Genetic Resistance in Melon PI 313970 to Cucurbit yellow stunting disorder virus

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Abstract. Melon (Cucumis melo L.) is a fresh vegetable and dessert fruit that may also be cooked or dried, processed for juice and flavoring, and the seeds of which are a source of high-quality cooking oil and high protein seed meal. Melon production throughout many parts of the world is now threatened by the crinivirus Cucurbit yellow stunting disorder virus (CYSDV) in tropical and subtropical areas favorable to its whitefly vector. CYSDV is transmitted by the sweetpotato whitefly, Bemisia tabaci Gennadius, biotypes A, B, and Q. CYSDV first appeared on melon in the 1980s in the United Arab Emirates and emerged on melon in the Yuma, AZ, and Imperial Valley, CA, regions and western Mexico during the Fall season of 2006 followed by Florida in 2007. PI 313970, C. melo var. acutangus, a salad-type melon from India, expressed high-level resistance to CYSDV in Yuma and Imperial Valley in Fall 2006, but it was not immune; the virus was detected in asymptomatic plants. Inheritance of resistance to CYSDV in PI 313970 was studied in three naturally infected, replicated field tests in Imperial Valley during the Fall seasons of 2007 and 2008 and the Spring season of 2009. Resistance in PI 313970 was recessive: all F1 PI 313970 (PI) × susceptible ‘Top Mark’ (TM) and BC1 TM individuals were susceptible, and the F2 and BC2 segregates 3:1 and 1:1 susceptible to resistance, respectively. Frequency distributions of CYSDV symptom severity ratings suggested a single recessive gene in PI 313970 for resistance to CYSDV. PI 313970 was, however, observed to be variable for resistance; a few plants in each test expressed distinct symptoms of CYSDV infection and its frequency distributions overlapped those of ‘Top Mark’. This variation may represent genetic variation suitable for uniform reaction to infection by CYSDV or phenotypic variation in the resistant reaction. The genetic relationship between the genes for resistance to CYSDV in PI 313970 (recessive) and TGR-1551 (dominant) is not known.

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monogenic dominant resistance to a Spanish strain of CYSDV (López-Sesé and Gómez-Guillamón, 2000), but when challenged with a Texas strain of CYSDV, it expressed intermediate resistance and the F2 segregation data did not fit a mono- or digenic, dominant gene model (Sinclair, 2003).

Reactions of 19 melon cultigens (cultivars, breeding lines, and PI) to CYSDV in Imperial Valley, CA, and Yuma, AZ, in Fall 2006 confirmed a previous report (López-Sesé and Gómez-Guillamón, 2000) of susceptible reactions of several of these cultigens to CYSDV inoculation but revealed a new putative source of genetic resistance to CYSDV: PI 313970, var. *acutifolius* Naudin, a salad-type melon from India (McCready and Wintermantel, 2008). PI 313970 was previously reported resistant to SPWF-B (Boisset et al., 2003).

We report the inheritance of host plant resistance in PI 313970 to CYSDV in the Fall seasons of 2006 and 2007 and the Spring season of 2009. The three studies were carried out in naturally infected field plantings at the University of California, Desert Research and Education Center (DREC), Holtville, CA. The fall season in Imperial Valley typically has high SPWF-B feeding pressure in contrast to low pressure in the spring season (McCready et al., 2010).

**Materials and Methods**

PI 313970 (PI) was crossed with ‘Top Mark’ (TM; var. *reticulatus*), a western U.S., shipping-type, orange-fleshed muskmelon. Seed of the parents, F1, F2, and respective backcross generations were made from controlled self- and sib-pollinations in a greenhouse at Salinas, CA (McCready et al., 1992; Robinson and Decker-Walters, 1997). Different progenies of each generation were used in the three tests. ‘Top Mark’ was obtained from a commercial source.

The plants in all three tests were grown using standard commercial practices with irrigation provided as needed by subsurface drip (20-cm depth). Admire® (imidacloprid) was applied once at the label rate through the drip 5 d after the initial irrigation. Bed width (2.0 m) and number of seed per plot (five) were consistent through the three tests, but plot length, seed spacing, numbers of entries, and replications varied among the tests. The 2007 test included one plot of each parent and F1, 31 plots of BCPI. The test was planted on 2 Apr. and evaluated 82 dpp on 23 June CYSDV symptoms were evaluated using a 1 to 10 scale (Table 1).

