may show both types of symptoms. Eventually, infected plants become severely stunted with reduced fruit numbers and size, with most fruit unfit for market (162). Reservoir hosts are generally weeds or crop plants of the family Malvaceae.

Management strategies before, during, and after the growing season. There are no commercially available cultivars with resistance to OYVD or OLCD. However, resistance has been identified in some cultivars and wild species and is being used in breeding programs (155, 162). To reduce losses, it is important to select fields far from those currently under okra production and to avoid planting periods with peak whitefly populations. In northern India, early planted okra fields showed lower begomovirus disease incidences (4.1%) compared with later planted crops (92.3%) (36). This difference likely reflects the dynamics of whitefly populations and demonstrates the importance of planting date. During the growing season, whitefly populations should be monitored and, when appropriate, managed by application of different types of insecticides. After harvest, okra plants should be destroyed and fields kept free of malvaceous volunteers and weeds. The use of an okra-free period should also be considered after the main growing season.

CASE STUDIES: MASTREVIRUS

Maize Streak Disease

Maize streak disease (MSD) is one of the most devastating diseases of maize worldwide (6, 82). Economic losses associated with MSD vary from US$120 to US$480 million per year, depending on the susceptibility of the cultivars being grown and the age at which plants are infected (119). MSD is caused by MSV, the type species of the genus *Mastrevirus* (161). The disease is widely distributed in Africa, and epidemics have occurred at 5- to 15-year intervals in more than 20 countries (6, 115, 119, 122).

MSV is transmitted by several species of leafhoppers in the genus *Cicadulina*, especially *C. mbila* (Hemiptera: Cicadellidae) (**Figure 3*c,d***). Transmission is circulative (persistent) and nonpropagative (92, 115, 146). MSV has been subdivided into 11 strains (MSV-A through MSV-K) (161). MSV-A is the most virulent and economically important strain (82).

Symptoms and epidemiology. Symptoms include reduction in plant growth, leaf size, and yield as well as leaves with yellow to light green streaks and striations (82) (**Figure 2*b***). Plants infected early in development are severely stunted, produce undersized and misshaped cobs, and have little or no yield. At later stages of growth, plants are substantially less affected (150). The host range of MSV includes many cultivated and noncultivated graminaceous species (82, 119).

Management strategies. One of the challenges of MSD management is that different strategies must be developed for the types of farms producing maize in Africa. These range from very large commercial farms that employ modern technologies to subsistence farmers with small fields (e.g., 0.5–2.0 hectares) and little access to modern technologies. Thus, this makes development and implementation of regional IPM strategies difficult.

Before the growing season. The measures that can be used include planting resistant cultivars; selection of the time of planting and field location of maize crops; and intercropping, which creates barriers that slow MSV spread (82, 119). Developing maize cultivars with resistance to MSV has been a long-term objective of several public and private breeding programs. International research centers, including the International Maize and Wheat Improvement Center (CIMMYT) and the International Institute of Tropical Agriculture (IITA), have generated cultivars that show reduced MSD incidence and severity (142). In South Africa and Zimbabwe, the government subsidizes the price of MSD-resistant hybrid maize seed to make it more affordable for small farmers. The MSV-resistant hybrid seed is provided to farmers through seed companies (82). Many conventionally

bred resistant cultivars possess the *Msv-1* gene, the first MSV resistance gene to be genetically mapped (94). It is a single and partially dominant gene, making it relatively easy to use in a breeding program (93). Transgenic maize with resistance to MSD was generated with a dominant-negative mutant strategy that targeted the Rep protein (160). This and other transgenes have not yet been introduced into commercial maize cultivars.

During the growing season. Farmers should inspect fields regularly and rogue maize plants and grassy weeds showing streak symptoms (82, 119). This should be done early in the growing season (up to 30–45 days after planting), when it is most effective. Application of insecticides to manage the leafhopper vector is an important and commonly used measure. This may involve a combination of contact and systemic insecticides, e.g., aldicarb, carbofuran, and imidacloprid (82). Carbofuran can be applied to seeds or to the furrow during maize planting or as foliar sprays, whereas imidacloprid can be applied as seed and soil treatments or as foliar sprays (82, 115, 161).

