Resistance to Cucurbit Yellowing Stunting Disorder Virus (CYSDV) in *Cucumis melo* L.

A.I. López-Sesé and M.L. Gómez-Guillamón¹

Experimental Station 'La Mayora', C.S.I.C., 29750, Algarrobo-Costa, Málaga, Spain

Additional index words. genetics, germplasm, inheritance, resistance gene, whitefly, *Bemisia tabaci*

Abstract. Forty-four accessions of *Cucumis melo* L. and related wild species were tested for reaction to a yellowing disease, incited by the recently identified cucurbit yellowing stunting disorder virus (CYSDV), under natural and controlled-inoculation conditions. The *C. melo* TGR-1551 accession and one *Cucumis metuliferus* Naud. accession were asymptomatic. The segregation ratios obtained following controlled inoculations of the family produced by crossing TGR-1551 with the susceptible Spanish cv. Piel de Sapo revealed that the resistance to CYSDV in TGR-1551 is conditioned by a dominant allele at one locus. The name *Cucurbit yellow stunting* and symbol *Cys* is proposed for this locus.

In southeast Spain, and in other Mediterranean countries, melon (*Cucumis melo*) crops grown in plastic greenhouses are severely affected by a yellowing disease; the causal agent was recently identified as the cucurbit yellowing stunting disorder virus (CYSDV), transmitted specifically by *Bemisia tabaci* Gennadius (Célix et al., 1996). Symptoms of this disease characteristically start with the appearance of interveinal chlorotic spots on the oldest leaves. These spots enlarge and eventually fuse together and the entire leaf, except the veins, becomes yellow (Gómez-Guillamón and Camero, 1993). Fruit number and weight are seriously reduced, and fruit quality is affected by this disease with a 30% to 40% reduction in yield.

The use of genetic resistance against pathogens is an effective strategy to limit the incidence of plant diseases (Browning, 1980); for viral diseases it is, in some cases, the only strategy (Fraser, 1990; Gray and Moyer, 1993). No resistance to CYSDV has been reported in melon, and chemical treatments against its vector do not eliminate the risk of spreading the disease because of the efficiency of *B. tabaci* in transmitting the virus (Duffus, 1995; López-Sesé, 1997).

To identify sources of resistance against the causal agent of the yellowing disease, several accessions of *C. melo* and related wild species were evaluated under natural- and controlled-infection conditions. Inheritance of resistance was determined for one *C. melo* accession.

Received for publication 11 Dec. 1998. Accepted for publication 1 June 1999. This work was financed by the CICYT project AGF92-0456-CO201. We also wish to thank Mr. David W. Schofield for translating the manuscript. The cost of publishing this paper was defrayed in part by the payment of page charges. Under postal regulations, this paper therefore must be hereby marked advertisement solely to indicate this fact.

¹To whom reprint requests should be addressed. E-mail: guillamon@eelm.csic.es

Materials and Methods

Evaluations of resistance against yellowing disease under conditions of natural infection. Forty-two accessions of *C. melo* from Spain and other countries, one accession of *Cucumis metuliferus* Naud., and one accession of *Cucumis anguria* L. var. *longipes* (Table 1) were grown in a polyethylene greenhouse at the Experimental Station 'La Mayora'. The melon cv. Piel de Sapo (*C. melo* ssp. *melo* Inodorus group) was used as a yellowing-susceptible control. Plants were grown in sandy soil typically used for greenhouse melon and vegetable production in southern Spain, with drip irrigation, and were trained for growth on a net. The sidewall windows of the greenhouse remained open during the test in order to permit free entry and unrestricted access of whiteflies (*Bemisia tabaci* Gennadius) to the plants. The planting frame was 1 × 0.8 m² per plant. Two replications, both with a randomized layout of the 42 accessions, were used, and 10 plants of each accession were evaluated in each replication.

