Epidemiology of criniviruses: an emerging problem in world agriculture

Ioannis E. Tzanetakis1*, Robert R. Martin2 and William M. Wintermantel3*

1 Department of Plant Pathology, Division of Agriculture, University of Arkansas, Fayetteville, AR, USA
2 Horticultural Crops Research Laboratory, United States Department of Agriculture-Agricultural Research Service, Corvallis, OR, USA
3 Crop Improvement and Protection Research Unit, United States Department of Agriculture-Agricultural Research Service, Salinas, CA, USA

INTRODUCTION
The genus Crinivirus is one of the three genera in the family Closteroviridae and includes viruses with segmented genomes, transmitted by whiteflies (Martelli et al., 2011). Details on the molecular biology of the criniviruses are presented in the Kiss et al. (2013) article and for the most part will not be duplicated in this communication. Instead this article will focus on virus epidemiology.

Criniviruses are emerging worldwide, with the first member of the genus, Beet pseudo-yellows virus (BPYV) identified in the 1960s (Duffus, 1965). Since then there has been a steady increase in the number of new species with most identified over the past 20 years (Winter et al., 1992; Celis et al., 1996; Duffus et al., 1996a,b; Liu et al., 1997; Wieler et al., 1998a; Salazar et al., 2000; Wieler and Duffus, 2001; Martin et al., 2004; Martin et al., 2008; Tzanetakis et al., 2004; Oikawa et al., 2010).

Crinivirus genomic RNAs are encapsidated into long flexuous rods averaging between 650 and 1000 nm in length (Liu et al., 1997; Kreuzet al., 2002), and have large bipartite or tripartite genomes of positive-sense single-stranded RNA totaling approximately 15.3–17.7 kb. Genome organization is similar across the genus, but there are also apparent differences among species. RNA1 encodes proteins that are associated predominantly with replication, whereas RNA2 (or RNAs 2 and 3 for Potato yellow stripe virus [PYVV]) encodes up to 10 proteins with a range of functions including but not limited to virus encapsidation, cell-to-cell movement, and vector transmission. Most genomic RNAs have common or highly conserved nucleotides at the 3′ end ranging from 4 to 11 nucleotides in length. The 3′ untranslated regions for each virus other than Lettuce infectious yellows virus (LIYV) share a region of approximately 150 nucleotides with a high degree of genetic conservatism between the genomic RNAs.

Crinivirus transmission is species-specific and performed exclusively by whiteflies in the genera Trialeurodes and Bemisia in a semi-persistent manner; the reason they are identified with increasing frequency in tropical and subtropical climates where whitefly populations are present. They often cause symptoms that are readily mistaken for physiological or nutritional disorders or pesticide phytotoxicity. Typically, infection is associated with a loss of photosynthetic capability, often characterized by inter-veinal yellowing of leaves, leaf brittleness, reduced plant vigor, yield reductions, and early senescence, depending on the host plant affected. Some plants may exhibit an interveinal reddening rather than yellowing. Others may exhibit chlorotic mottle on some leaves, usually progressing into interveinal discoloration. Symptoms generally first appear 3–4 weeks after infection, and are most apparent on the older areas of the plant, whereas new
growth appears normal. For example, a tomato plant infected with a crinivirus may show extensive interveinal yellowing on leaves near the base, developing interveinal chlorosis on leaves in the middle of the plant, but no symptoms near the apex (Figure 1). Similarly, an infected cucumber plant may appear healthy near the growing point of the vines, but exhibit progressively more severe interveinal yellowing toward the crown (Figure 1). In both cases it is not uncommon for brittle, symptomatic leaves to snap when bent.

An interesting characteristic of many of the criniviruses studied to date is their ability to interact with other viruses in plants and alter symptoms. Studies have shown host-specific competition between crinivirus species that influence accumulation of other viruses present in the plant and consequently symptom severity (Karyeija et al., 2000; Susaimuthu et al., 2008; Wintermantel et al., 2008). Other viruses interact with distantly related or unrelated co-infecting viruses, resulting in increased disease severity whereas single crinivirus infections may remain asymptomatic (Karyeija et al., 2000; Tzanetakis et al., 2004, 2006b).

Management of criniviruses is predominantly through management of their whitefly vectors. Criniviruses routinely emerge in areas with regularly occurring or persistent whitefly populations, or as vector populations migrate or are moved to new regions. An effective vector control regimen can slow spread or reduce severity of infections; however, such methods will not prevent infection as most criniviruses can be transmitted within the relatively short acquisition and transmission periods of a few hours (Wisler and Duffus, 2001). Sources of host plant resistance have been identified to some criniviruses (McCreight, 1987, 2000; Lopez-Sesé and Gomez-Guillamon, 2000; Aguilar et al., 2006; Eid et al., 2006; Garcia-Cano et al., 2010; McCreight and Wintermantel, 2011) and efforts to identify additional sources are in progress. This may offer potential for effective control and reduced pesticide application as resistance is incorporated into commercial cultivars. Recent studies have also shown that deterrence may effectively reduce whitefly and subsequently virus pressure within fields. For example, acylsucrose expressed through type IV glandular trichomes on tomato have been shown to interfere with the ability of whiteflies to settle and feed steadily, and this can significantly reduce primary and secondary spread of the Begomovirus, Tomato yellow leaf curl virus (Rodriguez-Lopez et al., 2011, 2012). Although no conclusive studies have been completed with criniviruses, preliminary studies on tomatoes expressing acyl sugars demonstrated delayed Tomato infectious chlorosis virus (TICV) symptom development in the field by as much as a month compared with controls (Mutschler and Wintermantel, 2006).

In this communication we provide information on the recent advances in crinivirus epidemiology and associated diseases. Viruses will be presented according to their phylogenetic grouping (Wintermantel et al., 2009b; Table 1) as members of each group tend to have similar vectors and host ranges (Table 1).