After the growing season. Following harvest, maize stubble, grassy weeds, and volunteers must be promptly removed and destroyed to reduce MSV inoculum sources for the next planting (82). Crop rotation can be used to manage MSD, but this requires regional coordination among farmers to be most effective (6, 82). Rotation of maize with nonhost broadleaf crops is recommended, as this leads to the greatest decrease in leafhopper populations (82, 161). Crop rotation is particularly important for smallholder farmers who often have limited access to seed of resistant maize cultivars and insecticides (6, 82).

Wheat Dwarf Disease

Wheat dwarf disease (WDD) can be severe and cause economic losses in cereals. The disease is caused by WDV, a member of the genus *Mastrevirus*. WDD-like symptoms, in association with high populations of potential leafhopper vectors, were reported in the county of Frankenstein and other parts of the Silesia province in Prussia (present-day Poland) as early as 1863 (79). The disease was first described in 1961 in western regions of the former Czechoslovak Socialist Republic (180). It was later detected in several locations throughout Europe as well as in parts of Africa, the Middle East (e.g., Iran and Turkey), and Asia (20, 88, 157). WDV is transmitted by the leafhopper *Psammotettix alienus* (Hemiptera: Cicadellidae) in a circulative (persistent) and nonpropagative manner (**Figure 3*e***).

Symptoms and epidemiology. WDV causes symptoms that include stunting, yellowing and streaking of leaves, and reduced head formation (157) (**Figure 2*i***). The host range of WDV includes cereals such as wheat and barley and several other noncultivated grass species, many of which can act as virus reservoirs (157). WDV has been subdivided into five strains (WDV-A–E), each of which has a different geographical range. There are also differences in host range among WDV strains. The most common and economically important strains are WDV-A and WDV-E, which preferentially infect barley and wheat, respectively (110, 145).

The leafhopper vector overwinters as eggs, and wingless nymphs are responsible for the spread of WDV within fields in spring and early summer, before wheat has reached the stage of growth where plants are resistant to WDV (108, 109, 181). The age and stage of growth when plants are infected are major determinants of WDD symptom severity (109). Wheat plants infected at later stages of development are less susceptible or even resistant. When conditions are favorable for disease spread, all plants in a field may be infected, resulting in substantial yield losses (108).

Management strategies. Because genetic resistance to WDD is limited, other measures must be used for WDD management. The development and implementation of a monitoring and predictive system in the fall have helped make decisions regarding the need for implementation of management measures for the upcoming growing season.

Before the growing season. Most barley and wheat cultivars are susceptible, with the exception of two partially resistant wheat cultivars (22, 181). A recent screening of close relatives of wheat revealed potential sources of resistance that could be used for future breeding programs (129). An alternative strategy is to engineer transgenic resistance to WDD in barley via artificial microRNAs (87). Early developing wheat cultivars are less affected by WDD, as they reach the resistant growth stage before leafhoppers transmit the virus in spring/early summer (109). The time of planting is also an important factor in the incidence and severity of WDD (109). When winter wheat is planted later in the fall, inoculum for primary infections will be reduced because adult leafhoppers are inactive at average temperatures below 10°C (108).

During and after the growing season. Application of insecticides can be used to manage the leafhopper vector and reduce the spread of WDD. Forecasts based on leafhopper and WDV surveys in the fall indicate what measures should be used in the next crop, including recommendations regarding the application of insecticides to prevent secondary spread by nymphs (117). This allows for the application of insecticides only when there is a high risk. Infected volunteers and regrowth from stubble of old harvested crops, as a result of reduced tillage, can act as resorvoirs for WDV and the leafhopper vector (108, 117). The spider *Tibellus oblongus* is a potential biological control agent for *P. alienus*, which is a preferred prey species, at least under experimental conditions (153).