Plants were also individually examined for foliar symptoms of two other diseases that may occur on melon in the spring and fall seasons in Imperial Valley: *Cucurbit leaf crumple virus* (CuLCrV; *Geminiviridae, Bemovirus*) in the fall (Guzman et al., 2000) and *Podosphaera xanthii* (Castagne) Baum & Shishkoff, a causal agent of melon powdery mildew (McCready, 2006) in the spring and fall. Observation of symptoms of other pathogens present in the field was necessary to determine if these might influence evaluation of CYSDV resistance. CuLCrV was noted as present or absent. Powdery mildew was evaluated using a 1 to 9 scale previously described and resistant blisters were noted as present or absent (McCready, 2006; Sediﬁrová et al., 2009).

Correlation of CYSDV infection with the presence of CYSDV-like symptoms was confirmed in 2007 at the time of symptom evaluation with reverse transcription–polymerase chain reaction (RT-PCR) and nucleic acid hybridization using major coat protein and HSP70 gene-specific probes and primers (Kuo et al., 2007). Each diagnostic sample consisted of a single 0.1-g piece from the interveinal area of a leaf on the main stem or one of the first lateral branches that exhibited early-to-moderate CYSDV symptoms. Presence of CuLCrV was confirmed visually based on symptoms. Symptoms of CuLCrV infection are distinct from those of CYSDV, can appear by 10 dpp, and are most evident at the terminal buds, whereas symptoms of CYSDV infection appear in expanded crown leaves and develop acropetally (McCready et al., 2008).

### Results

#### Experimental plots in the Fall 2007 and 2008 (Fall seasons) but was lower in 2008 (Spring season) test (McCready et al., 2010). The heavy SPWF-B pressure in the fall season appeared to suppress expression of CYSDV symptoms yet adversely affect plant growth in contrast to lower SPWF-B pressure in spring where CYSDV symptom expression and plant growth were inversely correlated for ‘Top Mark’ and PI 313970 (McCready et al., 2010). CYSDV infection was uniform across each test regardless of SPWF population level as indicated by uniform expression of CYSDV symptoms in the test and surrounding border rows and guard plots (data not shown). Presence of CYSDV was confirmed by RT-PCR (data not shown).

In Fall 2007, frequencies of CYSDV symptoms in ‘Top Mark’, F1, F2, and BC*TM* were nearly 100% at 49 dpp (data not shown), but symptoms were not sufficiently developed to assess with the 1 to 4 scale. In contrast, PI 313970 was virtually asymptomatic 49 dpp, only one of 35 plants exhibited symptoms, and 42 of 79 BCPI segregants exhibited symptoms. By 70 dpp, all plants of ‘Top Mark’, F1, F2, and BC*TM* were rated 4 for CYSDV symptom severity, and ratings on PI 313970 and BCPI ranged from 1 to 4 (Fig. 1–A).

The Fall 2008 test was similar to the 2007 test in that CYSDV symptoms were not sufficiently expressed 50 dpp (data not shown), but by 63 dpp ranged across the 1 to 9 rating scale (Fig. 1B). In the Spring 2009 test, symptoms ranged across the 1 to 10 scale 82 dpp (Fig. 1C).

Nearly all plants in 2007 exhibited symptoms of CuLCrV at 49 dpp, but at 70 dpp, the frequency of plants that exhibited CuLCrV symptoms was lower: 67% and 28% of ‘Top Mark’ and PI 313970, respectively (F2: 52%, BC*TM*: 54%, BCPI: 9%). Several cultivated cucurbit species exhibited recovery, most likely mediated by gene silencing and methylation. This was previously shown from particle bombardment-mediated infection by CuLCrV in which recovery was inversely correlated with virus titer and with melon and watermelon (*Citrullus lanatus* (Thunb.)