Yellowing incidence was recorded just before fruit harvest, when the disease was extensively established in the crop. Both presence or absence of yellowing and severity were recorded using a four-point scale: 0 = no symptoms; 1 = slight yellowing; 2 = moderate yellowing; 3 = severe yellowing. Whitefly infestation per plant was recorded according to the following scale: 0 = no whiteflies, 1 = a few individuals on most of the leaves, 2 = whiteflies covering less than a half of the leaf on most of the leaves, 3 = a high number of whiteflies covering more than a half of the abaxial leaf surface on most leaves.

Evaluations of resistance against yellowing disease using controlled inoculation. Those accessions whose plants showed no yellowing symptoms in response to natural infection were then tested for resistance to yellowing under controlled inoculation. Seeds of each

accession were planted in plastic pots inside a whitefly-proof glass greenhouse. Five seedlings of each accession were selected and inoculated when the first true leaves were fully expanded.

Inoculations were carried out with *B. tabaci* biotype B by using leaf cages, following the method of López-Sesé (1997). The source of inoculum was 'Piel de Sapo' plants that showed characteristic yellowing symptoms. This inoculum source was the same used for the isolation and identification of CYSDV (Célix et al., 1996).

Non-viruliferous whitefly colonies obtained from healthy tomato plants were maintained on melon plants in cages covered with whitefly-proof netting within a closed, air-conditioned glass greenhouse. For inoculation with CYSDV, groups of 60 whiteflies were allowed a 24-h acquisition feed before a 48-h inoculation period. Five plants of each accession were inoculated and, after inoculation, were placed in a whitefly-proof cage within an air-conditioned glass greenhouse. Fifteen days post-inoculation, the leaves on which the whiteflies had fed and laid their eggs were removed. Presence or absence of symptoms, and the date that symptoms first appeared, were recorded twice weekly for 4 weeks.

Evaluation of resistance by grafting. Grafting is an efficient method of transmission of CYSDV (López-Sesé, 1997) and was performed with those accessions that showed no yellowing symptoms in the controlled-inoculation test. This approach would distinguish resistance to the virus per se from resistance to transmission by *B. tabaci*. 'Piel de Sapo' plants that showed obvious yellowing symptoms were used as virus-infected stocks on which were grafted scions from putative-resistant *C. melo* TGR-1551 and *C. metuliferus*, or healthy plants of 'Piel de Sapo' as the susceptible control. In addition, five plants of each of these accessions were grafted onto non-inoculated healthy melon plants that served as virus-free controls. All plants were kept in whitefly-proof cages within an air-conditioned glass greenhouse.

Determination of the genetics of resistance against CYSDV. To study the genetics of resistance, one of the accessions that demonstrated resistance to CYSDV was crossed with the susceptible 'Piel de Sapo' by manual pollinations to generate families P₁, P₂, F₁, F₂, BC_s (backcross to the susceptible parent) and BC_r (backcross to the resistant parent).

Twelve seedlings of each parent (P₁ and P₂) and F₁, 120 seedlings of the F₂ generation, and 40 plants of each of the backcrosses (BC_s and BC_r) were inoculated with *B. tabaci* by using leaf cages, following the method of López-Sesé (1997). The source of inoculum of CYSDV was 'Piel de Sapo' plants that showed characteristic yellowing symptoms. Inoculations were done as described above. As controls, five non-inoculated plants of each of the parents, F₁, F₂, and backcrosses were kept under the same conditions during the inoculation period. Afterwards, they were transferred to a separate, protected, glass greenhouse to await the appearance of symptoms. After 3 weeks, the inoculated plants that showed no

yellowing symptoms were subjected to a second round of inoculations to reduce the chances of escapes.

The data were analysed by the χ^2 statistical test (Sokal and Rohlf, 1981) with Yates correction in those cases where the expected number in a class was less than five. The genetic models that conformed most closely to the results were subjected to tests of contrast.

Results

Evaluations of resistance against yellowing disease under conditions of natural infection. Under natural inoculation conditions, all the plants of the susceptible 'Piel de Sapo' showed clear symptoms of yellowing. None of the plants of *C. melo* accessions PI 414723, Kogane 9-G-0 Makuwa, Kanro Makuwa, Ginsen Makuwa, TGR-1551, TGR-1920, TGR-1937, and TGR-554, or of the wild species *C. metuliferus* and *C. anguria* var. *longipes*, showed yellowing symptoms (Table 1). Population levels of *B. tabaci* were rated 1 on 26 accessions, 1–2 on 16 accessions, and 2 on one accession (Table 1).