Chickpea Stunt Disease

The mastrevirus *Chickpea chlorotic dwarf virus* (CpCDV) is a part of the disease complex that causes chickpea stunt disease. CpCDV was first identified in India (69) and is transmitted by the leafhopper *Orosius orientalis*. This virus occurs across Africa, the Middle East, and South Asia; it is now spreading to new areas.

Symptoms and epidemiology. The major host of CpCDV is chickpea (*Cicer arietinum*). In chickpea, the virus causes stunting, foliar chlorosis or reddening (depending on the chickpea cultivar), and reduced leaflet size (**Figure 2*j***). The virus also affects other grain legumes, including lentil (*Lens culinaris*).

Management strategies. Chickpea is a cool-weather (winter) crop grown in semi-arid regions in open fields and is usually rain-fed. Seed is usually planted in September/October and harvested in early spring. Until recently, CpCDV was not considered a major component of chickpea stunt disease, and most management measures have focused on the other viruses involved in the disease. Delaying the planting date to avoid high leafhopper populations and planting tolerant chickpea cultivars have reduced vector populations and virus buildup. This has proven useful in preventing losses due to CpCDV (66). The use of shorter intervals between irrigations (less water stress) also reduced losses due to the disease.

The long-term management of CpCDV remains problematic and almost entirely depends on conventional breeding/selection. Unfortunately, this is complicated by mixed infections with other viruses. Therefore, it can be difficult to distinguish resistance to CpCDV from symptoms induced by these other viruses. An *Agrobacterium*-mediated inoculation system to screen chickpea

germplasm for resistance to CpCDV has been developed, but it has yet to be adopted on a wide scale (81).

CASE STUDY: CURLY TOP DISEASE CAUSED BY THE CURTOVIRUS *BEET CURLY TOP VIRUS*

CTD is caused by BCTV, the type species and major member of the genus *Curtovirus*. BCTV is a complex species, with at least 11 recognized strains, including some that are host-specialized. However, unlike most geminiviruses, BCTV has a broad host range of more than 300 species in 44 families, including crops and weeds (23). The vector of BCTV is the BLH (*C. tenellus*, Hemiptera, Cicadellidae), and transmission is circulative (persistent) and nonpropagative (**Figure 3*f***). BCTV and CTD have been economically important in the western United States since the early 1900s, when the virus was introduced along with the BLH vector. Initially, CTD caused substantial losses to sugar beet production in the western United States, but the development and planting of resistant cultivars greatly reduced these losses. Subsequently, CTD has caused more economic losses in processing tomato crops, especially in California, and common bean crops in Idaho. In the United States, the disease is particularly important in California, Idaho, New Mexico, and Utah. It also has caused losses in central and northern Mexico (especially in pepper crops) and the eastern Mediterranean region and parts of Central Asia (mostly in sugar beet).

Symptoms and Epidemiology

Symptoms in sugar beet include upward curling and twisting of leaves as well as swelling, enations, and phloem necrosis of veins. Sugar beet roots from BCTV-infected plants are stunted and the vascular tissue is necrotic, which reduces yield and quality. Sugar beet is a preferred host of the virus and the BLH. In tomato, plants are stunted and distorted and leaves are light green to dull yellow, rolled upward, and thickened, and veins develop a distinctive purple coloration (**Figure 2*k***). Tomato plants infected at a young age may die, whereas fruits on plants infected later in development are small and ripen prematurely. In contrast to sugar beet, tomato is a nonpreferred host of the BLH (the insect will not reproduce on tomato), but high incidences of CTD can develop in tomato fields when high populations of viruliferous leafhoppers pass through during spring migrations from the foothills.

