

2011 Gene List for Melon

Catherine Dogimont

INRA, UR1052, Unité de Génétique et d'Amélioration des Fruits et Légumes, BP 94, 84143 Montfavet cedex (France)
catherine.dogimont@avignon.inra.fr

Melon (*Cucumis melo* L.) is an economically important, cross-pollinated species. Melon has $2n = 24$ chromosomes and a relatively small genome (450 Mb), about three times larger than the *Arabidopsis thaliana* genome and similar to the rice genome (Arumanagathan and Earle, 1991). Melon has high intra-specific genetic variation and morphologic diversity. A great variety of genetic and molecular studies have been conducted on important agronomical traits, such as resistance to pathogens and insects, and floral and fruit traits.

The following list is the latest version of the gene list for melon. Previous gene lists were organized by Pitrat: (Pitrat, 2006), (Pitrat, 2002), (Pitrat, 1998), (Pitrat, 1994), (Pitrat, 1990), (Pitrat, 1986), (Committee, 1982). This current list has been modified from previous lists in that (1) it provides an update of the known genes and QTLs, and (2) it adds an expanded description for reported genes including sources of resistance and resistance genes, phenotypes of mutants and traits related to seeds, seedlings, plant morphology and architecture, flowers and fruits. Locations of the reported genes on the melon genetic map and linked markers useful for marker assisted selection were reported where available.

Since the first molecular marker-based melon map published in 1996 (Baudracco-Arnas and Pitrat, 1996), several genetic maps of melon have been published by several research teams, using several segregating populations. 2011 will be the year of the publication of an integrated map of melon in the framework of the International Cucurbit Genomic Initiative (Díaz et al., 2011). The integrated map has been constructed by merging data from eight independent mapping populations using genetically diverse parental lines. It spans 1150 cM distributed across the 12 melon linkage groups and comprises more than 1500 markers. Individual maps and the integrated map are available at www.icugi.org. The linkage groups were named according to Perin et al., 2002. The same nomenclature will be adopted hereafter. QTLs for 62 traits including virus resistance, fruit shape, fruit weight, sugar content have been located on this integrated map (Díaz et al., 2011).

The list of melon sequences was not updated, as melon ESTs and full cDNAs are increasing extraordinarily (www.icugi.org). A physical map of the melon genome, anchored to the genetic map, has been estab-

lished (Gonzalez et al., 2010) and the complete melon genome sequence is expected by the end of the year.

Host Plant Resistance genes

Considerable attention has been given to resistance genes in melon. Genes for resistance to viruses, insects, fungi and oomycetes have been reported.

Viral Diseases

The first source for resistance to *Zucchini yellow mosaic virus* (ZYMV, Potyvirus), and for a long time the only known source, was the Indian accession PI 414723 (Pitrat et al., 1996). The resistance proved to be strain-specific and was not effective against a second pathotype of the virus. The screening of about 60 cultivars from Iran allowed the identification of three immune cultivars: Magolalena Vertbrod, Soski and Bahramabadi (Arzani and Ahoonmanesh, 2000). Among 200 melons collected in Sudan, resistance sources to ZYMV were found, mainly in wild forms (Mohamed, 1999).

Resistance to ZYMV in PI 414723 was reported to be controlled by a single dominant gene, *Zym* (Pitrat and Lecoq, 1984), which mapped to the linkage group II (former LG 4), linked to the gene *a* (*andromonoecious*) (Pitrat, 1991; Perin et al., 2002). Using the ZYMV-Nat strain (pathotype 1), Danin-Poleg et al. (1997) found that three genes were needed to confer the resistance in PI 414723 (*Zym-1*, *Zym-2* and *Zym-3*). Molecular markers linked to the resistance were identified by bulk segregant analysis (Danin-Poleg et al., 2000; Danin-Poleg et al., 2002).

A semi-dominant gene named *Fn*, independent of *Zym*, was reported to control in 'Doublon' plant wilting and necrosis after inoculation with strains of the F pathotype of ZYMV (Risser et al., 1981). The *Fn* gene was located in the linkage group V (formerly 2), at 12 cM of the *Vat* gene, conferring *Aphis gossypii* resistance (Pitrat, 1991).

Necrosis after inoculation with *Watermelon mosaic virus-Morocco* (Potyvirus) was reported to be controlled by a single dominant gene *Nm* in 'Védreantais' (*nm* in 'Ouzbèque') (Quiot-Douine et al., 1988).

Papaya ringspot virus- watermelon type (PRSV, formerly called WMV-1, Potyvirus) resistance was reported

in the Indian accessions PI 180280 (Webb and Bohn, 1962; Webb, 1979), PI 180283 (Quiot et al., 1971), PI 414723 (Anagnostou and Kyle, 1996), and PI 124112 (McCreight and Fashing-Burdette, 1996) and in TGR-1551 (C-105) from Zimbabwe (Gómez-Guillamón et al., 1998). Resistance to PRSV-W is conferred by a single dominant gene, *Pro*, in PI 180280 (Webb, 1979) as well as in the lines B66-5 and WMR 29, derived from PI 180280 (Pitrat and Lecoq, 1983). An allele at the same locus was shown to incite a lethal necrotic response against French strains of PRSV-W in PI 180283 and in 72025, derived from PI 180283 (Pitrat and Lecoq, 1983). These alleles were called *Pro*¹ and *Pro*², *Pro*¹ being dominant over *Pro*² (Pitrat, 1986). *Pro* has been mapped to the linkage group IX (former 5) (Pitrat, 1991; Perin et al., 2002), closely linked to the gene *Fom-1* conferring resistance to *Fusarium oxysporum* races 0 and 2 (Pitrat, 1991; Perin et al., 2002; Brotman et al., 2005). A single dominant gene, *Pro-2*, was also reported to control an incompatible reaction of PI 124112 after inoculation with PRSV (McCreight and Fashing-Burdette, 1996).

Partial resistance to *Watermelon mosaic virus* (WMV, formerly WMV-2, Potyvirus) has been reported in melon line 91213, which was selected from PI 371795 and related to PI 414723 (Moyer et al., 1985; Gray et al., 1988; Moyer, 1989), the Korean accession PI 161375 (Pitrat, 1978), and in the accessions from Iran (Latifah-1, Tashkandi and Khorasgani) and in an exotic line (Galicum) (Arzani and Ahoonmanesh, 2000). Partial resistance was reported in breeding lines obtained by successive backcrossing with selection from PI 414723; inoculated plants develop mosaic symptoms on inoculated leaves but recover from symptoms and virus infection in the youngest leaves. This partial resistance was reported to be controlled by a single dominant gene, *Wmr*, linked to the ZYMV resistance gene, *Zym* (Gilbert et al., 1994; Anagnostou et al., 2000). PI 414723 was observed to be highly susceptible to WMV after inoculation with European strains of WMV (Dogimont et al., unpublished data; Gómez-Guillamón, 1998). The accession TGR-1551 was reported to exhibit very mild symptoms and a very reduced titer of virus; this partial resistance was essentially determined by a recessive gene (Diaz-Pendon et al., 2005). Still unnamed, we propose to name it *wmr-2*.

Several cultivars originating from Asia and belonging to Oriental pickling melon (var. *conomon*) and to Oriental melon (var. *makuwa*) were reported to be highly resistant to *Cucumber mosaic virus* (CMV, Cucumovirus) (Enzie, 1943; Webb and Bohn, 1962; Risser et al., 1977; Hirai and Amemiya, 1989; Daryono et al., 2003; Diaz et al., 2003). Interestingly, some accessions from Iran were also found resistant to CMV (Arzani and Ahoonmanesh, 2000) as well as the Indian IC274014 (Dhillon et al., 2007).

Resistance to the CMV-B2 strain in the accession Yamatouri was reported to be controlled in a single dominant manner. SCAR markers linked to the gene, named *Creb-2*, were identified (Daryono et al., 2010). CMV resistance was first reported to be controlled by three recessive genes in the cross Freeman Cucumber x Noy Amid (Karchi et al., 1975). Seven QTLs were shown to be involved in resistance to three different strains of CMV in the cross Védraçais x PI 161375 (Dogimont et al., 2000); one of them, located in linkage group XII explains a large part of the resistance to the strain P9 (Dogimont et al., 2000; Essafi et al., 2009).

Among about 500 accessions tested, resistance to *Cucurbit aphid borne yellows virus* (CABYV, Polerovirus, transmitted by aphids on a persistent manner) was reported in the Indian accessions 90625 (= PI 313970), Faizabadi Phoont, PI 124112, PI 282448, and PI 414723, in the Korean accession PI 255478 and in PI 124440 from South Africa (Dogimont et al., 1996). Resistance to CABYV in PI 124112 is conferred by two independent complementary recessive genes, named *cab-1* and *cab-2* (Dogimont et al., 1997).

Partial resistance to the *Beet pseudo yellows virus* (BPYV, Crinivirus), transmitted by the whitefly *Trialeurodes vaporariorum*, was reported in a few accessions of Asian origin: Nagata Kim Makuwa, PI 161375, Cma, a wild melon collected in Northern Korea and a Spanish landrace Tendral type (Esteva et al., 1989; Nuez et al., 1991). The resistance of Cma, expressed as a delayed and milder infection, resulted from the cumulative effect of an antixenosis against the vector and resistance to the virus (Soria et al., 1996; Nuez et al., 1999). Study of segregating families under natural infection suggested that the partial resistance to BPYV in Nagata Kim Makuwa, PI 161375 and Cma was controlled by single genes, partially dominant in Nagata Kim Makuwa (gene *My*) and Cma, and partially recessive in PI 161375 (Esteva and Nuez, 1992; Nuez et al., 1999).

Resistance to *Cucurbit yellow stunting disorder virus* (CYSDV, Crinivirus) was reported in the accession TGR-1551 (C-105), from Zimbabwe, under natural infection in Spain and when subjected to controlled inoculation by viruliferous *Bemisia tabaci* and by grafting (Lopez-Sese and Gomez-Guillamon, 2000). Delayed and only slight symptoms were reported in a few accessions under natural infection conditions in the United Arab Emirates (Jupiter, Muskotaly, PI 403994) and in Spain (Hassan et al., 1991; Lopez-Sese and Gomez-Guillamon, 2000). Partial resistance to CYSDV was also reported in PI 313970 in the United States (McCreight and Wintermantel, 2008). In progenies obtained from the cross between TGR-1551 and a susceptible Spanish Piel de Sapo cultivar, the resistance was shown to be controlled by a single domi-

nant gene, called *Cys* (Lopez-Sese and Gomez-Guillamon, 2000).

A large melon germplasm was tested for *Lettuce infectious yellows virus* (LIYV, Crinivirus) resistance in natural infection by *Bemisia tabaci* biotype A in California. A snake melon originating from Saudi Arabia was shown to exhibit very mild LIYV symptoms (McCreight, 1991; McCreight, 1992). After successive field tests and confirmation in controlled-inoculation greenhouse tests, the Indian accession PI 313970, was shown to be the most interesting source of resistance to LIYV, although an occasional plant of this accession may appear symptomatic, or have a positive ELISA for LIYV (McCreight, 1998, 2000). Resistance to LIYV in PI 313970 was shown controlled by a single dominant allele at the locus designated *Liy* (McCreight, 2000).

Melon breeding line MR-1 and PI 124112, PI 179901, PI 234607, PI 313970 and PI 414723 were reported to exhibit a partial resistance to *Cucurbit leaf crumple virus* (CuLCrV), a geminivirus transmitted by *B. tabaci* biotype B, while PI 236355 was found to be completely resistant. A single recessive gene, named *culcro*, was reported to control resistance in PI 313970, and likely in the other resistant accessions (McCreight et al., 2008).

Gonzalez-Garza et al. reported three phenotypes when they inoculated various melon cultivars with *Melon necrotic spot virus* (MNSV, Carmovirus) (Gonzalez-Garza et al., 1979): - cultivars susceptible to systemic infection showing local lesions on the inoculated leaves followed by systemic necrotic spotting, necrotic streaks on stems, conducting finally infected plants to collapse; - cultivars showing local lesions but no systemic symptoms: 53% of the accessions tested; - immune lines remaining free of symptoms ('Improved Gulfstream', 'Perlita', 'Planters Jumbo', 'PMR 5', WMR 29 and breeding line PMR Honeydew).

