Randomly Amplified Polymorphic DNA Markers Linked to Fusarium Wilt Resistance in Diverse Melons

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Abstract. Three randomly amplified polymorphic DNA (RAPD) markers (E07, G17, and 596) linked to the Fom-2 gene, which confers resistance to race 0 and 1 of Fusarium oxysporum f. sp. melonis, were evaluated by RAPD-polymerase chain reaction for their linkage to Fusarium wilt resistance/susceptibility in diverse melon cultivars (48 resistant, 41 susceptible). Primer 596 was identified in the multiple disease-resistant breeding line MD8654 as a resistance source, while Fom-1 and G17 (1.05 kb) correctly matched phenotypes in 88% and 81% of the cultivars. The validity of the RAPD scores was verified by Southern hybridization analysis for sequence homology and bulked segregant analysis of a selected cross population for the linkage. These results will facilitate the introgression of resistance genes into susceptible lines from multiple sources in marker-assisted selection.

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Materials and Methods

Germlasm. Eighty-nine melon genotypes (Table 1), including parental lines (Group A, B), cultivars (Group C, D), and F1 hybrids (Group E, F), representing several melon classes from diverse locations were screened with three RAPD primers E07, G17, and 596. Crops between ‘Vedrantais’ (susceptible) and PI 161375 (resistant) were made by M. Pitrat (INRA, Montfavet, France) for the F1 generation, which was selfed for the F2 population. Individual F2 plants were selfed and F2 progenies were inoculated with Fusarium wilt. Homogeneous resistant or susceptible F2 were recorded to track the homoyzogous F2 plants. To confirm the linkage between the RAPD markers E07 and G17 and disease response in genotypes other than the parental line ‘Vedrantais’, crosses between ‘Ananas Yokneam’ (susceptible) and MR-1 (resistant) were made to produce an F2 population. Then the F2 progenies of F1 individuals were determined by scoring both RFLP and CAPS markers (Zheng et al., 1999) to make bulked DNA pools for bulked segregant analysis. In addition, 17 selfed families (Table 2) from a backcross program (BC, S) using breeding line MD8654 as a resistance source, were also scored. Seeds for the BC, S, families were provided by B. Moraghan (Asgrow Seed Co., San Joaquin Breeding Station, Arvin, Calif.). Pedigree information is proprietary.

Fungal culture, host inoculation, disease scoring for Fusarium wilt. The disease phenotypes of melon cultivars and F1 hybrids (Table 1) were determined as follows. The evaluation of Fusarium resistance of the parental lines ‘Vedrantais’ × PI 161375, and their F1 and F2 progenies, as well as of the resistant cultivars listed in Table 1 (except MR-1 and ‘Vine Peach’), was conducted by M. Pitrat by using a Fusarium isolate FOM 26 (race 1). The mycelia were cultured on potato dextrose agar (PDA) plates and conidial suspensions were used to roots dip as described by Risser and Mas (1965). Roots of 20 seedlings of each F1 family were dipped in a conidial suspension before transplanting to sand. Susceptible plants died 2 weeks after inoculation, whereas resistant ones remained green. The disease phenotypes of MR-1 and ‘Vine Peach’ and all of the susceptible cultivars were cited from published screening experiments (Baudracco-Arnas and Pitrat, 1996; Wechter et al., 1995; Zink, 1991; 1992; Zink and Gulber, 1985; Zink and Thomas, 1990). The disease phenotypes for the F1 hybrids were determined by several seed companies cited from seed catalog descriptions (Asgrow Seed Co.; Harris Moran, San Juan Bautista, Calif.; Sakata Seed America, Lehigh,
Table 1. Presence of RAPD markers in diverse melon (Cucumis melo L.) cultigens with different reactions to Fusarium oxysporium f. sp. melonis races 0 and 1.