The BLH is a migratory desert insect in the western United States. During late fall, female BLHs migrate to the foothills of western mountain ranges and overwinter on various weed hosts in uncultivated areas. These include Russian thistle (*Salsola* spp.), filaree (*Erodium* spp.), peppergrass (*Lepidium nitidum*), and numerous other species (23, 37, 44). Furthermore, when infected with BCTV, these weed hosts are typically symptomless and have low viral titers. In California, overwintered female BLHs lay eggs from late winter to early spring, and adults from this generation, some of which have acquired BCTV, migrate to the Central Valley floor in March-May, depending on weather conditions. There are three to five generations on the valley floor, mostly on plants of the family Chenopodiaceae. Weather conditions dictate the extent of the annual outbreaks of BLH and BCTV (23, 37, 42).

Management Strategies for Sugar Beet

The major crops affected by CTD are sugar beet and tomato. Because of differences between sugar beet and tomato production systems in terms of BCTV, management strategies are presented separately.

Before the growing season. Sugar beet cultivars with conventional resistance have been generated by mass selection and hybridization of diverse sugar beet lines placed under high virus pressure (136). Currently, resistant cultivars are commercially available and can be used to reduce losses due to CTD. This resistance is quantitative (i.e., involves multiple genes) and has been robust (i.e., resistance-breaking strains have not emerged). However, it has been difficult to introgress this resistance into preferred high yielding cultivars (136, 168). Furthermore, cultivars with BCTV resistance may develop symptoms when plants are infected at a young age (8 or fewer leaves). Currently, growers prefer susceptible high yielding cultivars despite the risk of losses from CTD.

Effective insecticide-based management, together with early planting dates, has led to the abandonment of resistant cultivars in many regions (80). Seed treatments with the neonicotinoids clothianidin and thiamethoxam, which protect plants from BLHs for at least 60 days, greatly reduced early season incidences and severity of CTD (166, 167). In some regions, seed treatment is required to compensate for the planting of cultivars with low to moderate levels of resistance (166, 167). The timing of planting is also important. Early planting allows for maximum plant growth before leafhoppers migrate to the valley floor in mid- to late spring and transmit BCTV.

During and after the growing season. The use of neonicotinoid insecticides as seed treatments has revolutionized the management of BCTV in sugar beet in the Pacific Northwest of the United States (166, 167). However, it will be important to use a combination of different active ingredients to reduce the risks of selecting for insecticide resistance in the BLH. Insecticide applications to older plants can also be used, but in many cases this has been discontinued owing to the cost and questions about the efficacy of such treatments. To reduce inoculum sources for the next season, sanitation should be used in between harvest and planting of the new crop. Fields should be free of weeds, especially those in the family Chenopodiaceae, and volunteer sugar beet plants.

Special integrated pest management program for *Beet curly top virus* in processing tomatoes in the Central Valley of California. CTD is an economically important disease of processing tomato in certain parts of central California, e.g., Fresno, Kern, Kings, and Merced counties. There are no curly top–resistant tomato cultivars commercially available, and most currently grown cultivars are very susceptible to BCTV. Although the BLH does not reproduce on tomato, it transmits BCTV during probing of the phloem. Furthermore, because sugar beet is no longer grown in the Central Valley of California, there has been a selection of BCTV strains that are more severe in tomato (e.g., CO and LH71) (37).

A grower-funded program managed by the California Department of Food and Agriculture monitors BLH with sweep nets. Researchers at the University of California, Davis, determine the incidence of BCTV and viral strains in these BLHs. In addition, the incidence of BCTV infection in tomato and other crops and weeds is determined during the growing season and in weeds in overwintering locations in the foothills in the late fall through early spring. Forecasting the severity of CTD for the next growing season is done on the basis of (*a*) the number of BLHs captured and (*b*) the levels of BCTV determined in these BLHs with a PCR test (37). High BLH populations (>5 BLHs/sweep) and high incidences of BCTV in these BLHs (>50% of BLH samples have a strong positive signal) indicate the threshold for economic impact in tomato (e.g., >10% CTD incidence). On the basis of the BLH population and distribution, the California Department of Food and Agriculture conducts an insecticide (malathion) spray program via aircraft and ground rigs that target locations with high BLH populations. Growers also have the option of applying insecticide, e.g., cyazypyr. Finally, although BCTV-resistant cultivars are not currently commercially available, it has been established by agroinoculation that tomato breeding lines with certain combinations of *Ty* genes, which confer resistance to the begomovirus TYLCV, are also resistant

to BCTV. Efforts are ongoing by seed companies to breed BCTV-resistant processing tomato cultivars.

