Cucurbit Viruses: The Classics and the Emerging

Hervé Lecoq
INRA, Station de Pathologie Végétale, Domaine Saint Maurice, BP 94, 84143 Montfavet cédex, France

Gail Wisler
USDA-ARS, U.S. Agricultural Research Station, 1636 E. Alisal Street, Salinas, CA 93905

Michel Pitrat
INRA, Station de Génétique et d'Amélioration des Fruits et Légumes, Domaine Saint Maurice, BP 94, 84143 Montfavet cédex, France

ADDITIONAL INDEX WORDS. cucumber, melon, squash, watermelon, cucumovirus, potyvirus, crinivirus, geminivirus, aphid, beetle, leafhopper, thrips, whitefly, nematode, fungus, vector, resistance

ABSTRACT. Viral diseases cause important economic losses in cucurbit crops throughout the world. In the major growing regions, cucurbit viruses represent a complex and changing pathosystem. Several viruses often develop, concomitantly or successively, severe epidemics within a single crop. Among the 35 well-characterized viruses infecting cultivated *Cucurbitaceae* some have been known for a long time (the classics) while others have been spreading and causing serious damage only recently (the 'emerging'). A brief description is provided for each of these viruses along with their distribution, and discussion of the threat they pose to cucurbit crop production. The availability of resistances to these viruses in the four major cultivated cucurbit species (cucumber, melon, squash and watermelon) is also discussed.

Cucurbit crops are affected by a great number of viral diseases: more than 35 well-characterized viruses belonging to the major plant virus groups have been found naturally infecting cultivated *Cucurbitaceae* (Brunt et al., 1996; Lovisolo, 1980; Provvidenti, 1986; Provvidenti, 1996). This virus diversity results in part from the genetic and ecological diversity of their cucurbit hosts. There are more than 10 cultivated cucurbit species, and for each species there are many distinct landraces or cultivars. Also, cucurbits are grown throughout the world in a great variety of agroecosystems ranging from the highly sophisticated soilless cucumber production in heated glasshouses of Northern Europe to the more traditional rainfed cultivation of watermelon in the Sudano-Sahelian region of Africa. These various environments may provide more or less favorable conditions for specific viruses or for their vectors.

Typical viral symptoms in cucurbits generally fall within three major categories (Blancard et al., 1991). 1) Mosaics on leaves that are often associated with different types of deformations: leaf size reduction, rugosity, lacinations or enations. Fruits may also develop a range of discolorations and deformations rendering them unmarketable. 2) Yellows affecting generally the older leaves, but eventually all the leaves may be yellowed, often accompanied by leaf thickening. Fruit production may be significantly reduced, but fruit quality is generally not affected. 3) Necrosis either as necrotic spots of different sizes on leaves or as a generalized necrosis that eventually causes the plant to die. Fruits generally do not reach maturity, and may also develop necrotic symptoms. Necrotic reactions sometimes occur only in certain virus strain/host cultivar combinations, especially with potyviruses. Often, in the fields, plants are infected by more than one virus leading to a combination of these three major types of symptoms.

Probably, more than for any other crops or pathogens, cucurbit viruses constitute a complex and changing pathosystem. This was very well emphasized in the publication of Nameth et al.

(1986) entitled 'Cucurbit viruses of California: an ever-changing problem,' which analyzed the dynamic changes, within a few decades, of the economically important viruses occurring in California. This title could be applied to many other cucurbit production areas around the world. As an illustration, in a comprehensive and detailed review on cucurbit viruses, Lovisolo listed, in 1980, 23 viruses naturally infecting cultivated cucurbits in the world. Since then, at least 16 'new' viruses have been described that infect cucurbits, some of which are widespread and may cause major yield reductions, while others remain restricted to limited geographical regions or to specific cropping systems.

Some of these 'new' viruses were probably present in cultivated cucurbits for a long time, but remained unnoticed because their symptoms were masked by other viruses or were attributed to other causes (mainly nutritional or physiological disorders). This probably applies to cucurbit aphid borne yellows virus (CABYV), a widespread luteovirus causing yellowing symptoms on the older leaves of the plants. CABYV was first identified in 1988, but it was later found to have been very common in samples that had been collected in 1982 (Lecoq et al., 1992).

Others of these new viruses may represent typical 'emerging' viruses. Some became suddenly widespread because they followed the rapid increase and dissemination of their natural vectors. The geminiviruses or criniviruses which are transmitted by the whitefly *Bemisia tabaci* (Wisler et al., 1998) are a good example of this type of 'new' virus. The situation of zucchini yellow mosaic virus (ZYMV) is probably unique in cucurbits (Desbiez and Lecoq, 1997). This potyvirus, transmitted by many aphid species in a nonpersistent manner, was described in the mid-1970s as occurring locally in Northern Italy. Within a decade, ZYMV had spread to all the major cucurbit growing areas in the world. The seemingly rapid spread of ZYMV occurred by means that are still unknown. The spread of ZYMV cannot be related to an extension of the distribution area of its aphid vectors which were already prevalent wherever cucurbits were grown.

Cucurbit virus control strategies are based on the implementation of prophylactic measures mainly aimed at preventing or delaying virus spread by their vectors, and on the use of resistant cultivars when commercially available. For viruses transmitted by airborne vectors, control measures include the use of healthy seeds, protection of nurseries from insect vectors, weed eradication as a means to eliminate virus or vector reservoirs before planting, plastic mulch, and covering the plants with insect-proof nets or floating covers. Control measures for viruses transmitted by soilborne vectors include the use of healthy seeds, soil sterilization, and fungicide addition to nutrient solutions. Disinfection of pruning or harvesting tools prevents dissemination of mechanically transmitted viruses (Blancard et al., 1991).

The use of virus-resistant cultivars is probably the easiest way to control viral diseases at the farmer's level. Breeding for resistance still mainly relies upon germplasm evaluation and introgression of the resistance gene(s) into commercially acceptable cultivars. In recent years, biotechnology has emerged as a new means for creating virus-resistant cultivars. It is significant to note that squash cultivars resistant to ZYMV and watermelon mosaic virus 2 (WMV2) were the first virus-resistant, transgenic plants to be commercially cultivated in the U.S. (Medley, 1994; Tricoli et al., 1995).

In this paper we will describe briefly the major viruses infecting cucurbits with a special emphasis on these 'new' viruses potentially or actually causing major problems in the fields. The viruses will be presented according to their vectors and to their taxonomic position (Table 1). Resistance genes or resistance sources will be indicated when available for the four major cultivated cucurbit species *Cucumis melo* (melon) (Table 2), *Cucumis sativus* (cucumber) (Table 3), *Citrullus lanatus* (watermelon) (Table 4), *Cucurbita pepo* (squash) (Table 5).

Due to the abundance of the literature on cucurbit viruses, and resistances to these viruses, we will refer as much as possible to general reviews or books from which more detailed references can be obtained. Information on individual viruses was obtained from Brunt (1996), Lovisolo (1980), Provvidenti (1986), and Provvidenti (1996) unless stated otherwise. Information on resistances

was obtained from Pitrat (1998), Provvidenti (1986), Provvidenti (1990), Provvidenti (1993), Provvidenti and Hampton (1992), and Wehner (1997) unless stated otherwise.

Viruses transmitted by aphids

CUCUMBER MOSAIC CUCUMOVIRUS (CMV). CMV is a small isometric virus with particles circa 29 nm in diameter, which is very efficiently transmitted by more than 60 aphid species in a nonpersistent manner. CMV has a divided genome constituted of three single stranded RNA molecules. CMV has a broad host range, infecting more than 775 different species in more than 86 different families. Two major subgroups (type I and type II) have been differentiated according to serological and molecular properties. A diversity of strains of the virus has been described differing in symptomatology, host range, virulence towards resistance genes, aphid transmissibility. Strains producing white or yellow mosaic symptoms can be very useful for rapid differentiation of susceptible plants in screening tests for resistance.

CMV, the first mosaic disease reported in cucurbits in the U.S., has been known since the beginning of the 20th century. CMV causes very severe economic losses in melon, cucumber and squash, but is rarely encountered in watermelon. CMV induces plant stunting and mosaic, size reduction and distortion of leaves. Symptoms on fruit are discolorations and occasionally deformations. CMV is still probably one of the most common viruses infecting cucurbits worldwide, especially in temperate or Mediterranean climatic conditions. In tropical or subtropical regions, CMV is less frequent in cultivated cucurbits although it

Table 1. Major viruses infecting cultivated *Cucurbitaceae*: means of spread, geographical distribution and host range.