| Cultigen/F1 hybrid | Source | E07 (1.25 kb) | G17 (1.05 kb) | 596 (1.6 kb) | 596 (1.55 kb) |
|--------------------|--------|--------------|--------------|--------------|--------------|
| **Group A: Resistance parental lines** |
| PI 161375 | Korea | – | – | + |
| MR-1/PI 124111.1 | India | – | – | + |
| **Group B: Susceptible parental lines** |
| Vedrantais | France | + | + | – | – |
| Ananas Yokneam | Hollars | + | + | – | – |
| **Group C: Resistant cultigens** |
| Aodaishi Morou | Japan | – | – | – | – |
| Charentais Fom-2 | France | – | – | + | – |
| Chenggam | Korea | – | – | – | + |
| CM 17187/PI 446928 | Israel | – | – | – | + |
| CM 17188 | Israel | – | – | – | + |
| Freeman’s cucumber | Japan | – | – | + | – |
| Ginsen Makwa | Japan | – | – | – | + |
| Isabelle | France | – | – | – | + |
| K 2005 | China | – | – | – | + |
| Kanro Makua | Japan | – | – | – | + |
| Kogane 9 Go Makua | Japan | – | – | – | + |
| Kogane Sennari Makwa | Japan | – | – | + | – |
| LJ 33430 | TW Whitaker | + | + | – | – |
| LJ 90279/ PI 157083 | China | – | – | – | + |
| LJ 90389 | TW Whitaker | – | – | – | + |
| Meshed | Iran | – | – | – | – |
| Miel Blanc | China | – | – | – | + |
| Nambukin | China | – | – | – | + |
| Nyumelon | Japan | – | – | – | – |
| Ogon 9 | Japan | – | – | – | + |
| Ouzbeque 1 | Japan | – | – | – | – |
| Perlica 1.5 | Guadeloupe | – | – | – | + |
| Persia 202 | Iran | + | + | – | – |
| PI 157084 | China | – | – | – | + |
| PI 125915 | Afghanistan | – | – | – | + |
| PI 164723 | India | – | – | – | + |
| PI 223637 | Iran | – | – | – | – |
| Samarcande | USSR | – | – | – | – |
| Semosouri Varamin | Iran | – | – | – | – |
| Shiroubi Okyama | Japan | – | – | – | + |
| Showa Kogane Nashi Makua | Japan | – | – | – | + |
| Sisi | Iran | ?/– | + | – | – |
| Tokio Mamnuth | Japan | – | – | – | + |
| Vine Peach | Hollars | – | + | + | – |
| **Group D: Susceptible cultigens** |
| Casaba Golden Beauty | Hollars | + | – | – | – |
| Charentais T | F. Zink | + | + | – | – |
| Crenshaw | Hollars | + | – | – | – |
| D21 1005 | E. Cox | + | – | – | – |
| D21 1014 | E. Cox | + | – | – | – |
| Delicious 51 | Hollars | + | – | – | – |
| Doubion | F. Zink | + | + | – | – |
| Dulce | R.T. Correa | + | + | – | – |
| Honey Dew Green Flesh | Hollars | + | + | – | – |
| Honey Dew Orange Flesh | Hollars | + | + | – | – |
| Iroquois | Hollars | + | + | – | – |
| Israel Ogen | Hollars | + | + | – | – |
| Marygold | Hollars | + | + | – | – |
| Mondo | Nunhems | + | – | – | – |
| Perleta | R.T. Correa | + | + | – | – |
| Perleta 45/21 | R.T. Correa | + | + | – | – |
| Persian | Hollars | + | + | – | – |
| Santa Clause | Hollars | + | + | – | – |
| TAM Dew Improved | R.T. Correa | + | + | – | – |
| TAM Mayan Sweet | R.T. Correa | + | + | – | – |
| TAM Perleta 45 | R.T. Correa | + | + | – | – |
| TAM Sun | B. Scully | + | + | – | – |
| TAM Yellow Canary | R.T. Correa | + | + | – | – |
| TAM Uvalde | R.T. Correa | + | + | – | – |
| Topmark | Hollars | + | + | – | – |
| UC Topmark | UC Davis | + | + | – | – |

Table 2. Co-segregation of RAPD markers in BC1S1 families with different reactions to Fusarium oxysporium f. sp. melonis races 0 and 1.