CASE STUDY: GRAPEVINE RED BLOTCH DISEASE CAUSED BY THE GRABLOVIRUS *GRAPEVINE RED BLOTCH VIRUS*

Until 2011, geminiviruses had been mainly reported from annual or biennial crops. In recent years, RCA and next-generation sequencing revealed several new circular ssDNA viruses that resemble geminiviruses from perennial crops. Among these is *Grapevine red blotch virus* (GRBV), which was detected in wine grapes exhibiting a leaf roll–like disease in California. GRBV has now been established as the causal agent of grapevine red blotch disease (GRBD) (169, 193).

Because of its strong association with poor wine quality, GRBV has drawn much attention since it was first identified in 2012. In the United States, GRBV is present in most wine grape production regions, and it also has been detected in grapevines in Canada. In addition to wine grapes, GRBV has been detected in table grapes and wild *Vitis* spp. plants around vineyards (10, 16, 137). The primary economic impact is the loss of revenue due to adverse effects on wine quality. The presence of the three-cornered alfalfa treehopper (*Spissistilus festinus*, Hemiptera, Membracidae) in vineyards with GRBD and the detection of GRBV in the treehopper have implicated this insect as a potential vector (15, 39) (**Figure 3*g***). Indeed, subsequent transmission studies established that this treehopper can transmit GRBV to grapevines.

Symptoms and Epidemiology

Symptoms of GRBD are irregular red blotches on leaves and red veins (**Figure 2*l***) that appear in late summer through the fall. The fruit has substantially reduced soluble solids (9, 169). However, similar symptoms are also caused by other abiotic and biotic factors. In white-fruited cultivars, GRBD symptoms resemble nutritional disorders. The symptoms induced by GRBV and leaf roll disease (caused by a number of viruses in two genera of the family *Closteroviridae*) are similar, but leaves infected with GRBV do not roll downward and those infected with leaf roll viruses do not have red veins. Nonetheless, diagnosis based solely on symptoms can be very difficult. To confirm symptomatic infections and to detect GRBV in symptomless plants, PCR-based assays are conducted with DNA extracts from petioles (121, 169). At the present time, there are many aspects of the epidemiology of GRBD that are unknown and need to be investigated.

Management Strategies

GRBV is one of the few geminiviruses that infect a perennial crop (a vineyard can remain in production for decades). This means that some measures used for management of geminivirus disease of annual crops, e.g., host-free periods, cannot be used. It remains to be determined whether GRBV is primarily spread in association with grapevine propagative materials or whether an insect vector plays an important role (or both). Regardless, the elimination of GRBV from the propagation line is essential to provide growers with GRBV-free planting materials. Many believe that this one step will result in a significant reduction in the incidence of this virus.

Before the growing season. Grapevine materials with resistance to GRBV have not been identified, but the development of an agroinoculation system for GRBV should facilitate screening of grapevine germplasm and wild species for resistance (193). Transgenic approaches have been successful in generating resistance to many geminiviruses, and such strategies should be evaluated

for GRBV. However, the wine industry is concerned about regulatory issues and public perception associated with transgenic grapevines.

A primary source of GRBV is infected grapevine planting stock in the supply chain. This includes public and private grapevine foundations and nursery production blocks. Because grapevine is a clonally propagated perennial crop, a key management measure for GRBD is planting virus-free propagative material. Molecular detection techniques such as PCR and quantitative PCR are used to detect virus-infected propagative material and plants, which are then eliminated. Due to the requirement of seasonal virus testing of large volumes of vegetative material, the cost of detection can be substantial. Thus, there is a need to develop high-throughput low-cost detection techniques for GRBV, such as an enzyme-linked immunosorbent assay. The ultimate goal is the elimination of GRBV from the propagative stock pipelines that produce plants for the establishment of commercial vineyards.