Genus and virus	First report	Means of spread	Distribution	Host range: Cucurbits	Host range: Noncucurbits
Carmovirus					
Cucumber leaf spot virus, CLSV	1982	fungi, seeds	Europe	wide	wide
Cucumber soil-borne virus, CuSBV	1983	fungi	Lebanon	wide	wide
Melon necrotic spot virus, MNSV	1966	fungi, seeds, contact	worldwide	wide	narrow
Comovirus					
Squash mosaic virus, SqMV	1941	beetles, seeds, contact	worldwide	wide	narrow
Crinivirus					
Beet pseudo-yellows virus, BPYV	1965	whiteflies	worldwide	wide	wide
Cucurbit yellow stunt disorder virus, CYSDV	1982	whiteflies	Mediterranean Basin, Middle East	intermediate	narrow
Lettuce infectious yellows virus, LIYV	1982	whiteflies	North America	wide	wide
Cucumovirus					
Cucumber mosaic virus, CMV	1916	aphids, (seeds ?)	worldwide	wide	wide
Geminivirus, subgroup II					
Beet curly top virus, BCTV	1909	leafhoppers	worldwide	wide	wide
Geminivirus, subgroup III					
Squash leaf curl virus, SLCV	1983	whiteflies	North America	wide	narrow
Watermelon chlorotic stunt virus, WmCSV	1987	whiteflies	Red Sea region	intermediate	unknown
Ipomovirus					
Squash yellow leaf curl virus, SYLCV	1998	whiteflies	Sultanate of Oman	narrow	none
Luteovirus					
Cucurbit aphid borne yellows virus, CABYV	1992	aphids	worldwide	wide	intermediate
Necrovirus					
Tobacco necrosis virus, TNV	1935	fungi	worldwide	wide	wide
Nepovirus					
Artichoke yellow ringspot virus, AYRSV	1973	nematodes (?)	Italy, Greece	wide	wide
Tobacco ringspot virus, TRSV	1927	nematodes, seeds	worldwide	wide	wide
Tomato black ring virus, TBRV	1946	nematodes	worldwide	wide	wide
Tomato ringspot virus, ToRSV	1936	nematodes	worldwide	wide	wide
Ourmiavirus					
Melon Ourmia virus, OuMV	1988	unknown	Iran	intermediate	wide

may be found in weeds or other crops nearby (Quiot et al., 1983). Differences in the type of spread may occur in different cucurbit species: CMV epidemics were observed to be very rapid in Southern Europe and generalized in melons crops while in zucchini squash they were often slow to develop and limited to a few plants.

There have been some reports of seed transmission of CMV in cucurbits, but this is apparently rare and it is probably of little importance on virus epidemiology in crop production. The abundance and diversity of weed reservoirs and aphid vectors for CMV are probably responsible for the early appearance of the virus in cucurbit crops.

CMV diagnosis can be easily achieved within one to few days through two means. 1) Biological assays using local or systemic infections of *Vigna unguiculata*, or systemic infection of *Nicotiana tabacum* are easily done and do not require highly sophisticated facilities or equipment. 2) Classical serological methods such as the double antibody sandwich variant of the enzyme linked immunosorbent assay (DAS-ELISA), or the immunodiffusion test with sodium dodecyl sulfate (SDS-ID) (Clark and Adams, 1977; Purcifull et al., 1988) are also relatively easy to perform. DAS-ELISA diagnostic kits are commercially available for CMV (They include polyclonal or monoclonal antibodies necessary for differentiating strains of the two subgroups of CMV).

Various levels of resistance to CMV are present in some cucumber, melon and squash commercial cultivars. Resistance to CMV transmission by one of its major vectors, *Aphis gossypii*, has been introduced into commercial melon cultivars. This resistance is also efficient against potyviruses and to

Table 1. Continued.

Genus and virus	First report	Means of spread	Distribution	Host range: Cucurbits	Host range: Noncucurbits
Potyvirus					
Clover yellow vein virus, ClYVV	1965	aphids	worldwide	narrow	wide
Melon vein-banding mosaic virus, MVBMV	1993	aphids	Taiwan	wide	intermediate
Papaya ringspot virus, PRSV	1949	aphids	worldwide, tropical and subtropical	wide	narrow
Telfairia mosaic virus, TeMV	1975	aphids, seeds	Nigeria	wide	wide
Watermelon mosaic virus Morocco strain, WMV-M	1974	aphids	Africa and Mediterranean Basin	wide	narrow
Watermelon mosaic virus 2, WMV 2	1954	aphids	worldwide, temperate and Mediterranean	wide	wide
Zucchini yellow fleck virus, ZYFV	1981	aphids	Mediterranean Basin	wide	narrow
Zucchini yellow mosaic virus, ZYMV	1981	aphids, seeds, contact	worldwide	wide	intermediate
Rhabdovirus					
Cucumber toad-skin virus, CTSV	1982	unknown	Mediterranean Basin	narrow	wide
Tobamovirus					
Cucumber green mottle mosaic virus, CGMMV	1935	contact, seeds	Europe, Asia	wide	narrow
Tombusvirus					
Cucumber necrosis virus, CNV	1959	fungi, contact	Canada	wide	wide
Tospovirus					
Watermelon silver mottle virus, WSMV	1982	thrips	Asia, Brazil	wide	wide
Tymovirus					
Chayote mosaic virus, ChMV	1997	unknown	Costa Rica	unknown	unknown
Melon rugose mosaic virus, MRMV	1981	unknown	Red Sea region	intermediate	narrow
Unassigned viruses					
Cucumber vein yellowing virus, CVYV	1960	whiteflies	Middle East	wide	narrow
Squash necrosis virus, SqNV	1983	fungi	Brazil	wide	wide

Table 2. Virus and vector resistance in melon (*Cucumis melo*).

Virus or vector	Resistance source	Genetic control	Strain specificity	Commercial cultivars[z]
Carmovirus				
MNSV	Gulfstream, PI 161375	*nsv*	no	yes
Crinivirus				
CYSDV	TGR 1551	Monogenic dominant	unknown	no
LIYV	V001, PI 313970, MR-1	unknown	unknown	
Cucumovirus				
CMV	Freeman's Cucumber, PI 161375	Oligogenic recessive	yes	few
Luteovirus				
CABYV	PI 124112	*cab-1* and *cab-2*	unknown	no
	PI 414723, PI 255478	unknown	unknown	
Potyvirus				
PRSV	PI 180280	*Prv*[1]	no	yes
	PI 180283	*Prv*[2]	yes	
	PI 124112	Monogenic dominant	unknown	
WMV2	PI 414723	*Wmr*	yes	no
WMV-M	WMR 29	unknown	unknown	yes
ZYFV	PI 414723	unknown	unknown	no
ZYMV	PI 414723	*Zym*	yes	no
	PI 414723	Oligogenic dominant	unknown	
Tobamovirus				
CGMMV	Phoot, Kachri	Polygenic recessive	unknown	no
Tymovirus				
MRMV	WMR29	unknown	unknown	yes
Virus vector				
A. gossypii	PI 161375	*Vat*	no	yes
	PI 414723	*Ag*	no	

[z]Resistances in commercial cultivars are not always derived from sources described in the literature.

Table 3. Virus resistance in cucumber (*Cucumis sativus*).

Virus	Resistance source	Genetic control	Strain specificity	Commercial cultivars[z]
Cucumovirus				
CMV	Tokyo Long Green	Oligogenic (3 genes?)	no	yes
	Chinese Long	Oligogenic	no	
	Taichung Mou Gua	unknown	unknown	
Luteovirus				
CABYV	Taichung Mou Gua	unknown	unknown	no
Potyvirus				
PRSV	Surinam	*wmv-1-1 (=prsv)*	unknown	yes
	Taichung Mou Gua	*Prsv-2*	unknown	
WMV2	Kyoto 3-feet	*Wmv*	unknown	yes
	Taichung Mou Gua	*wmv-2* and *wmv-3*	unknown	
WMV-M	Taichung Mou Gua	*mwm* (similar or linked to *zym*)	unknown	no (?)[y]
ZYFV	Taichung Mou Gua	*zyf* (similar or linked to *zym*)	no	no (?)[y]
ZYMV	Taichung Mou Gua	*zym*	no	yes
Tobamovirus				
CGMMV	Natsufushinari	unknown	unknown	no
No group				
CVYV	unknown	unknown	unknown	yes

[z]Resistances in commercial cultivars are not always derived from sources described in the literature.
[y]Some cultivars that are resistant to ZYMV might also be resistant to ZYFV and WMV-M due to the close linkage between *zym*, *zyf*, and *mwm* genes.

a lesser extent against CABYV. Biotechnology has provided new opportunities to diversify resistance mechanisms to CMV. Significant levels of resistance to CMV have been obtained in squash, melon and cucumber using the coat protein mediated resistance (Fuchs et al., 1997; Gonsalves et al., 1992). Another promising strategy uses ribozymes which are small RNA molecules that possess specific cleavage activities on target RNAs. Ribozymes directed towards sequences coding for the CMV coat protein have proved to be effective in protecting melons against several strains of the virus (Plages, 1997).