**GROUP-1**

**ABUTILON YELLOWS VIRUS**

Abutilon yellows virus (AYV) is a partially characterized crinivirus originally identified from the common weed velvetleaf (Abutilon theophrasti Medic.) collected from Illinois in 1977 (Liu et al., 1997). AYV has flexuous filamentous particles of 12 nm in diameter, approximately 850–900 nm in length (Liu et al., 1997, 2000) but the genome remains uncharacterized; with the exception of the
Abutilon yellows virus was the first crinivirus known to be transmitted exclusively by *T. abutilonea* Haldeman (banded-wing whitefly) but there is limited information on its host range and geographic distribution. No crop plants have been identified as hosts; however, the virus can infect members of the Malvaceae, and the experimental solanaceous species, *Nicotiana clevelandii* Haldeman (banded-wing whitefly) is found (Wisler et al., 1998a). The range of *T. abutilonea* has increased dramatically in recent years with the movement of plant material as has BPYV. Both virus and vector have become serious problems for greenhouse production of vegetables, fruits, and ornamentals worldwide. BPYV is transmitted very efficiently by its vector (Wisler et al., 1998a; Tzanetakis et al., 2003). In California, where the vector has become naturalized, BPYV is now restricted to Duchesne clones UC-10 and UC-11 (Tzanetakis et al., 2003). In addition, once introduced into areas where *T. vaporariorum* does well outside the protected environment of greenhouses, the vector has often become naturalized and BPYV often becomes problematic in field-grown crops, as was the case in the western United States (Wintermantel, 2004). Additionally, once introduced into areas where *T. vaporariorum* does well outside the protected environment of greenhouses, the vector has often become naturalized and BPYV often becomes problematic in field-grown crops, as was the case in the western United States (Wintermantel, 2004). Another unique feature of BPYV is its broad host range infecting plants in at least 12 plant families including many vegetable, ornamental, and berry crops. Typical symptoms include interveinal chlorosis as leaves mature (Figure 1), reduced growth and fruit size, and early senescence in cucurbits (Wisler et al., 1996a). BPYV was first reported in a rosaceous host, strawberry in 2002 and is one of the criniviruses that can induce strawberry pallidosis disease in Fragaria virginiana Duchesne clones UC-10 and UC-11 (Tzanetakis et al., 2003). In California, where the vector has become naturalized, BPYV is now quite common in strawberry (Martin and Tzanetakis, 2013). It was also reported from blackberry in the southeastern United States in plants that exhibited symptoms of blackberry yellow vein disease (BYVD; Tzanetakis and Martin, 2004b). At present, BPYV is rare

Abutilon yellows virus (AYV) X
Bean pseudos-yellows virus (BPV) X
Bean yellow disorder virus (BYTD) X
Blackberry yellow vein associated virus (BYYVA) X
Cucurbit chlorotic yellow virus (CCYV) X
Cucurbit yellow stunting disorder virus (CYSDV) X
Dodicia vein chlorosis virus (DVCV) X
Lettuce chlorosis virus (LCV) X
Lettuce infectious yellow virus (LYV) X
Lettuce infectious yellows virus (LIYV) X
Potato vein yellow virus (PYYV) X
Strawberry pallidosis associated virus (SPaV) X
Sweet potato chlorotic stunt virus (SPCSV) X
Tomato infectious chlorosis virus (TICV) X
Tomato infectious chlorosis virus (TICV) X

**Table 1** Crinivirus species and their known vectors.

| Virus | Whitefly vector |
|-------|-----------------|
| AYV   | X               |
| BPV   | X               |
| BYTD  | X               |
| BYYVA | X X             |
| CCYV  | X               |
| CYSDV | X X             |
| DVCV  | X X             |
| LCV   | X               |
| LIYV  | X               |
| PYYV  | X               |
| SPaV  | X               |
| SPCSV | X               |
| TICV  | X X X X         |

*Ref: Benouis tabaci biotype A, BtB, B. tabaci biotype B; BtQ, B. tabaci biotype Q; Baf, B. fruticicola; Tab, T. abutilonea; Tvp, T. vaporariorum.*
Blackberry yellow vein disease was first observed in the North and South Carolina in 2000 and has since become the most important component of the epidemiology of BYV\textsubscript{aV}. The virus has been detected throughout the southeastern United States, in California and Oklahoma and as far north as Illinois and West Virginia, but with surprisingly low incidence in Georgia and Florida. Overall, 145 of 234 samples of cultivated and native blackberries that exhibited BYVD symptoms tested positive for BYV\textsubscript{aV} (Poudel et al., 2013). Given the complexity of BYV\textsubscript{aV} there have not been efforts to introduce resistance for to BYV\textsubscript{aV}.

**Diodia vein chlorosis virus**

Virginia buttonweed (Diodia virginiana L.) is a member of the Rubiaceae (coffee family). Its natural habitat is in wetlands of
FIGURE 3 | (A) Blackberry infected with Blackberry yellow vein associated virus and Blackberry chlorotic ring spot virus showing symptoms of yellow vein disease; (B) Diodia virginiana infected with Diodia vein chlorosis virus showing vein netting symptoms; (C) potato infected with Potato yellow vein virus showing yellow vein disease symptoms; (D) strawberry decline symptoms of leaf reddening and dieback associated with Beet pseudo-yellows virus and Strawberry pallidosis associated virus co-infection with other viruses.

The Americas, extending between the 45th parallels of both continents. It propagates in a prolific manner through stolons and seed, making it one of the most noxious weeds of turfgrass. Several Virginia buttonweed populations in the southern United States show distinct vein chlorosis or vein netting symptoms, typical of virus infection (Figure 3). Larsen et al. (1991) studied the disease and discovered virus aggregates in infected material similar to those found in closterovirus-infected plants. The putative virus produced double-stranded RNA was similar in size to that of LIYV and T. abutilonea was experimentally verified as a vector. All these properties indicated that Diodia vein chlorosis virus (DVCV) is a member of the genus Crinivirus but no molecular information was available until recently when an isolate from a clone of a plant used in the Larsen et al.’s (1991) study was sequenced (Tzanetakis et al., 2011). DVCV genome is composed of 16.2 kb with RNA1 coding for the replication-associated polyprotein and RNA2 having the normal array of eight genes found in most members of the genus. Phylogenetic analysis clearly placed DVCV in group 1 of the genus. Given that all members of the group have been proven transmissible with T. vaporariorum, this was evaluated for DVCV. Indeed, both T. abutilonea and T. vaporariorum transmit the virus with efficiencies of over 36 and 12%, respectively when plants were inoculated with 50 whiteflies after 48-h AAP and IAP (Tzanetakis et al., 2011). The phylogenetic placement of DVCV, its vectors and the co-habitat of D. virginiana and berry crops resulted in a decision to conduct a series of experiments to determine the ability of the virus to infect strawberry and blackberry. Those experiments failed to identify additional hosts for DVCV other than D. virginiana. Given that the only known host for DVCV is a weed, control measures are not employed for this virus other than the elimination of Virginia buttonweed through the use of herbicides.

