Molecular Markers Associated with Morphological Traits in Watermelon

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Abstract. Morphological traits were examined in an F3 generation derived from a cross between C. lanatus var. lanatus (L. Thunb.), Matsum. & Nakai) and C. lanatus var. citroides. At least three genes, C (yellow), I (inhibitory to C), and y (yellow) vs. y (white), with epistatic and inhibitory actions were found to govern the inheritance of fruit flesh color. The high frequency of yellow-fleshed fruit and low frequencies of white and red fruits can be explained by the presence of a new allele (y recessive to y) in the multiple allele series at the Y locus. The low frequency of tan colored seeds in segregating populations could be explained by at least three genes governing inheritance of seed-coat color. Single factor analysis of variance was conducted for each pairwise combination of random amplified polymorphic DNA (RAPD) locus and fruit or seed characteristics. Several RAPD loci were identified to be loosely linked to morphological characteristics.

Variations in morphological characteristics among cultivated watermelons [Citrus lanatus var. lanatus, 2n = 2x = 22] are relatively low (Biles et al., 1989; Hashizume et al., 1996), while the cultivated and wild form (C. lanatus var. citroides) differ in fruit and seed characteristics, and important disease resistance genes. Within the species, fruits range from small, hard, bitter, white, and inedible to large, succulent, red or yellow, and sweet (Chakravarty, 1990; Lee et al., 1996; Robinson and Decker-Walters, 1997). Some of the morphological characteristics most well studied in watermelon are fruit flesh color (Poole, 1944; Henderson, 1989; Henderson et al., 1998), flower color (Kwon and Dane, 1999), seed characteristics (Porter, 1937; Poole et al., 1941), bitterness (Chambliss et al., 1968; Navot et al., 1990), and fruit weight and shape (Poole, 1945).

The Mendelian inheritance of some traits in watermelon has been demonstrated (Table 1). The action of several genes govern fruit flesh color. Shimotsum (1963) reported a two-gene model (Wf [white] and y [yellow]) controlling flesh color in an interspecific Citrus. A white fleshed F, and ratio of 12 white, 3 yellow, and 1 red in the F1, indicated epistatic interaction between Wf and b. Hashizume et al. (1996) in a cross between a cultivated red-fleshed watermelon and a white-fleshed wild African form observed a yellow fleshed F1, and backcross (BC) population with fruit color segregation of 1 yellow : 1 white. Henderson et al. (1998) detected three genes involved in the inheritance of flesh color of cultivated watermelon varieties: C (yellow) vs. c (red); a multiple allelic series Y (red flesh) dominant to y (orange) and dominant to y (pale yellow); and I (inhibitory to C) vs. i (non-inhibitory to C), and y (yellow) vs. y (white), with epistatic and inhibitory actions were found to govern the inheritance of fruit flesh color. The high frequency of yellow-fleshed fruit and low frequencies of white and red fruits can be explained by the presence of a new allele (y recessive to y) in the multiple allele series at the Y locus. The low frequency of tan colored seeds in segregating populations could be explained by at least three genes governing inheritance of seed-coat color. Single factor analysis of variance was conducted for each pairwise combination of random amplified polymorphic DNA (RAPD) locus and fruit or seed characteristics. Several RAPD loci were identified to be loosely linked to morphological characteristics.

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by a final extension at 72°C. Lambda DNA digested with PstI was run on 1.2% agarose gels and stained with ethidium bromide. The gels were run in TE buffer and quantified using a Hoefer DyNA Quant 200® fluorometer (Hoefer Pharmacia Biotech, San Francisco, CA). The DNA concentration was adjusted as required for a given application and stored at –20°C until needed.

**RAPD analysis.** One hundred-eighteen decamer RAPD primers from Operon Technologies (OP; Alameda, Calif.) and the Univ. of British Columbia (UBC; Vancouver, BC Canada), along with five 12-mer RAPD primers identified by Hashizume et al. (1993; 1996) were screened for their ability to detect polymorphisms between NHM and PI 296341. Selected primers were used to amplify polymorphic bands in four individuals from each F2 family (Hawkins et al., 2001). The tissue was ground in liquid nitrogen and either immediately used for DNA extraction or stored at –80°C. The extracted DNA was suspended in TE buffer and quantified using a Hoefer DyNA Quant 200® fluorometer (Hoefer Pharmacia Biotech, San Francisco, CA). The DNA concentration was adjusted as required for a given application and stored at –20°C until needed.