Potyviruses. Several distinct members of the Potyvirus Genus infect cultivated cucurbits. Three of them, papaya ringspot virus (PRSV), WMV2 and ZYMV have a worldwide distribution, and cause important yield reductions in melon, cucumber, squash and watermelon. Other potyviruses have either a restricted geographical distributions, for example watermelon mosaic virus Morocco strain (WMV-M), zucchini yellow fleck virus (ZYFV), and melon vein banding mosaic virus (MVBMV), or a wide geographical distribution but have little overall economic effect on yield, for example clover yellow vein virus (ClYVV). Several distinct cucurbit potyviruses from Africa are only partially characterized; extension of their geographical distribution, changes in cultivars, or changes in growing conditions could increase the importance of one or more of them in the future.

Potyviruses have flexuous particles circa 700 to 900 nm long and 12 nm in diameter. Their genome is a unique single stranded RNA molecule that is translated as a polyprotein that is subsequently cleaved by their own encoded proteases to form various functional proteins. Potyviruses are transmitted efficiently by aphids in a nonpersistent manner. Their host range varies among the members of the group, some being relatively wide (such as WMV2) while others are restricted to only a few species outside the *Cucurbitaceae* (such as PRSV).

Potyviruses are highly variable; a number of strains differing in pathogenicity, aphid transmissibility, and antigenic properties have been reported for many of them. Potyvirus strains that have lost their aphid transmissibility can be very useful for screening for resistance. Indeed, some have the same pathogenicity as aphid transmissible isolates, but do not present the risk of accidental spread by aphids in greenhouses or growth chambers.

Potyvirus diagnosis is generally based on classical serological methods (DAS-ELISA, ID-SDS), although cross-reactions might be observed depending upon the virus strains and the antiserum. DAS-ELISA diagnostic kits are commercially available for the most important cucurbit potyviruses: PRSV, WMV2, ZYMV, WMV-M, and ZYFV.

Papaya ringspot virus (PRSV) (formerly watermelon mosaic virus 1, WMV1). PRSV causes severe mosaic and deformations on leaves and fruits of melon, cucumber, squash and watermelon. Some strains of PRSV can induce systemic necrotic spots and

Table 4. Virus resistance in watermelon (*Citrullus lanatus*).

Virus	Resistance source	Genetic control	Strain specificity	Commercial cultivar[y]
Potyvirus				
WMV2	PI 494528,[z] PI 494532[z] PI 189316,[z] PI 189317[z] PI 248178,[z] Egunz	Unknown	Unknown	No
ZYMV	PI 494528,[z] PI 494532[z] PI 482261 Egunz	Unknown One recessive *Zym*	Unknown Yes Unknown	No
Crinivirus				
CYSDV	PI 386015,[z] PI 386016[z]	Unknown	Unknown	No
Geminivirus				
WmCSV	*C. lanatus*	Unknown	Unknown	No

[z]*Citrullus colocynthis.*
[y]Resistances in commercial cultivars are not always derived from sources described in the literature.

systemic necrosis in some melon cultivars. PRSV epidemics are common in tropical or subtropical areas where the wild cucurbit species reservoirs of the virus are prevalent. Epidemics are occasionally observed in temperate regions such as northeastern U.S. or central Europe. PRSV strains have been differentiated either on a host range basis (the papaya strain, PRSV-P, infects papaya and cucurbits; the watermelon strain, PRSV-W, infects cucurbits but not papaya), or on a serological and molecular basis (the tigré strain, PRSV-T).

Several other cucurbit potyviruses (WMV-M, ZYFV and some other only partially characterized potyviruses from Africa) are biologically related to PRSV: they share similar host ranges and might be controlled by the same host plant resistance genes (Quiot-Douine et al., 1990). They are, however, now considered as distinct entities. Resistance to PRSV is available in melon, cucumber and various *Cucurbita* species.

Watermelon mosaic virus 2 (WMV2). WMV2 has been reported for more than 40 years in different parts of the world. WMV2 and PRSV were once thought to be strains of the same virus; now they are recognized as distinct entities based on serological, molecular and biological properties. WMV2 is probably one of the cucurbit viruses whose symptomatology varies the most among cultivars of its host species. It may induce very severe vein banding, green mosaic, deformation and plant stunting in some melon, cucumber, and squash cultivars while it may be almost symptomless in others. WMV2 is prevalent in Mediterranean and temperate regions while it is only rarely detected in tropical areas. Various levels of resistance to WMV2 have been identified in cucumber (Wai and Grumet, 1995), melon (Gilbert et al., 1994), watermelon (Gillaspie and Wright, 1996), and squash. Some of the resistances appear to be strain specific. A good level of resistance has been

Table 5. Virus resistance in *Cucurbita* sp.

Virus	Resistance source[z]	Genetic control	Strain specificity	Commercial cultivar[y]
Comovirus				
SqMV	ECD, OKE	unknown	unknown	no
Cucumovirus				
CMV	PI 176959	unknown	unknown	yes
	Cinderella	2 recessive?	yes	
	OKE	partially dominant	unknown	
	ECD, FOE, CYL, DIG	unknown	unknown	
	Nigerian[x]	partially dominant	unknown	
Nepovirus				
ToRSV	CYL, DIG	unknown	unknown	no
TRSV	Seneca Butterbar	unknown	unknown	no
	Waltham Buttercup[w]			
	ECD, FOE, OKE, CYL			
Potyvirus				
ClYVV	many	unknown	unknown	yes
PRSV	Nigeria[x]	recessive	unknown	no
	ECD, FOE	unknown	unknown	
WMV2	Nigeria[x]	unknown	no	yes
	Menina[x]	Monogenic dominant	no	
	Pai Yu[w]	unknown	unknown	
	ECD, FOE	unknown	unknown	
ZYMV	Nigeria[x]	Monogenic partially dominant	no	yes
	Menina[x]	Monogenic dominant	no	
	ECD	Monogenic dominant	unknown	
Geminivirus				
SqLCV	*C. moschata*	unknown	unknown	no

[z]OKE = *C. okeechobeensis* (= *C. martinezii*); ECD = *C. ecuadorensis*; FCA = *C. ficifolia*; FOE = *C. foetidissima*; CYL = *C. cylindrata*; DIG = *C. digitata*.
[y]Resistances in commercial cultivars are not always derived from sources described in the literature.
[x]*C. moschata*.
[w]*C. maxima*.

obtained in squash and melon using the coat protein mediated resistance approach (Fuchs and Gonsalves, 1995; Fuchs et al., 1997).

Zucchini yellow mosaic virus (ZYMV). ZYMV was the first of a series of cucurbit 'emerging' viruses which threatened the cucurbit industry in different parts of the world in the early 1980s. First identified in Italy and France, ZYMV spread within a decade to the major cucurbit producing regions worldwide. Recently, ZYMV was reported to have colonized new regions or islands (Desbiez and Lecoq, 1997).

ZYMV induces extremely severe mosaic and deformations on leaves and fruits of all cultivated cucurbits. In particular, various "bump" shapes are observed on mature fruits, making them unmarketable. Economic effect of the virus can be dramatic, early infections generally leading to a complete yield loss.

A number of ZYMV variants have been described. Some strains may induce a lethal wilting in melons possessing the *Fn* gene. Other strains will induce cracks on the fruits, or mosaic and hardening of the fruit flesh. One variant of ZYMV inducing very mild symptoms has been found to be very efficient in protecting plants against severe forms of the virus. It is now used as a cross-protecting agent in different parts of the world.

Seed transmission of ZYMV has been reported to occur at very low rate in zucchini squash. Attempts to experimentally observe seed transmission of ZYMV have, however, been generally unsuccessful. This suggests that ZYMV seed transmission is a very rare or erratic event, although it could well be the means by which it was spread throughout the world.

Resistance to ZYMV has been found in cucumber, melon, watermelon, *Cucurbita moschata* and wild *Cucurbita* species. The resistances in melon and watermelon appear to be strain specific. A good level of resistance has been achieved in squash and melon using the coat protein mediated resistance approach (Fuchs and Gonsalves, 1995; Fuchs et al., 1997).