Among a broad germplasm collection of melons inoculated with MNSV (532 accessions), Pitrat et al. (1996) found 7% immune accessions. The resistance was confirmed to be quite common in American cantaloupe cultivars (22 resistant accessions representing 28 % out the North American accessions tested). Some resistant accessions were found originating also from Far East and India. One recessive gene, *nsv*, controls the resistance to MNSV (Coudriet et al., 1981). First described in the American cultivar Gulfstream, the same gene was shown to be present in other American germplasm ('PMR 5', 'Planters Jumbo', VA 435) and the Asian accession PI 161375 (Coudriet et al., 1981; Pitrat, 1991). *nsv* was mapped on the linkage group XII (formerly 7) (Pitrat, 1991; Baudracco-Arnas and Pitrat, 1996; Perin et al., 2002). The fine mapping and the cloning of the gene revealed that the resistance corresponds to a single nucleotide

substitution in the translation initiation factor eIF4E (Morales et al., 2002; Morales et al., 2005; Nieto et al., 2006). The same substitution was found in all the MNSV resistant accessions, suggesting that the resistance has a unique origin (Nieto et al., 2007).

Two independent dominant genes, named *Mnr-1* and *Mnr-2*, were reported to control resistance to systemic infection of MNSV in Doublon; *Mnr-1* is linked to *nsv* at 19 cM (Mallor et al., 2003).

No complete sources of resistance to *Squash mosaic virus* (SqMV, Comovirus) have been reported in melons. Tolerance was, however, observed in accessions originating from India, Afghanistan, China and Pakistan (Webb and Bohn, 1962; Provvidenti, 1989, 1993). The Korean and Chinese accessions PI 161375 and China 51 (var. *makuwa*) were described to develop delayed mosaic symptoms, reduced virus multiplication, and, interestingly, complete resistance to seed transmission of SqMV (Maestro-Tejada, 1992; Provvidenti, 1998). Resistance to seed transmission was shown to be effective against four different strains of SqMV (Provvidenti, 1998). Tolerance to foliar symptoms incited by a melon strain of SqMV was shown to be controlled by a single recessive gene in China 51, but appeared to be partially dominant against a squash pathotype of SqMV (Provvidenti, 1998). Unnamed so far, we propose to name the gene *sqmv*.

Partial resistance (restriction to the virus movement) to the SH isolate of *Cucumber green mottle mosaic virus* (CGMMV, Tobamovirus) was reported in the makuwa type Chang Bougi accession (Sugiyama et al., 2006). The resistance was controlled by two complementary, recessive genes, called *cgmmv-1* and *cgmmv-2* (Sugiyama et al., 2007).

Resistance to a complex of viruses from Egypt in PI 378062 was reported to be controlled by a single dominant gene, named *Imy*, *Interveinal mottling and yellowing resistance* (Hassan et al., 1998).

Insect resistance

Resistance to the melon-cotton aphid, *Aphis gossypii* (Homoptera: Aphididae), was first reported by Kishaba and Bohn. A dominant gene, *Ag*, was reported to control antixenosis, antibiosis under controlled no-choice tests and free-curling tolerance in LJ 90634, later called PI 414723 (Kishaba et al., 1971, 1976). Pitrat and Lecoq (1980; 1986) reported resistance in PI 161375 and in PI 414723 to several viruses when they are transmitted by *A. gossypii*. The resistance is vector-specific (only *A. gossypii*), and non-specific to viruses (CMV, ZYMV, WMV...). It co-segregates with antixenosis described previously. Resistance to viruses when they are transmitted by *A. gossypii*, is controlled by a single gene, named *Vat* (*Virus aphid transmission*). The *Vat* locus was

mapped to a subtelomeric position on the linkage group V (formely 2) (Pitrat, 1991; Baudracco-Arnas and Pitrat, 1996; Brotman et al., 2002; Perin et al., 2002). A single gene was cloned by positional cloning, which confers both aphid resistance and virus resistance when they are transmitted by *A. gossypii*. The gene was shown to encode a CC-NBS-LRR protein (Dogimont et al., 2004; Pauquet et al., 2004; Dogimont et al., 2010). Four additive and two couples of epistatic QTLs affecting behaviour and biotic potential of *A. gossypii* were mapped in recombinant inbred lines derived from the cross Védraçais x PI 161375; amongst them, a major QTL, which affects both behavior and biotic potential of *A. gossypii*, corresponds to the *Vat* gene (Boissot et al., 2010).

A single dominant gene, named *Lt*, was reported to control resistance to the leafminer *Liriomyza trifolii* (Diptera : Agromyzidae) in the old French cultivar Nantais Oblong (Dogimont et al., 1999). Resistant plants exhibit fewer mines and a very high larval mortality. The resistance is inefficient towards *L. huidobrensis*.

Two complementary recessive genes (*dc-1* and *dc-2*) for resistance to the melon fruit fly, *Bractocera cucurbitae* (formely *Dacus cucurbitae*, Diptera: Tephritidae) were reported by (Sambandam and Chelliah, 1972).

A monogenic recessive resistance to cucumber beetles was reported in C922-174-B in crosses among non-bitter genotypes. The gene named *cb₁* (= *cb*) was shown to be efficient towards three species of Coleoptera: the banded beetle *Diabrotica balteata*, the spotted beetle *D. undecimpunctata howardi* and the striped beetle *Acalymna vittatum* (Nugent et al., 1984). In AR Top Mark, resistance to *D. undecimpunctata howardi* was also reported to be recessive and linked to the bitterness trait, controlled by the dominant gene *Bi* (Lee and Janick, 1978) that makes the melon attractive to the spotted beetle (Nugent et al., 1984).

A dominant gene, named *Af*, was reported to control resistance to the red pumpkin beetle (*Aulacophora foveicollis*, Coleoptera: Chrysomelidae) in Casaba (Vashistha and Choudhury, 1974).

Fungal Diseases

Fusarium wilt resistance. Three genes were reported to control resistance to *Fusarium oxysporum* f.sp. *melonis*. A single dominant gene, *Fom-1*, controls resistance to *F. oxysporum* races 0 and 2; it was reported in the old French cultivar Doublon (Risser, 1973; Risser et al., 1976). *Fom-1* was mapped at a distal end of the linkage group IX (formely 5), at 2 cM from the PRSV resistance gene, *Pro²* (Perin et al., 2002). Molecular markers for *Fom-1*, useful for marker assisted selection, were developed (Brotman

et al., 2005; Oumouloud et al., 2008; Tezuka et al., 2009; Tezuka et al., 2011). A single dominant gene, *Fom-2*, controls resistance to *F. oxysporum* races 0 and 1; it was reported in CM17187 (Risser, 1973; Risser et al., 1976). *Fom-2* was mapped to the linkage group XI (Perin et al., 2002). The gene *Fom-2* was cloned and reported to encode a NBS-LRR type R protein of the non-TIR subfamily (Joobeur et al., 2004). Molecular markers linked to *Fom-2* were developed (Zheng et al., 1999; Zheng and Wolff, 2000), but their use was not completely satisfying because of recombination (Sensoy et al., 2007). New promising molecular markers were recently designed within the gene (Wang et al., 2011). Resistance to *F. oxysporum* races 0, 1 and 2 is quite frequent (Alvarez et al., 2005). The *Fom-3* gene was reported in Perlita FR; it confers the same phenotype as *Fom-1* but segregates independently from *Fom-1* (Zink and Gubler, 1985).

Resistance to *F. oxysporum* races 0 and 2 in the Spanish var. *cantalupensis* accession Tortuga was reported to be controlled by two independent genes, one dominant and the other one recessive. The dominant likely is *Fom-1*; the recessive one was named *fom-4* (Oumouloud et al., 2010).

A major recessive gene, named *fom1.2a*, was reported to confer resistance to *F. oxysporum* race 1.2 in the Israeli breeding line BIZ. The gene was located at a distal end of the LG II (opposite to the gene *a*, *andromonoecious*) (Herman et al., 2008). A second recessive gene was previously reported to segregate in the same population (Herman and Perl-Treves, 2007). In contrast, nine QTLs were reported to control the recessive resistance to race 1.2 in the French breeding line Isabelle, derived from the Far East resistant accession Ogon 9 (Perchepied and Pitrat, 2004; Perchepied et al., 2005). The resistance of the var. *cantalupensis* accession BG-5384 from Portugal to *F. oxysporum* race 1.2 (Y pathotype) was also reported to be polygenic and recessive (Chikh-Rouhou et al., 2008; Chikh-Rouhou et al., 2010).

Powdery mildew resistance. Several dominant resistance genes to powdery mildew were reported in melon. Genetic relationship between these genes is still confused, as is the definition of powdery mildew races (McCreight, 2006; Lebeda et al., 2011). Mapping of powdery resistance genes and QTLs in several crosses has thus far located them in six distinct melon linkage groups.

Jagger et al. (1938) reported a dominant resistance gene, *Pm-1*, to powdery mildew in 'PMR 45'. In the original paper, *Pm-1* was reported to confer resistance to *Erysiphe cichoracearum* but the pathogen was misidentified and was later determined to have been *Podosphaera xanthii*. *Pm-1* likely corresponds to the gene

Pm-A, which confers resistance to *P. xanthii* race 1 in 'PMR 45', described in Epinat et al. (1993). The powdery mildew resistance gene from 'PMR 45', introgressed into a yellow-fleshed breeding line, was reported to be located in the linkage group IX, loosely linked to the PRSV resistance gene, *Prv* (Teixeira et al., 2008).

A single dominant gene, *Pm-x*, confers resistance to *P. xanthii* race 1 and 2 (at least) in PI 414723; it was located in the linkage group II, linked to the ZYMV resistance gene *Zym* and to the andromonoecious gene *a* (Pitrat, 1991; Perin et al., 2002).

A single dominant gene was reported in WMR 29, *Pm-w*, which confers resistance to *P. xanthii* races 1, 2 and 3 (Pitrat, 1991). It likely corresponds to *Pm-B* in Epinat et al. (1993). It was located in the linkage group V (formerly 2), closely linked to the *Vat* locus (Pitrat, 1991; Perin et al., 2002).

Harwood and Markarian (1968) reported two dominant genes in PI 124112, *Pm-4* and *Pm-5*. These genes may correspond to the two genes of PI 124112 reported in Perchepped et al. (2005), *PmV.1* and *PmXII.1*. *PmV.1* confers resistance to *P. xanthii* races 1, 2, and 3 and was located in the linkage group V, closely linked to the *Vat* locus. *Pm-XII.1* confers resistance to *P. xanthii* races 1, 2 and 5 and to *Golovinomyces cichoracearum* race 1 and was mapped to the linkage group XII. It may correspond to one of the two genes, *Pm-F* and *Pm-G*, which were reported to interact for controlling resistance to *G. cichoracearum* in PI 124112 (Epinat et al., 1993).

Two genes were reported in 'PMR 5', *Pm-1* and *Pm-2* (Bohn and Whitaker, 1964). Allelism tests clearly showed that 'PMR 5' has the same gene as 'PMR 45' to control *P. xanthii* race 1. *Pm-2* likely corresponds to *Pm-C*, which confers resistance to *P. xanthii* race 2 in interaction with *Pm-1* (Epinat et al., 1993). Two genes, *Pm-C* (*Pm-2*) and *Pm-E*, were suggested to interact in 'PMR 5' to control resistance to *G. cichoracearum* (Epinat et al., 1993). Recently, two QTLs of resistance to *P. xanthii* race 1 and N1 were located in the linkage groups II and XII in recombinant inbred lines derived from the cross PMAR No.5 x Harukei No.3 (Fukino et al., 2006; Fukino et al., 2008). These two QTLs may correspond to the same genomic regions as reported in PI 124112, with different alleles. PMAR No.5 (= AR 5) was obtained from an aphid resistant line and successive backcrosses to 'PMR 5' (McCreight et al., 1984). The results obtained by (Fukino et al., 2006; Fukino et al., 2008) suggest that powdery mildew resistance genes in PMAR No.5 may be different from those in 'PMR 5', as *Pm-1* is expected to be located in the linkage group IX (Teixeira et al., 2008).

Harwood and Markarian (1968) reported a single dominant resistance gene in PI 124111, *Pm-3*. Kenigsbuch

and Cohen (1989) reported a second gene in PI 124111, *Pm-6*, independent from *Pm-3*, which confers resistance to *P. xanthii* race 2. Their relationship with the other powdery mildew resistance genes is unknown.

Resistance to the Chinese race of *P. xanthii* (with a unique reaction pattern of the commonly used melon race differentials) in the Indian accession PI 134198 was reported to be controlled by a single dominant gene, designated *Pm-8*, which was suggested to be located in the linkage group VII (Liu et al., 2010).

Resistance to *P. xanthii* races 1, 2 and 5 in TGR-1551 was reported to be controlled by two independent genes, one dominant and one recessive, each one conferring resistance to all three races (Gómez-Guillamón et al., 2006; Yuste-Lisbona et al., 2008). The dominant gene, *Pm-R*, was recently located in the linkage group V, closely linked to the *Vat* and *Pm-w* loci (Yuste-Lisbona et al., 2011); the recessive gene was putatively located in the linkage group VIII, with a LOD score lower than the threshold score (Yuste-Lisbona et al., 2011).