Evaluations of resistance against yellowing disease using controlled inoculation. Under controlled-inoculation conditions, all 'Piel de Sapo' plants developed characteristic yellowing symptoms. No serological or nucleic acid techniques were used to confirm infection, but the symptoms in the inoculated plants were similar to symptoms shown by the plant sources of the inoculum identified as CYSDV (Célix et al., 1996). Some 80% or more of the plants of PI 414723, Kogane 9-G-0 Makuwa, Ginsen Makuwa, and *C. anguria* var. *longipes* developed yellowing symptoms, while 60% of the plants of TGR-554 and TGR-1920 exhibited yellowing symptoms (Table 2). Thirty-six percent of Kanro Makuwa plants developed symptoms. Only 20% of the TGR-1937 accession showed yellowing symptoms (Table 2), and in this case, the symptoms were milder; only small areas of the leaf surfaces were affected. Symptoms appeared 30 d after inoculation with CYSDV in all the accessions except TGR-1920 and TGR-1937, when the yellowing symptoms appeared 40 and 45 d after inoculation, respectively (Table 2).

None of the inoculated Zimbabwean TGR-1551 or *C. metuliferus* accession plants showed yellowing symptoms (Table 2). Symptoms were not observed on susceptible melons when asymptomatic plants of TGR-1551 and *C. metuliferus* were used as sources of inoculum.

Evaluation of resistance by grafting. None of the TGR-1551 plants or *C. metuliferus* plants grafted onto infected plants of 'Piel de Sapo', showed yellowing symptoms. Healthy 'Piel de Sapo' plants grafted on infected plants, however, showed obvious symptoms of yellowing disease.

Determination of the genetics of resistance against CYSDV. None of the plants of the resistant TGR-1551 parental or of the F_1 inoculated with CYSDV showed yellowing symptoms. All plants of the susceptible 'Piel de Sapo' showed clear symptoms of yellowing that started to appear 24 d after inoculation

Table 1. Evaluation of accessions of *Cucumis melo* and other *Cucumis* species for resistance to yellowing disease following natural inoculation by whiteflies in a plastic greenhouse

Nº Reg. ^z	Accession	Origin	Resistance to:	
			CYSDV ^y	Whitefly ^x
C-2	Tendral negro	Spain	2	1–2
C-5	De olor antiguo	Spain	1	1
C-8	Negro	Spain	1	1
C-14	Redondo amarillo	Spain	2	1–2
C-20	Amarillo Soto	Spain	2	1
C-21	Amarillo Domingo	Spain	1	2
C-23	Verde de Holguera	Spain	2	1
C-40	Shiroubi Okayama	Japan	1	1
C-41	Freeman's cucumber	Japan	1	1
C-56	De tajadas señaladas	Spain	2–3	1
C-68	Casero rayado	Spain	1	1–2
C-69	Mollerusa-1	Spain	2	1
C-75	Mollerusa-7	Spain	1	1–2
C-87	<i>C. melo</i> ssp. <i>agrestis</i>	UPV ^v	1	1
C-105	TGR-1551	Zimbabwe	0	1
C-108	Amarillo Onteniente	Spain	2	1–2
C-109	Amarillo Santo Tomé	Spain	1	1–2
C-111	Bolas	Spain	1	1
C-113	Amarillo exportación	Spain	2	1–2
C-117	TGR-1920	Zimbabwe	0	1
C-122	Mochuelo	Spain	1	1
C-124	TGR-1937	Zimbabwe	0	1
C-125	TGR-554	Zimbabwe	0	1
C-133	Ardales-5	Spain	2	1–2
C-136	Ardales-7	Spain	1	1
C-142	Ardales-11-1	Spain	1	1
C-145	Ardales-14	Spain	2	1–2
C-151	Ardales-19-1	Spain	1	1
C-157	PI 414723	India	0	1
C-185	India CUM-225	India	1	1–2
C-209	Temprano Roget	Spain	1	1–2
C-212	Kogane 9-G-0 Makuwa	Japan	0	1
C-213	Kanro Makuwa	Japan	0	1
C-214	Nagata Kim Makuwa	Japan	1	1
C-215	Ginsen Makuwa	Japan	0	1–2
C-269	PI 157084	China	0	1
C-276	PI 124111 2 B	India	1	1–2
C-280	NR8996	Libia	2	1
C-334	PAK 214-3	Pakistan	2	1
C-335	PAK 214-4	Pakistan	2–3	1
C-339	PAK 224-8	Pakistan	2	1–2
C-76	<i>C. metuliferus</i>	UPV	0	1–2
C-165	<i>C. anguria</i> var. <i>longipes</i>	UPV	0	1–2