Watermelon mosaic virus Morocco strain (WMV-M). WMV-M was first described in Morocco as a strain of WMV2. Serological and molecular studies revealed later that it is a distinct member of the potyvirus group. WMV-M and closely serologically related strains are widespread in the African continent where it may be one of the most damaging viruses in cucurbits. Symptoms are very severe in cucumber, squash and watermelon, and include mosaic and deformations in leaves and fruits. In most melons cultivars, symptoms are systemic necrotic spots that are often followed by a complete collapse and necrosis of the plant.

WMV-M has been reported from Southern Spain (Quiot-Douine et al., 1990) and more recently from Italy (Roggero et al., 1998). If this virus continues to spread in the Mediterranean basin it could become a major threat to cucurbit production in that region. Resistance to WMV-M has been observed in cucumber (Kabelka and Grumet, 1997) and melon.

Zucchini yellow fleck virus (ZYFV). ZYFV is quite widely spread in the Mediterranean basin, although it is not very common in cultivated cucurbits. In contrast, ZYFV is commonly found in the wild cucurbit *Ecballium elaterium*. Occasionally, severe ZYFV epidemics have been reported in cucumber or watermelon where it induces mosaic symptoms and stunting. In squash, symptoms on leaves are yellow spotting that are much less severe than the symptoms caused by other potyviruses, but fruit deformations are often observed. In melons, symptoms are limited to a few systemic necrotic spots. Resistance to ZYFV has been found in cucumber (Gilbert-Albertini et al., 1995) and melon.

Other potyviruses. Melon vein banding mosaic virus (MVBMV) is a distinct potyvirus reported from Taiwan where it seems to have a limited distribution (Huang et al., 1993). It induces severe mosaic symptoms in melon, squash, cucumber and watermelon. No resistance has been described so far for this virus.

Telfairia mosaic virus (TeMV) is prevalent in *Telfairia occidentalis* in Nigeria. TeMV has not yet been reported in other cucurbit species, although some could be infected following mechanical inoculation.

Clover yellow vein virus (ClYVV) induces pronounced yellow spotting in various *C. pepo* genotypes including 'Yellow Summer Squash' and 'Patisson'. It does not seem to have a signifi-

cant overall incidence on fruit yield in cucurbits. Since ClYVV has a worldwide distribution, special care should, however, be paid not to inadvertently introduce susceptibility to ClYVV during the course of breeding programs.

Cucurbit aphid borne yellows virus (CABYV). CABYV is comprised of small isometric particles circa 25 nm in diameter with a single stranded RNA genome. It was the first luteovirus described naturally infecting cucurbits, and one of several viruses inducing yellowing symptoms in these crops (Lecoq et al., 1992). CABYV has a phloem-limited distribution in plants. Typical symptoms in cucumber, melon, squash and watermelon include yellowing and thickening of the older leaves. There is a wide range of symptom intensity in susceptible cultivars that varies from a yellowing limited to only a few older leaves to a complete discoloration of the plants. Although CABYV doesn't affect fruit quality it may significantly reduce fruit production.

CABYV is transmitted in a persistent manner by at least two aphid species (*Aphis gossypii* and *Myzus persicae*). It also infects a number of weeds that might be reservoirs of the virus. CABYV has been found to be widespread throughout the world. In France, epidemics were observed to be very rapid, probably due to the abundance of vector and virus sources.

Resistance has been found in cucumber, melon and squash (Lecoq et al., 1994).

Viruses transmitted by whiteflies

CRINIVIRUSES. Three viruses which belong to the new Crinivirus Genus, Closteroviridae Family, infect greenhouse and/or field grown cucurbits (Wisler et al., 1998). These are beet pseudo-yellows virus (BPYV), cucurbit yellow stunting disorder virus (CYSDV) and lettuce infectious yellows virus (LIYV). The basis for this new genus is the presence of long, highly flexuous particles, a linear, positive sense single stranded RNA genome that is divided into two molecules, and transmission by whiteflies in a semipersistent manner. A feature that is unique to all members of the Family Closteroviridae described to date is the presence of the heat shock protein HSP70. Closteroviridae express their large genomes via polyprotein processing, translational frameshifting, and production of multiple subgenomic RNAs.

Criniviruses induce pronounced interveinal yellowing symptoms, generally limited to the older leaves. These symptoms are usually confused with physiological and/or nutritional disorders, natural senescence, and even pesticide phytotoxicity. For this reason, crinivirus infections are difficult for growers, diagnosticians, and researchers to recognize. A characteristic feature that distinguishes infection by this group of viruses from the disorders listed above is the thickening and brittleness of symptomatic leaves. Leaves of infected plants "snap" when they are crushed in one's hand. Diagnosis of and distinction between criniviruses infecting cucurbits is based on several different factors. Nucleic acid probes are available to BPYV, CYSDV, and LIYV. Reliable antisera have been produced to LIYV, but not to BPYV or CYSDV. Most of the criniviruses have been found in the past eight years, and more are being found. It is, therefore, important to verify the results from molecular or serological diagnosis with biological tests that require inoculation by whiteflies, both *Bemisia tabaci* and *Trialeurodes* sp. of specific, diagnostic indicator plants.

Beet pseudo-yellows virus (BPYV). BPYV was the first whitefly-transmitted crinivirus described. The greenhouse whitefly (*Trialeurodes vaporariorum*) is the only known vector, and it is one of the most important greenhouse pests throughout the world. BPYV persists in the insect vector for seven days. In addition to cucurbits, BPYV has a wide host range, which includes ornamentals, vegetable crops and weeds. Since its first description in California, BPYV has subsequently been found throughout the world. This is likely due to the international movement of susceptible hosts, in particular vegetatively propagated greenhouse ornamentals, and their resident vectors. BPYV has not been well characterized, and although the particle length is within the range of criniviruses (700 to 950 nm) and its HSP70 gene has been sequenced, two RNA molecules have yet to be identified (Wisler et al., 1998).

BPYV has been responsible for significant

economic losses in cucurbit crops in North America, Europe, and Asia. The increase in greenhouse production of melons and other vegetable crops has provided prime conditions for the greenhouse whitefly vector in areas where it would not normally survive, and for spread of BPYV. Symptoms of BPYV in cucurbits appear first as chlorotic, angular spots on older leaves. The interveinal areas of leaves eventually become completely chlorotic in contrast to the veins that remain green. As infection progresses, the plants become progressively more yellow, although young leaves appear normal.

Several other viruses have been reported to infect cucurbits that may be the same or similar to BPYV: muskmelon yellows virus, cucumber yellows virus, cucumber chlorotic spot virus, melon yellows virus, and cucumber infectious chlorosis virus. Further studies are needed to determine if these are truly different viruses or strains of BPYV.

Cucurbit yellow stunting disorder virus (CYSDV). A disease of cucurbit crops induced by CYSDV was first detected in the United Arab Emirates in 1982, but is now widespread in the Mediterranean Basin (Celix et al., 1996; Hassan and Duffus, 1991). CYSDV causes symptoms indistinguishable from those caused by BPYV. CYSDV can be readily distinguished from BPYV by differences in vector specificity, time of retention in the whitefly vector, host range, and molecular probes.

CYSDV is transmitted efficiently by *B. tabaci* biotype B and relatively inefficiently by *B. tabaci* biotype A, and not at all by the greenhouse whitefly. CYSDV has persisted in its vector for nine days in serial, daily transfers. Although the greenhouse whitefly is found throughout the world, CYSDV has to date only been found in the Old World. This situation could, however, change.

Sources of resistance to CYSDV have been identified in melon (Gomez-Guillamon et al., 1995) and watermelon (Hassan et al., 1991).

Lettuce infectious yellows virus (LIYV). A distinct virus of lettuce, sugarbeet, cucurbits, and other weed and crop hosts, LIYV was found in the desert regions of California and Arizona in 1981 (Wisler et al., 1998). LIYV is type member of the Genus Crinivirus. It is transmitted relatively efficiently by *B. tabaci* biotype A, but very inefficiently by *B. tabaci* biotype B. In contrast to CYSDV, which has been found only in the Middle East and Spain, LIYV was primarily restricted to the desert southwest U.S. Like BPYV, LIYV has a wide host range that includes 45 species in 15 plant families.

LIYV is considered to be a major virus of cucurbits, lettuce and sugarbeets. The infection of Fall melon crops was a significant factor in the LIYV epiphytotics that occurred in the winter lettuce crops in the desert southwest U.S. in the 1980s. Fall melons in this region, which are planted beginning in July through mid-August, served as a major reservoir for LIYV for the Winter lettuce crops, and provided the link between old and new lettuce crops. Melons and cotton were the major hosts for increasing the size of the whitefly population, while melons were the major source of LIYV for lettuce.