In the same manner, resistance to *P. xanthii* in PI 313970 or 90625 was reported to be controlled by dominant, co-dominant, and recessive genes (McCreight, 2003; McCreight and Coffey, 2007; Pitrat and Besombes, 2008). Recently, PI 313970 resistance to the race S, a new strain of *P. xanthii* from Eastern-USA, virulent on all the commonly used resistance differentials, was reported to be controlled by a single recessive gene, named *pm-S*. The relationship of *pm-S* with the previously reported resistance genes in PI 313970 is unknown (McCreight and Coffey, 2011).

Other fungi. Several genes have been described to control resistance to gummy stem blight, caused by *Didymella bryoniae* (asexual form *Mycosphaerella citrullina*). Four independent dominant genes, *Gsb-1* through *Gsb-4*, were reported to confer a high level of resistance in PI 140471, PI 157082, PI 511890, and PI 482398 (Prasad and Norton, 1967; Frantz and Jahn, 2004). In the latter accession, a recessive gene, *gsb-5*, independent from *Gsb-1*, *Gsb-2*, *Gsb-3* and *Gsb-4* was also reported (Frantz and Jahn, 2004). A single dominant gene (previously named *Mc-2*), was reported to confer a moderate level of resistance in C-1 and C-8 (Prasad and Norton, 1967); we propose to rename it *Gsb-6*.

A single dominant gene, *Ac*, was reported to control resistance to *Alternaria cucumerina* in the line MR-1 (Thomas et al., 1990). A semi-dominant gene, *Mvd*, was reported to control partial resistance to melon vine decline caused by *Acremonium cucurbitacearum* and *Monosporascus cannonballus* in the wild type accession Pat 81 (Iglesias et al., 2000).

Oomycete resistance

Sources of resistance to downy mildew caused by the oomycete *Pseudoperonospora cubensis* were reported in several Indian accessions (Dhillon et al., 2007; Fergany et al., 2011). Downy mildew resistance was reported to be controlled by two partially dominant, complementary genes, *Pc-1* and *Pc-2*, in the Indian accession PI 124111 (Cohen et al., 1985; Thomas et al., 1988; Kenigsbuch and Cohen, 1992). This accession was reported to be resistant to the six known pathotypes of downy mildew (Cohen et al., 2003). Two complementary, dominant genes (*Pc-4* and *Pc-1* or *Pc-2*) were also reported to control resistance to downy mildew in another Indian accession PI 124112 (Kenigsbuch and Cohen, 1992). Nine QTLs for resistance to *P. cubensis* were located on a melon map developed from the cross 'Védraçais' x PI 124112. Among them, a major QTL, *Pc-XII.1*, was located in the linkage group XII, closely linked to the powdery mildew resistance QTL *Pm-XII.1*, which confers resistance to *P. xanthii* races 1, 2 and 5 and *G. cichoracearum* race 1 (Perchepped et al., 2005). A single dominant gene of partial resistance, *Pc-3*, was reported in the Indian accession PI 414723 (Epinat and Pitrat, 1989). The gene *Pc-5* was reported to interact with the modifier gene, *M-Pc-*, to control downy mildew resistance in the line 5-4-2-1; in presence of *M-Pc-5*, the resistance conferred by the gene *Pc-5* is dominant, while in absence of *M-Pc-5*, the resistance is recessive (Angelov and Krasteva, 2000).

Seed and Seedling Genes

Three genes were reported to control seed coat color: the *r* gene (*red stem*) controls brown seed color and a red stem in PI 157083 (30569) (Bohn, 1968; McCreight and Bohn, 1979). The gene *Wt* (*White testa*) controls white seed testa color and is dominant to yellow or tan seed coat color (Hagiwara and Kamimura, 1936). A *White testa* gene (*Wt-2*) was also reported in PI 414723, dominant to yellow seed testa color and mapped to the linkage group IV (Périn et al., 1999). The pine-seed shape of the seeds of PI 161375 is controlled by a single recessive gene, *pin*, *pine-seed shape*, which was mapped to the linkage group III (Perin et al., 2002). This trait is common in melon in the pinonet Spanish type. The presence of a gelatinous sheath around the seeds (versus absence) was reported to be controlled by a single dominant gene *Gs*, *Gelatinous sheath* (Ganesan, 1988).

Several chlorophyll deficient mutants were reported in melon. A single recessive gene, *alb*, (*albino*) controls the white cotyledon, lethal mutant in Trystorp (Besombes et al., 1999). The dominant pale cotyledons mutant *Pa*, *Pale*, is a lethal mutation as *PaPa* are albinos

and die early, while *PaPa+* have yellow cotyledons and leaves (McCreight and Bohn, 1979); *Pa* was shown to be linked to the *gl* (*glabrous*) and *r* (*red stem*) mutant genes (Pitrat, 1991). A single recessive gene, *yg* (*yellow green*), controls light green cotyledons and leaves in the line 26231 (Whitaker, 1952); it was located in the linkage group XI (former 6) (Pitrat, 1991). An allele of *yg*, first described as *lg* (*light green*) in the cross Dulce x TAM-Uvalde, was renamed *yg^w* (*yellow green Weslaco*) (Cox, 1985; Cox and Harding, 1986). A single recessive gene, *f* (*flava*), controls bronze yellow cotyledons and leaves and a reduced plant growth in the Chinese accession K2005 (Pitrat et al., 1986); it was reported to be closely linked to the *lmi* (*long main stem internode*) gene (Pitrat, 1991). A recessive mutant with a yellow ring on the cotyledons that later disappears, leaving the plants a normal green, was named *h* (*halo*) (Nugent and Hoffman, 1974); it was shown to be linked to the genes *a* (*andromonoecious*), *Pm-x* (*Powdery mildew resistance x*) and *Zym* (*Zucchini yellow mosaic virus resistance*) and was then located in the linkage group II (former 4) (Pitrat, 1991; Perin et al., 2002). Three recessive *virescent* genes *v*, *v-2* and *v-3* control pale cream cotyledons and hypocotyls, which turn green later; the younger leaves are light green while the older ones are normal green (Hoffman and Nugent, 1973; Dyutin, 1979; Pitrat et al., 1995); the *v-3* gene was shown to be independent to *v* (Pitrat et al., 1995). Two yellow virescent recessive mutant genes, *yv* (*yellow virescent*) and *yv-2*, were reported (allelism unknown); they control pale cotyledons, yellow green young leaves and tendrils and green older leaves, associated with a severely reduced plant growth (Zink, 1977; Pitrat et al., 1991).

The incapacity of a mutant to efficiently absorb Fe (iron) and Mn (manganese) was reported to be controlled by a recessive gene, *fe*; the mutant chlorotic leaves with green veins turn to green when iron is added to the nutrient solution (Nugent and Bhella, 1988; Jolley et al., 1991).

A single recessive gene, *ech* (*exaggerated curvature of the hook*), was shown to control the triple response of seedling germination in the dark in the presence of ethylene. Seedlings exhibit a very strong, 360° hook curvature of hypocotyls in PI 161375 (*ech*), while they exhibit a moderate, 180° curvature in 'Védraçais' and PI 414723 (*Ech*). The *ech* gene was mapped to the linkage group I (Perin et al., 2002).

Seedling bitterness due to the presence of cucurbitacins, common in honeydew or Charentais type, was shown to be dominant over non-bitter, found in most American cantaloupes, and controlled by a single gene *Bi* (*Bitter*) (Lee and Janick, 1978).

A single recessive *delayed lethal* mutant, *dlet* (formerly *dl*) was described by Zink (1990); it exhibits a re-

duced growth, necrotic lesions on leaves leading to premature death.

Leaf and Foliage Genes

Several genes control leaf and foliage traits in melon. Two linked dominant genes, *Ala* (*Acute leaf apex*) and *L* (*Lobed leaves*) were reported to control leaf shape in 'Main Rock' (*Ala* and *L*) crossed with 'PV Green' (*ala* and *l*) (Ganesan and Sambandam, 1985). Highly indented leaves, instead of round, are controlled by a single recessive gene, *dl* (*dissected leaf*), in URRS 4 (Dyutin, 1967). An allele of *dl* in 'Cantaloup de Bellegarde', previously described as *cut leaf*, was named *dl^v*, *dissected leaf Velich* (Velich and Fulop, 1970). A second gene *dl-2* (*dissected leaf-2*), allelism unknown, was reported as "hojas hendidas" (Esquinas Alcazar, 1975). A single dominant gene, *Sfl*, was reported to control the *subtended floral leaf* trait; the leaves bearing hermaphrodite/pistillate flowers in their axis, are sessile, small and enclosing the flowers in 'Makuwa', *Sfl*, while normal in 'Annamalai', *sfl* (Ganesan and Sambandam, 1979). Cox (1985) reported two recessive leaf mutant genes, *brittle leaf dwarf* (*bd*) and *curled leaf* (*cl*), which both affect the female fertility. Spoon-shaped leaves with upward curling of the leaf margins were reported to be controlled by a single recessive gene, named *cf* (*cochleare folium*) in a spontaneous mutant in 'Galia' (Lecouviour et al., 1995). A single recessive gene, *gl* (*glabrous*), was reported to control completely hairless plants in Arizona *glA* (Foster, 1963). A single recessive gene, *r* (*red stem*), controls in PI 157083 (30569) a red striped hypocotyl and red stem, especially at internodes, that is photosensitive, and reddish or tan seed coat color (Bohn, 1968; McCreight and Bohn, 1979). The genes *gl* and *r* were shown to be linked in a same linkage group (LG 3) comprising also *Pa* (*Pale*) and *ms-1* (*male sterile-1*) (McCreight, 1983; Pitrat, 1991).

Plant Architecture Genes

A single gene, recessive or incompletely dominant, called *slb*, *short lateral branching* (formerly *sb*) was suggested to control the short lateral branching trait in LB-1, a wild melon from Russia (Ohara et al., 2001). In 2008, (Fukino et al., 2008) reported two QTL for short lateral branching in a cross between a breeding line Nou 4 derived from LB-1 and the normal branching 'Earl's Favourite' (Harukei 3). The QTL mapped to LG VII and LG XI, explained, respectively, 14.8 % (The allele of Harukei 3 contributed to shorter length branches) and 42.2% (The allele of 'Nou 4' contributed to shorter length branches). A mutant lacking lateral branches, named *ab*, *abrachiate*, was reported; it produces only male flowers (Foster and Bond, 1967).

A single recessive gene, *lmi* (*long main-stem internode*), controls a long hypocotyl and a long internode length (about 20 cm) in the main stem but does not affect internode length of lateral branches in 48764 (McCreight, 1983). Three recessive genes that controlled short-internodes, *si-1*, *si-2*, *si-3* (*short internode-1, -2, -3*), were reported in three independent melon lines, UC Topmark Bush, Persia 202, and 'Maindwarf' (Denna, 1962; Paris et al., 1984; Knavel, 1990). *si-1* plants display a bush phenotype, with an extremely compact growing habit and very short (about 1 cm) internode length (Denna, 1962; Zink, 1977); *si-1* is linked to the gene *yv*, *yellow virescent* (Pitrat, 1991). Internodes of *si-2* and *si-3* plants are short but less compact than *si-1* plants. In *si-2* plants, the first internodes are short, leading to a 'bird's nest' phenotype; later internodes are not modified. In *si-3* plants, internode length is reduced at all plant development stages. Fasciation of the main stem (reaching up to 15 cm) in the Charentais type line Vilmorin 104 was controlled by a single recessive gene, named *fas*, *fascinated* (Gabillard and Pitrat, 1988).

Flower Genes

Sex determination in melon is controlled by two major genes, *a* and *g*. The *andromonoecious* gene *a* (Rosa, 1928; Poole and Grimball, 1939; Wall, 1967) controls the monoecious versus andromonoecious sex type in melon. The gene mapped to the linkage group II (Perin et al., 2002; Silberstein et al., 2003). The gene was recently cloned and was shown to encode an ACC synthase gene, *CmACS7*. The transition between monoecy and andromonoecy is conferred by a single substitution, which leads to an inactive form of this key enzyme in the ethylene biosynthesis (Boualem et al., 2008). Molecular markers linked to the gene (Noguera et al., 2005; Sinclair et al., 2006; Kim et al., 2010) and within the gene are available (Boualem et al., 2008).