**PCR was performed in a 25-µL volume containing 10 mM Tris-HCl, 3.0 mM MgCl2, 10 mM KCl (pH 8.3), 0.2 mM of each nucleotide (Perkin Elmer, Branchburg, N.J.), 5 µmol decamer or 12mer oligonucleotide primers, 10 ng genomic DNA and 1 unit of AmpliTaq® polymerase (Perkin Elmer, Branchburg, N.J.). The thermocyclers were programmed as follows: initial denaturation at 94°C for 10 min, 35 cycles of 94°C for 45 s, 36°C for 45 s, and 72°C for 2 min, followed by a final extension at 72°C for 5 min. Amplification products were electrophoresed on 1.2% agarose gels and detected by staining with ethidium bromide. The gels were photographed under UV light with Polaroid film 667. Lambda DNA digested with PstI was used as a molecular size marker. Each band was named by the primer used and its size in basepairs (bp’s), such that OPF16 was the 1600 bp band amplified by Operon primer F16.

**Statistical analysis.** Data from four individuals in 28 F2 families were analyzed based on single factor ANOVA for each pairwise combination of marker loci and quantitative trait (SAS Institute, 1996). F2 test statistics (F < 0.05) were used to determine if significant differences in trait expression were associated with differences in marker locus-genotypic classes (Edwards et al., 1987). Heritability estimates were calculated for percentage of soluble solids, fruit weight, seed length and width, and fruit weight, length, width, and shape. Heritability was calculated using the following formula:

\[ h^2 = \frac{\sigma_g^2}{\sigma_e^2 + \sigma_g^2}, \]

where \( k \) is the number of replications and \( \sigma_g^2 \) is the residual (Liu, 1997).

**Results and Discussion**

**Seed characteristics.** Seeds from NHM are on average 1.1–1.6 mm, brown to black in color, while PI 296341 seeds are on average 1.0×0.6 mm, reddish-brown, stippled, with a few cracks on the seed coat. F1 seeds are dark brown with some tan cracks and range in size from 1.0 to 1.1×0.6 mm to 0.7 cm. Seed lengths in F2 and F3 progeny varied from 0.7 to 1.3 cm, seed width from 0.4 to 0.9 cm (Fig. 1. A and B). In the presence of a RAPD marker, a wide variety of seed patterns was observed, including the tan, red and plum, and stippled characteristics described by Poole et al. (1941). When seeds from the F2 were scored as black vs. nonblack, 50 F2 plants produced black seeds, 19 nonblack, resulting in good fit to a 3:1 segregation ratio (\( \chi^2 = 0.24, df = 1 \)). Similarly, when the seeds were scored as nonstippled vs. stippled, 53 F3 plants had a ratio of 3 nonstippled vs. 1 stippled (\( \chi^2 = 0.12, df = 1 \)). The low frequency of tan seeds in the F2 and 4% in the F3 generation, a recessive trait, can be explained by the interactions of at least three genes that govern the inheritance of seed color in this population. The frequency of black-seeded phenotypes and stippled seeded phenotypes increased from the F1 (43% black, 12% stippled) to the F2 (55% black, 40% stippled). In the F3 generation, seeds had a more uniform appearance among the families, but showed more differences in pattern within the family. One trait, cracked seed coat, was noticeably absent. This trait was examined by El-Hafez et al. (1981; 1985) and found to be controlled by a recessive gene (cr). Cracked seeds were observed on PI 296341, F1, and several F2 seeds, but seldom appeared in the F3, which points toward epistatic interactions between loci involved in the inheritance of seed-coat colors. Phenotypes of watermelon seed-coat colors are difficult to classify because of variable degrees of expression. Since this trait is under multigenic control, advanced generations such as recombinant inbred lines, in addition to reciprocal backcrosses are needed to further deduce its complex inheritance. The elucidation of the inheritance of seed-coat color has also proved problematic in other plant species. Using 80 F2 individuals, Kaga et al. (1996) were unable to clarify the mechanism of seed color expression in azuki bean because of complicated segregation patterns.

**Fruit characteristics.** Individual F1 fruit, fruit weights ranged from 0.4 to 4.5 kg, fruit lengths varied from 10 to 23.4 cm, while fruit widths ranged from 4.3 to 21.3 cm, and their soluble solids ranged from 3% to 7% (Figure 1 C, D, E, and F). Fruits of PI 296341 were white (155A from the RHS color chart), NHM red, and the F1 yellow (8C). Thirty-nine of the F1 lines produced yellow fruits only, four red fruits only, four white only. 17 segregated as yellow and white, five segregated as yellow and red, one segregated red and white, and one segregated red, yellow and white. In some fruit, the color red was found localized to areas around the seeds. The presence of yel-
low fruit in the F1 and high incidence of yellow fruit in the F3 generation indicates that yellow is dominant over white and red.