The *Bemisia* populations changed from biotype A to biotype B during the 1980s and early 1990s in the sunbelt states of the U.S., and throughout tropical and subtropical zones worldwide. In recent years, LIYV has not been found to any significant degree in the desert southwest U.S. due to the predominance of the B biotype along with its poor efficiency in transmitting LIYV. Instead, lettuce chlorosis virus (LCV), another crinivirus which is transmitted very efficiently by Biotype B, has been found increasingly in these areas. The important difference in the epidemiology of LCV is that, unlike LIYV, cucurbits are not a host for LCV (Wisler et al., 1998).

In spite of this atypical shift in vectors populations, LIYV caused major economic losses due to infections of lettuce, sugarbeets and melons. In 1981 alone, the losses were estimated to be $20 million. Resistance has been identified in melon (McCreight, 1998).

Geminiviruses. Whitefly transmitted geminiviruses infecting cucurbits belong to the subgroup III bipartite members of the Geminiviridae. The geminate particles measure 20 × 30 nm and contain a circular single-stranded DNA molecule consisting of distinct A and B components: Squash leaf curl virus (SCLV) and watermelon chlorotic stunt virus (WmCSV) are transmitted by *B. tabaci* in a persistent manner. The acquisition period for whiteflies is several

hours, followed by a latent period of 12 or more h. After that, the whiteflies can transmit for several weeks, approximately the life of the insect.

Unlike the criniviruses, geminiviruses produce distinctive symptoms with a bright yellow mosaic or mottle and leaf curling, severe stunting, fruit distortion, and yield reduction in susceptible cultivars.

Squash leaf curl virus (SLCV). The original isolate of SLCV described from California infects beans and squashes only. Watermelon curly mottle virus (WCMoV), which may be considered to be a strain of SLCV, has an expanded host range that includes melons and cucumbers (Brown et al., 1995).

Serological and nucleic acid hybridization assays indicate some cross-reactivity between SLCV and other group III geminiviruses, but not with members of subgroup I or II which are leafhopper-transmitted. Molecular probes have been used to diagnose SLCV from field samples, and a monoclonal antibody has been produced which is broadly reactive with SLCV but reacts with some other whitefly-transmitted geminiviruses.

The restricted host range of SLCV permits control of it by implementation of a host-free period and removal of weed reservoirs of the virus and the whitefly vector. Tolerance and resistance to SLCV has been described in several cucurbit species including *Cucurbita moschata*, *C. ficifolia*, and *Luffa* sp. Preliminary studies indicate that resistance developed in *C. pepo* is controlled by a single, dominant gene (Montes-Garcia et al., 1998).

Watermelon chlorotic stunt virus (WmCSV). WmCSV, first described in Yemen, is now causing devastating epidemics in other countries of the Red Sea region (Jones et al., 1988). Symptoms are very severe in watermelon and melons. Once infected, watermelon plants develop only yellow leaves and have very short internodes, which gives them a characteristic look as a result of the old green leaves in the center of the plant and completely yellow and stunted young shoots at the periphery. Infected plants produce only a few misshapen fruits. In melons, symptoms are a bright yellow mosaic or mottle, leaf deformations and stunting.

WmCSV can be readily detected by DAS-ELISA or by acid hybridization assays (such as the 'leaf squash' technique).

Different levels of tolerance to WmCSV have been observed in *Citrullus lanatus* landraces from Sudan (Omara et al., 1997).

Cucumber vein yellowing virus (CVYV). CVYV has not yet been assigned to any plant virus genus. CVYV has rod-shaped particles circa 750 nm long and has been reported to have a double stranded DNA genome. The virus is transmitted by *B. tabaci* in a semipersistent manner. CVYV is common in the southeastern part of the Mediterranean Basin. CVYV infects several cucurbit species, but it is mostly reported from cucumber in which it causes severe vein clearing on young leaves and yellowing on the older leaves. Several commercial cucumber cultivars are resistant to CVYV.

Squash yellow leaf curl virus (SYLCV). SYLCV has been recently described in the Sultanate of Oman (Zouba et al., 1998). SYLCV is transmitted by *B. tabaci* in a semipersistent manner, and has properties similar to those of potyviruses: size and shape of particles (flexuous particles circa 700 to 750 nm long) and typical pinwheels cytoplasmic inclusions. In that respect, SYLCV could be a tentative new member of the Ipomovirus Genus. SYLCV infects only squash and *Luffa* sp., where it induces yellow spots, diffuse veinal yellowing and curling of young leaves.

Viruses transmitted by leafhoppers, beetles and thrips

Beet curly top geminivirus (BCTV). BCTV has geminate particles circa 20 nm x 30 nm and a monopartite single stranded DNA genome. BCTV is transmitted in a persistent manner by leafhoppers (*Circulifer* sp.). It causes severe diseases in cucurbits. Symptoms include leaf curling, distortion, and yellowing. Infected plants are stunted and fruit malformed. BCTV occurs in the eastern Mediterranean Basin and in the U.S., where it once was a major problem in cucurbit crops. Probably due to a better general control of the virus in other crops (such as sugarbeet), BCTV seems to have now a lower economic importance in cucurbits. Different susceptibility levels were reported in squash and melons.

Squash mosaic comovirus (SqMV). SqMV has isometric particles circa 30 nm in diameter, and a divided genome consisting of two single stranded RNA molecules. SqMV is transmitted by several beetle species, mechanically during pruning operations, and also by the seeds. Seed transmission is probably the means by which SqMV has spread throughout the world. Although it infects many cucurbit species, SqMV is economically important mostly in melon and squash. Symptoms on leaves include mosaic, vein banding and deformations. Mosaic may be also very severe on fruits of some melon cultivars. Two major groups of strains have been differentiated based on their biological, serological, or seed transmission properties.

SqMV can be easily detected by serological methods (DAS-ELISA, SDS-ID). Detection of SqMV in seed lots by DAS-ELISA is possible, but does not correlate well with the actual seed transmission rates. Indeed, SqMV seed transmission occurs through embryonic infection; DAS-ELISA can detect the virus in teguments or papery layers of individual seeds while their embryos are not infected. DAS-ELISA diagnostic kits are commercially available for SqMV.

Production of virus-free seeds will prevent early contaminations in the fields and limit virus spread. Some melon and squash cultivars develop only mild symptoms, but these cultivars should not be used in breeding programs if not associated with the absence of seed transmission. Hypersensitive resistance has been observed in the wild species *Cucumis metuliferus*. Coat protein mediated resistance has proved to be efficient in melon.

Watermelon silver mottle tospovirus (WSMV). WSMV has quasispherical enveloped particles 75 to 100 nm in diameter, containing three linear single stranded RNA molecules (Yeh and Chang, 1995; Yeh et al., 1992). Tospovirus genomic organization is complex: the large RNA (L-RNA) has a negative polarity while the two others (S-RNA and M-RNA) use ambisense coding strategies. WSMV is transmitted in a circulative manner by *Thrips palmi*. By analogy with what is known for tomato spotted wilt virus (TSWV), it is anticipated that WSMV could multiply in its vector, and that the virus might be only acquired at the larvae stage. This has yet to be confirmed for WSMV. WSMV has been described in Japan, Taiwan and possibly Brazil. Symptoms in watermelon include severe stunting, mottling, yellow spotting and deformation of leaves, tip necrosis, and dieback. Fruits are malformed and show silver mottling. Severe epidemics have also been observed in melon. No resistance has yet been described for WSMV.

Viruses transmitted by nematodes

Several distinct nepoviruses infect cucurbit crops. They have small isometric particles circa 28 nm in diameter, and a divided genome consisting of two single stranded RNA molecules. These viruses generally have a broad host range outside the cucurbits that includes annual and perennial plants. Each is transmitted by a few specific nematode species; some are also transmitted by seeds. Generally, due to the limited mobility of their vectors, nepoviruses will not develop rapid epidemics, generalized to a whole region. Epidemics can occur and be drastic if cucurbits are grown in soils that are very highly infested by the vectors. Strains have been differentiated based on their biological and serological properties.

Diagnosis can be readily achieved through classical serological methods (DAS-ELISA, standard-ID). DAS-ELISA diagnostic kits are available for the most important nepoviruses infecting cucurbits.