**POTATO YELLOW VEIN VIRUS**

Potato, a plant native to South America, is a host of several viruses. Many are asymptomatic in single infections, and as many cause devastating diseases that lead to major losses (Salazar, 2006). Plants affected by potato yellow vein disease can suffer losses reaching as much as 50%. Typical symptoms include vein yellowing that gives leaves the appearance of a yellow net (Figure 3). The disease was first identified in 1943 and has since been reported in Venezuela, Columbia, Ecuador, and Peru (Diazmore, 1963; Salazar, 2006). It was not until the turn of the century that the putative causal agent was identified and characterized (Salazar...
Crinivirus

Strawberries (family Rosaceae) are known to be natural hosts for several of which occur wherever the crop is grown and can cause significant losses (Spiegel and Martin, 1998). Pallidosis disease is a significant problem, with the virus being transmitted by aphids. The agent was transmitted by T. vapariorum and named PYVV. Virus purification and cloning of the HSP70 gene of the virus indicated that is a member of the genus Crinivirus (Salazar et al., 2000). PYVV is a unique crinivirus as the only member of the genus with a tripartite genome. RNA1 is organized similarly to other criniviruses, encoding the replication-associated proteins and small peptide with a transmembrane domain. RNA2 encodes five proteins that are found in the 5′ terminus of the crinivirus orthologous molecule whereas RNA3 has three ORFs commonly found at the 5′ terminus of RNA2 in other crinivirus species, indicating that PYVV is probably a product of an ancestral virus segmentation in which the ancestral RNA2 segregated into PYVV RNAs 2 and 3 (Liveratos et al., 2004) although, in phylogenetic terms, it appears ancestral to the bipartite members of group-1 (Figure 2). The host range of the virus is rather restricted, and includes species in the genera Solanum, Polygnum, Rumex, Tagetes, Catharanthus, and Malva able to sustain virus replication whereas many common crinivirus indicators including Nectarisana, Datura, and Physalis species are resistant to infection (Salazar et al., 2000; Guzman and Rodriguez, 2010). The limited host range of the virus is reinforced by the fact that studied isolates present rather limited diversity (Offer et al., 2004; Guzmán et al., 2006; Rodríguez-Burgos et al., 2010). PYVV is closely associated with yellow vein disease symptoms but Koch’s postulates have not been fulfilled as the virus can remain asymptomatic in potato. The importance of the disease, the ease of transmission, as recorded with the transmission of the virus in greenhouses in the UK, in combination with the asexual propagation and the cosmobital growth of the potato industry has made the development of advanced detection methods obligatory for the industry, and there are reports of sensitive detection protocols available (López et al., 2006). Virus control is based on insecticide use and strict quarantine directives that would not allow virus spread outside the countries where it is currently present.

**Strawberry Pallidosis Associated Virus**

Strawberries (family Rosaceae) are known to be natural hosts for about 30 viruses (Martin and Tzanetakis, 2006; Tzanetakis, 2010). Several of which occur wherever the crop is grown and can cause significant losses (Spiegel and Martin, 1998). Pallidosis disease initially was identified in the United States during the 1950s (Frazier and Stabbbs, 1969). Symptoms on indicator plants of F. virginiana clones “UC-10” or “UC-11,” can include leaf distortion, chlorosis, and some dwarfing, though under less than optimal conditions for symptom development it is easy to overlook symptoms. Two viruses have been consistently associated with the disease, BPYV and Strawberry pallidosis associated virus (SPaV). Sequencing of the genome of SPaV confirmed it as a crinivirus (Tzanetakis et al., 2005). It contains two RNAs, both approximately 8 kb, with typical crinivirus genome organization. SPaV is most closely related to BPYV and AVY based on phylogenetic analysis (Tzanetakis et al., 2005).

There have been reports of severe strains of the pallidosis agents that are lethal on indicators. Graft transmission of multiple isolates from the eastern and western United States caused only mild symptoms and it is most likely that these “severe strains” actually represented mixed virus infections involving not only a crinivirus, but likely another partner virus (Hokanson et al., 2000; Tzanetakis et al., 2004).

**Strawberry pallidosis associated virus** is transmitted by T. vapariorum, although somewhat inefficiently compared to BPYV (Tzanetakis et al., 2006b). Surprisingly, SPaV was more common in strawberry than BPYV in field settings. Both viruses were found in the majority of plants that exhibited decline symptoms due to mixed virus infections in California in the 2002–2003 periods (Figure 3). The decline epidemic was estimated to cause losses of about 50 million dollars for the two seasons (Martin and Tzanetakis, 2013). Plants were infected with at least one of the two criniviruses (BPYV or SPaV) and one of the common aphid-transmitted strawberry viruses (Strawberry crinkle virus, Strawberry vein banding virus, Strawberry mottle virus, or Strawberry mild yellow edge virus), incidence of SPaV was as high as 90% compared to 40% for BPYV. In plants from the Mid-Atlantic states that indexed positive for pallidosis disease based on symptoms, 37 of 38 plants were positive for SPaV and only about 25% were positive for BPYV (Tzanetakis et al., 2006b). Either virus can cause pallidosis symptoms in indicator plants. In other comparisons, SPaV was always more common in strawberry plants in side-by-side field comparisons than BPYV. This suggests that in nature there are other factors that contribute to virus transmission efficiency than what is typically measured in greenhouse or growth chamber studies. It is possible that the colony of whiteflies used in the greenhouse studies is better adapted to transmission of BPYV than SPaV or there are other, yet to be identified, vectors that are more efficient for transmission of SPaV. SPaV had a very limited host range in greenhouse studies, where it did not infect Urtica urens L., but was found in an Urtica species in the field in an area with high T. vapariorum populations, though this could have been a different Urtica species (Tzanetakis et al., 2006b). The virus has been reported in strawberry production areas throughout the Americas, Australia, and Egypt (Wintermantel et al., 2006; Ragab et al., 2009; Constable et al., 2010; Martin and Tzanetakis, 2013). Both BPYV and SPaV are asymptomatic in single or mixed infections in “Hood” and “Northeast” strawberry (Tzanetakis, 2004). Given the annual plasticulture that has been adapted in most production areas in the world it is imperative that plants do not become infected within the nursery system. Infections with the strawberry criniviruses may be asymptomatic but when plants accumulate additional viruses in the field, they can decline rapidly. The titre of SPaV declines in summer and for this reason testing for this virus is recommended in spring or late fall using younger but fully expanded leaves (Tzanetakis et al., 2004). As in the case of BYVV, the symptomsless single infections and the complexity of disease-causing virus complexes have discouraged work toward identification of accessions which preclude virus replication.

**GROUP-2 BEAN YELLOW DISORDER VIRUS**

Legumes (family Fabaceae) are infected by numerous viruses, several of which cause significant losses with many regularly identified in new locations around the world (de Oliveira et al., 2011; Zhou et al., 2011). This was also the case of a disease observed in common bean (Phaseolus vulgaris L.) in Spain in...
2003. Symptoms were similar to nutritional disorders with yellowing of the leaf blade, whereas pods appeared malformed. Leaves were brittle and whitefly transmission with B. tabaci/Gennadius yielded reproducible symptoms. These observations pointed to a crinivirus infection. Confirmation came with the cloning of the HSP70h gene of the virus, which was named Bean yellow disorder virus (BYDV; Segundo et al., 2004). An extended study in greenhouses in Spain, the only country the virus is known to exist, showed BYDV incidence of about 6%, indicating that the virus was an emerging problem for bean growers (Segundo et al., 2008). BYDV genome is 17.5 kb; encoding four proteins in RNA1 and nine in RNA2 (Martin et al., 2008). Phylogenetic analysis indicated the close relationship of BYDV with vegetable-infecting criniviruses that are efficiently transmitted by B. tabaci (Martin et al., 2008). Transmission experiments revealed efficiencies that exceeded 35% using single whiteflies with 24 h AAP and IAP, respectively. A much more surprising result was the retention ability of B. tabaci which reached 2 weeks when most other criniviruses are retained for less than a week (Martin et al., 2011). More than 30 species belonging to the families Asteraceae, Boraginaceae, Cucurbitaceae, Fabaceae, Geraniaceae, Lamiaceae, Malvaceae, Scrophulariaceae, Solanaceae, Thymelaeaceae, and Verbenaceae were evaluated as hosts but only four legume species (P. vulgaris, Pisum sativum, L.Lens culinaris Medik., and Vicia faba L.) were able to sustain virus replication. Given the high incidence of the virus in greenhouses, control measures have primarily focused in these production systems. Beans grown in screenhouses had 14 times fewer whiteflies per plant. The incidence of the virus under greenhouse protection never exceeded 12.5% unlike that in conventional greenhouses which reached over 80% (Janssen et al., 2011). Given the incidence of the virus in the confined environment of a greenhouse, the physical barrier of fine mesh screenhouses appears to be the most efficient approach to minimize vector presence and associated virus transmission.