The *gynoecious*, *g*, gene controls the transition of monoecious plants to gynoecious plants carrying only female flowers. The gene was mapped to a distal end of the linkage group V, opposite to the *Vat* gene. Positional cloning of the gene showed that the gene *G* encodes for a transcription factor of the WIP family, *CmWIP1*. The gynoecious allele *g* corresponds to the insertion of a transposable element, which epigenetically represses the expression of *CmWIP1* (Martin et al., 2009). A third gene, named *gy* (*gynomonoecious*, previously also called *n* or *M*), interacts with *a* and *g* to produce stable gynoecious plants in the gynoecious line WI 998 (Kenigsbuch and Cohen, 1987, 1990).

Five single recessive genes of male-sterility including *ms-1* to *ms-5* were reported in melon (Bohn and Whitaker, 1949; Bohn and Principe, 1964; Lozanov,

1983; McCreight and Elmstrom, 1984; Lecouviour et al., 1990) in (Pitrat, 1991, 2002). Each of these genes displays a unique phenotype. The five sterility genes were located in five different linkage groups (Pitrat, 1991; Park et al., 2009). McCreight (1983) and Pitrat (1991) reported loose linkages between red stem (*r*) and the *ms-1* gene, and between yellow green leaves (*yg*) and the *ms-2* gene, respectively. Park et al. (2009) mapped the *ms-3* gene to the linkage group 9 of the linkage map Deltex x TGR-1551, which corresponds to the linkage group VII.

A *Macrocalyx* dominant gene, *Mca*, was reported to control the presence of large, leafy sepals in staminate and hermaphrodite flowers in the Japanese cultivar Makuwa (Ganesan and Sambandam, 1979). Two recessive genes were reported to modify the color of petals; *gp* (*green petals*) and *gyc* (*greenish yellow corolla*) control the presence of a green corolla with venation or the presence of a greenish yellow corolla, instead of the normal yellow corolla (Mockaitis and Kivilaan, 1965; Zink, 1986).

Rosa (1928) reported that tricarpellary ovary was monogenically inherited over pentacarpellary ovary found in Cassaba melons; the gene, named *p* (*pentamerous*) was mapped to the linkage group XII, closely linked to the major QTL for CMV resistance (Dogimont et al., 2000; Perin et al., 2002; Essafi et al., 2009). A single recessive gene, *n* (*nectarless*), was reported to control the absence of nectar in all flowers in the mutant 40099 (Bohn, 1961).

Fruit Genes

Fruit shape was reported to be controlled by a single gene *O* (*Oval shape*), dominant to round, and associated with *andromonoecious* gene *a* (Wall, 1967). As early as 1928, Rosa (1928) noted the association of elongate fruit with pistillate flowers (monoecious plants) and globular fruit with perfect flowers (andromonoecious plants) in segregating populations. More recently, several fruit shape QTL were mapped in several populations to at least five linkage groups; one of them co-localized with the *a* locus on the linkage group II (Perin et al., 2002; Monforte et al., 2004; Eduardo et al., 2007; Fernandez-Silva et al., 2010; Díaz et al., 2011). Spherical fruit shape was also reported to be controlled by a single gene, *sp* (*spherical fruit shape*), recessive to an obtuse fruit shape (Lumsden, 1914; Bains and Kang, 1963); this gene may be the same as the gene *O*.

A single dominant gene, *Ec* (*Empty cavity*), was reported to control the presence of separated carpels at fruit maturity, leaving a cavity in PI 414723 fruit (*ec* in 'Védrantais') (Périn et al., 1999). The *Ec* gene was mapped to the linkage group III (Perin et al., 2002).

External fruit appearance. Rind color of melon fruit varieties includes white, yellow, orange, or green, and can be variegated. The white color of immature fruits was reported to be dominant to green immature fruits and controlled by a single gene, *Wi*, *White color of immature fruit* (Kubicki, 1962). The white color of mature fruits was, in contrast, reported to be controlled gene *w*, *white*, recessive to dark green fruit skin in a cross between Honeydew (*w*) and Smiths' Perfect cantaloupe (*W*, dark green) (Hughes, 1948). Melon rind color was shown to be based on different combinations of three major pigments, chlorophyll, carotenoids and naringerin-chalcone, a flavonoid pigment responsible for the yellow color of mature fruits in Yellow Canari melon type (Tadmor et al., 2010). Accumulation of naringerin-chalcone was reported to be inherited as a monogenic dominant trait in the cross 'Noy Amid' (yellow rind) x 'Tendral Verde Tardio' (dark green rind); accumulation of chlorophyll and carotenoids segregates jointly as a single dominant gene, independent to naringerin-chalcone accumulation (Tadmor et al., 2010). We propose to name *Nca* the gene, which regulates *naringerin-chalcone accumulation* (versus non-accumulation), and *Chl* and *Car* the two linked genes, which control *chlorophyll* and *carotenoid accumulation* in the rind of mature fruit, respectively. In addition, minor genes likely control quantitative variation of the accumulation of these pigments. A polygenic control of the external fruit color was reported in the cross 'Piel de Sapo' x PI 161375 (Whitaker and Davis, 1962; Monforte et al., 2004; Eduardo et al., 2007; Obando et al., 2008).

Vein tracts, formerly and incorrectly referred to as sutures, on the fruit rind was reported to be controlled by a single recessive gene *s*, *sutures* (Bains and Kang, 1963; Davis 1970). The same inheritance was found in two crosses: 'Védrantais' (*s-2*, presence of sutures) x PI 161375 (*S-2*, without sutures) and 'Védrantais' x PI 414723 (*S-2*). The *s-2* gene was mapped to the linkage group XI (Perin et al., 2002). Stripes on the rind was reported to have a monogenic recessive inheritance (gene *st* for *striped epicarp*) by (Hagiwara and Kamimura, 1936). The presence of stripes on young fruits of 'Dulce' (before netting development) was also reported to be controlled by a single recessive gene, *st-2* (*striped epicarp-2*), in the cross Dulce (*st-2*) x PI 414723 (*St-2*, non-striped) (Danin-Poleg et al., 2002); the gene *st-2* was mapped to the linkage group XI. Further studies would be required to clarify the relationship between *st-2* and *s-2*, also located in the linkage group XI.

The ridge fruit surface was reported to be controlled by a single gene, *ri* (*ridge* in C68), recessive to ridgeless (*Ri* in 'Pearl') (Takada et al., 1975). The speckled epidermis of the fruit is controlled by a single recessive

sive gene, *spk* (*speckled fruit epidemis*) in PI 414723 (*Spk* in 'Védrantais') and was mapped to the linkage group VII (Perin et al., 2002). A single gene, *Mt* (*Mottled rind pattern*), was reported to control a mottled rind in 'Annamalai', dominant to uniform color *mt* in 'Makuwa' (Ganesan, 1988). The presence of dark spots (about 1 cm in diam.) on the rind (versus no spots) has a monogenic recessive inheritance in crosses Védrantais (*Mt-2*) x PI 161375 (*mt-2*) and Védrantais (*Mt-2*) x PI 414723 (*mt-2*), as the F₁ fruits have a uniform color rind (Périn et al., 1999); it was erroneously named *Mt-2* in the previous gene list. *mt-2* was mapped to the linkage group II (Perin et al., 2002).

A single dominant gene governing the development of net tissue, regardless of the degree of netting was reported in BIZ in a cross with smooth-skinned PI 414723 (Herman et al., 2008). We propose to name the gene *Rn* (*Rind netting*) instead of *N*. The gene was mapped to the linkage group II, closely linked to *fom1.2a* for Fusarium wilt resistance; additional minor loci likely affect the density of the net (Herman et al., 2008). Several QTL for the height and the width of the net in 'Deltex' were detected in a cross between netted 'Deltex' and net-free TGR-1551 (Park et al., 2009).

Melon fruit flesh color has been proposed to be controlled by two genes, *gf* for *green flesh* in Honeydew, recessive to orange flesh (*Gf* in Smiths' Perfect cantaloupe) (Hughes, 1948) and *wf* for *white flesh* (Iman et al., 1972). Genetic control of melon mesocarp color has, however, not been clearly elucidated and likely differs among market types. Clayberg (1992) confirmed that green and white mesocarps are recessive to orange and indicated that *gf* and *wf* interact epistatically. Mesocarp color (orange vs. green) segregated as a single recessive gene in recombinant inbred lines derived from orange flesh Védrantais x green flesh PI 161375 (Perin et al., 2002) and orange flesh AR 5 x green flesh Harukai N°3 (Fukino et al., 2008). The segregating gene, named *gf*, proposed to be renamed *wf*, mapped to the linkage group IX. In F₂ and doubled haploid lines derived from the cross between green mesocarp PI 161375 and white mesocarp Piel de Sapo T111, individuals with orange mesocarp were observed at a low frequency (Monforte et al., 2004); a single recessive gene segregated, if orange mesocarp phenotype was excluded and mapped to the linkage group VIII (formerly G1) (Monforte et al., 2004). Several QTL for fruit flesh color were described in near isogenic lines derived from the same cross (Eduardo et al., 2007; Obando et al., 2008). Recently, three QTL associated with color variation (white, green, orange) with putative epistatic interaction were identified in the cross between the white-fleshed Chinese line Q3-2-2 and orange-fleshed 'Top Mark' (Cuevas et al., 2009; Cuevas et

al., 2010). Five QTL associated with beta-carotene content, which is related to color intensity of the mesocarp, were identified in the cross between two orange-fleshed genotypes, USDA 846-1 and 'Top Mark' (Cuevas et al., 2008).

Sweet melon cultivars are characterized by high sucrose and low acid levels in mature fruit flesh. A single, incompletely recessive gene, *suc*, controlled accumulation of sucrose in the cross between the low sucrose Faqqous (var. *flexuosus*) and the high sucrose 'Noy Yizre'el' (Burger et al., 2002). Several QTL associated with total soluble solid content and sugar content have been described in several populations (Monforte et al., 2004; Sinclair et al., 2006; Park et al., 2009; Harel-Beja, 2010).

A dominant gene, *So* (*Sour*) was reported to control high acidity in melon fruit (Kubicki, 1962). A single dominant gene, *So-2* (*Sour-2*) for *sour taste* of the mature fruit, was also reported in PI 414723 (Périn et al., 1999; Burger et al., 2003). A single recessive gene, *pH*, was reported to control fruit flesh acidity in PI 414723. Low pH value in PI 414723 was dominant to high pH value in 'Dulce'. The *pH* gene was mapped to the linkage group VIII (Danin-Poleg et al., 2002); it likely corresponds to *So-2*.

While ripe melon fruits usually do not have a bitter taste, young fruits are divided into two types: bitter and non-bitter. A single dominant gene, *Bif-1* (*Bitter fruit-1*, formerly *Bif*), was reported to control the strong bitter taste of tender fruits in Indian wild melon (Parthasarathy and Sambandam, 1981). A monogenic dominant inheritance for the bitterness of young fruits was confirmed in wild melons from Africa and China (Ma et al., 1997). The cross of non-bitter melon lines (var. *conomon* and var. *makuwa*) with var. *inodorus* and var. *cantalupensis*) yielded, however, bitter young melons, which suggests complementary gene action of two independent genes, *Bif-2* and *Bif-3* (*Bif-2_ Bif-3_* are bitter; *bif-2bif-2 Bif-3_* and *Bif-2_ bif-3bif-3* are non-bitter) (Ma et al., 1997). One of them may be the same as *Bif-1*. The relationship with the gene *Bi* controlling seedling bitterness (Lee and Janick, 1978) is unknown.

While the single dominant gene *Mealy*, *Me*, was reported to control mealy flesh texture by Ganesan (1988) in an accession named *C. callosus* crossed with a crisp-fleshed 'Makuwa', a monogenic recessive inheritance was found for the mealy flesh texture in the var. *momordica* accession PI 414723 (*me-2*) crossed by 'Védrantais' (*Me-2*) (Périn et al., 1999) (included erroneously as *Me-2* in the previous gene list, it is now included as *me-2*). A monogenic recessive inheritance was reported for the juicy character of melon fruit flesh; the gene was named *juicy flesh*, symbolized *jf* (Chadha et al., 1972). A single gene was reported to control the

musky flavor of *C. melo callosus* (*Mu*, *Musky*), dominant to the mild flavor in 'Makuwa' or 'Annamalai' (*mu*) (Ganesan, 1988).

Fruit abscission at maturity was reported to be controlled by two independent loci in two independent studies. In absence of allelism tests, the genes were named abscission layer *Al-1* and *Al-2* in C68, *al-1* and *al-2* in 'Pearl' (Takada et al., 1975), and *Al-3* and *Al-4* in the climacteric Charentais type 'Védrantais' (Perin et al., 2002). *Al-3* and *Al-4* were mapped to the linkage groups VIII and IX in a recombinant inbred population derived from a cross between 'Védrantais' and the non-climacteric PI 161375 (Perin et al., 2002). A single dominant

gene, *Al-5*, was reported to control fruit abscission layer formation in the climacteric western shipper type 'TAM Uvalde' in the cross with the non-climacteric Casaba type 'TAM Yellow Canary' (Zheng et al., 2002).