| BC1S1 family | Infected plants (%) | Marker |
|--------------|---------------------|--------|
| | E07 (1.25 kb) | G17 (1.05 kb) |
| 1 | 5 | 0 | – | – |
| 2 | 14 | 0 | – | – |
| 3 | 25 | 0 | – | – |
| 4 | 29 | 0 | – | – |
| 5 | 33 | 0 | – | – |
| 6 | 34 | 0 | – | – |
| 7 | 35 | 0 | – | – |
| 8 | 36 | 0 | – | – |
| 9 | 38 | 0 | – | – |
| 10 | 39 | 0 | – | – |
| 11 | 40 | 0 | – | – |
| 12 | 37 | 0 | – | – |
| 13 | 37 | 0 | – | – |
| 14 | 39 | 0 | – | – |
| 15 | 40 | 0 | – | – |
| 16 | 41 | 0 | – | – |
| 17 | 42 | 0 | – | – |
| 18 | 43 | 0 | – | – |
| 19 | 44 | 0 | – | – |
| 20 | 45 | 0 | – | – |
| 21 | 46 | 0 | – | – |
| 22 | 47 | 0 | – | – |
| 23 | 48 | 0 | – | – |
| 24 | 49 | 0 | – | – |
| 25 | 50 | 0 | – | – |
| 26 | 51 | 0 | – | – |
| 27 | 52 | 0 | – | – |
| 28 | 53 | 0 | – | – |
| 29 | 54 | 0 | – | – |
| 30 | 55 | 0 | – | – |
| 31 | 56 | 0 | – | – |
| 32 | 57 | 0 | – | – |
| 33 | 58 | 0 | – | – |
| 34 | 59 | 0 | – | – |
| 35 | 60 | 0 | – | – |
| 36 | 61 | 0 | – | – |
| 37 | 62 | 0 | – | – |
| 38 | 63 | 0 | – | – |
| 39 | 64 | 0 | – | – |
| 40 | 65 | 0 | – | – |
| 41 | 66 | 0 | – | – |
| 42 | 67 | 0 | – | – |
| 43 | 68 | 0 | – | – |
| 44 | 69 | 0 | – | – |
| 45 | 70 | 0 | – | – |
| 46 | 71 | 0 | – | – |
| 47 | 72 | 0 | – | – |
| 48 | 73 | 0 | – | – |
| 49 | 74 | 0 | – | – |
| 50 | 75 | 0 | – | – |
| 51 | 76 | 0 | – | – |
| 52 | 77 | 0 | – | – |
| 53 | 78 | 0 | – | – |
| 54 | 79 | 0 | – | – |
| 55 | 80 | 0 | – | – |
| 56 | 81 | 0 | – | – |
| 57 | 82 | 0 | – | – |
| 58 | 83 | 0 | – | – |
| 59 | 84 | 0 | – | – |
| 60 | 85 | 0 | – | – |
| 61 | 86 | 0 | – | – |
| 62 | 87 | 0 | – | – |
| 63 | 88 | 0 | – | – |
| 64 | 89 | 0 | – | – |
| 65 | 90 | 0 | – | – |
| 66 | 91 | 0 | – | – |
| 67 | 92 | 0 | – | – |
| 68 | 93 | 0 | – | – |
| 69 | 94 | 0 | – | – |
| 70 | 95 | 0 | – | – |
| 71 | 96 | 0 | – | – |
| 72 | 97 | 0 | – | – |
| 73 | 98 | 0 | – | – |
| 74 | 99 | 0 | – | – |
| 75 | 100 | 0 | – | – |

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DNA samples. For the bulk DNAs from the F₂ population of the cross between ‘Vedrantais’ × PI 161375, the resistant bulk contained equal amounts of 46 homozygous resistant DNA samples, and the susceptible bulk DNAs contained equal amounts of 47 homozygous susceptible DNA samples.