CUCURBIT CHLOROTIC YELLOWS VIRUS

Cucurbits are grown throughout the world and are exposed to a wide array of production environments and pests. These crops are known to be infected by more than 60 viruses (Leocq and Desbiez, 2012), and several are discovered each year (Brown et al., 2013). Until resistance is incorporated into commercial cultivars, control will require insecticide treatment of whitefly-infested areas. Many whiteflies transmit cucurbit yellows, however, more recent studies have demonstrated CYSDV infections result in reduced plant vigor, and can significantly reduce fruit sugar production, resulting in poor tasting, unmarketable fruit. The host range of CYSDV was originally believed to be restricted to members of the Cucurbitaceae (Celix et al., 1996); however, more recent studies have demonstrated CYSDV can infect plant species from at least nine families (Wintermantel et al., 2009a). Although cucurbits are the predominant and most significant agricultural hosts of the virus, common bean can be severely affected, resulting in severe stunting and virtual elimination of yield when infected at an early age. Lettuce is another host of the virus (Wider et al., 1998a), and can be a reservoir for transmission to other crops, but symptoms are mild and agronomically insignificant (Wintermantel et al., 2009a). Numerous common weeds are also hosts of the virus, but in most cases these plants are symptomless and vary in their ability to serve as effective virus reservoirs for transmission to crop hosts (Wintermantel et al., 2009a).
When vector populations are high it is virtually impossible to prevent infection of cucurbits. When CYSDV emerged in the American Southwest nearly all cucurbit production was affected during the first year due to the presence of excessively high vector populations.

Studies conducted on isolates collected over geographically distinct regions (Rubio et al., 2001) as well as local populations (Marco and Aranda, 2005), demonstrated most isolates are highly conserved genetically. Proteins show significant variation and among them the coat protein region seems to exhibit the most substantial variability, illustrating the divergence of a cluster of isolates from Saudi Arabia from other isolates identified from throughout the world (Rubio et al., 2001). Examination over time of a CYSDV collection from a localized region in Spain demonstrated an exceptionally high level of conservation within the virus population compared with other plant viruses (Marco and Aranda, 2005). It is speculated that genetic bottlenecks may influence the low genetic diversity within local populations. Similarly, genetic bottlenecks may also influence emergence of unique variants as observed for Arabian isolates (Marco and Aranda, 2005).

Management of CYSDV is predominantly through insecticide based vector control, which reduces vector numbers and results in slower rates of symptom development, but does not prevent virus transmission. Increasing efforts are focusing on development of virus resistance, particularly in cucumber and melon (Lopez-Sesé and Gomez-Guillamon, 2000; Marco et al., 2003; Aguilar et al., 2006; Eid et al., 2006; McCreight and Wintermantel, 2011), in which new sources of resistance to the virus have been identified in recent years. Efforts are progressing toward characterization of resistance in both hosts and toward combining resistance sources in melon.

**LETTUCE CHLOROSIS VIRUS**

Yellowing symptoms, normally associated with the crinivirus, LIYV, were observed in vegetable fields in the southwestern United States in the 1990s. At that point in time LIYV had virtually been eliminated following displacement of its primary vector, *B. tabaci* biotype A. This fact lead Duffus et al. (1996b) to investigate the possibility that other viruses might be present in the region, and ultimately to the discovery of *Lettuce chlorosis virus* (LChV). The virus is transmitted by *B. tabaci* biotypes A and B with similar efficiencies. Whiteflies can acquire and transmit the virus with AAP/IAP of 1 h each. Transmission was more efficient after 24 h of feeding whereas retention did not exceed 4 days. The host range includes at least 31 species belonging to 13 families, with several noteworthy hosts including spinach, sugar beet, and several weed species commonly found in the southwestern United States (Duffus et al., 1996b; McLain et al., 1998). The two genomic RNAs of the virus are contain in individual particles of 800–850 × 12 nm. The 17-kb genome is arranged similarly to that of other members of group-2, encoding four proteins in RNA1 and 10 in RNA2 (Salem et al., 2009). Insecticide applications can minimize virus incidence, something that is particularly important in early season where LChV can have a significant impact in lettuce yield (McLain et al., 1998). Infected lettuce can exhibit foliar yellowing, but also head deformation if infection occurs early. LChV has not spread
to areas outside the southwest United States and is not usually a significant production threat, probably as a result of lettuce-free periods and the inability of the virus to infect other significant crop hosts during the fall season when whitely populations are elevated.

**SWEET POTATO CHLOROTIC STUNT VIRUS**

Sweet potato is one of the most nutritious vegetables, rich in vitamins and microelements and one of the most important staple foods available today in sub-Saharan Africa (Luebestedt and Thotappilly, 2009). Virus-like diseases of sweet potato have been reported for more than 50 years in Africa with several aphid-

**TOMATO CHLOROSIS VIRUS**

Tomato chlorosis virus (ToCV) was originally identified in 1996 from greenhouse-grown tomatoes (Lycopersicon esculentum Mill.) from Florida (Wäster et al., 1998), and exhibits a moderate host range of at least 24 plant species from seven different families (Wintermantel and Wäster, 2006). Symptoms on tomato include interveinal chlorosis, leaf browning, and limited necrotic flecking or leaf bronzing, and are nearly identical to those associated with infection by TCV (Figure 4), although genetically the two viruses vary significantly. Several methods are now available to differentiate ToCV from TCV, including RT-PCR (Wintermantel and Hladky, 2010; Papajannis et al., 2011), molecular probes (Garcia-Cano et al., 2010), or virus-specific antisera (Duffus et al., 1996; Jacquesmondi et al., 2009; Wintermantel, unpublished).