Organogenic competence varies among melon genotypes. *In vitro* shoot regeneration capacity was reported to be controlled by two independent genes, partially dominant, *Org-1* and *Org-2* (*Organogenic* response) (Molina and Nuez, 1996). A single dominant gene, *Org-3*, was reported to control the high regeneration competence in the line BU-21/3, in crosses with the low regeneration competent lines 'PMR 45' and 'Ananas Yokneam' (Galperin et al., 2003).

Table 1. Reported host plant resistance and morphological genes of melon, including genes symbol, synonyms, descriptions, and linkage groups.^z

Gene symbol				
Preferred	Synonym	Character	LG ^y	References
a	M	andromonoecious. Mostly staminate, fewer perfect flowers; on A_ plants, pistillate flowers have no stamens; epistatic to g.	4, II	(Rosa, 1928 ; Poole and Grimball, 1939; Wall, 1967)
<i>ab</i>	-	<i>abrachiate</i> . Lacking lateral branches. Interacts with <i>a</i> and <i>g</i> , e.g., <i>abab aa G_</i> plants produce only staminate flowers.		(Foster and Bond, 1967)
Ac	-	<i>Alternaria cucumerina</i> resistance, in MR-1.		(Thomas et al., 1990)
<i>Af</i>	-	<i>Aulacophora foveicollis</i> resistance. Resistance to the red pumpkin beetle.		(Vashistha and Choudhury, 1974)
Ag	-	<i>Aphis gossypii</i> tolerance. Freedom of leaf curling following aphid infestation; in PI 414723.		(Bohn et al., 1973)
<i>Ala</i>	-	<i>Acute leaf apex</i> . Dominant over obtuse apex, linked with <i>Lobed</i> leaf, <i>Ala</i> in Maine Rock, <i>ala</i> in PV Green.		(Ganesan and Sambandam, 1985)
alb	-	<i>albino</i>. White cotyledons, lethal mutant; in Trystorp.		(Besombes et al., 1999)
<i>Al-1</i>	<i>Al₁</i>	<i>Abscission layer-1</i> . One of two dominant genes for abscission layer formation, <i>Al-1Al-2</i> in C68, <i>al-1al-2</i> in Pearl. See <i>Al-2</i> .		(Takada et al., 1975)
<i>Al-2</i>	<i>Al₂</i>	<i>Abscission layer-2</i> . One of two dominant genes for abscission layer formation. See <i>Al-1</i> .		(Takada et al., 1975)
Al-3		<i>Abscission layer-3</i>. One dominant gene for abscission layer formation in PI 161375. Relationship with <i>Al-1</i> or <i>Al-2</i> is unknown	VIII	(Perin et al., 2002)
Al-4		<i>Abscission layer-4</i>. One dominant gene for abscission layer formation in PI 161375. Relationship with <i>Al-1</i> or <i>Al-2</i> is unknown	IX	(Perin et al., 2002)
Al-5	-	<i>Abscission layer-5</i>. One dominant gene for abscission layer formation; full-slip in TAM Uvalde.		(Zheng et al., 2002)
<i>bd</i>	-	<i>brittle dwarf</i> . Rosette growth with thick leaf. Male fertile, female sterile; in TAM-Perlita 45.		(Cox, 1985)
Bi	-	<i>Bitter</i>. Bitter seedling. Common in honeydew or in Charentais type while most American cantaloupes are <i>bi</i>.		(Lee and Janick, 1978)
<i>Bif-1</i>	<i>Bif</i>	<i>Bitter fruit-1</i> . Bitterness of tender fruit in wild melon. Relation with <i>Bi</i> is unknown.		(Parthasarathy and Sambandam, 1981)
<i>Bif-2</i>	-	<i>Bitter fruit-2</i> . One of two complementary independent genes for bitter taste in young fruit: <i>Bif-2_ Bif-3_</i> are bitter. Relationships with <i>Bi</i> and <i>Bif-1</i> are unknown.		(Ma et al., 1997)
<i>Bif-3</i>	-	<i>Bitter fruit-3</i> . One of two complementary independent genes for bitter taste in young fruit: <i>Bif-2_ Bif-3_</i> are bitter. Relationships with <i>Bi</i> and <i>Bif-1</i> are unknown.		(Ma et al., 1997)
cab-1	-	<i>cucurbit aphid borne yellows virus resistance-1</i>. One of two complementary independent genes for resistance to this polerovirus: <i>cab-1cab-1 cab-2cab-2</i> plants are resistant; in PI 124112.		(Dogimont et al., 1997)
cab-2	-	<i>cucurbit aphid borne yellows virus resistance-2</i>. One of two complementary independent genes for resistance to this polerovirus: <i>cab-1cab-1 cab-2cab-2</i> plants are resistant; in PI 124112.		(Dogimont et al., 1997)
Car		<i>Carotenoids accumulation</i> in the rind of mature fruit. A single dominant gene for the accumulation of these pigments in the rind of mature fruit versus non accumulation; <i>Car</i> in Tendral Verde Tardio, <i>car</i> in Noy Amid (Canary type); linked to <i>Chl</i>.		(Tadmor et al., 2010)

Gene symbol				
Preferred	Synonym	Character	LG ^y	References
<i>cb</i>	<i>cbj</i>	<i>cucumber beetle</i> resistance. Interacts with <i>Bi</i> , the nonbitter <i>bibi cbcb</i> being the more resistant; in C922-174-B.		(Nugent et al., 1984)
<i>cf</i>	-	<i>cochleare folium</i>. Spoon-shaped leaf with upward curling of the leaf margins; spontaneous mutant in Galia.		(Lecouviour et al., 1995)
<i>cgmmv-1</i>		<i>cucumber green mottle mosaic virus resistance-1</i> . One of two complementary genes for resistance to this tobamovirus in Chang Bougi.		(Sugiyama et al., 2007)
<i>cgmmv-2</i>		<i>cucumber green mottle mosaic virus resistance-2</i> . One of two complementary genes for resistance to this tobamovirus in Chang Bougi.		(Sugiyama et al., 2007)
<i>Chl</i>		<i>Chlorophyll accumulation</i> in the rind of mature fruit. A single dominant gene for chlorophyll accumulation in the rind of mature fruit versus non-accumulation; <i>Chl</i> in Tendral Verde Tardio, <i>chl</i> in Noy Amid, (Canary type), linked to <i>Car</i>.		(Tadmor et al., 2010)
<i>cl</i>	-	<i>curled leaf</i> . Elongated leaves that curl upward and inward. Usually male and female sterile.		(Cox, 1985)
<i>Creb-2</i>		<i>Cucumber mosaic virus resistance</i> . A single dominant gene for resistance to this cucumovirus in Yamatouri		(Daryono et al., 2010)
<i>culcrv</i>		<i>cucurbit leaf crumple virus resistance</i>. A single recessive gene for resistance to this geminivirus transmitted by whitefly in PI 313970.		(McCreight et al., 2008)
<i>Cys</i>	-	<i>Cucurbit yellow stunting</i> disorder virus resistance. One dominant gene for resistance to this crinivirus in TGR-1551.		(Lopez-Sese and Gomez-Guillamon, 2000)
<i>dc-1</i>	-	<i>Dacus cucurbitae-1</i> resistance. One of two complementary recessive genes for resistance to the melon fruitfly. See <i>dc-2</i> .		(Sambandam and Chelliah, 1972)
<i>dc-2</i>	-	<i>Dacus cucurbitae-2</i> resistance. One of two complementary recessive genes for resistance to the melon fruitfly. See <i>dc-1</i> .		(Sambandam and Chelliah, 1972)
<i>dl</i>	-	<i>dissected leaf</i>. Highly indented leaves in URSS 4.		(Dyutin, 1967)
<i>dl'</i>	<i>cl</i>	<i>dissected leaf Velich</i>. First described as <i>cut leaf</i> in Cantaloup de Bellegarde. Allelic to <i>dl</i>.		(Velich and Fulop, 1970)
<i>dl-2</i>	-	<i>dissected leaf-2</i> . First described as « hojas hendidas ».		(Esquinas Alcazar, 1975)
<i>dlet</i>	<i>dl</i>	<i>delayed lethal</i> . Reduced growth, necrotic lesions on leaves and premature death.		(Zink, 1990)
<i>Ec</i>	-	<i>Empty cavity</i>. Carpels are separated at fruit maturity leaving a cavity; <i>Ec</i> in PI 414723, <i>ec</i> in Védraçais.	III	(Périn et al., 1999)
<i>ech</i>	-	<i>exaggerated curvature of the hook</i>. Triple response of seedlings germinating in darkness in presence of ethylene; <i>ech</i> in PI 161375, <i>Ech</i> in Védraçais.	I	(Perin et al., 2002)
<i>f</i>	-	<i>flava</i>. Chlorophyll deficient mutant. Growth rate reduced in K 2005.	8	(Pitrat et al., 1986)
<i>fas</i>	-	<i>fasciated stem</i>, in Vilmorin 104.		(Gabillard and Pitrat, 1988)
<i>fe</i>	-	<i>fe</i> (iron) inefficient mutant. Chlorotic leaves with green veins that turn green when adding iron in the nutrient solution.		(Nugent and Bhella, 1988; Jolley et al., 1991)
<i>Fn</i>	-	<i>Flaccida necrosis</i>. Semi-dominant gene for wilting and necrosis reactions to F pathotype of <i>Zucchini yellow mosaic virus</i>; <i>Fn</i> in Doublon, <i>fn</i> in Védraçais).	2, V	(Risser et al., 1981)
<i>Fom-1</i>	<i>Fom₁</i>	<i>Fusarium oxysporum melonis</i> resistance. Resistance to races 0 and 2 and susceptibility to races 1 and 1.2 of <i>Fusarium wilt</i>; <i>Fom-1</i> in Doublon, <i>fom-1</i> in Charentais T.	5, IX	(Risser, 1973)