Polymerase chain reaction (PCR). All PCR conditions were optimized and modified from protocols of Baudracco-Arnas and Pitrat (1996) and Wéchter et al. (1995) by using a different source of Taq polymerase. Concentrations of all DNA samples were diluted to 10 ng µL⁻¹. All PCR amplifications were carried out in 25-µL reaction volumes in 600-µL tubes. Each reaction mixture contained 13.38 µL of ddH₂O (Sigma, St. Louis), 2.5 µL of 10 × buffer A, 2.5 µL of 25 mM MgCl₂, 0.13 µL of Taq polymerase, 2.0 µL of 2.5 mM dNTP (New England Biolabs, Beverly, Mass.), 2.5 mL of 6 ng µL⁻¹ primers for E07 and G17 or 30 ng µL⁻¹ for primer 596, and 2.0 mL DNA (10 ng µL⁻¹). Two drops of mineral oil were added to the top of each tube. All three primers were synthesized by New England Biolabs in LB medium plates (1% tryptone, 0.5% yeast extract, 1% NaCl, 1.5% agar, pH 7.0). The plasmids were isolated and digested with EcoRI to check for the inserts by gel electrophoresis. Clone(s) containing the inserts that corresponded to PCR products were saved for use in preparing DNA probes.

### Results

Conservation of RAPD markers in diverse genotypes. Scores of the four RAPD markers in diverse melon cultivars were listed in Table 1. The RAPD markers of 1.25-kb and 1.05-kb fragments resulting from primers E07 and G17 were not only confirmed in the susceptibleparent ‘Vedrantais’ (Baudracco-Arnas and Pitrat, 1996), but were also found in other susceptible cultivars. (Fig. 1A and B). Similarly, a polymorphic fragment of 1.6 kb linked to resistance in MR-1 (Wéchter et al., 1995) was confirmed by using primer 596 (Fig. 1C). It was also detected in resistant lines ‘Desio’ and ‘Vine Peach’. A 1.55-kb polymorphic fragment resulting from primer 596 was found to associate with resistance. Among the 11 cultivars in which phenotype did not match with RAPD-E07 scores, eight were resistant F₁ hybrids (‘Aril’, ‘Corin’, ‘Daimiel’, ‘Lutina’, ‘Pandor’, ‘Preco’, ‘Solo’, and ‘Toledo’) that had the susceptible-linked E07-1.25-kb RAPD marker. Among the other three lines (LJ 34340, Persia 202, and ‘Sisi’), LJ 34340 was later found to segregate for resistance (M. Pitrat, personal communication.). For primer G17, five out of 14 mismatched genotypes, two F₁ hybrids (‘Corin’ and ‘Preco’) and three lines (LJ 34340, Persia 202, and ‘Vine Peach’), were resistant phenotypically but were scored as susceptible for the marker, i.e., showed a 1.05-kb fragment. The other nine genotypes, including one F₁ hybrid (‘Delada’) and eight lines (‘Casaba Golden Beauty’, ‘Creshnaw’,...
D21 1005, ‘Iroquois’, ‘Israel Ogen’, ‘Marygold’, ‘Mondo’, and ‘TAM Dew Improved’), were susceptible phenotypically but were scored as resistant for the G17 RAPD marker, i.e., absence of a 1.05-kb fragment. Four genotypes, including three susceptible cultivars (‘TAM Mayan Sweet’, ‘TAM Yellow Canary’, and ‘TAM Uvalde’) and one resistant F₁ hybrid (‘Accent’), were difficult to score because they showed a 1.05-kb fragment that was not as intense as those observed in the other susceptible lines. These accesses were later verified by Southern hybridization. For primers E07 and G17, most of the mismatching between marker genotype and disease phenotype could be accounted for by the known hybridism because of the dominance of the RAPD marker. Only two lines (LJ 34340 and Persia 202) were mismatched for both markers. The fragment linked to susceptibility from primer E07 (1.25-kb) and G17 (1.05-kb) correctly matched the phenotype in 78 (88%) and 72 (81%) of the 89 cultivars, respectively (Table 3). When combining E07 and G17 scores (at least one matching), the marker genotype(s) matched the disease reaction phenotypes in 95% of the melon lines and hybrids tested (Table 3). For primer G17, the newly identified 1.55-kb fragment was found in 22 out of 36 (61%) of the resistant lines (Table 1, Group C) but in none of the susceptible lines (Table 1, Group D), with a 70% match with phenotype overall (Table 3).