Tobacco ringspot virus (TRSV). TRSV has mostly been reported to infect cucurbits in North America where it is endemic. TRSV has, however, been reported in other crops in different parts of the world where it spread probably through dissemination of infected plant material. It is transmitted by the dagger nematode and related species (*Xiphinema* sp.). TRSV is also seed transmitted in melon and cucumber. Symptoms are severe on newly infected plants (bright yellow mosaic and leaf distortion), then they tend to become milder with a recovery stage. Fruit are distorted and their production is reduced.

Resistance to TRSV has only been reported in cultivated and wild *Cucurbita* sp. and in *Cucumis anguria*.

Tomato ringspot virus (ToRSV). ToRSV has a

similar distribution to TRSV. It is also transmitted by the dagger nematode and related species (*Xiphinema* sp.). Symptoms are severe particularly on squash. Plants react first with a bright yellow mosaic that is followed by a recovery stage. Fruit production is reduced and of poor quality. Symptoms are milder in other cucurbits. Resistance to ToRSV has only been reported in wild *Cucurbita* sp.

Other nepoviruses. Tomato black ring virus (TBRV) is transmitted by *Longidorus* sp. It was reported in cucurbits only in Europe where it has been occasionally associated with severe diseases in squash and cucumber. Artichoke yellow ringspot virus (AYRSV) has been shown to cause a severe stunting and yellow mosaic in cucumber. AYRSV has been reported only from Greece and Italy.

Viruses transmitted by fungi

Melon necrotic spot carmovirus (MNSV). MNSV has small isometric particles circa 30 nm in diameter and a single stranded RNA molecule. MNSV is transmitted by the chytrid fungus, *Olpidium bornovanus*; the virus being carried externally by the fungal zoospores. Alternatively, MNSV can be transmitted mechanically through pruning operations and possibly by some chewing insects. MNSV was found in the U.S., Europe, the Mediterranean Basin and Japan; it has a host range mostly limited to cucurbits. MNSV has been reported naturally infecting melon, cucumber and watermelon. Typical symptoms are systemic necrotic spots, streaks on petioles and stems that may eventually lead to the plant necrosis. The intensity of symptoms varies greatly according to the cultivar and the season. In Europe, the more severe symptoms are often observed in spring and fall crops. Strains have been differentiated on symptomatological or serological basis.

Recently, MNSV has been shown to be seed transmitted (Campbell et al., 1996). The mechanism involved has been called vector-assisted seed transmission. The virus is carried on the seed (probably in the seed coat or papery layers), released in the soil during germination, acquired and then introduced to the plant by zoospores of the fungus that are present in the potting soil used for the seedlings. Since *O. bornovanus* is a very resistant and ubiquitous fungus, this original seed transmission process may be very efficient, and has probably contributed to dissemination of MNSV throughout the world. MNSV diagnosis can be easily achieved through biological assays (local lesions in melon or cucumbers within 7 days) or by DAS-ELISA. DAS-ELISA diagnostic kits are commercially available for MNSV. Resistance has been identified in melon.

Tobacco necrosis virus (TNV). TNV has small isometric particles circa 26 nm in diameter; its genome is a single stranded RNA molecule. TNV is transmitted by the chytrid fungus *O. brassicae* and is carried externally by the zoospores. TNV has a very wide experimental host range, and is present worldwide. It has been found occurring naturally in cucumber and squash grown in glasshouses or plastic tunnels. Symptoms consist of necrotic spots that may coalesce and lead to the complete drying of the leaves or of the whole plant.

TNV diagnosis can be easily achieved through biological assays (local lesions in various hosts within 1 to 2 days) or by DAS-ELISA. DAS-ELISA diagnostic kits are commercially available for TNV. No resistance has been described in cucurbits.

Other viruses transmitted by fungi. Several other cucurbit viruses have been shown to be transmitted by *O. bornovanus*. They belong either to the tombusvirus or carmovirus groups. They generally have limited areas of distribution, and only occasionally do they cause damage to crops. If the seed transmission mechanism described for MNSV is also efficient for these viruses, larger dissemination could eventually occur through germplasm exchange. All these viruses induce necrotic symptoms similar to those observed for MNSV or TNV. Cucumber necrosis tombusvirus (CNV) has been reported in Canada, cucumber leaf spot carmovirus (CLSV) in Europe, squash necrosis virus (SqNV) in Brazil, and cucumber soilborne carmovirus (CuSBV) in Lebanon. These viruses are transmitted only by some host specific strains of *O. bornovanus*, except CNV which has, as MNSV, been transmitted by all *O. bornovanus* strains tested so far (Campbell et al., 1995). No resistance has been described for these viruses.

Viruses with unknown vectors

Cucumber green mottle mosaic tobamovirus

(CGMMV). CGMMV has rod-shaped particles circa 300 nm long and 18 nm in diameter , and a single stranded RNA genome. It is easily transmitted mechanically (handling of the plants, leaf contact), through soil or substrate contamination, and by seeds in cucumber, watermelon and bottlegourd. CGMMV contamination of the seeds seems to be mostly external. The virus can be eliminated from seeds by dry heat treatment at 70 °C for 3 days without impairment of seed germination rates. CGMMV induces mosaic of varying intensity on the leaves; fruit are often reduced in size and deformed. CGMMV has been reported in Europe and Asia.

Control of CGMMV includes the use of virus-free seeds, careful cleaning of the greenhouse structure before planting, and disinfection of pruning tools. CGMMV can be easily detected by standard serological tests. DAS-ELISA diagnostic kits are commercially available for CGMMV. No resistance has been found in cucumber, except for some genotypes that do not develop foliar symptoms. Resistance has been found in melon and in *C. anguria*.

MELON RUGOSE MOSAIC AND CHAYOTE MOSAIC TYMOVIRUSES (MRMV AND CHMV). MRMV and ChMV are recently described tymoviruses for which no vector has yet been identified. They have isometric particles circa 30 nm and a single stranded RNA genome. They have at present restricted distribution areas: MRSV being reported only from the Red Sea region, and ChMV from Central America (Hord et al., 1997). MRMV has a restricted host range within the cucurbits, and reaches very high titers in infected plants. MRMV and ChMV produce severe mosaic symptoms in their natural hosts, melon and *Secchium edule*, respectively. MRMV is seed transmitted in melon (Mahgoub et al., 1997).

MRMV and ChMV can be easily detected through standard serological procedures (DAS-ELISA, standard ID). Resistance to MRMV has been found in melon (Mahgoub et al., 1997).

CUCUMBER TOAD SKIN RHABDOVIRUS (CTSV). CTSV has enveloped, bullet-shaped virus particles circa 100 to 400 nm long and 50 to 100 nm in diameter. It could be a strain of eggplant mottled dwarf virus (EMDV). CTSV induces very severe vein yellowing, leaf curling and plant stunting in cucumber. Fruits show severe mosaic, cracks and deformations. Attempts to transmit the CTSV by aphids have failed. CTSV occurs in the Mediterranean Basin. It is relatively widespread, but it generally infects only few plants (less than 10 %) within a field. CTSV has a limited host range within the cucurbits, but relatively wide host range outside of cucurbits.

MELON OURMIA OURMIAVIRUS (OUMV). OuMV is the type member of the Ourmiavirus Genus. It was isolated from a melon plant from Iran, the only country where this virus has been reported to date. Particles have a bacilliform shape 30 to 37 × 18 nm, and contain three single stranded RNA molecules. OuMV induces chlorotic spots and irregular ringspots in melon leaves. It infects several *Cucumis* sp., and has a wide host range outside of cucurbits.

Conclusions

This overview on viruses infecting cucurbits highlights the diversity and sometimes the severity of the diseases caused by this complex pathosystem. Cucurbit viruses are transmitted by vectors that are strikingly different in their ecology and ability for dissemination of viruses. Understanding virus–vector relationships is of major importance not only for monitoring the progress of epidemics, but also for developing prophylactic control measures.

Cucurbit virus control methods can be divided according to their specificities. Some are applicable to all viruses (use of virus-free seeds or healthy seedlings, elimination of virus or vector reservoirs), other will be efficient for viruses that have the same vectors and virus–vector relationships (plastic mulching, insect-proof nets), and some will be specific for only one virus (cross protection).

Breeding for resistance is highly specific, and may sometimes be efficient for only some strains of one virus. Important efforts have been made in recent years by virologists and plant breeders to identify sources of resistance in germplasm collections. It is worth noting that some resistances are used by plant breeders and resistant cultivars are released without publication of the source or the inheritance of resistance. For instance, many CVYV-resistant cucumber cultivars have been released in the Middle East but there is no description of this resistance.