<table>
<thead>
<tr>
<th>Year</th>
<th>Symptoms</th>
<th>2007</th>
<th>2008</th>
<th>2009</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Asymptomatic</td>
<td>1</td>
<td>Asymptomatic</td>
<td>1</td>
</tr>
<tr>
<td>2</td>
<td>Mild</td>
<td>2</td>
<td>Possible</td>
<td>2</td>
</tr>
<tr>
<td>3</td>
<td>Restricted to crown leaves</td>
<td>3</td>
<td>Faint at crown</td>
<td>3</td>
</tr>
<tr>
<td>4</td>
<td>Extensive from crown to tips</td>
<td>4</td>
<td>Obvious at crown</td>
<td>4</td>
</tr>
<tr>
<td>5</td>
<td>Bright at crown</td>
<td>5</td>
<td>Some tips symptomatic</td>
<td>5</td>
</tr>
<tr>
<td>6</td>
<td>Advancing toward tips</td>
<td>6</td>
<td>Advancing toward tips, bright</td>
<td>6</td>
</tr>
<tr>
<td>7</td>
<td>Some tips symptomatic</td>
<td>7</td>
<td>60% to 70%</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>All tips symptomatic</td>
<td>8</td>
<td>71% to 80%</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>All tips symptomatic</td>
<td>9</td>
<td>81% to 90%</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>All tips symptomatic</td>
<td>10</td>
<td>91% to 100%</td>
<td></td>
</tr>
</tbody>
</table>

*The 2007 and 2008 scales characterized symptom expression; their approximate equivalence is indicated by their vertical placement within the table. The semiquantitative scale used in 2009 estimated the percentage of foliage that expressed symptoms without regard to symptom characteristics.*

### Table 1. Rating scales used for evaluation of foliar symptoms of *Cucurbit yellow stuntling disorder virus* in naturally infected field tests in 2007, 2008, and 2009, Holtville, CA.*
Matsum & Nakaj showing stronger recovery
than zucchini (Cucurbita pepo L.) and pump-
kin (Cucurbita maxima Duchesne) (Hagen
et al., 2008). Some individual plants in five of
16 melon cultivars recovered from SPWF-
B-mediated CuLCrV infection in greenhouse
tests (McCreight et al., 2008). PI 313970,
which also recovered from symptom expres-
sion in greenhouse tests, possesses a single
recessive gene for partial resistance to CuLCrV
(McCreight et al., 2008). Recovery has been
reported in other host–Begomovirus associ-
atives (see Hagen et al., 2008, for a discussion)
and is Begomovirus-specific (Carrillo-Tripp
et al., 2007). In Fall 2008, all plants exhibited
CuLCrV symptoms 49 dpp, but in contrast to
2007, the frequencies of plants exhibiting
symptoms 70 dpp remained high: 96% and
70% for ‘Top Mark’ and PI 313970, respec-
tively, and ranged from 79% (BCPI) to 87%
(F2) in their offspring in three seasons at Holtville, CA, during Fall 2007 (A), Fall 2008 at 63 dpp (B), and Spring 2009 (C) seasons using different rating scales as indicated by the X-axes. In 2007 (A), a 1 to 4 scale was used, where 1, asymptomatic; 2, mild; 3, crown; 4, extensive symptoms. In 2008 (B), a 1 to 9 scale was used, where 1, asymptomatic; 2, possible symptoms; 3, faint symptoms at crown; 4, obvious symptoms at crown; 5, bright symptoms at crown; 6, symptoms advancing toward tips; 7, symptoms further advanced toward crown; 8, some tips yellowed; 9 all tips symptomatic. In 2009 (C), a 1 (0% to 10% symptomatic) to 10 (91% to 100%) scale was used to estimate the proportion of foliage that exhibited CYSDV symptoms.

Fig. 1. Frequency distributions of Cucurbit yellow stunting disorder virus symptom severity ratings of CYSDV-resistant PI 313970, susceptible ‘Top Mark’, and their offspring in three seasons at Holtville, CA, during Fall 2007 (A), Fall 2008 at 63 dpp (B), and Spring 2009 (C) seasons using different rating scales as indicated by the X-axes. In 2007 (A), a 1 to 4 scale was used, where 1, asymptomatic; 2, mild; 3, crown; 4, extensive symptoms. In 2008 (B), a 1 to 9 scale was used, where 1, asymptomatic; 2, possible symptoms; 3, faint symptoms at crown; 4, obvious symptoms at crown; 5, bright symptoms at crown; 6, symptoms advancing toward tips; 7, symptoms further advanced toward crown; 8, some tips yellowed; 9 all tips symptomatic. In 2009 (C), a 1 (0% to 10% symptomatic) to 10 (91% to 100%) scale was used to estimate the proportion of foliage that exhibited CYSDV symptoms.