During the growing season. In commercial vineyards, roguing of symptomatic grapevines, together with a general vector management strategy, e.g., an application of a systemic insecticide such as a neonicotinoid, is currently recommended. As the importance of the three-cornered alfalfa treehopper in GRBD epidemiology remains to be established, there are no specific management recommendations for this insect. However, preliminary results based on the observation of GRBV at the edges of blocks next to riparian areas or irrigation ponds have suggested that winged insects spread the virus. At present, GRBV infection is restricted to *Vitis* spp. and infection of other plants from different families on vineyard floors has not been detected (16, 137).

EXAMPLES OF RESISTANCE-BREAKING STRAINS OF GEMINIVIRUSES

Viruses Causing Cotton Leaf Curl Diseases

A major setback to the management of CLCuD was the appearance of a resistance-breaking virus complex in South Asia in 2001. Conventional breeding/selection in the 1990s led to the development of resistant lines and cultivars, resulting in the virtual absence of cotton plants with CLCuD by 2000 (a period of approximately five years). However, in 2001, CLCuD reappeared in the same area as the initial epidemic, and plants of the resistant cultivars were developing severe disease symptoms, suggesting the emergence of a resistance-breaking strain or virus. The resistance-breaking virus spread across central Pakistan and into northwestern India, where it caused economic losses. Resistance breaking was associated with a recombinant begomovirus strain derived from CLCuKoV-Burewala. This strain turned out to be derived from two species, CLCuKoV and CLCuMuV, that were prominent prior to the deployment of resistant cultivars. In addition, the resistance-breaking strain was associated with a recombinant version of the betasatellite that was associated with these viruses, CLCuMuB (11, 12). Remarkably, the resistance was overcome in little more than five years. This has had serious implications for the management of CLCuD with conventional resistance and suggests that resistance based on a (presumed) single mechanism is not durable.

An Emerging and Resistance-Breaking *Tomato yellow leaf curl virus* Recombinant

In 1997, tomato yellow leaf curl disease was observed for the first time in Morocco (139), and because all tomato cultivars were susceptible, farmers had to use insect-proof screenhouses and insecticides to manage *B. tabaci* populations and TYLCV. In 2003–2004, cultivars with the *Ty-1*

resistance gene were deployed. These provided high levels of resistance and allowed for tomato production in open fields. By 2010, these resistant cultivars had replaced the previously grown susceptible cultivars. However, a recombinant TYLCV strain (TYLCV-IS76) with sequences of TYLCV/*Tomato yellow leaf curl Sardinia virus*, replaced its parental virus in southern Morocco and caused tomato yellow leaf curl disease on *Ty-1* cultivars (21). This revealed the emergence of resistance-breaking strains of TYLCV, and, according to experimental studies, the replacement was due to its positive selection by the *Ty-1* resistant cultivars (21a).