^zNº Reg.: accession number in the Germplasm Bank of the Experimental Station 'La Mayora' (CSIC), Málaga, Spain.

^yYellowing incidence rated as follows: 0 = no symptoms, 1 = slight yellowing, 2 = moderate yellowing, 3 = severe yellowing.

^xWhitefly infestation rated as follows: 0 = no whiteflies, 1 = a few individuals on most of the leaves, 2 = whiteflies covering less than a half on the leaf on most of the leaves of plants, 3 = a high number of whiteflies covering more than a half of leaf on most of leaves.

^vUPV: donated by the Germplasm Bank of Polytechnics Univ. of Valencia, Spain.

Table 2. Percentage of symptomatic plants of *Cucumis* species obtained after inoculation using viruliferous whiteflies, and time to appearance of symptoms.

Accession	Symptomatic (%) ^z	Time (d)
Piel de Sapo	100.0	30
TGR-1551	0.0	---
TGR-1920	60.0	45
TGR-1937	20.0	40
TGR-554	60.0	30
PI 414723	81.8	30
Kogane 9-G-0 Makuwa	80.0	30
Kanro Makuwa	36.4	30
Ginsen Makuwa	90.9	30
<i>C. metuliferus</i>	0.0	---
<i>C. anguria</i> var. <i>longipes</i>	90.9	30

^zInfection (%) as percentage of inoculated plants showing characteristic yellowing symptoms.

(Table 3). About one-quarter of the inoculated plants of the F_2 generation, and half of the plants of the backcross to the susceptible parental (BC_s) showed the characteristic symptoms of disease (Table 3). The symptoms were uniform and consistent; the resistant and susceptible plants were clearly differentiated. The proportion of resistant to susceptible plants in the F_2 and the BC_s families were good fits to the expected 3:1 and 1:1 ratio, respectively (Table 3).

Discussion

Some of the accessions tested responded differently to inoculations. Under natural inoculation conditions, all 'Piel de Sapo' plants showed symptoms of yellowing, while plants of Ginsen Makuwa and *C. anguria* var. *longipes* were asymptomatic. This indicates that Ginsen Makuwa and *C. anguria* var. *longipes* accessions escaped inoculation under natural-inoculation conditions. PI 414723 also appeared to be resistant under natural infection, with none of the plants showing symptoms of yellowing. However, nearly 82% of the plants inoculated under controlled conditions developed yellowing symptoms, indicating susceptibility to CYSDV. Under natural-inoculation conditions the numbers of whiteflies on the plants were very low, suggesting that plants of PI 414723 may have some degree of resistance to whitefly infestation, and that the resistance factors modify whitefly behavior (antixenosis), or affect their physiological processes, such as feeding or egg laying (antibiosis). This apparent resistance to the insect vector of the virus would explain the apparent resistance to the yellowing causal agent shown by accession PI 414723 following natural inoculation. Moreover, PI 414723 has been described as resistant to aphids and also to virus transmission by these insects (Pitrat and Lecoq, 1982).