Interestingly, in many instances a single accession has provided resistances to several different viruses. This is the case in melons where PI 414723 is

resistant to CABYV, PRSV, ZYMV and *A. gossypii* (Pitrat et al., 1996), in squash where *Cucurbita moschata* 'Nigeria' is resistant to CMV, PRSV, WMV2 and ZYMV (Provvidenti, 1986), and in cucumber where 'Taichung Mou Gua' is resistant to CABYV, CMV, PRSV, WMV2, WMV-M, ZYFV and ZYMV (Gilbert-Albertini et al., 1995; Kabelka and Grumet, 1997; Lecoq et al., 1994; Provvidenti, 1986).

Some of the resistances to potyviruses are conferred by one gene or very closely linked genes: resistance to ZYMV, ZYFV and WMV-M in the cucumber 'Taichung Mou Gua', and resistance to ZYMV and WMV2 in *C. moschata* 'Menina'. In other cases, linkages are observed between genes involved in resistance to different viruses: for instance CMV and MNSV in melon. The genetic maps of cucurbits are not yet accurate enough to determine if there is a linkage or only one gene, particularly in the case of oligogenic resistances. Progress must be made in this area. Where it is confirmed that some genes are involved in the control of very different viruses, they should be cloned in order to permit study of their functions.

Screening efforts should be continued to diversify resistance genes in order to provide high levels of resistance and prevent selection of more virulent strains. If sources of resistance are quite abundant for aphid-borne viruses, they still remain scarce for viruses transmitted by other vectors. The increase in the number of economically important cucurbit viruses emphasizes the need for breeding for multiple resistance (Kyle, 1995).

Transgenosis provides a new avenue for creating virus-resistant cultivars. Different strategies have been proved promising in cucurbits: coat protein mediated resistance and ribozymes. The possibility of creating transgenic plants with multiple virus resistance is particularly interesting for cucurbits in which mixed infections often occur in open fields. In the future, classical breeding and biotechnology will probably combine their efforts to construct composite resistances that provide resistance to a broad spectrum of viruses and be more durable.

Some cucurbit viruses (the 'classics') have been recognized for some time as and still are major cucurbit pests. Such are CMV, PRSV and WMV2 on a worldwide basis, or TRSV, BCTV and MNSV in certain countries. Other viruses (the 'emerging') have appeared as major problems only recently, and have spread rapidly in the crops. Such are ZYMV or BPYV at a worldwide level or LIYV, CYSDV or SLCV at a regional level. The severity of the symptoms caused by these viruses rules out the possibility that they might have been present in the crops but unnoticed before their discovery. The hypothesis of a *'de novo'* sudden occurrence of these viruses, after a recombination event for instance, has not been proven for any of these viruses. More likely is the possibility that these viruses were already occurring in restricted ecological niches, where they remained unnoticed. They might have spread after their accidental introduction into important cucurbit growing regions or after the expanded distribution of their vectors. Changes in cultural practices, and in particular the development of protected crops (plastic tunnels) may offer more facilities to viruses or to their vectors that permit them to reach high populations year-round.

Some viruses presently having restricted geographical distributions have the potential to become widely spread if they have an efficient vector and good ecological fitness. Such is the case for WmCSV, which is presently limited to the Red Sea region; it might spread in the coming years to the whole Mediterranean Basin region where *B. tabaci* is prevalent. This occurred with tomato yellow leaf curl geminivirus in tomato, and CYSDV in cucurbits during the last decade. WMV-M, which is presently spreading in the Mediterranean Basin, could also be as 'successful' as ZYMV was in the early 1980s.

These considerations emphasize the need for increased work on virus diagnosis, including for those viruses that are not yet widespread. In particular, it would be beneficial to have a better knowledge of the viruses that occur in centers of origin and diversification of the major cucurbit species where some of the future 'emerging' cucurbit viruses may already occur. International cooperation for production and exchange of antisera or molecular probes is essential in order to conduct regular surveys to keep the knowledge of the local virus pathosystem compositions up to date. An efficient international cooperation and the development of simple diagnostic methods (DAS-ELISA, ID-SDS) contributed greatly to the identification of ZYMV on a

worldwide basis within a few years. The development of commercially available DAS-ELISA kits provides a means to develop more accurate evaluations of the virus situations in different countries, and will aid virus resistance breeding programs. There are still major viruses for which efforts have to be continued to develop simple serological diagnostic methods.

There is also a need for a better understanding of the viral pathogenicity determinants and of the mechanisms of virus evolution. This knowledge will permit us to elucidate virus resistance mechanisms at the molecular level. It will also contribute to understanding the processes leading to the occurrence of new virus strains able to infect resistant plants. Altogether, this will help to evaluate durability of newly found or created resistance genes, and in the future, will contribute to construction of 'composite' resistances that will not be easily overcome by 'emerging' virulent virus strains.

Literature cited

Blancard, D., H. Lecoq, and M. Pitrat. 1991. Maladies des cucurbitacées. Observer, identifier, lutter. PHM and INRA-Editions, Paris, France.

Brown, J.K., K. Wendt, and S.D. Wyatt. 1995. Genetic variability of Squash Leaf Curl Virus isolates by DNA hybridization and component-specific polymerase chain reaction, p. 5–11. In: G.E. Lester and J.R. Dunlap (eds.). Cucurbitaceae '94: Evaluation and Enhancement of Cucurbit Germplasm, 1–4 Nov. 1994. South Padre Island, Texas. Gateway Printing, Edinburg, Texas.

Brunt, A.A., K. Crabtree, M.J. Dallwitz, A.J. Gibbs, and L. Watson. 1996. Viruses of plants. CAB International. Wallingford, Oxon, U.K.

Campbell, R.N., S.T. Sim, and H. Lecoq. 1995. Virus transmission by host-specific strains of *Olpidium bornovanus* and *Olpidium brassicae*. European J. Plant Pathol. 101:273–282.

Campbell, R.N., C. Wipf-Scheibel, and H. Lecoq. 1996. Vector-assisted seed transmission of melon necrotic spot virus in melon. Phytopathology 86:1294–1298.

Celix, A., A. Lopez-Sese, N. Almarza, M. Gomez-Guillamon, and E. Rodriguez-Cerezo. 1996. Characterization of cucurbit yellow stunting disorder virus, a *Bemisia tabaci*-transmitted Closterovirus. Phytopathology 86:1370–1376.

Clark, M.F. and A.N. Adams. 1977. Characteristics of microplate method of enzyme-linked immunosorbent assay for the detection of plant viruses. J. Gen. Virol. 34:475–483.

Desbiez, C. and H. Lecoq. 1997. Zucchini yellow mosaic virus. *Plant Pathol.* 46:809–829.

Fuchs, M. and D. Gonsalves. 1995. Resistance of transgenic hybrid squash ZW-20 expressing the coat protein genes of zucchini yellow mosaic virus and watermelon mosaic virus 2 to mixed infections by both potyviruses. Bio/Technology 13:1466–1473.

Fuchs, M., J. McFerson, D. Tricoli, J. McMaster, R. Deng, M. Boeshore, J. Reynolds, P. Russell, H. Quemada, and D. Gonsalves. 1997. Cantaloupe line CZW-30 containing coat protein genes of cucumber mosaic virus, zucchini yellow mosaic virus, and watermelon mosaic virus-2 is resistant to these aphid-borne viruses in the field. Mol. Breeding 3:279–290.

Montes-Garcia, C.E., S. Garza-Ortega, and J.K. Brown. 1998. Inheritance of the resistance to squash leaf curl in *Cucurbita pepo* L. Cucurbitaceae '98. p. 328–330.

Gilbert, R.Z., M.M. Kyle, H.M. Munger, and S.M. Gray. 1994. Inheritance of resistance to watermelon mosaic virus in *Cucumis melo* L. HortScience 29:107–110.

Gilbert-Albertini, F., M. Pitrat, and H. Lecoq. 1995. Inheritance of resistance to Zucchini Yellow Fleck Virus in *Cucumis sativus* L. HortScience 30:336–337.

Gillaspie, A.J. and J. Wright. 1996. Evaluation of *Citrullus lanatus* germplasm for resistance to watermelon mosaic virus 2 strains. Ga. Agr. Expt. Sta. Res. Rpt. 638.

Gomez-Guillamon, M.L., J.A. Tores, C. Soria, and A.I.L. Sese. 1995. Screening for resistance to *Sphaerotheca fuliginea* and to two yellowing diseases in *Cucumis melo* and related *Cucumis* species, p. 205–208. In: G.E. Lester and J.R. Dunlap (eds.). Cucurbitaceae '94: Evaluation and Enhancement of Cucurbit Germplasm, 1–4 Nov. 1994. Gateway Printing, Edinburg, Texas.