SUMMARY

This review highlights the global importance of geminivirus diseases, which continue to spread worldwide, and efforts to manage these diseases. Furthermore, new geminivirus species and genera continue to be described, further expanding the geographic distribution and host range of these viruses. Extensive efforts to understand the biology of these viruses and the epidemiology of geminivirus diseases have allowed for considerable progress in managing geminivirus diseases. The most desirable measure entails breeding and deployment of geminivirus-resistant cultivars. However, such cultivars are not available for some crops (e.g., cucurbits, grapevines, and peppers) and, in the case of some resistant cultivars, resistance-breaking geminivirus strains have emerged (e.g., cotton and tomato). There is also a need for systems to deliver seeds of geminivirus-resistant cultivars to smallholder farmers, who tend to experience more extensive losses because of these diseases. Protected culture has allowed for effective management of geminivirus diseases in high-value vegetable crops, including locations where disease pressure is high. In open fields, row covers can protect young plants from early infection, but additional measures are often needed after covers are removed. The narrow host range of many geminiviruses allows two-to-three-month regional host-free periods to be an effective management measure. Application of insecticides to manage insect vectors continues to be an important management measure for geminivirus diseases, and promising new chemistries are available (e.g., cyazypyr). However, insecticide-resistant insect vectors, especially *B. tabaci*, are an ongoing problem. Thus, although substantial progress has been made, effective management of geminivirus diseases remains challenging, especially in tropical and subtropical regions where subsistence farmers grow overlapping crops and apply excessive amounts of insecticide. Clearly, no single measure will provide long-term sustainable management, and an IPM approach is required. Finally, new approaches employing technologies such as gene editing need to be utilized to facilitate the development of horticulturally desirable cultivars with resistance to geminivirus diseases.

SUMMARY POINTS

1. IPM is the most desirable and effective approach for managing geminivirus diseases. This approach can be broken down into measures used before, during, and after the growing season. The combination of measures used in IPM programs depends on the crop and cropping system, geographical region, and knowledge of the virus-vector biology and disease epidemiology.
2. There is a need to develop new and novel ways to deliver and implement IPM programs for different types of production, ranging from protected culture to open-field production on large commercial farms to small subsistence farms. It is important to know the most effective IPM programs for each case.

3. Although the planting of resistant cultivars is one of the most desirable measures for management of geminivirus diseases, resistance-breaking strains have overcome (broken) this resistance in a number of cases, sometimes relatively rapidly. This further highlights the importance of using an IPM program with multiple management measures and raises questions about the best breeding strategies to minimize the selection of resistance-breaking strains.
4. Geminiviruses can occur in mixed infections with other economically important plant viruses (e.g., RNA viruses such as criniviruses, ipomoviruses, potyviruses, and tospoviruses), and some combinations result in synergistic interactions and more severe disease. This adds additional challenges in terms of diagnostics and management.
5. Application of insecticides to manage insect vectors of geminiviruses will continue to be a relevant management measure. Ideally, insecticide applications will be implemented with an IPM approach, i.e., based on monitoring vector populations and making applications only when thresholds have been reached. Finally, it is important to continue to develop new chemistries that allow for rotation of different classes of insecticides as well as alternatives for organic production and that are compatible with biological control.

FUTURE ISSUES

1. Smartphone technologies show tremendous potential for diagnostics and delivery of IPM approaches, especially in less-developed countries.
2. Are there other new approaches that allow for the delivery of IPM programs, especially to subsistence farmers? This eventually must be done at the local level.
3. What is the future of GMO technology for managing geminivirus diseases (and agriculture in general)? Can GMOs be compatible with IPM programs, and can the complex regulatory and social issues impacting their commercialization be resolved?
4. Is CRISPR-Cas going to provide a non-GMO gene-editing technology that will allow for engineering of crops with resistance to geminivirus diseases? Will this provide a more rapid response to the challenge of resistance-breaking strains/viruses? Will this be more acceptable for IPM?
5. Are geminiviruses truly circulative (persistent) and nonpropagative in their insect vectors, or are certain insect vector–geminivirus combinations, e.g., *B. tabaci*–TYLCV, evolving to be circulative (persistent), propagative, and transovarially transmitted? What, if any, are the epidemiological implications?
6. The same questions can be asked regarding the recent report that TYLCV is seed-transmitted in tomato. Is this an anomaly, or can it explain the long-distance spread of some geminiviruses? What, if any, is the epidemiological importance of seed transmission?
7. Are unknown geminiviruses present in wild reservoirs a future threat to agriculture? Can the use of next-generation sequencing help reveal the extent of this threat?
8. Are there ways to reinforce or strengthen regulatory policies that will limit long-distance spread of geminiviruses without interfering with international trade?

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