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<i>Fom-2</i>	<i>Fom1,2</i>	<i>Fusarium oxysporum melonis</i> resistance. Resistance to races 0 and 1 and susceptibility to races 2 and 1.2 of <i>Fusarium wilt</i> ; <i>Fom-2</i> in CM 17187, <i>fom-2</i> in Charentais T.	6, XI	(Risser, 1973)
<i>Fom-3</i>	-	<i>Fusarium oxysporum melonis</i> resistance. Same phenotype as <i>Fom-1</i> but segregates independently from <i>Fom-1</i> ; <i>Fom-3</i> in Perlita FR, <i>fom-3</i> in Charentais T.		(Zink and Gubler, 1985)
<i>fom1.2a</i>		<i>Fusarium oxysporum melonis</i> resistance. Resistance to race 1.2 of <i>Fusarium wilt</i> ; <i>fom1.2a</i> in BIZ, <i>Fom1.2a</i> in PI 414723.	II	(Herman et al., 2008)
<i>fom-4</i>		<i>Fusarium oxysporum melonis</i> resistance. Resistance to race 0 and 2 of <i>Fusarium wilt</i> , <i>fom-4</i> in Tortuga, likely associated with <i>Fom-1</i> .		(Oumouloud et al., 2010)
<i>g</i>	-	gynoecious . Controls the presence of one (<i>g</i>) or two (<i>G</i>) types of flowers on one plant. Epistatic to <i>a</i> : <i>A_ G_</i> monoecious; <i>A_ gg</i> gynoecious; <i>aa G_</i> andromonoecious; <i>aa gg</i> hermaphrodite.		(Poole and Grimball, 1939)
<i>gf</i>	-	green flesh color . Recessive to salmon, <i>gf</i> in honeydew, <i>Gf</i> in Smiths' Perfect cantaloupe.	VIII	(Hughes, 1948)
<i>gl</i>	-	glabrous . Trichomes lacking in Arizona glA.	3	(Foster, 1963)
<i>gp</i>	-	<i>green petals</i> . Corolla leaf like in color and venation.		(Mockaitis and Kivilaan, 1965)
<i>Gs</i>	-	<i>Gelatinous sheath</i> around the seeds. Dominant to absence of gelatinous sheath.		(Ganesan, 1988)
<i>Gsb-1</i>	<i>Mc</i>	Gummy stem blight resistance-1 . High degree of resistance to <i>Didymella bryoniae</i> (= <i>Mycosphaerella citrullina</i>) in PI 140471.		(Prasad and Norton, 1967; Frantz and Jahn, 2004)
<i>Gsb-2</i>	<i>Mc-3</i>	<i>Gummy stem blight</i> resistance-2. High level of resistance to <i>Didymella bryoniae</i> (= <i>Mycosphaerella citrullina</i>) in PI 157082), independent from <i>Gsb-1</i> , <i>Gsb-3</i> , <i>Gsb-4</i> and <i>gsb-5</i> .		(Zuniga et al., 1999; Frantz and Jahn, 2004)
<i>Gsb-3</i>	<i>Mc-4-</i>	Gummy stem blight resistance-3 . High level of resistance to <i>Didymella bryoniae</i> (= <i>Mycosphaerella citrullina</i>) in PI 511890, independent from <i>Gsb-1</i> , <i>Gsb-2</i> , <i>Gsb-4</i> and <i>gsb-5</i> .		(Zuniga et al., 1999; Frantz and Jahn, 2004)
<i>Gsb-4</i>	-	Gummy stem blight resistance-4 . High level of resistance to <i>Didymella bryoniae</i> (= <i>Mycosphaerella citrullina</i>) in PI 482398, independent from <i>Gsb-1</i> , <i>Gsb-2</i> , <i>Gsb-3</i> and <i>gsb-5</i> .		(Frantz and Jahn, 2004)
<i>gsb-5</i>	-	gummy stem blight resistance-5 . High level of resistance to <i>Didymella bryoniae</i> (= <i>Mycosphaerella citrullina</i>) in PI 482399, independent from <i>Gsb-1</i> , <i>Gsb-2</i> , <i>Gsb-3</i> and <i>Gsb-4</i> .		(Frantz and Jahn, 2004)
<i>Gsb-6</i>	<i>Mcⁱ</i> , <i>Mc-2</i>	<i>Mycosphaerella citrullina</i> resistance-2. Moderate degree of resistance to gummy stem blight in C-1 and C-8.		(Prasad and Norton, 1967)
<i>gyc</i>	-	greenish yellow corolla .		(Zink, 1986)
<i>gy</i>	<i>n, M</i>	gynomonoecious . Interacts with <i>a</i> and <i>g</i> to produce stable gynoecious plants (<i>A_ g gy gy</i>) in WI 998.		(Kenigsbuch and Cohen, 1987, 1990)
<i>h</i>	-	halo cotyledons . Yellow halo on the cotyledons, later turning green.	4, II	(Nugent and Hoffman, 1974)
<i>lmy</i>	-	<i>Interveinal mottling and yellowing</i> resistance. Resistance to a complex of viruses, in PI 378062.		(Hassan et al., 1998)
<i>jf</i>	-	<i>juicy flesh</i> . Segregates discretely in a monogenic ratio in segregating generations.		(Chadha et al., 1972)
<i>L</i>	-	<i>Lobed leaf</i> . Dominant on non lobed, linked with <i>Acute leaf apex L</i> in Maine Rock, <i>l</i> in P.V. Green.		(Ganesan and Sambandam, 1985)

Gene symbol				
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<i>Liy</i>	-	Lettuce infectious yellows virus resistance. One dominant gene for resistance to this crinivirus in PI 313970.		(McCreight, 2000)
<i>lmi</i>	-	long mainstem internode. Affects internode length of the main stem but not of the lateral ones in 48764.	8	(McCreight, 1983)
<i>Lt</i>	-	Liriomyza trifolii (leafminer) resistance in Nantais Oblong.		(Dogimont et al., 1999)
<i>M-Pc-5</i>	-	<i>Modifier of Pc-5. Gene Pc-5 for downy mildew resistance is dominant in presence of M-Pc-5, recessive in the absence of M-Pc-5.</i>		(Angelov and Krasteva, 2000)
<i>Mca</i>	-	<i>Macrocalyx. Large, leaf like structure of the sepals in staminate and hermaphrodite flowers; Mca in makuwa, mca in Annamalai.</i>		(Ganesan and Sambandam, 1979)
<i>Me</i>	-	<i>Mealy flesh texture. Dominant to crisp flesh; Me in C. callosus, me in makuwa.</i>		(Ganesan, 1988)
<i>me-2</i>	-	mealy flesh texture-2 in PI 414723.		(Périn et al., 1999)
<i>Mnr-1</i>	<i>Mnr1</i>	Melon necrotic resistance 1. One of two dominant genes for resistance to Melon necrotic spot virus (MNSV) located at 19 cM from nsv; Mnr-1 in Doublon, mnr-1 in ANC-42.	XII	(Mallor Gimenez et al., 2003)
<i>Mnr-2</i>	<i>Mnr2</i>	Melon necrotic resistance 2. One of two dominant genes for resistance to Melon necrotic spot virus (MNSV) independent from Mnr-1; Mnr-2 in Doublon, mnr-2 in ANC-42.		(Mallor Gimenez et al., 2003)
<i>ms-1</i>	<i>ms¹</i>	male sterile-1. Inehiscent anthers with empty pollen walls in tetrad stage.	3	(Bohn and Whitaker, 1949)
<i>ms-2</i>	<i>ms²</i>	male sterile-2. Anthers indehiscent, containing mostly empty pollen walls, growth rate reduced.	6, XI	(Bohn and Principe, 1964)
<i>ms-3</i>	<i>ms-L</i>	male sterile-3. Waxy and translucent indehiscent anthers, containing two types of empty pollen sacs.	12, VII	(McCreight and Elmstrom, 1984)
<i>ms-4</i>	-	male sterile-4. Small indehiscent anthers. First male flowers abort at bud stage in Bulgaria 7.	9	(Lozanov, 1983)
<i>ms-5</i>	-	male sterile-5. Small indehiscent anthers. Empty pollen in Jivaro, Fox.	13	(Lecouviour et al., 1990)
<i>Mt</i>	-	<i>Mottled rind pattern. Dominant to uniform color. Epistatic with Y (not expressed in Y_) and st (Mt_ st st and Mt_ St_ mottled; mt mt st st striped, mt mt St_ uniform); Mt in Annamalai, mt in Makuwa.</i>		(Ganesan, 1988)
<i>mt-2</i>	-	mottled rind pattern in PI 161375.	II	(Périn et al., 1999)
<i>Mu</i>	-	<i>Musky flavor (olfactory). Dominant on mild flavor; Mu in C. melo callosus, mu in Makuwa or Annamalai.</i>		(Ganesan, 1988)
<i>Mvd</i>	-	<i>Melon vine decline resistance in Pat 81. Semi-dominant gene for partial resistance to Acremonium cucurbitacearum and Monosporascus cannonballus.,</i>		(Iglesias et al., 2000)
<i>My</i>	-	Melon yellows virus resistance. Semi-dominant gene for partial resistance to this crinivirus, in Nagata Kin Makuwa.		(Esteva and Nuez, 1992; Nuez et al., 1999)
<i>n</i>	-	nectarless. Nectaries lacking in all flowers of 40099.		(Bohn, 1961)
<i>Nca</i>		naringerin-chalcone accumulation in the rind of mature fruit. A single dominant gene for the accumulation of this flavonoid pigment in the rind of mature fruit versus non-accumulation; Nca in Noy Amid, Canary type, nca in Tendral Verde Tardio.		(Tadmor et al., 2010)
<i>Nm</i>	-	Necrosis with Morocco strains of Watermelon mosaic virus, a potyvirus; Nm in Védreantais, nm in Ouzbèque.		(Quiot-Douine et al., 1988)

Gene symbol				
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<i>nsv</i>	-	Melon <i>necrotic spot virus</i> resistance. A single recessive gene for resistance to this carmovirus in Gulfstream, Planters Jumbo.	7, XII	(Coudriet et al., 1981)
O	-	Oval fruit shape. Dominant to round, associated with a .		(Wall, 1967)
<i>Org-1</i>	-	<i>Organogenic</i> response for <i>in vitro</i> shoot regeneration. Partially dominant. Interacts with an additive model with <i>Org-2</i> .		(Molina and Nuez, 1996)
<i>Org-2</i>	-	<i>Organogenic</i> response for <i>in vitro</i> shoot regeneration. Partially dominant. Interacts with an additive model with <i>Org-1</i> .		(Molina and Nuez, 1996)
<i>Org-3</i>	-	<i>Organogenic</i> response for <i>in vitro</i> regeneration. Dominant allele for high response in BU-12/3, recessive allele in PMR 45 or Ananas Yokneam. Probably different from <i>Org-1</i> and <i>Org-2</i> .		(Galperin et al., 2003)
p	-	pentamerous . Five carpels and stamens; recessive to trimerous; in Casaba.	XII	(Rosa, 1928)
Pa	-	Pale green foliage. PaPa plants are white (lethal); Papa are yellow; in 30567.	3	(McCreight and Bohn, 1979)
Pc-1	-	Pseudoperonospora cubensis resistance. One of two complementary incompletely dominant genes for downy mildew resistance in PI 124111. See Pc-2 .		(Cohen et al., 1985; Thomas et al., 1988)
Pc-2	-	Pseudoperonospora cubensis resistance. One of two complementary incompletely dominant genes for downy mildew resistance in PI 124111). See Pc-1 .		(Cohen et al., 1985; Thomas et al., 1988)
Pc-3	-	Pseudoperonospora cubensis resistance. Partial resistance to downy mildew in PI 414723.		(Epinat and Pitrat, 1989)
Pc-4	-	Pseudoperonospora cubensis resistance. One of two complementary genes for downy mildew resistance in PI 124112. Interacts with Pc-1 or Pc-2 .		(Kenigsbuch and Cohen, 1992)
Pc-5	-	Pseudoperonospora cubensis resistance. One gene in Line 5-4-2-1 which interacts with M-Pc-5 in the susceptible line K15-6; Pc-5 is dominant in presence of M-Pc-5 , recessive in the absence of M-Pc-5 .		(Angelov and Krasteva, 2000)
pH	-	pH (acidity) of the mature fruit flesh. Low pH value in PI 414723 dominant to high pH value in Dulce.	VIII	(Danin-Poleg et al., 2002)
pin	-	pine-seed shape in PI 161375.	III	(Perin et al., 2002)
Pm-1	Pm¹ Pm-A ?	Powdery mildew resistance-1. Resistance to race 1 of <i>Podosphaera xanthi</i> in PMR 45.		(Jagger et al., 1938)
Pm-2	Pm² Pm-C ?	Powdery mildew resistance-2. Interacts with Pm-1 ; Resistance to race 2 of <i>Podosphaera xanthii</i> in PMR 5 with Pm-1 .		(Bohn and Whitaker, 1964)
Pm-3	Pm³	Powdery mildew resistance-3. Resistance to race 1 of <i>Podosphaera xanthii</i> in PI 124111.	7	(Harwood and Markarian, 1968, 1968)
Pm-4	Pm⁴	Powdery mildew resistance-4. Resistance to <i>Podosphaera xanthii</i> in PI 124112.		(Harwood and Markarian, 1968, 1968)
Pm-5	Pm⁵	Powdery mildew resistance-5. Resistance to <i>Podosphaera xanthii</i> in PI 124112.		(Harwood and Markarian, 1968, 1968)
Pm-6	-	Powdery mildew resistance-6. Resistance to <i>Podosphaera xanthii</i> race 2 in PI 124111.		(Kenigsbuch and Cohen, 1989)
Pm-7	-	Powdery mildew resistance-7. Resistance to <i>Podosphaera xanthii</i> race 1 in PI 414723.		(Anagnostou et al., 2000)