Gonsalves, D., P. Chee, R. Provvidenti, R. Seem, and J.L. Slightom. 1992. Comparison of coat protein-mediated and genetically derived resistance in cucumber to infection by cucumber mosaic virus under field conditions with natural challenge inoculations by vectors. Bio/Technology 10:1562–1570.

Hassan, A.A. and J.E. Duffus. 1991. A review of a yellowing and stunting disorder of cucurbits in the United Arab Emirates. Emirates J. Agr. Sci. 2:1–16.

Hassan, A.A., N.E. Quronfilah, U.A. Obaji, M.A. Al-Rays, and M.S. Wafi. 1991. Screening of domestic and wild *Citrullus* germplasm for resistance to the yellow stunting disorder in the United Arab Emirates. Cucurbit Genetic Coop. Rpt. 14:98–101.

Hord, M., W. Villalobos, A.V. Macaya-Lizano, and C. Rivera. 1997. Chayote mosaic, a new disease in *Sechium edule* caused by a tymovirus. Plant Dis. 81:374–378.

Huang, C.H., L. Chang, and J.H. Tsang. 1993. The partial characterization of Melon Vein-banding Mosaic Virus, a newly recognized virus infecting cucurbits in Taiwan. Plant Pathol. 42:100–107.

Jones, P., M.H.A. Sattar, and N. Al Kaff. 1988. The incidence of virus disease in watermelon and sweetmelon crops in the People Democratic Republic of Yemen and its impact on cropping policy. Aspects Appl. Biol. 17:203–207.

Kabelka, E. and R. Grumet. 1997. Inheritance of resistance to the Moroccan watermelon mosaic virus in the cucumber line TMG-1 and cosegregation with zucchini yellow mosaic virus resistance. Euphytica 95:237–242.

Kyle, M. 1995. Breeding cucurbits for multiple disease resistance, p. 55–59. In: G.E. Lester and J.R. Dunlap (eds.). Cucurbitaceae '94: Evaluation and Enhancement of Cucurbit Germplasm, 1–4 Nov. 1994. Gateway Printing, Edinburg, Texas.

Lecoq, H., D. Bourdin, C. Wipf Scheibel, M. Bon, H. Lot, O. Lemaire and E. Herrbach. 1992. A new yellowing disease of cucurbits caused by a luteovirus, cucurbit aphid borne yellows virus. Plant Pathol. 41:749–761.

Lecoq, H., F. Gilbert-Albertini, C. Wipf-Scheibel, M. Pitrat, D. Bourdin, H. Belkhala, N. Katis, and M. Yilmaz. 1994. Occurrence of a new yellowing disease of cucurbits in the Mediterranean Basin caused by a luteovirus, Cucurbit Aphid-Borne Yellows Virus, and prospects for control, p. 461–463. In: 9th Congress of the Mediterranean Phytopathological Union. Turkish Phytopathological Society. Kusadasi-Aydin, Turkey.

Lovisolo, O. 1980. Virus and viroid diseases of cucurbits. Acta Hort. 88:33–82.

Mahgoub, H., C. Wipf-Scheibel, B. Delecolle, M. Pitrat, G. Dafalla, and H. Lecoq. 1997. Melon rugose mosaic virus: characterization of an isolate from Sudan and seed transmission in melon. Plant Dis. 81:656–660.

McCreight, J.D. 1998. Resistance to lettuce infectious yellows virus in melon. HortScience 33:533.

Medley, T. 1994. Availability of determination of nonregulated status for virus resistant squash. Fed. Reg. 59:64187–64188.

Nameth, S.T., J.A. Dodds, A.O. Paulus, and F.F. Laemmlen. 1986. Cucurbit viruses of California: an ever-changing problem. Plant Dis. 70:8–11.

Omara, S., R. Mohielden, G. Dafalla, H. Lecoq, A. Kheyr Pour, and B. Gronenborn. 1997. Resistance to watermelon chlorotic stunt geminivirus in local and exotic germplasm, p. 196. In: W. Khoury and B. Bayaa (eds.). 6th Arab Congress of Plant Protection. Beirut, Lebanon.

Pitrat, M. 1998. Gene list for *Cucumis melo* L. Cucurbit Genet. Coop. Rpt. 21:69–81.

Pitrat, M., G. Risser, F. Bertrand, D. Blancard, and H. Lecoq. 1996. Evaluation of a melon collection for disease resistances, p. 49–58. In: M.L. Gómez-Guillamón, C. Soria, J. Cuartero, J.A. Torés, R. Fernández-Muñoz (eds.). Cucurbits towards 2000. Proceedings of the VIth Eucarpia Meeting on Cucurbit Genetics and Breeding, 28–30 May, Malaga, Spain.

Plages, J.N. 1997. L'avenir des variétés génétiquement modifiées pour la résistance aux virus (un exemple développé par Limagrain). Comptes-rendus de l'Académie d'Agriculture de France 83:161–164.

Provvidenti, R. 1986. Viral diseases of cucurbits and sources of resistance. Food & Fert. Technol. Cntr., Taiwan, Tech. Bul.

Provvidenti, R. 1990. Viral diseases and genetic sources of resistance in *Cucurbita* species, p. 427–435. In: D. M. Bates, R. W. Robinson, and C. Jeffrey (eds.). Biology and utilization of Cucurbitaceae. Cornell Univ. Press, Ithaca, N.Y.

Provvidenti, R. 1993. Resistance to viral diseases of cucurbits, p. 8–43. In: M.M. Kyle (ed.). Resistance to viral diseases of vegetables: Genetics and breeding. Timber Press, Portland, Ore.

Provvidenti, R. 1996. Diseases caused by viruses, p. 37–45. In: T.A. Zitter, D.L. Hopkins, and C.E. Thomas (eds.). Compendium of cucurbit diseases. APS Press, St. Paul, Minn.

Provvidenti, R. and R.O. Hampton. 1992. Sources of resistance to viruses in the Potyviridae. Arch. Virol. Suppl. 5:189–211.

Purcifull, D.E., G.W. Simone, C.A. Baker, and E. Hiebert. 1988. Immunodiffusion tests for six viruses that infect cucurbits in Florida. Proc. Fla. State Hort. Soc. 101:400–403.

Quiot, J.B., G. Labonne, and L. Quiot-Douine. 1983. The comparative ecology of cucumber mosaic virus in Mediterranean and tropical regions, p. 177–183. In: R.T. Plumb and J.M. Tresh (eds.). Plant virus epidemiology. Blackwell Scientific Publications. Oxford, U.K.

Quiot-Douine, L., H. Lecoq, J.B. Quiot, M. Pitrat, and G. Labonne. 1990. Serological and biological variability of virus isolates related to strains of papaya ringspot virus. Phytopathology 80:256–263.

Roggero, P., G. Dellavalle, and V. Lisa. 1998. First Report of Moroccan Watermelon Mosaic Potyvirus in Zucchini in Italy. Plant Dis. 82:351.

Tricoli, D., K. Carney, P. Russell, J. McMaster, D. Groff, K. Hadden, P. Himmel, J. Hubbard, M. Boeshore, J. Reynolds, and H. Quemada. 1995. Field evaluation of transgenic squash containing single or multiple virus coat protein gene constructs for resistance to cucumber mosaic virus, watermelon mosaic virus 2, and/or zucchini yellow mosaic virus. Bio/Technology 13:1458–1465.

Wai, T. and R. Grumet. 1995. Inheritance of resistance to watermelon mosaic virus in the cucumber line TMG-1: Tissue-specific expression and relationship to zucchini yellow mosaic virus resistance. Theor. Appl. Genet. 91:699–706.

Wehner, T.C. 1997. 1997 Gene list for cucumber. Cucurbit Genet. Coop. Rpt. 20:66–88.

Wisler, G.C., J.E. Duffus, H.-Y. Liu, and R.H. Li. 1998. Ecology and epidemiology of whitefly-transmitted closteroviruses. Plant Dis. 82:270–280.

Yeh, S.D. and T.F. Chang. 1995. Nucleotide sequence of the N gene of watermelon silver mottle virus, a proposed new member of the genus tospovirus. Phytopathology 85:58–64.

Yeh, S.D., Y.C. Lin, Y.H. Cheng, C.L. Jih, M.J. Chen, and C.C. Chen. 1992. Identification of tomato spotted wilt-like virus on watermelon in Taiwan. Plant Dis. 76:835–840.

Zouba, A., M. Lopez, and H. Anger. 1998. Squash yellow leaf curl virus: a new whitefly-transmitted poty-like virus. Plant Dis. 82:475–478.