These data confirmed the observation in 2006 that PI 313970 was resistant to CYSDV, although not immune (McCreight and Coffey, 2011). There was no correlation between CYSDV and powdery mildew symptom severity ratings in either the F2 (r = −0.02) or the BCPI (r = 0.004).

Analysis of variance of CYSDV symptom severity ratings was highly significant (P < 0.0001) in each year. Mean symptom severity of PI 313970 and ‘Top Mark’ differed significantly (P = 0.05) in each season regardless of the rating scale used (Tables 1 and 2).

PI 313970 exhibited significantly less CYSDV symptoms than the F1, F2, and BC TM in all 3 years and the BCPI in 2007 and 2009. The BC TM was significantly more susceptible than the BCPI in 2007 and 2008 but not in 2009 (Table 2).

Frequency distributions of CYSDV symptom severity ratings in 2007 suggested PI 313970 is heterogeneous for resistance to CYSDV; its ratings ranged from 1 to 4 and overlapped ‘Top Mark’ (Fig. 1A). Selection for resistance to CYSDV in progeny from crosses with PI 313970 would be facilitated by initially selecting within PI 313970 for uniform reaction to CYSDV infection before crossing with susceptible parents. The F2 and BC TM were uniformly susceptible (Fig. 1), but the distribution of the BCPI was negatively skewed (Fig. 1A).

Discussion

These data confirmed the observation in 2006 that PI 313970 was resistant to CYSDV, although not immune (McCreight and Wintermantel, 2008). Susceptibility of the F1 in each test suggested that resistance to CYSDV in PI 313970 is recessive (Table 2). This is in contrast to the report of dominant resistance to a Spanish isolate of CYSDV in TGR-1551 (López-Sesé and Gómez-Guilamón, 2000), although genetic control of resistance in TGR-1551 may be more complex or strain-specific (Sinclair, 2003). These data also indicate resistance to CYSDV is genetically distinct from resistance to Lettuce infectious yellows virus (LIYV), a crinivirus transmitted by SPWF biotype A that is controlled by a dominant gene in PI 313970 (McCreight, 2000).

Resistance to CuLCrV in PI 313970 was recessive in a greenhouse test in Salinas and field tests at DREC (McCreight et al., 2008). These field data from DREC suggest that resistances to CYSDV and CuLCrV traits are not allelic. All 35 plants of PI 313970 in 2007 exhibited symptoms of CuLCrV infection at 49 dpp; however, only 10 of them exhibited symptoms of CuLCrV infection 70 dpp and these were distributed in all four CYSDV symptom severity classes. The 2008 CuLCrV data (not shown) at both 49 and 70 dpp were similar to the data at 49 dpp in 2007. This suggested that many plants exhibited recovery from CuLCrV infection in 2007, a trait common among some host plant–begomovirus interactions, including CuLCrV (Carrillo-Tripp et al., 2007; Hagen et al., 2008). Recovery did not occur in 2008, and CuLCrV was not present in 2009. Some individual plants in five of 16 melon cultivars recovered from CuLCrV infection in previous greenhouse tests (McCreight et al., 2008). PI 313970, which also recovered from symptom expression in greenhouse tests, possesses a single recessive gene for partial resistance to CuLCrV (McCreight et al., 2008). Factors influencing recovery of plants from begomovirus infection