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<i>Pm-8</i>		<i>Powdery mildew</i> resistance-8.. Resistance to <i>Podosphaera xanthii</i> race pxCh1 in PI 134198.		(Liu et al., 2010)
<i>Pm-E</i>	-	<i>Powdery mildew</i> resistance-E. Interacts with <i>Pm-C</i> in PMR 5 for <i>Golovinomyces cichoracearum</i> resistance.		(Epinat et al., 1993)
<i>Pm-F</i>	-	<i>Powdery mildew</i> resistance-F. Interacts with <i>Pm-G</i> in PI 124112 for <i>Golovinomyces cichoracearum</i> resistance.		(Epinat et al., 1993)
<i>Pm-G</i>	-	<i>Powdery mildew</i> resistance-G. Interacts with <i>Pm-F</i> in PI 124112 for <i>Golovinomyces cichoracearum</i> resistance.		(Epinat et al., 1993)
<i>Pm-H</i>	-	<i>Powdery mildew</i> resistance-H. Resistance to <i>Golovinomyces cichoracearum</i> and susceptibility to <i>Podosphaera xanthii</i> in Nantais oblong.		(Epinat et al., 1993)
<i>Pm-R</i>	-	<i>Powdery mildew</i> resistance-R. Resistance to <i>Podosphaera xanthii</i> races 1, 2, and 5 in TGR-1551.	V	(Yuste-Lisbona et al., 2011)
<i>pm-S</i>		<i>powdery mildew resistance-S</i> . Resistance to <i>Podosphaera xanthii</i> race S in PI 313970. Recessive to susceptibility in Top Mark.		(McCreight and Coffey, 2011)
<i>Pm-w</i>	<i>Pm-B ?</i>	<i>Powdery mildew</i> resistance-w. Resistance to <i>Podosphaera xanthii</i> race 2 in WMR 29.	2, V	(Pitrat, 1991)
<i>Pm-x</i>	-	<i>Powdery mildew</i> resistance-x. Resistance to <i>Podosphaera xanthii</i> in PI 414723.	4, II	(Pitrat, 1991)
<i>Pm-y</i>	-	<i>Powdery mildew</i> resistance-y. Resistance to <i>Podosphaera xanthii</i> in VA 435.	7, XII	(Pitrat, 1991)
<i>Pm-z</i>		<i>Powdery mildew</i> resistance-z. Resistance to <i>Podosphaera xanthii</i> races 1 and 2US in PI 313970.		(McCreight, 2003)
<i>PmV.1</i>	-	<i>Powdery mildew</i> resistance V.1. Resistance to <i>Podosphaera xanthii</i> races 1, 2 and 3 in PI 124112.	V	(Perchepped et al., 2005)
<i>PmXII.1</i>		<i>Powdery mildew</i> resistance XII.I. Resistance to <i>Podosphaera xanthii</i> races 1, 2 and 5 and to <i>Golovinomyces cichoracearum</i> race 1 in PI 124112	XII	(Perchepped et al., 2005)
<i>Prv¹</i>	<i>Wmv</i>	<i>Papaya Ringspot virus</i> resistance ¹ . Resistance to W strain of this potyvirus (formerly <i>Watermelon mosaic virus 1</i>) in PI 180280, and B66-5, WMR 29, which were derived from PI 180280. Dominant to <i>Prv²</i> .	5, IX	(Webb, 1979; Pitrat and Lecoq, 1983)
<i>Prv²</i>	-	<i>Papaya Ringspot virus</i> resistance ² . Allele at the same locus as <i>Prv¹</i> but different reaction with some strains of the virus; in 72-025, which was derived from PI 180283. Recessive to <i>Prv¹</i> .	5, IX	{Kaan, 1973}; Pitrat, 1983)
<i>Prv-2</i>	-	<i>Papaya Ringspot virus</i> resistance-2. Relationship with <i>Prv</i> is unknown; in PI 124112		(McCreight and Fashing-Burdette, 1996)
<i>r</i>	-	<i>red stem</i> . Red pigment under epidermis of stems, especially at nodes, and reddish or tan seed color; in PI 157083.	3	(Bohn, 1968; McCreight and Bohn, 1979)
<i>ri</i>	-	<i>ridge</i> . Ridged fruit surface, recessive to ridgeless. (<i>ri</i> in C68, <i>Ri</i> in Pearl).		(Takada et al., 1975)
<i>Rn</i>	<i>N</i>	<i>Rind netting</i> . Netted fruit surface, regardless of the degree of netting; <i>Rn</i> in B12 dominant to smooth, non-netted rind, <i>m</i> in PI 414723.		(Herman et al., 2008)
<i>s</i>	-	<i>sutures</i> . Presence of vein tracts on the fruit (« sutures »); recessive to ribless.		(Bains and Kang, 1963)
<i>s-2</i>	-	<i>sutures-2</i> on the fruit rind of PI 161375. Relationship with <i>s</i> is unknown.	XI	(Périn et al., 1999)
<i>Sfl</i>	<i>S</i>	<i>Subtended floral leaf</i> . The floral leaf bearing the hermaphrodite flowers is sessile, small and encloses the flower; <i>Sfl</i> in makuwa, <i>sfl</i> in Annamalai.		(Ganesan and Sambandam, 1979)

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<i>si-1</i>	<i>b</i>	<i>short internode-1</i> . Extremely compact plant habit (bush type) in UC Topmark Bush.	1	(Denna, 1962)
<i>si-2</i>	-	<i>short internode-2</i> . Short internodes from 'birdnest' melon in Persia 202.		(Paris et al., 1984)
<i>si-3</i>	-	<i>short internode-3</i> . Short internodes in Maindwarf.		(Knavel, 1990)
<i>slb</i>	<i>sb</i>	<i>short lateral branching</i> . Reduction of the elongation of the lateral branches, in LB-1		(Ohara et al., 2001)
<i>So</i>	-	<i>Sour</i> taste. Dominant to sweet.		(Kubicki, 1962)
<i>So-2</i>	-	<i>Sour taste-2</i> . Relationship with <i>So</i> is unknown, in PI 414723.		(Périn et al., 1999)
<i>sp</i>	-	<i>spherical</i> fruit shape. Recessive to obtuse; dominance incomplete.		(Lumsden, 1914; Bains and Kang, 1963)
<i>spk</i>	-	<i>speckled</i> fruit epidermis; <i>spk</i> in PI 161375 and PI 414723, <i>Spk</i> in Védrantais.	VII	(Perin et al., 2002)
<i>sqmv</i>		<i>squash mosaic virus</i> resistance. A single recessive gene for resistance to this Comovirus in China 51.		(Provvidenti, 1998)
<i>st</i>	-	<i>striped</i> epicarp. Recessive to non-striped.		(Hagiwara and Kamimura, 1936)
<i>st-2</i>	<i>st</i>	<i>striped epicarp-2</i> . Present in Dulce, recessive to non-striped in PI 414723. Relationship with <i>st</i> is unknown.	XI	(Danin-Poleg et al., 2002)
<i>suc</i>		<i>sucrose</i> accumulation. Low sucrose level in Faqqous (<i>suc</i>), high sucrose in Noy Yizre'el (<i>Suc</i>). Incomplete recessivity.		(Burger et al., 2002)
<i>v</i>	-	<i>virescent</i> . Pale cream cotyledons and hypocotyls, and yellow green foliage, mainly young leaves.		(Hoffman and Nugent, 1973)
<i>v-2</i>	-	<i>virescent-2</i> .		(Dyutin, 1979)
<i>v-3</i>	-	<i>virescent-3</i> . White cotyledons which turn green, light green young leaves which are normal when they are older.		(Pitrat et al., 1995)
<i>Vat</i>	-	<i>Virus aphid transmission</i> resistance. Resistance to several viruses when transmitted by <i>Aphis gossypii</i> , in PI 161375.	2, V	(Pitrat and Lecoq, 1980)
<i>w</i>	-	<i>white</i> color of mature fruit. Recessive to dark green fruit skin; <i>w</i> in honeydew, <i>W</i> in Smiths' Perfect cantaloupe.		(Hughes, 1948)
<i>wf</i>	-	<i>white flesh</i> . Recessive to salmon. <i>Wf</i> epistatic to <i>Gf</i> .	IX	(Iman et al., 1972; Clayberg, 1992)
<i>Wi</i>	-	White color of <i>immature</i> fruit. Dominant to green.		(Kubicki, 1962)
<i>Wmr</i>	-	<i>Watermelon mosaic virus</i> (formerly <i>Watermelon mosaic virus 2</i>) resistance. A single dominant gene, in PI 414723	II	(Gilbert et al., 1994)
<i>wmr-2</i>		<i>Watermelon mosaic virus</i> (formerly <i>Watermelon mosaic virus 2</i>) resistance. A single recessive gene, in TGR-1551.		(Diaz-Pendon et al., 2005)
<i>Wt</i>	-	<i>White testa</i> . Dominant to yellow or tan seed coat color.		(Hagiwara and Kamimura, 1936)
<i>Wt-2</i>	-	<i>White testa-2</i> . Relationship with <i>Wt</i> unknown, in PI 414723.	IV	(Périn et al., 1999)
<i>Y</i>	-	<i>Yellow</i> epicarp. Dominant to white fruit skin.		(Hagiwara and Kamimura, 1936)

Gene symbol				
Preferred	Synonym	Character	LG ^y	References
<i>yg</i>	-	<i>yellow green</i> leaves. Reduced chlorophyll content.	6, XI	(Whitaker, 1952)
<i>yg^w</i>	<i>lg</i>	<i>yellow green Weslaco</i>. First described as <i>light green</i> in a cross Dulce x TAM-Uvalde. Allelic to <i>yg</i>.		(Cox and Harding, 1986)
<i>yv</i>	-	<i>yellow virescence</i>. Pale cotyledons; yellow green young leaves and tendrils; bright and yellow petals and yellow stigma; etiolated; older leaves becoming green.	1	(Zink, 1977)
<i>yv-2</i>	<i>yv-X</i>	<i>yellow virescence-2</i>. Young leaves yellow green, old leaves normal green	5, IX	(Pitrat, 1991)
<i>Zym</i>	<i>Zym-1</i>	<i>Zucchini Yellow Mosaic</i> virus resistance. Resistance to pathotype 0 of this potyvirus in PI 414723.	4, II	(Pitrat and Lecoq, 1984)
<i>Zym-2</i>	-	<i>Zucchini Yellow Mosaic</i> potyvirus resistance. One of three complementary genes (see <i>Zym</i> and <i>Zym-3</i>) for resistance to this potyvirus in PI 414723.		(DaninPoleg et al., 1997)
<i>Zym-3</i>	-	<i>Zucchini Yellow Mosaic</i> potyvirus resistance. One of three complementary genes (see <i>Zym</i> and <i>Zym-2</i>) for resistance to this potyvirus in PI 414723.		(DaninPoleg et al., 1997)

Maternally inherited genes.

<i>cyt-Yt</i>	-	<i>cytoplasmic yellow tip</i> . Chlorophyll deficient mutant with yellow young leaves, turning green when becoming older. Maternally inherited.		(Ray and McCreight, 1996)
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^z Genes maintained by the curators or very common in collections (like *andromonoecious* or *white testa*) are **emboldened**.

Genes that have been apparently lost or not maintained by curators, or have uncertain descriptions are normal in weight.

^yLinkage groups to which the genes belong are indicated as Arabic numbers, according to Pitrat (1991) and Roman numbers according to Périn et al. (2002).

Table 2. Reported melon isozyme genes, including gene symbol, synonym, description, location on the melon genome and reference.

Gene symbol		Gene description and type lines	LG ^z	References
Preferred	Synonym			
<i>Aco-1</i>	<i>Ac</i>	<i>Aconitase-1</i> . Isozyme variant with two alleles, each regulating one band, in PI 218071, PI 224769.	A	(Staub et al., 1998)
<i>Acp-1</i>	<i>APS-11, Ap-11</i>	<i>Acid phosphatase-1</i> . Isozyme variant with two codominant alleles, each regulating one band. The heterozygote has two bands.		(Esquinas Alcazar, 1981)
<i>Acp-2</i>	<i>Acp-1</i>	<i>Acid phosphatase-2</i> . Isozyme variant with two alleles, each regulating one band, in PI 194057, PI 224786. Relationship with <i>Acp-1</i> is unknown.		(Staub et al., 1998)
<i>Acp-4</i>	-	<i>Acid phosphatase-4</i> . Isozyme variant with two alleles, each regulating one band, in PI 183256, PI 224786. Relationship with <i>Acp-1</i> unknown, different from <i>Acp-2</i> .		(Staub et al., 1998)
<i>Ak-4</i>	-	<i>Adenylate kinase</i> . Isozyme variant with two alleles, each regulating one band, in PI 169334.		(Staub et al., 1998)
<i>Fdp-1</i>	-	<i>Fructose diphosphate-1</i> . Isozyme variant with two alleles, each regulating one band, in PI 218071, PI 224688.		(Staub et al., 1998)
<i>Fdp-2</i>	-	<i>Fructose diphosphate-2</i> . Isozyme variant with two alleles, each regulating one band, in PI 204691, PI 183256.		(Staub et al., 1998)
<i>Gpi</i>	-	<i>Glucosephosphate isomerase</i> . Isozyme variant with two alleles, each regulating one band, in PI 179680.		(Staub et al., 1998)
<i>Idh</i>	-	<i>Isocitrate dehydrogenase</i> . Isozyme variant with two alleles, each regulating one band, in PI 218070, PI 224688.	A	(Staub et al., 1998)
<i>Mdh-2</i>	-	<i>Malate dehydrogenase-2</i> . Isozyme variant with two alleles, each regulating one band, in PI 224688, PI 224769.	B	(Staub et al., 1998)
<i>Mdh-4</i>	-	<i>Malate dehydrogenase-4</i> . Isozyme variant with two alleles, each regulating one band, in PI 218070, PI 179923.	B	(Staub et al., 1998)
<i>Mdh-5</i>	-	<i>Malate dehydrogenase-5</i> . Isozyme variant with two alleles, each regulating one band, in PI 179923, PI 180283.	B	(Staub et al., 1998)
<i>Mdh-6</i>	-	<i>Malate dehydrogenase-6</i> . Isozyme variant with two alleles, each regulating one band, in PI 179923, PI 180283.	B	(Staub et al., 1998)
<i>Mpi-1</i>	-	<i>Mannosephosphate isomerase-1</i> . Isozyme variant with two alleles, each regulating one band, in PI 183257, PI 204691.	A	(Staub et al., 1998)
<i>Mpi-2</i>	-	<i>Mannosephosphate isomerase-2</i> . Isozyme variant with two alleles, each regulating one band, in PI 183257, PI 204691.	A	(Staub et al., 1998)
<i>Pep-gl</i>	-	<i>Peptidase with glycyl-leucine</i> . Isozyme variant with two alleles, each regulating one band, in PI 218070.	B	(Staub et al., 1998)
<i>Pep-la</i>	-	<i>Peptidase with leucyl-alanine</i> . Isozyme variant with two alleles, each regulating one band, in PI 183256.		(Staub et al., 1998)
<i>Pep-pap</i>	-	<i>Peptidase with phenylalanyl-proline</i> . Isozyme variant with two alleles, each regulating one band, in PI 183256.		(Staub et al., 1998)
<i>Pgd-1</i>	<i>6-PGDH-21</i> <i>Pgd-21</i>	<i>Phosphoglucose dehydrogenase-1</i> . Isozyme variant with two alleles, each regulating one band. The heterozygote has one intermediate band.		(Esquinas Alcazar, 1981)
<i>6-Pgd-2</i>	-	<i>6-Phosphogluconate dehydrogenase</i> . Isozyme variant with two alleles, each regulating one band, in PI 161375, Védreantais. Relationship with <i>Pgd-1</i> is unknown.	IX	(Baudracco-Arnas and Pitrat, 1996)