**Verification of RAPD score results by DNA gel blotting analysis.** PCR products with the same molecular size do not necessarily have the same origin and/or sequence when amplified from different genotypes. DNA gel blotting analyses were thus conducted to verify the RAPD scores in diverse melon genotypes. Southern hybridization results confirmed the RAPD scores for E07 marker on all melon cultivars and F₁ hybrids (Fig. 2). All melon cultivars and F₁ hybrids that showed E07-1.25-kb RAPD marker fragments and scored as positive (Fig. 1A; Table 1) produced a positive hybridization signal when probed with the clone-derived E07-1.25-kb fragment from the susceptible line ‘Vedrantais’. The Southern hybridization results not only verified the RAPD scores for G17 marker but also corrected one error of RAPD score in D21-1005. For this phenotypically susceptible genotype, it was scored as resistant by RAPD marker scoring because of the absence of the susceptible-linked G17-1.05-kb fragment. However, the Southern hybridization results showed a positive hybridization signal and was thus relabeled as susceptible.

**Confirmation of RAPD score results by bulk segregant analysis.** The 1.25-kb E07 fragments were amplified from both F₂ homozygous susceptible and heterozygous resistant bulks from both crosses, either between ‘Vedrantais’ × PI 161375 or ‘Ananas Yokneum’ × MR-1 (Fig. 3A). However, a much less intensive 1.25-kb fragment was detectable sometimes from homozygous resistant bulks compared with the fragments amplified from the F₂ homozygous susceptible and heterozygous resistant bulks. Primer G17 amplified a 1.05-kb fragment only from the F₂ susceptible and heterozygous resistant bulks from both crosses, but not from the homozygous resistant bulks (Fig. 3 B).

**Score of RAPD markers E07 and G17 in a segregating backcross population.** Although information about parental lines (from a private seed company) was not available, both RAPD markers predicted the resistance phenotype of the backcross families (Table 2). For the susceptible backcross families that contained different percentages of susceptible individuals in each segregating population, E07-RAPD scored more precisely than did G17-RAPD. The former had only one mismatch that

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**Fig. 1.** Ethidium bromide-stained gel of polymerase chain reaction (PCR) amplified products using decamer primer E07 (panel A), G17 (panel B), and 596 (panel C) and genomic DNAs of melon lines differing in resistance to Fusarium wilt caused by *Fusarium oxysporum* f. sp. *melonis* races 0 and 1. Lanes 1, 2, 3, 4, 8, 10, 12, and 15 are resistant genotypes ‘Charentais Fom-2’, LJ 90279, PI 446928, ‘Sisi’, PI 161375, MR-1, ‘Isabelle’, and ‘Freeman’s Cucumber’, respectively. Lanes 5, 6, 7, 9, 11, 13, 14, and 16 are susceptible genotypes ‘TAM Uvalde’, ‘Cruiser’, ‘Honey Dew Orange Flesh’, ‘Vedrantais’, ‘Iroquois’, ‘Delicious 51’, ‘Crenshaw’, and ‘Perlita’, respectively. The single arrow indicates the 1.25-kb (panel A), 1.05-kb (panel B), and 1.6-kb (panel C) polymorphic marker band. The double arrow indicates the 1.55-kb fragment (panel C) associated with resistance when primer 596 was used. M is a 1-kb DNA ladder from Gibco BRL, Life Technologies, Gaithersburg, Md.
Table 3. Prediction accuracy of three RAPD markers linked to the Fom-2 gene conferring resistance to Fusarium oxysporum f. sp. melonis races 0 and 1 in diverse melon cultivars. 

| RAPD marker | Match Count | Match % | Mismatch Count | Mismatch % |
|-------------|-------------|---------|----------------|------------|
| E07         | 78          | 88      | 11             | 12         |
| G17         | 72          | 81      | 17             | 19         |
| 596         | 62          | 70      | 27             | 30         |
| E07/G17     | 85          | 95      | 4              | 5          |

*Primer E07 and G17 are susceptible-linked markers, primer 596 is a resistant-associated marker of 1.55 kb. The genotypes of the three RAPD markers were fragments of 1.25, 1.05, and 1.55 kb, respectively. E07/G17 means the combined data from the two markers, i.e., one match with either primer was counted as correctly predicting phenotype. Disease reactions of melon lines and hybrids were tested as described in text.