<table>
<thead>
<tr>
<th>Generation</th>
<th>2007*</th>
<th>2008*</th>
<th>2009*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>70 dpp</td>
<td>n</td>
<td>50 dpp</td>
</tr>
<tr>
<td>Top Mark</td>
<td>35</td>
<td>4.0 a1</td>
<td>36</td>
</tr>
<tr>
<td>PI 313970</td>
<td>35</td>
<td>2.2 c</td>
<td>36</td>
</tr>
<tr>
<td>F1 TM x PI</td>
<td>36</td>
<td>4.0 a2</td>
<td>32</td>
</tr>
<tr>
<td>F1 PI x TM</td>
<td>33</td>
<td>4.5 a</td>
<td>32</td>
</tr>
<tr>
<td>F2</td>
<td>99</td>
<td>4.0 a</td>
<td>163</td>
</tr>
<tr>
<td>BC TM</td>
<td>44</td>
<td>4.0 a</td>
<td>64</td>
</tr>
<tr>
<td>BC PI</td>
<td>79</td>
<td>3.8 b</td>
<td>73</td>
</tr>
</tbody>
</table>

1 = asymptomatic; 2 = mild; 3 = crown; 4 = extensive symptoms.
1 to 9 scale where 1, asymptomatic; 2, possible symptoms; 3, faint symptoms at crown; 4, obvious symptoms at crown; 5, bright symptoms at crown; 6, symptoms advancing toward tips; 7, symptoms further advanced toward crown; 8, some tips yellowed; 9 all tips symptomatic.
1 (0% to 10%) to 10 (91% to 100%) scale estimated the proportion of foliage that exhibited symptoms.
Number of plants.
Days post-planting.
Within years, means followed by different letters are significantly (P = 0.05) different.

These data from three seasons suggest resistance to CYSDV in PI 313970 is controlled by a single recessive gene. The four different F1 and three different BC TM progenies included in the three tests were similar in their reactions to ‘Top Mark’, and the F2 and BC PI progenies segregated as expected for recessive resistance.

PI 313970 was, however, observed to be variable for resistance; a few plants in each test expressed distinct symptoms of CYSDV infection and its frequency distributions overlapped that of susceptible ‘Top Mark’. The variation may be random or represent genetic variation to SWPF-B infection. The F1 generation virus infection either at the phenotypic (symptom) or molecular level that either masks CuLCrV symptoms or more likely triggers the recovery phenotype. Clarification of the influence of coinfection by CYSDV on CuLCrV titer and host plant resistance to it in CYSDV-resistant plants will require more extensive study.

Resistance to CYSDV is likely independent of resistance to *Cucurbit aphid borne yellowing virus* (CABYV; *Luteoviridae, Polerovirus*) and *Watermelon chlorotic stunt virus* (WCSV; *Geminiviridae, Begomovirus*). CABYV was detected in some plants of PI 313970; although this may be an indication of heterogeneity in PI 313970 (Dogimont et al., 1996). Two recessive genes controlled resistance to CABYV in PI 124112 (Dogimont et al., 1997), and presumably also in PI 313970, but the genetic relationship between resistances to CYSDV and CABYV in PI 313970 is unknown. PI 313970 is one of six melon accessions reported to have complete resistance to WCSV as evidenced by absence of symptoms and no detectable virus (Yousif et al., 2007). Inheritance of complete resistance to WCSV and its relationship to CYSDV resistance in PI 313970 are, likewise, unknown.

Overlap of the frequency distributions of PI 313970 and ‘Top Mark’ may have been attributable in part to an artifact of the 1 to 4 rating scale used to assess symptoms severity in 2007. It was our observation that the 1 to 4 qualitative scale (Table 1) masked informative variation in symptom expression. In other words, phenotypic variation within Classes 2, 3, and 4 could be expressed on a more continuous scale. The 1 to 9 rating scale (Table 1) used in 2008 separated the parents further, but their distribution again overlapped (Fig. 1B). Although their means were significantly different, the observed variability within PI 313970 still suggested potential for selection of more uniform reaction to CYSDV infection. The distributions of their offspring and segregating generations followed the same general pattern of the 2007 distributions with the exception that the BC F1 was more evenly distributed (Fig. 1A–B).

The rating scale was changed in 2009 to a 1 to 10 scale to estimate percentage of symptomatic leaves per plant (Table 1). This scale simplified evaluation by ignoring symptom intensity, i.e., faint yellow vs. bright yellow, so that the focus was on the extent to which CYSDV symptoms were expressed throughout the plant. This test was remarkable from three aspects. First, as a result of the lower SPWF-B feeding pressure in the spring season, there was no confounding effect of severe SPWF-B feeding damage per se that since 1990 has been characteristic of fall melons in the desert southwest United States, particularly in Imperial Valley (McCreight et al., 1995, 2010; Wisler et al., 1998); such numbers of SPWF have not been reported from any other production area. Second, CYSDV can reach 100% rates of infection at low-level SPWF-B infestation (McCreight et al., 2010). Third, CuLCrV was not observed in the spring test (Kuo et al., 2007), preventing any potentially confounding influence CuLCrV might have on CYSDV symptom expression.