Gene symbol				
Preferred	Synonym	Gene description and type lines	LG^z	References
<i>Pgd-3</i>	<i>Pgd</i>	<i>6-Phosphogluconate dehydrogenase</i> . Isozyme variant with two alleles, each regulating one band, in PI 218070. Relationship with <i>Pgd-1</i> and <i>6-Pgd-2</i> is unknown.	A	(Staub et al., 1998)
<i>Pgi-1</i>	<i>PGI-11</i>	<i>Phosphoglucoisomerase-1</i> . Isozyme variant with two alleles, each regulating two bands. The heterozygote has three bands.		(Esquinas Alcazar, 1981)
<i>Pgi-2</i>	<i>PGI-21</i>	<i>Phosphoglucoisomerase-2</i> . Isozyme variant with two alleles, each regulating two bands. The heterozygote has three bands.		(Esquinas Alcazar, 1981)
<i>Pgm-1</i>	<i>PGM-21</i> <i>Pgm-21</i>	<i>Phosphoglucomutase-1</i> . Isozyme variant with two alleles, each regulating two bands. The heterozygotes has three bands.		(Esquinas Alcazar, 1981)
<i>Pgm-2</i>	<i>Pgm</i>	<i>Phosphoglucomutase</i> . Isozyme variant with two alleles, each regulating one band, in PI 218070, PI 179923. Relationship with <i>Pgm-1</i> is unknown.	A	(Staub et al., 1998)
<i>Px-1</i>	<i>PRX-11</i>	<i>Peroxidase-1</i> . Isozyme variant with two codominant alleles, each regulating a cluster of four adjacent bands. The heterozygote has five bands.		(Esquinas Alcazar, 1981)
<i>Px-2</i>	<i>Px2A</i> <i>Prx2</i>	<i>Peroxidase-2</i> . Isozyme variant with two codominant alleles, each regulating a cluster of three adjacent bands. The heterozygote has four bands.		(Dane, 1983; Chen et al., 1990)
<i>Skdh-1</i>	-	<i>Shikimate dehydrogenase-1</i> . Isozyme variant with two codominant alleles, each regulating one band. The heterozygote has three bands.		Chen et al., 1990) (Gang and Lee, 1998)

^zLinkage groups to which the genes belong are indicated as letters, according to Staub et al. (1998), and Roman numbers according to Périn et al. (2002).

Table 3. Quantitative traits loci, including description of the quantitative trait, number of QTL reported, parental lines of the cross used, and references.

Description of the quantitative trait, parental lines of the cross used	References
<p><i>Aphis gossypii</i> resistance Four additive and two couples of epistatic QTL affecting behaviour and biotic potential of <i>Aphis gossypii</i> in the cross Védraçais x PI 161375 (RILs).</p>	(Boissot et al., 2010)
<p><i>Bemisia tabaci</i> resistance Two QTL affecting the biotic potential of the whiteflies in the cross Védraçais x PI 161375 (RILs).</p>	(Boissot et al., 2010)
<p><i>Cucumber mosaic virus</i> resistance. Seven QTL are involved in resistance to three different CMV strains in the cross Védraçais x PI 161375 (RILs). A single QTL required for controlling CMV P9 and P104.82 strains in the cross Piel de Sapo x PI 161375 (LG XII).</p>	(Dogimont et al., 2000) (Essafi et al., 2009)
<p><i>Fusarium oxysporum</i> f.sp. <i>melonis</i> race 1.2 resistance Nine QTL described in the cross Védraçais x Isabelle.</p>	(Perchepped et al., 2005)
<p><i>Pseudoperonospora cubensis</i> resistance Nine QTL for resistance to downy mildew described in the cross Védraçais x PI 124112.</p>	(Perchepped et al., 2005)
<p><i>Podosphaera xanthii</i> resistance Two QTL for resistance to powdery mildew described in the cross TGR-1551 x Bola de Ora (F₂), a major one, dominant (LG V) and a minor one, recessive (LG VIII).</p>	(Yuste-Lisbona et al., 2011)
<p><i>Ovary shape</i> Six QTL for ovary length, eight QTL for ovary width and six QTL for the ratio ovary length/ovary width described in the cross Védraçais x PI 161375 (RILs). Five QTL for ovary shape in the cross Piel de Sapo x PI 161375 (NILs).</p>	(Perin et al., 2002) (Eduardo et al., 2007)
<p><i>Fruit shape</i> Four QTL for fruit length, 5 QTL for fruit width and 6 QTL for the ratio fruit length/fruit width described in the cross Védraçais x PI 161375. Four QTL for fruit length, one for fruit width and two for the ratio fruit length : fruit width described in the cross Védraçais x PI 414723, which are common to both crosses. Eight QTL for fruit shape described in the cross Piel de Sapo x PI 161375 (F₂ and DHLs). Eleven QTL for fruit length, 10 QTL for fruit width and 15 QTL for the ratio fruit length/fruit width described in the cross Piel de Sapo x PI 161375 (NILs). Two QTL for fruit length, 2 QTL for fruit width and QTL for the ratio fruit length/fruit width described in the PI 414723 x Dulce (RI).</p>	(Perin et al., 2002) (Perin et al., 2002) (Monforte et al., 2004) (Eduardo et al., 2007; (Fernandez-Silva et al., 2010) (Harel-Beja et al., 2010)
<p><i>Fruit weight</i> Six QTL described in the cross Piel de Sapo x PI 161375(F₂ and DHLs). Eleven QTL described in the cross Piel de Sapo x PI 161375 (NILs).</p>	(Monforte et al., 2004) (Eduardo et al., 2007)
<p><i>Fruit firmness</i> Two QTL for fruit firmness of the whole fruit described in the cross PI 414723 x Dulce (RI).</p>	(Harel-Beja et al., 2010)
<p><i>Rind traits</i> Three QTL for stripes, three QTL for sutures described in the cross PI 414723 x Dulce (RI).</p>	(Harel-Beja et al., 2010)
<p><i>External color of the fruit</i> Four QTL described in the cross Piel de Sapo x PI 161375 (F₂ and DHLs). Four QTL described in the cross Piel de Sapo x PI 161375 (NILs). Thirteen QTL for skin color and 12 QTL for ground spot color using the three color components in the cross Piel de Sapo x PI 161375 (NILs).</p>	(Monforte et al., 2004) (Eduardo et al., 2007) (Obando et al., 2008)
<p><i>Flesh color</i> Three QTL for orange flesh color described in the cross Piel de Sapo x PI 161375 (F₂ and DHLs). Four QTL for fruit flesh color described in the cross Piel de Sapo x PI 161375 (NILs). Sixteen QTL for flesh color and 10 QTL for juice color using the three color components in the cross Piel de Sapo x PI 161375 (NILs). Three QTL for flesh color described in the cross PI 414723 x Dulce (RI).</p>	(Monforte et al., 2004) (Eduardo et al., 2007) (Obando et al., 2008) (Harel-Beja et al., 2010)

Description of the quantitative trait, parental lines of the cross used	References
<p><i>Sugar content of fruit flesh in mature fruit</i> Five QTL for soluble solid content described in the cross Piel de Sapo x PI 161375 (F₂ and DHLs). QTL for sucrose, total soluble solids in the cross TAM Dulce x TGR-1551 (F₂). Fifteen QTL for soluble solid content in the cross Piel de Sapo x PI 161375 (NILs). Twenty-seven QTL for sugars, eight for fructose, six for glucose, four for sucrose, nine for sucrose equivalents in the cross Piel de Sapo x PI 161375 (NILs). Six QTL for sucrose, total soluble solids in the cross Deltex x TGR-1551 (F₂). Six QTL for sucrose, total soluble solids in the cross PI 414723 x Dulce (RI).</p>	<p>(Monforte et al., 2004) (Sinclair et al., 2006) (Eduardo et al., 2007) (Obando-Ulloa et al., 2009) (Park et al., 2009) (Harel-Beja et al., 2010)</p>
<p><i>Organic acid profile of fruit flesh in mature fruit</i> Twenty-one QTL for organic acids in the cross Piel de Sapo x PI 161375 (NILs).</p>	<p>(Obando-Ulloa et al., 2009)</p>
<p><i>Ascorbic acid</i> One QTL in the in the cross Deltex x TGR-1551 (F₂).</p>	<p>(Park et al., 2009)</p>
<p><i>Ethylene production in fruit (climacteric crisis).</i> Four QTL described in the cross Védraçais x PI 161375 (RILs). One QTL for ethylene production and climacteric response in the cross Piel de Sapo x PI 161375 (NILs), non-climacteric parental lines.</p>	<p>(Perin et al., 2002) (Moreno et al., 2008)</p>
<p><i>Fruit flesh firmness</i> Five QTL for flesh firmness in the cross Piel de Sapo x PI 161375 (NILs).</p>	<p>(Moreno et al., 2008)</p>
<p><i>Fruit flesh aroma profile</i> <i>Ester 3-hydroxy-2,4,4-trimethyl-pentyl 2-methylpropanoate</i>: Two QTL in the cross Piel de Sapo x PI 161375 (NILs). <i>(Z,Z)-3,6 nonadiena</i>, responsible for the cucumber-like aroma: One QTL in the cross Piel de Sapo x PI 161375 (NILs). <i>Octanal</i>: One QTL in the cross Piel de Sapo x PI 161375 (NILs).</p>	<p>(Obando-Ulloa et al., 2010)</p>
<p><i>Root growth and architecture</i> Seventeen QTL for root traits in the cross Piel de Sapo x PI 161375 (NILs).</p>	<p>(Fita et al., 2008)</p>
<p><i>Earliness.</i> Nine QTL described in the cross Piel de Sapo x PI 161375 (F₂ and DHLs). Three QTL for early fruit maturity in the cross Chinese line Q 3-2-2 x Top Mark (F₂-F₃).</p>	<p>(Monforte et al., 2004) (Cuevas et al., 2009)</p>
<p><i>Yield-related traits</i> Four QTL for primary branch number, five QTL for fruit number per plant, four QTL for fruit weight per plant, two QTL for average weight per fruit and one QTL for percentage of mature fruit per plot in the cross USDA 846-1 x Top Mark (RILs).</p>	<p>(Zalapa et al., 2007)</p>
<p><i>Postharvest life traits</i> Three QTL involved in reduced postharvest losses and 11 with a detrimental effect on fruits after storage in the cross Piel de Sapo x PI 161375 (NILs).</p>	<p>(Fernandez-Trujillo et al., 2007)</p>
<p><i>Sensory traits</i> Thirty-two QTL including global appreciation, sweetness, sourness in the cross Piel de Sapo x PI 161375 (NILs).</p>	<p>(Obando-Ulloa et al., 2009)</p>

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