The 16.8 kb genome of ToCV is typical of criniviruses and is encapsidated as long flexuous virions approximately 800–850 nm in length (Liu et al., 2000). RNA1 encodes four ORFs including proteins associated with virus replication, and suppression of gene silencing (Wintermantel et al., 2005; Canizares et al., 2008), and RNA2 encodes up to nine ORFs encoding proteins involved in a multitude of functions including virus encapsidation, cell-to-cell movement, membrane association, and whitely transmission (Stewart et al., 2010; Chen et al., 2011).

The host range of ToCV extends, in addition to tomato, to other solanaceous hosts including pepper (Lozano et al., 2003), potato (Fortes et al., 2012), and tomato (Trenado et al., 2007). Several weed species can also harbor ToCV (Font et al., 2004; Wintermantel and Wäster, 2006), and the presence of weed hosts near production areas can provide an alternate host for the virus between cropping seasons, as well as providing an acquisition source for whitely vectors that can carry the virus back to cultivated hosts.

**Tomato chlorosis virus** is unique among members of the genus as transmission by at least five different whitelys has been documented (Navas-Castillo et al., 2000; Wintermantel and Wäster, 2006). The virus AAP is short, but transmission occurs more readily when vector whitelys have IAP of several hours. Transmission efficiency varies among whitely species, with *T. abutilonense* and *B. tabaci* biotype A and *T. vapturium* transmit ToCV with much lower efficiency (Wintermantel and Wäster, 2006). *B. tabaci* biotype Q is also an efficient vector, and has emerged as the predominant vector in southern Europe (Navas-Castillo et al., 2000). Each vector also differs in its ability to retain the virus, with *T. abutilonense* able to transmit for up to 5 days following virus acquisition, whereas *B. tabaci* biotype B loses its ability to transmit ToCV after 3 days.
B. tabaci biotype A and T. vaporariorum lose their transmissibility after only a day (Wintermantel and Wider, 2006). ToCV has a relatively long latent period in infected host plants, often not inducing symptoms until 3 weeks after infection. If nursery plants are exposed to viruliferous vector populations at an early age, it is possible for ToCV-infected plants to be carried to new areas through movement of transplants prior to symptom development.

Management of ToCV is primarily through the management of vector populations using both chemical and cultural practices. Since criniviruses cannot spread without whitefly vectors, suppression of vector populations can keep crinivirus spread to a minimum. Although insecticides can reduce whitefly populations, such control methods are inefficient for virus control, since whiteflies can transmit viruses before being killed by an insecticide. In addition to vector control, it is important to limit availability of alternate host plants that can serve as virus reservoirs. Testing of nursery stock and ornamental host plants for the presence of these viruses can also reduce movement of ToCV to new areas. Importantly, resistance to ToCV was recently identified in crosses between Solanum lycopersicum (tomato) and S. peruvianum L., as well as S. chilense (Dunal) Reiche (Garcia-Cano et al., 2010). Intrigression of this resistance into cultivated tomato should greatly strengthen future management of ToCV.

GROUP 3: LETTUCE INFECTIOUS YELLOWS VIRUS

Lettuce infectious yellow virus is the most thoroughly studied virus in the genus Crinivirus. It was discovered in the southwestern desert agricultural regions of the United States in 1981 (Duffus and Flock, 1982), and was the first crinivirus sequenced (Klassen et al., 1995). Its 15.3 kb genome partially defined the characteristics of the genus.

Lettuce infectious yellow virus has a relatively large host range, infecting at least 45 species of plants in 15 families, and caused significant yield losses for lettuce, melon, and sugar beet. LIYV causes interveinal yellowing symptoms in melon and sugar beet, and a severe yellowing symptom on lettuce that gave the virus its name and resulted in widespread field yellowing (Figure 4). Unlike most other criniviruses affecting commercial agriculture, which have effectively been distributed around the world, LIYV remained predominantly confined to southwestern United States and northern Mexico. This is due to its close relationship with the B. tabaci biotype A, which shared a common geographical range with the virus (Brown and Nelson, 1986; Duffus et al., 1986). The virus persisted in the region throughout the 1980s, but quickly faded from prevalence with the emergence of the B. tabaci biotype B in the early 1990s (Cohen et al., 1992; Brown et al., 1995). As the B biotype became established, the A biotype gradually disappeared from fields, and along with it LIYV. Studies have shown a biological basis for this, with LIYV exhibiting over 100 times greater transmission using the B. tabaci biotype A than biotype B (Wider and Duffus, 2001). LIYV has not been identified in the American Southwest for well over a decade, and although it is possible the virus may still exist in long-term reservoir hosts, the likelihood that it would reemerge is slim, since it is transmitted poorly by current B. tabaci biotypes, and the A biotype is no longer present in the field.

TOMATO INFECTIOUS CHLOROSIS VIRUS

Tomato infectious chlorosis virus was discovered in tomato from southern California in 1993 (Duffus et al., 1994a) and has since been identified as a problem for tomato production in many parts of the world including Mexico, Europe, the Middle East, as well as East and Southeast Asia (Wintermantel et al., 2009b). Symptoms on tomato include, similarly to ToCV, interveinal yellowing (Figure 4) with leaves becoming thickened and crispy, breaking easily when bent. Yield is affected through decreased fruit size and number (Wider et al., 1996), as well as decreased plant longevity (Wintermantel, 2004).

Tomato infectious chlorosis virus virions consist of long flexuous rods varying from 850 to 900 nm in length (Liu et al., 2000) containing the two RNAs of about 8.3 and 7.9 kb. Similarity between TICV and other criniviruses varies throughout the genome but TICV is related much more closely to LIYV than to any other crinivirus, and together the two form a distinct clade within the genus (Wintermantel et al., 2009b).

The virus is transmitted exclusively by T. vaporariorum (Duffus et al., 1994a). TICV can be acquired and transmitted after a 1-h AAP; however, transmission efficiency increases steadily with longer AAPs. A 48-h AAP using 30 whiteflies per plant was most efficient and resulted in 94% transmission. Individual whiteflies given a 24-h AAP on infected source plants transmit TICV at an 8% rate, whereas an 83% transmission rate is found when plants are exposed to 40 viruliferous whiteflies each. Transmission by viruliferous whiteflies also varies over time with transmission using 30 viruliferous whiteflies per plant increasing from 16% transmission with 1 h transmission access periods to 80% when whiteflies are exposed to test plants for 48 h. TICV can persist in whiteflies for up to 4 days, but transmission efficiency drops off dramatically after 24 h (Duffus et al., 1996a).

Although tomato is considered the principal host of TICV, the virus also infects a number of important vegetable and ornamental host plants (Duffus et al., 1994a; Wider et al., 1996). Lettuce, potato, petunia, artichoke, ranunculus, and China aster can also be infected by TICV. Like other criniviruses, TICV symptoms take up to 5 weeks to develop, and during this period movement of infected plant material by the nursery industry or by commercial vendors can be responsible for distribution of TICV to new regions (Wider et al., 1994a). The virus can survive during non-crop seasons in a wide range of weed hosts near production areas and move into crops as whitefly populations develop and become active. Similarly, some ornamentals or alternate crops such as lettuce can serve as reservoirs for virus transmission to tomato (Duffus et al., 1996a; Wider et al., 1998a; Font et al., 2004).