CYSDV symptom severity frequency distributions of PI 313970 and ‘Top Mark’ increased in the 2009 test were further separated but still overlapped (Fig. 1C). The range of symptom expression in PI 313970, again, suggested potential genetic variation for selection of a uniform reaction to CYSDV. The range in ‘Top Mark’ may reflect differences in plant size at the time of infection, variation in SPWF-B distribution across the tests, and random variation in time of infection. Distributions of their offspring and segregating generations followed the same pattern observed in 2008 (Fig. 1B).

are only partially understood, and much remains to be determined. Recovery of plants infected with CuLCrV or other begomoviruses is likely mediated by methylation and gene silencing of virus gene expression, and plants exhibiting recovery generally have reduced virus titers in leaves exhibiting the recovery phenotype compared with non-recovered leaves (Hagen et al., 2008). Segregation of CuLCrV resistance did not fit the expected ratios in the F2 or BC PI generations in 2007 or 2008 experiments. This suggested a possible negative syneresis between CYSDV and CuLCrV either at the phenotypic (symptom) or molecular level that either masks CuLCrV symptoms or more likely triggers the recovery phenotype. Clarification of the influence of coinfection by CYSDV on CuLCrV titer and host plant resistance to it in CYSDV-resistant plants will require more extensive study.
in PI 313970. Breeding for resistance to CYSDV should, therefore, be done under low SPWF-B feeding pressure either by implementation of aggressive SPWF-B control measures or planting when SPWF-B control measures or planting when SPWF-B feeding pressure will be low and CYSDV infection will be consistently uniform. This situation can be ensured in controlled greenhouse tests.

The genetic relationship of the resistances to CYSDV in PI 313970 and TGR-1551 has not been reported. TGR-1551 expressed high-level resistance in Fall 2007 to CYSDV in a naturally infected field test in Imperial Valley, CA (McCreight and Wintermantel, 2008). Resistance in TGR-1551 was initially reported to be dominant (López-Seson and Gómez-Guillamón, 2000), but data from Texas suggest it may be codominant and complex (Sinclair, 2003). Alternatively, CYSDV resistance in TGR-1551 may be affected by environmental variation or be strain-specific, although there is limited genetic variability among most CYSDV isolates with the exception of isolates from Saudi Arabia (Rubió et al., 2001). The intriguing question is whether the combination of these distinctly different sources of resistance to CYSDV can provide a higher and more uniform level of resistance than either alone.

PI 313970 is a source of host plant resistance to an increasing number of disease and insect pests. This research adds resistance to CYSDV to a list of resistance of four other viruses: CABYV (Dogimont et al., 1996), CUlCrV (McCreight et al., 2007), LIYV (McCreight, 2000), and WCSV (Yousif et al., 2007). PI 313970 is also valuable for resistance to several insect pests of melon. It is resistant to one genotype and susceptible to a second genotype of the melon aphid, *Aphis gossypii* Glover (Boisset et al., 2008). It is resistant to the agromyzid leafminer, *Liriomyza sativae* Blanchard (munda Frick), expressed as mean number of mines per leaf and as percent mortality of larvae (Kennedy et al., 1978). Resistance in 90625 (PI 313970) to melonworm, *Diaphania hyalinata* L., was likely the result of antixenosis (Boisset et al., 2000). Of immediate interest is its reported resistance to SPWF-B (Boisset et al., 2003), although this can be overcome with heavy whitefly infestation. PI 313970 is a source of salt tolerance in melon (Shannon et al., 1984). PI 313970 is, furthermore, a source of unique, salt-tolerant melon (Dhillon et al., 2003). PI 313970 possesses many other host plant resistance genes that enable development of a multiple disease- and insect-resistant and heat and salt-tolerant melon (Dhillon et al., 2011).

**Literature Cited**