The results with Kanro Makuwa and TGR-1937 suggest that both accessions have a degree of resistance to either the virus or to the vector, because, under natural-inoculation conditions, plants of neither genotype developed symptoms, and under controlled-inoculation conditions, only a few plants showed symptoms of yellowing. Under natural-inoculation conditions, the whitefly densities on these accessions were very similar to those on most of the other accessions tested in this work, which indicates that both accessions seem to show resistance to the virus and could also have mechanisms of resistance to transmission of the virus by the insect vector, but not to the insect itself.

Under controlled-inoculation conditions, a high percentage (20%) of the plants of the TGR-1937 accession showed yellowing symptoms 40 d after inoculation (Table 2). Despite the high percentage (60%) of TGR-1920 plants that developed symptoms under controlled-inoculation conditions, symptoms appeared 45 d after inoculation (Table 2). The appearance of symptoms in plants of both TGR-1920 and TGR-1937 some 10 d after such symptoms appeared in plants of other accessions

Table 3. Segregation for resistance to cucurbit yellowing stunting disorder virus in crosses of the resistant accession, TGR-1551 (TGR), and the susceptible 'Piel de Sapo' (PS).

Pedigree	Generation	Observed number of		Expected	χ^2	P
		Resistant	Susceptible			
Piel de Sapo	P ₁	0	12	All susceptible		
TGR-1551	P ₂	12	0	All resistant		
TGR x PS	F ₁	12	0	All resistant		
TGR x PS	F ₂	95	25	3:1	1.11	0.29
F ₁ x PS	BC _s	18	22	1:1	0.40	0.53
F ₁ x TGR	BC _r	40	0	All resistant		

might be associated with the presence of mechanisms that inhibit either virus replication or movement or both. Soria and Gómez-Guillamón (1994) reported a similar response in a *C. melo* ssp. *agrestis* when it was tested against beet pseudo yellows virus (BPYV), a closterovirus that causes a yellowing disease transmitted by *Trialeurodes vaporariorum* Westwood. These results suggest that TGR-1937 could have several mechanisms of resistance to CYSDV and BPYV, and probably also to virus transmission.

C. melo TGR-1551 and the *C. metuliferus* accession did not show yellowing symptoms under natural or controlled-inoculation conditions. Both accessions had whitefly densities similar to those on the other accessions. The fact that none of the grafted TGR-1551 plants or *C. metuliferus* plants showed yellowing symptoms suggests that these accessions are virus-resistant. They may also possess resistance to transmission of the virus by *Bemisia tabaci* biotype B. Although a search of the literature revealed no reference to resistance to the transmission of virus by whitefly in melons, resistance exists to transmission of virus by aphids. The *Vat* gene impedes the colonization of the plant by *Aphis gossypii* Glover and the transmission by this aphid of some viruses, such as the cucumber mosaic virus (CMV) and zucchini yellow mosaic virus (ZYMV) (Pitrat and Lecoq, 1982).

The resistance observed in *C. melo* TGR-1551 and the *C. metuliferus* accession could also be related to the existence of mechanisms that inhibit vascular transport of the pathogen, as indicated by resistance to infection following grafting to infected 'Piel de Sapo' plants. This type of resistance would affect long-distance movement of viral particles. Resistance could also involve cellular membrane changes that impede the diffusion or transport of infective virus particles from cell to cell, or an inhibition of virus particle replication in the tissues of the resistant host plant, as has been described for virus diseases in some cucurbit species (Gray et al., 1988), or even a combination of these two mechanisms. The resistant mechanisms acting in the *C. melo* TGR-1551 and *C. metuliferus* accessions need to be further clarified.

Resistance to papaya ringspot virus in *C. metuliferus* was reported (Provvidenti and Robinson, 1974) as well as to squash mosaic virus (Provvidenti, 1986), watermelon mosaic virus (Provvidenti and Robinson, 1977), and to a yellowing virus transmitted by *T. vaporariorum* (Soria, 1991). However, sexual incompatibility prevents exploitation

of these resistances for melon by traditional plant breeding methods. TGR-1551 accession belongs to *C. melo* species, so there should be no impediment to using it as a donor of resistance against CYSDV to the other cultivated melon groups (Esquinas-Alcázar and Gulick, 1983). No reports are presently available that describe a *C. melo* genotype with resistance against CYSDV.