Management of TICV, like other criniviruses, involves both chemical and cultural practices. Since criniviruses cannot spread without whitefly vectors, suppression of vector populations can keep crinivirus spread to a minimum. In addition to vector control, it is important to limit availability of alternate host plants that can serve as virus reservoirs. Although insecticides can reduce whitefly populations, such control methods are inefficient for virus control, since whiteflies can transmit viruses before being killed.
Citrus tristeza virus has changed the map of citrus production. Tomato (Mutschler and Wintermantel, 2006).

Discussion

Citrusviruses cause diseases of great economic importance. Citrus tristeza virus has changed the map of citrus production around the world and the Grapevine leafroll associated viruses have had a major impact on vine health and wine quality, both affecting multi-billion dollar industries worldwide. Citrusviruses have recently emerged as major pathogens in world agriculture, primarily because of the movement and establishment of their whitefly vectors in temperate regions around the world.

There are clear cases in which citrusviruses are the causal agents of devastating diseases such as CYSDV and BPTV in cucurbits or TCV and ToCV in tomato. In addition, there are many cases in which citrusviruses have been the underlying problem behind major epidemics even though they were not originally recognized as such. The examples of SPVd, strawberry decline, and BYVD illustrate how citrusviruses can be asymptomatic in single infections and yet cause serious diseases in the presence of virus complexes with major impacts on plant health and yield. Furthermore, even citrusviruses normally regarded as symptomatic can be asymptomatic in some hosts. Most members of the genus also require a minimum of 3 weeks for symptoms to become apparent. During this time infected plants can be moved to new areas or even new countries without evidence of infection. This fact has major implications at many levels; especially for viruses infecting clonally propagated crops (BPPV, BYVdV, PTVV, SPaV, and SPCSV) or crops associated with grafted transplants (CYSDV and CCYV).

In today's global trading environment there is constant germplasm exchange among individuals and organizations. The previous examples of citrusvirus-driven epidemics should become lessons for the future and provide the impetus to improve plant certification schemes. This will facilitate increasing international trade in plant and plant products while decreasing the unintentional movement of plant pathogens. Given that some of the aforementioned viruses remain confined in specific geographic areas (i.e., BYVdV in the United States, PTVV in northwestern South America) it is still feasible to minimize their future impact by eliminating movement of infected material into areas where these viruses are not present. It is also important to establish vector exclusion strategies at the nursery or propagation field level. It has been common practice in certification schemes that plants are only visually inspected at the certified plant (G4) level. Using strawberry or blackberry as an example, neither BPPV, BYVdV nor SPaV cause symptoms in single infections in modern berry cultivars. However, when singly infected plants are planted in the field they often become infected with additional viruses and the resulting mixed infections can lead to serious epidemics. Exclusion and testing at the G4 level or prior to distribution can enhance longevity and profitability of the crops within regions and prevent or reduce accidental introduction of viruses into new production areas.

Given the relatively recent identification of citrusviruses as economically important disease agents, work has primarily focused on characterization, epidemiology, and in certain cases chemical control of vectors. Still, the ultimate control strategy for any pathogen is strong, stable genetic resistance. Resistance using modern methods such as RNA interference is probably the most straightforward and durable approach to prevent infection by viruses, but public resistance to genetically modified plants especially in crops that are labeled as “healthy food” or “superfoods” such as fruits and vegetables, the primary hosts for citrusviruses, has minimized the application of this technology. For the majority of the citrusviruses little or no work has been directed toward identification of resistance using traditional screening of germplasm resources and/or breeding to incorporate such sources into commercially acceptable cultivars. In the few cases where resistance has been identified it is almost always found in wild accessions, which requires many generations of backcrossing before the relevant genes are incorporated into marketable varieties. That is not to say progress is not being made. Sources of resistance to LfIV were identified in both lettuce and melon (McCreight, 1987, 2000), although the demise of LFIV as an agricultural threat due to shifting vector population dynamics largely rendered advancement of the material a moot point. Other efforts however offer real potential for effective crinivirus management. A source of resistance to ToCV was recently identified in tomato (Garcia-Canto et al., 2010), and two independent and complementary sources of resistance to CYSDV have been found in melon (Lope-Ses and Gomez-Guillamon, 2000; McCreight and Wintermantel, 2011). Sequencing of the genomes of many crops affected by citrusviruses, identification of resistance sources, and the use of marker-assisted selection will speed up the incorporation of these and likely other resistance traits into commercially relevant cultivars.

Criniviruses are transmitted in a semi-persistent manner and chemical control of vectors has not always been effective for virus disease management. In addition, the development of resistance to insecticides in insect populations and the effect of insecticides on whitefly predators may have a negative impact on vector and virus control, particularly in systems using broad integrated pest management approaches. Consequently, it may be appropriate to consider a more generic approach, such as identification of resistance against whitefly vectors. There have been several cases in which insect resistance has been identified in plants (Mutschler and Wintermantel, 2006). In many cases this has been more effective and long-lived than virus resistance, possibly due to the ability of the viruses to drift toward resistance-breaking populations. In addition, vector resistance may be effective in controlling several viruses that are transmitted by a common vector. As an extreme example, aphid resistance to Amphorophora agathonica (Hottes) had been effective for over 30 years in controlling three aphid-borne viruses in raspberry in the North America, before new biotypes of the vector developed that overcame the resistance (Hall et al., 2009). Forms of resistance against insects can function in a number of ways, including acting as feeding deterrents, physical barriers, or oviposition inhibitors. Some plant secondary metabolites dissuade insects from settling on plants, preventing the steady feeding that can lead to toxicity or virus transmission. Others may prevent oviposition, reducing vector populations (Mutschler and Wintermantel, 2006). Studies are just beginning to
address the potential of resistance to insect feeding on control of whitefly-transmitted viruses (Mutschler and Wintermantel, 2006; Rodriguez-Lopez et al., 2011). Appropriate and effective utilization of such approaches will require specific research to confirm that methods effective in controlling one pest do not exacerbate problems with another. Integrating vector control with other means of pest and disease management, however, offers the potential to strengthen durability and effectiveness of control for not only criniviruses, but a number of insect-borne pathogens.

There have been numerous significant breakthroughs in understanding criniviruses, the diseases they cause, and their epidemiology. However, a great deal more work is needed on virus understanding, the diseases they cause, and their potential to strengthen durability and effectiveness of control for not only criniviruses, but a number of insect-borne pathogens.

REFERENCES

Ateka, E. M., Njeru, R. W., Kibaru, A., Aguilar, J. M., Abad, J., and Aranda, P. E., Sobh, H., and Abou-Alicai, T., Fenby, N. S., Gibson, R. Abou-Jawdah, Y., Sobh, H., Fayad, long-term reliability of crinivirus management. Such efforts will among others are not yet disease and viruses, and introgression of resistance genes into commercially acceptable germplasm. These should be priority areas for long-term reliability of crinivirus management. Such efforts will

Brown, J. K., Froshich, D. R., and Rosell, B. C. (1995). The sweetpotato or silvery whitefly-borne Sweetpotato nematode virus: a species complex? Am. J. Entomol. 40, 511–514.