Discussion

Our data present a comprehensive test and show the utility of dominant- and susceptible-linked RAPD markers for Fusarium wilt in diverse melon cultivars. RAPD markers E07 and G17 had a high degree of conservation among the melon cultivars and F1 hybrids from diverse locations and origins. The validity of the RAPD scores in diverse melon cultivars was confirmed by Southern hybridization (Thormann et al., 1994), in which only the RAPD fragments with sequences identical to or highly similar to that of the probe (from the original parental line) resulted in positive hybridization. Identified originally from a segregating population of 'Vedrantais' × PI 161375, the validity of the RAPD markers E07 and G17 were further substantiated by bulked segregant analysis in an additional selected segregation population derived from 'Ananas Yokneam' × MR-1. A linkage must exist in other melon cultivars that had positive scores in both RAPD and Southern hybridization.

All mismatches for E07 RAPD were derived from phenotypically resistant lines that were scored as susceptible, i.e., showing the susceptible-linked 1.25-kb fragment. The majority of these were F2 resistant hybrids. If one parent carried Fom-2, then the F2 was heterozygous and disease-resistant. Theoretically, the susceptible-linked marker RAPD E07-1.25 kb would be detected in a heterozygous plant, while the phenotype would be resistant. Three other genotypes (LJ 34340, Persia 202, and 'Vine Peach') that were mismatched with the E07 RAPD marker were not hybrids but could be heterozygous for Fom-2 gene because of recombination. Indeed, one of the genotypes (LJ 34340) was later identified to be segregant for Fusarium wilt resistance. The primer E07 was 1.6 ± 0.9 cm away from the resistance gene Fom-2 (Baudracco-Arnas and Pitrat, 1995; Pitrat, 1991), which would result in a 2% to 5% recombination frequency. Based on this map distance, we would expect 2% to 5% of the mismatches to be due to recombination events. The fact that two different genes (Fom-2 and Fom-3) present in different cultivars of melon and control resistance to the same race of F. oxysporum f. sp. melonis (Zink and Gubler, 1985) may also contribute to the mismatch. Thus, the susceptible linked primer E07 was adequate and suggestive of its potential. Primer G17 was 4.5 ± 1.5 cm from Fom-2 (Baudracco-Arnas and Pitrat, 1996; Pitrat, 1991), and would produce a 6% to 12% recombination frequency. As described above for E07, the RAPD marker G17 also worked very well in our test.

Our data suggest that problems associated with PCR failure could be eliminated by comparing the whole PCR profile between the susceptible and resistant samples in the gel picture, instead of the target fragment only. However, the fact that RAPD-PCRs are sub-
jected to different sources or batches of *Tag* polymerases, tissue ages, PCR conditions, and even different PCR runs (Staub et al., 1996) was observed in this study. Ironically, for RAPD primer 596, an additional polymorphic fragment of 1.55 kb associated with resistance was identified by using a *Tag* polymerase differed from the original investigators who reported a 1.60-kb RAPD linked to resistance in melon line MR-1 (Wechter et al., 1995). The newly identified RAPD marker (1.55 kb) was found in 23 of the 36 (64%) resistant melon cultivars, but in none of the susceptible cultivars tested (Zheng et al., unpublished data). Primer E07 produced a 1.25-kb marker band with variable intensity in some cultivars or BC$_2$ families, that had DNA pooled from varying percentages of diseased plants in the population. The reduced intensity (varied in degrees) of 1.25 kb marker band was not due to PCR partial failure, because other bands in the PCR profiles were about the same as those with the typical E07-1.25-kb fragment. The multiple copies of the E07 fragment located across the melon genome, as revealed by genomic Southern blotting of DNA. BioTechniques 15:260–262.

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