The results of the genetic analysis support the hypothesis that the resistance to CYSDV in TGR-1551 is controlled by a dominant allele at one locus (Table 3). In accordance with the rules of nomenclature proposed by Robinson et al. (1976) that are incorporated in the latest version of gene nomenclature for the Cucurbitaceae (Cucurbit Genetics Cooperative Gene List Committee, 1982), we propose the name *Cucurbit yellow stunting* symbol *Cys* for the locus in *C. melo* TGR-1551 that conditions the resistance against CYSDV. This is the first gene described that conditions resistance to a whitefly-transmitted virus of melon (Lecoq et al., 1998; Pitrat, 1998). It is the second gene for resistance to a virus yellows disease of melon; PI 124112 has two complementary, recessive genes for resistance to cucurbit aphid-borne yellowing virus (Dogimont et al., 1997).

TGR-1551 carries antixenotic and antibiotic resistance to *B. tabaci*, since the reproduction rate and the viability of the females of this insect on plants of TGR-1551 are significantly lower than on plants of commercial Spanish cultivars, e.g., Bola de Oro (Soria et al., 1999). This character could also contribute to reducing the populations of the sweetpotato whitefly in the crops. In southeastern Spain, cucumber (*Cucumis sativus* L.) and melon crops are grown in the same or adjacent areas and they overlap in their timing of cultivation. Both cucurbit species are hosts of CYSDV (Célix et al., 1996), and every year each species acts as an inoculum source or reservoir of this virus for the other species. Resistance in melons would decrease the incidence of CYSDV in cucumber crops, because there would be no infected melon plants to serve as sources of inoculum in the area. This resistance to CYSDV, in theory at least, would be easy to introduce into other melon genotypes to produce resistant commercial hybrids. Fruit of TGR-1551 are small-medium sized (700 g), of elongated shape, with yellow-orange, smooth and hard skin, white flesh, and low content of soluble solids (8 °Brix). Certain undesirable hereditary characteristics of TGR-1551 observed in its hybrids with 'Piel de Sapo' and 'Bola de Oro' types, i.e., excessive

length, hard skin, and low soluble-solids content (Sesé et al., 1997) should be easily eliminated in a backcrossing program to combine resistance with desired horticultural qualities.