Brown, J. K., Mills-Lequin, K. and Idris, A. M. (2011). Phylogenetic analysis of Melon yellow leaf curl virus from Guatemala: another emergent species in the Squash leaf curl virus clade. Virus Res. 158, 237–262.

Brown, J. K., and Novel, M. R. (1986). Whitefly-borne viruses of melons and cucurbits: A review. Phytopathology 76, 238–250.

Caballero, M. C., Naras-Chauhan, I. and Morales, E. (2008). Multiple supertrees of RNA silencing encoded by both genomic RNAs of the criniviruses. Tomato chlorotic virus. Virology 359, 168–179.

Aguilar, J. M., Abad, J., and Aranda, M. A. (2008). Resistance to Cucurbit yellow stunting disorder virus in cucumber. Plant Dis. 92, 503–506.

Alicia, X., Forby, N. S., Gibson, R. W., Adilpua, E., Heten, J. H., Fuentes, S., Jester, W., and Moyer, W. G. (2009). Occurrence of two serotypes of Sweet potato chlorotic stunt virus in Asia and their associated differences in coat protein and HSP70 homologue gene sequences. Plant Pathol. 48, 718–726.

Abou-Jawdah, Y., Sobh, H., and Abou-Alicai, T. (2012). First report of Cucurbit yellow chlorotic virus on cucumber in Lebanon. Plant Dis. 96, 172–173.

Ashik, E. M., Nyema, R. W., Kifirau, A. G., Kinuthia, J. W., Bax, E., Gibson, R. W., et al. (2004). Identification and distribution of viruses infecting sweet potato in Kenya. Ann. Appl. Biol. 144, 317–328.

Beitel, B., Bernal, J. J., Sáez, E., Woudt, B., Beitia, F., and Rodríguez-Castellanos, M. A. (2006). Resistance to Cucurbit yellow stunting disorder virus, a Brazilian tospovirus transmitted by Tospovirus. Phytopathology 96, 1370–1376.

Chau, P. Y. (2003). Lettuce chlorosis virus: p22 acquisition. Virus Genes 35, 385–389.

Duffus, J. E., Liu, H.-Y., and Walke, G. C. (1996a). Tomato infectious chlorosis virus – a new whitefly-transmitted closterovirus. J. Plant Pathol. 82, 86–90.

Espinosa, S., Abou-Jawdah, Y., El-Melihit, M., Sobh, H., and Aranda, M. A. (2008). Resistance to Cucurbit yellow stunting disorder virus in cucumber. Plant Dis. 92, 503–506.

Fuentes, S., Jester, W., and Moyer, W. G. (2009). Occurrence of two serotypes of Sweet potato chlorotic stunt virus in Asia and their associated differences in coat protein and HSP70 homologue gene sequences. Plant Pathol. 48, 718–726.

Alicia, X., Forby, N. S., Gibson, R. W., Adilpua, E., Heten, J. H., Fuentes, S., Jester, W., and Moyer, W. G. (2009). Occurrence of two serotypes of Sweet potato chlorotic stunt virus in Asia and their associated differences in coat protein and HSP70 homologue gene sequences. Plant Pathol. 48, 718–726.

Alicia, X., Forby, N. S., Gibson, R. W., Adilpua, E., Heten, J. H., Fuentes, S., Jester, W., and Moyer, W. G. (2009). Occurrence of two serotypes of Sweet potato chlorotic stunt virus in Asia and their associated differences in coat protein and HSP70 homologue gene sequences. Plant Pathol. 48, 718–726.

Alicia, X., Forby, N. S., Gibson, R. W., Adilpua, E., Heten, J. H., Fuentes, S., Jester, W., and Moyer, W. G. (2009). Occurrence of two serotypes of Sweet potato chlorotic stunt virus in Asia and their associated differences in coat protein and HSP70 homologue gene sequences. Plant Pathol. 48, 718–726.

Alicia, X., Forby, N. S., Gibson, R. W., Adilpua, E., Heten, J. H., Fuentes, S., Jester, W., and Moyer, W. G. (2009). Occurrence of two serotypes of Sweet potato chlorotic stunt virus in Asia and their associated differences in coat protein and HSP70 homologue gene sequences. Plant Pathol. 48, 718–726.
Franzen, N. W., and Stebbins, L. L. (1949). Paleheads—a new virus dis- ease of strawberry. Plant Dis. Rep. 33, 524–528.

Gumara, H., Fuentes, S., Morales, F. J., Gómez, R., Malahamp, C., and Burt, I. (2010). Bacteriophage-like virus, a vector of Sweet potato chlorotic stunt virus. Plant Dis. 94, 509–514.

Garcia-Cano, A., Navar-Castillo, J., Moriones, E., and Fernández- Muñoz, R. (2010). Resistance to Tomato chlorotic virus in wild tomato species that impair virus accumulation and disease symptom expression. Phytopathology 100, 582–587.

Gibson, R. W., Mpenube, I., Altic, T., Caray, E. A., Awakwaghio, H. O. M., Sol, S. E., et al. (1998). Symptoms, aetiology and serological analysis of sweet potato virus disease in Uganda. Paper Trop. 47, 99–102.

Guo, S. J., Liu, Y. H., Wang, Y. H., Huang, W. G., Gu, H. F., Xu, L., et al. (2011). First report of Cucumber yellow viruses in cucumber, melon, and watermelon in China. Plant Dis. 95, 73.

Guzman, M., and Rodriguez, P. (2010). Susceptibility of Solanum phureja (Luz edulcorado) to Potato yellow virus. Agrovet. Colomb. 28, 219–224.

Guizani, M., Ruiz, E., Armingino, N., and Cozzii, R. H. A. (2006). Occurrence and variability of Potato yellow virus in three departments of Colombia. J. Phytopathol. 154, 748–750.

Grosskopf, Y., Okanaki, S., Furuta, A., Esh, T., Mihobe, M., Kano, K., et al. (2009). Chlorotic yellow disease of melon caused by Cucurbit chlorotic yellow viruses, a new crinivirus. Jpn J. Hort. Sci. 71, 109–111. (In Japanese with English abstract).

Hokanson, S. C., Martin, R. R., and Tzanetakis, I. E. (2011). Bean yellow stripe disease (crinivirus) (genus Crinivirus) in North America. Plant Dis. 95, 101.

Koyací, R. F., Gibson, R. W., and Valkonen, J. T. P. (1998). Resistance to vectorette virus disease (SPVD) in wild bean in greenhouse by means of internal screenhouses. Acta Hort. 497, 279–280.\n
Kao, J., Liu, L., Tian, T., Rublo, L., and Falb, B. W. (2000). First report of Cucurbit yellow stunting disorder viruses in French bean in greenhouse by means of internal screenhouses. Acta Hort. 520, 99–110.