Henderson et al. (1998) suggested that at least three genes control the inheritance of fruit flesh color. In addition to the genes \( C \) (yellow) vs. \( c \) (red) and \( i \) (inhibitory to \( C \)) vs. \( I \) (noninhibitory to \( C \)), described by Henderson et al. (1998), it is proposed that a new allele be added to the multiple allelic sequence of \( Y \). This gene is designated \( yw \), conditions white flesh, and is recessive to \( y \) (canary yellow flesh). The \( C \) locus is epistatic to \( y \), such that \( C-y- \), \( C-yw yw \), \( ccyw yw \) are yellow-fleshed, and \( ccy- \) is red-fleshed. White-fleshed fruits occur when the \( i \) locus inhibits the \( C \) locus in the presence of \( yw yw \) (Table 2). The proposed parental genotypes are \( IIccyy \) (red) for New Hampshire Midget, \( iiCCyw yw \) (white) for PI 296341 and \( IiCcyyw \) (yellow) for the F1. In 17 F3 lines yellow and white fruit were detected (possible F2 genotypes: \( I-ccyy \), \( I-C-yw \), \( I-Ccyy \), \( iCcyy \), \( iCcyy \), \( iCcy- \), \( iCC- \), \( iCy- \), \( icc- \), \( icy- \), \( iccyy \), \( iccy- \), \( iCcy- \), \( iCy- \), \( icCyy \), \( icCcy \), \( iccCyy \)), five F3 lines produced yellow and red fruit (\( I-ccyy \), \( I-C-yw \), \( I-Ccyy \), \( iCcy- \), \( iCy- \)), five F3 lines produced yellow, white and red fruit (\( I-ccy- \) or \( I-Cy- \)), and one F3 line produced red and white fruit (\( I-Ccy- \)). A total of 41 red and 45 white fruit were observed in the F3, which resulted in a good fit to the expected segregation ratio of 365 yellow : 75 red : 72 white (\( \chi^2 = 0.75, P < 0.05, df = 2 \); Table 2). The proposed theory of three loci with epistatic and inhibitory actions governing the inheritance of fruit color in this population accounts for the large number of yellow-fleshed fruits and the scarcity of red and white-fleshed fruits in the F3.

**RAPD analysis.** Those primers that did not provide a reproducible amplification product or were monomorphic, were excluded from this investigation. RAPD loci exhibiting the expected 5 presence : 3 absence Mendelian segregation ratio in the F3 were compared against each morphological trait using single factor analysis of variance (ANOVA) (Table 3). Several RAPD marker loci were found to be linked to fruit color, fruit length, soluble solids, fruit width, seed length, fruit weight, and seed color (Table 3). The heritability of fruit width, fruit length, fruit weight, seed length, seed width, and soluble solids was calculated and ranged from 0.33 for seed length, 0.38 for fruit shape, 0.39 for soluble solids, 0.40 for fruit length, 0.47 for fruit width, 0.49 for fruit length, 0.50 for seed width. Most of the RAPD loci and consequently the quantitative trait loci (QTLs) governing the morphological characteristics could not be assigned to specific genomic regions. Only seed length and width could be assigned to linkage group 3. The low heritability estimates detected for the various morphological characteristics (Fig. 1) and the dominant nature of the molecular marker hindered identification of RAPD loci linked to the traits. QTLs affecting cucumber fruit shape (length : diameter ratio) were identified by Dijkhuizen and Staub (1999). Differences in QTL number and location could be attributed to disparities in population size, dominance, and the amount of genotypic information available. Fruit length, diameter, and to a lesser extent shape are developmentally dependent, and thus map placement of QTL was affected by the physiological stage of fruit development (Dijkhuizen and Staub, 1999). Only through the development of a more detailed linkage map of the watermelon genome, will it be possible to precisely assign QTLs for the various morphological characteristics to specific genomic regions.

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Table 2: Proposed genotypes and phenotypes for fruit flesh color observed in F₂ watermelon population derived from a cross between the Fusarium wilt-susceptible NHM ("ccy") and the Fusarium wilt-resistant PI 296341 ("CCy2y").

| Proposed genotypes | Expected phenotypes | Expected F₂ family | Expected|F₂ phenotypes|Observed F₂ phenotypes |
|-------------------|---------------------|---------------------|---------|--------------|---------------------|
| I-C-yw             | yellow              | 27 (Y : W = 3:1)     | 125     | 196 yellow   |                     |
| I-cyw              | red                 | 9 (Y : W = 6:1)      | 75      | 41 red       |                     |
| I-cyyw             | yellow              | 3 (Y : W = 3:1)      | 45      |              |                     |
| I-ccyw             | yellow              | 9 (Y : W = 3:1)      | 75      |              |                     |
| I-cccyw            | white               | 3 white only         | 45      | 45 white     |                     |
| I-ccy-             | yellow              | 27 (Y : R : W = 51:9:4) | 125 | 196 yellow   |                     |

\( \chi^2 = \text{yellow, } R = \text{red, } W = \text{white.} \)

*Significance levels determined by F-test based on the single-factor ANOVA for each pairwise comparison of a quantitative trait and marker locus

**Unassigned marker.

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