Literature Cited

- Browning, J.A. 1980. Genetic protective mechanisms of plant-pathogen populations: Their co-evolution and use in breeding for resistance, p. 52–76. In: M.K. Harris (ed.). *Biology and breeding for resistance to arthropods and pathogens in agricultural plants*. Texas A&M Univ. Press, College Station.
- Célix, A., A. López-Sesé, N. Almaraz, M.L. Gómez-Guillamón, and E. Rodríguez-Cerezo. 1996. Characterization of cucurbit yellow stunting disorder virus, a new *Bemisia tabaci*-transmitted closterovirus. *Phytopathology* 86:1370–1376.
- Cucurbit Genetics Cooperative Gene List Committee. Update of cucurbit gene list and nomenclature rules, 1982. *Cucurbit Genet. Coop. Rpt.* 5:62–66.
- Dogimont, C., A. Bussemakers, J. Martin, S. Slama, H. Lecoq, and M. Pitrat. 1997. Two complementary recessive genes conferring resistance to cucurbit aphid borne yellows luteovirus in an Indian melon line (*Cucumis melo* L.). *Euphytica* 96:391–395.
- Duffus, J.E. 1995. Whitefly-transmitted yellowing viruses of the Cucurbitaceae, p. 12–16. 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.
- Esquinas-Alcázar, J.T. and P.J. Gulick. 1983. Genetic resources of Cucurbitaceae. A.G.P.G.R.: I.B.P.G.R./83/84:20. Rome.
- Fraser, R.S.S. 1990. The genetics of resistance to plant viruses. *Annu. Rev. Phytopathol.* 28:179–200.
- Gómez-Guillamón, M.L. and R. Camero. 1993. ¿Es *Bemisia tabaci* el vector de un nuevo virus de amarillito? II Congreso Ibérico de Ciencias Hortícolas. Apr. 1993, Zaragoza. *Actas Hort.* 10:1356–1358.
- Gray, S.M. and J.W. Moyer. 1993. Genetic resistance that reduces disease severity and disease incidence, p. 196–217. In: M.M. Kyle (ed.). *Resistance to viral disease of vegetable. Genetics and breeding*. Timber Press, Portland.
- Gray, S.M., J.W. Moyer, and G.G. Kennedy. 1988. Resistance in *Cucumis melo* to watermelon mosaic virus 2 correlated with reduced virus movement within leaves. *Phytopathology* 78:1043–1047.
- Hassan, A.A. and J.E. Duffus. 1991. A review of a yellowing and stunting disorder of cucurbits in the United Arab Emirates. *J. Agr. Sci.* 2:1–16.
- Lecoq, H., G. Wisler, and M. Pitrat. 1998. Cucurbit viruses: The classic and the emerging, p. 126–142. In: J.D. McCreight (ed.). *Cucurbitaceae '98: Evaluation and enhancement of cucurbit germplasm*. ASHS Press, Alexandria, Va.
- López-Sesé, A.I. 1997. Virus del enanismo amarillo de las cucurbitáceas (CYSDV): técnicas de inoculación artificial y resistencia al virus y a su vector, *Bemisia tabaci* Gennadius, en melón, *Cucumis melo* L. PhD Diss., Univ. Málaga, Spain.
- Pitrat, M. 1998. 1998 gene list for melon. *Cucurbit Genet. Coop. Rpt.* 21:69–81.
- Pitrat, M. and H. Lecoq. 1982. Relations génétiques entre les résistances par non-acceptation et par antibiose du melon à *Aphis gossypii*. Recherche de liaisons avec d'autres gènes. *Agronomie* 2 (Gene nomenclature for the Cucurbitaceae, 1996):503–508.
- Provvidenti, R. 1986. Viral diseases of cucurbits and sources of resistance. *Bul.* 96. Food and Fert. Tech. Center, Taipei, Taiwan.
- Provvidenti, R. and R.W. Robinson. 1974. Resistance to squash mosaic virus and watermelon mosaic virus 1 in *Cucumis metuliferus*. *Plant Dis. Rptr.* 58:735–738.
- Provvidenti, R. and R.W. Robinson. 1977. Inheritance of resistance to watermelon mosaic virus 1 in *Cucumis metuliferus*. *J. Hered.* 68:56–57.
- Robinson, R.W., H.M. Munger, T.W. Whitaker, and G.W. Bohn. 1976. Genes of the Cucurbitaceae. *HortScience* 11:554–568.
- Sesé, A.I.L., F. Sánchez, and M.L. Gómez-Guillamón. 1997. Evaluation of melon F₁ hybrids resistant to cucurbit yellowing and stunting disorder virus (CYSDV), p. 81. In: K. Abak (ed.). *Abstract book of the 1st Intl. ISHS Symp. on Cucurbits*. 20–23 May 1997. Adana, Turkey.
- Sokal, R.R. and F.J. Rohlf. 1981. *Biometry*. W.H. Freeman, San Francisco.
- Soria, C. 1991. Amarilleo del melón (*Cucumis melo* L.). Causas, técnicas de inoculación y fuentes de resistencia. PhD Diss., Univ. Málaga, Spain.
- Soria, C. and M.L. Gómez-Guillamón. 1994. Resistance of *Cucumis melo* var. *agrestis* against melon-yellowing disease. *Cucurbit Genet. Coop. Rpt.* 17:74–75.
- Soria, C., A.I. López-Sesé, and M.L. Gómez-Guillamón. 1999. Resistance of *Cucumis melo* against *Bemisia tabaci* (Homoptera: Aleyrodidae). *Environ. Entomol.* 28(5):831–835.