Kawaji, H., Liang, H., Li, J., and Choi, T. C. (2010). First report of Cucurbit chlorotic yellow viruses infecting cucumbers in Taiwan. Paper Trop. 94, 1148.

Jänsch, C., Uhlemann, D., Gauld, A., Gauld, L., and Goggin, P. (2000). Serological and molecular detection of Tomato chlorotic virus and Tomato infectious disease virus in tomato. Plant Pathol. 49, 220–223.

Janssen, D., García, M. C., Bement, A., Pascual, F., García, T., Botstein, G., et al. (2011). Control of Bemisia tabaci in bean yellow-diseased virus in French bean in greenhouses by means of internal screenhouses. Acta Hort. 927, 279–289.

Kao, J., Liu, L., Tian, Y., and Falb, B. W. (2000). First report of Cucurbit yellow stunting disorder viruses in French bean in greenhouse by means of internal screenhouses. Acta Hort. 520, 99–110.

Kawaji, H., Liang, H., Li, J., and Choi, T. C. (2010). First report of Cucurbit chlorotic yellow viruses infecting cucumbers in Taiwan. Paper Trop. 94, 1148.

Kawaji, H., Liang, H., Li, J., and Choi, T. C. (2010). First report of Cucurbit chlorotic yellow viruses infecting cucumbers in Taiwan. Paper Trop. 94, 1148.

Kawaji, H., Liang, H., Li, J., and Choi, T. C. (2010). First report of Cucurbit chlorotic yellow viruses infecting cucumbers in Taiwan. Paper Trop. 94, 1148.

Kawaji, H., Liang, H., Li, J., and Choi, T. C. (2010). First report of Cucurbit chlorotic yellow viruses infecting cucumbers in Taiwan. Paper Trop. 94, 1148.

Kawaji, H., Liang, H., Li, J., and Choi, T. C. (2010). First report of Cucurbit chlorotic yellow viruses infecting cucumbers in Taiwan. Paper Trop. 94, 1148.

Kawaji, H., Liang, H., Li, J., and Choi, T. C. (2010). First report of Cucurbit chlorotic yellow viruses infecting cucumbers in Taiwan. Paper Trop. 94, 1148.
Martin, R. R., Tzanetakis, I. E., Gerg-
trich, R. C., Fernandez, G., and
Pone, Z. (2004). Blackberry yellow
virus associated with a new crinivirus
found in blackberry. Acta Hortic. 636, 157–162.

McCoigh, J. D. (1987). Resistance in
wild lettuce to lettuce infectious yel-
low virus. Frontiers in Microbiology 2, 646–642.

McCoigh, J. D. (2000). Inheritance of
resistance to Lettuce infectious yel-
low virus in melon. Frontiers in Hortic. 35, 1118–1210.

McCoigh, J. D., and Wintermantel, W.
M. (2011). Genetic resistance in
chlorsis virus.

Ragab, M., El-Dougdoug, K., Mousa,
A., and Fereres, A. (2010). Occurrence and inci-
dence of viruses infecting green beans
by the complete genome sequence
of a strawberry isolate.

Riccardi cucurbits.

Rican cucurbits.

Tanzania, I. E., and Martin, R. R. (2008).
Emerging crinivirus disease (SPVD), and its practi-
cal implications. Acta Hortic. 679, 272–279.

Tanzania, I. E., and Martin, R. R. (2005). Nucleotide sequence,
Arch. Virol. 150, 150–151.

Tanzania, I. E., and Martin, R. R. (2006a). Nucleotide sequence
of a virus associated with straw-
berry plants forces

Tanzania, I. E., and Martin, R. R. (2004b). First report of Beet pseudo-
yellows virus. J. Plant Pathol. 86, 385–389.

Tanzania, I. E., and Martin, R. R. (2003). Nucleotide sequence,
Arch. Virol. 150, 150–151.

Tanzania, I. E., and Martin, R. R. (2004). Complete nucleotide
sequence of a strawberry isolate of Beet pseudo-yellows virus. Virus Genes 28, 239–246.

Tanzania, I. E., and Martin, R. R. (2005). First report of Beet pseudo-
yellows virus in blackberry in the United States. Plant Dis. 89, 225.

Tanzania, I. E., Reed, J., and Martin,
R. R. (2001). Nucleotide sequence,
genome organization and phylog-
etic analysis of Strawberry phloem-associated virus, a new member of the genus Crinivirus. Acta Hortic. 55, 517.

Tanzania, I. E., and Martin, R. R. (2008). Complete nucleotide
sequence of a strawberry isolate of Beet pseudo-yellows virus. J. Plant Pathol. 90, 1191–1201.

Tanzania, I. E., Martin, R. R. (2000). A new natural host of T omato chloro-
sis virus disease in Taiwan and evalu-
ation of T omato infectious virus.

Tanzania, I. E. (2010). Emerging
strawberry virus and virus-like dis-
es in the world. Julius Kultur Archi 427, 41–45.

Tanzania, I. E., Bergstrom, M., Keller, K. H., Haukani, S. C.,
Ricardo, E., and Martin, R. R. (2006a). Nucleotide sequence,
Arch. Virol. 150, 150–151.

Tanzania, I. E., and Martin, R. R. (2006b). Nucleotide sequence
of a virus associated with straw-
berry plants forces

Tanzania, I. E., and Martin, R. R. (2004). Complete nucleotide
sequence of a strawberry isolate of Beet pseudo-yellows virus. Virus Genes 28, 239–246.

Tanzania, I. E., and Martin, R. R. (2005). First report of Beet pseudo-
yellows virus in blackberry in the United States. Plant Dis. 89, 225.

Tanzania, I. E., Reed, J., and Martin,
R. R. (2001). Nucleotide sequence,
genome organization and phylog-
etic analysis of Strawberry phloem-associated virus, a new member of the genus Crinivirus. Acta Hortic. 55, 517.
Crinivirus epidemiology

Wintermantel, W. M. (2004). Emergence of greenhouse whitefly (Trialeurodes vaporariorum) as threats to vegetable and fruit production in world agriculture. Front. Microbiol. 4, 119. doi: 10.3389/fmicb.2013.00119

Conflict of Interest Statement: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Received: 08 February 2013; accepted: 28 April 2013; published online: 16 May 2013.

Citation: Tzanetakis IE, Martin RR and Wintermantel WM (2013) Epidemiology of criniviruses: an emerging problem in world agriculture. Front. Microbiol. 4:119. doi: 10.3389/fmicb.2013.00119

This article was submitted to Frontiers in Virology, a specialty of Frontiers in Microbiology.

Copyright © 2013 Tzanetakis, Martin and Wintermantel. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits use, distribution and reproduction in other forums, provided the original authors and source are credited and subject to any copyright notices concerning any third-party graphics etc.