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Genetic mapping of *jointless-2* to tomato chromosome 12 using RFLP and RAPD markers

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Abstract Abscission zones are specialized regions in plants, usually located at the base of most plant parts, such as flowers, fruit and leaves, where organs are shed. Although a great deal of information is known about the physiological and biochemical events that lead to organ shedding, very little is known of the molecular events that lead to the formation of the abscission zone itself. In tomato, two recessive mutations have been discovered that completely suppress the formation of flower and fruit pedicel abscission zones, i.e., *jointless* (*j*) and *jointless-2* (*j-2*), both tentatively localized to chromosome 11 about 30 cM apart. Because the study of the control of abscission zone development is important for both basic and applied research we are using a map-based cloning approach to identify the jointless genes. The first step in any positional cloning experiment is to establish segregating mapping populations for the target gene and identify closely linked molecular markers that flank the locus. In this study, bulked segregant analysis was used to identify a RAPD marker associated with the *j-2* locus, RPD140. To determine the chromosome location of RPD140, we converted it to an RFLP marker that was then mapped on the Cornell reference tomato map in a marker-dense region of chromosome 12. To verify that the *j-2* locus was located on tomato chromosome 12, we used nine chromosome 12 RFLP markers linked with RPD140 to map the *j-2* gene in an interspecific F_2 mapping population of 151 plants segregating for *j-2*. The *j-2* gene was localized to a 3.0-cM interval between RPD140 and TG618 on tomato chromosome 12.

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Introduction

Abscission is a developmentally regulated process whereby plants shed their organs, such as leaves, fruit, flowers, and floral parts, at the abscission zone structures. Because of the importance of abscission, especially flower and fruit abscission, in agricultural production, there is a long history of abscission studies by plant anatomists and physiologists. However, despite extensive research on the process of cell separation between cells of fully developed abscission zones, the process by which certain cells differentiate into an abscission zone is still not well understood. In our laboratory, we are using a genetic approach to study the first step in the abscission process by attempting to clone two genes from tomato, *jointless* (*j*) and *jointless-2* (*j-2*), that completely suppress the formation of pedicel abscission zones. *Jointless* was isolated as a spontaneous mutation in an *Lycopersicon esculentum* cv. Rogue (Butler 1936), whereas *jointless-2* was discovered on the Galapagos islands of South America in the wild species *L. cheesmanii* LA166 (Rick 1956).

In tomato, the abscission zone structure is located in the middle of the flower pedicel and is easily recognized by its indentation, called the "joint", as shown in Fig. 1 (Butler 1936; Roberts et al. 1984; Wing et al. 1994). In large-scale tomato production, fruit are harvested mechanically by picking the fruit with distal pedicels and calyxes still attached, requiring an additional processing step for their removal. The absence of an abscission zone (Fig. 1) makes jointless tomatoes highly desirable for the tomato processing industry (Zhang et al. 1994). Additionally, the jointless pedicel can be used to overcome stem retention on the fruit (Reynard 1961) during loading and transportation, which thereby prevents puncture injury to other tomato fruit that otherwise may lead to

Fig. 1A, B Phenotypic comparison between wild-type and *jointless-2* tomato plants. **A** Wild-type tomato displays an abscission zone (''joint'') in the middle of the pedicel. **B** A *jointless-2* mutant (*L. cheesmanii* LA 166) displays an abscission zone minus phenotype

fruit rot microbial infection, resulting in postharvest losses (Zahara and Scheuerman 1988; Lukyanenko 1991).

To isolate the jointless genes, whose functions and protein products are unknown, we are using a map-based cloning strategy initiated with the genetic mapping of the target gene on the molecular genetic map. Both *j* and *j-2* were tentatively mapped approximately 30 cM apart to tomato chromosome 11 using classical approaches (Rick and Yoder 1988). The map position of the *j* locus was confirmed on chromosome 11 using RPD158, a marker isolated by random amplified polymorphic DNA (RAPD) screening against two isogenic DNA pools containing DNA from F_2 plants of an interspecific cross segregating for *j* (Wing et al. 1994). Several genes have been tagged in similar ways, such as the *Pto* gene conferring resistance to *Pseudomonas syringae pv*. tomato (Martin et al. 1991), the *Nor* mutation affecting fruit ripening in tomato (Giovannoni et al. 1991), the *Dm* genes conferring resistance against downy mildew in lettuce (Michelmore et al. 1991), and $Hs1^{pat-1}$ conferring resistance to cyst nematode in beet (Salentijn et al. 1995). In contrast to the *j* locus, the map position of the *j-2* locus on chromosome 11 has not yet been confirmed by molecular genetics even though it has been used widely for over 40 years in the tomato processing industry.

Here we report the identification of a molecular marker linked to *j-2* using bulked segregant analysis (BSA) with RAPDs and the genetic mapping of the *j-2* locus to a 3-cM interval on tomato chromosome 12.

Materials and methods

Plant materials

For RAPD analysis, seeds (population no. 83–375–8) from a cross between an F_1 hybrid *L. esculentum* (*L. esculentum* VF vendor 821510 × *L. esculentum* VF72–34 821460–2) *J-2*/*j-2* and *L. esculentum* VF calypso 841144 *j-2*/*j-2* were provided by Dr. Martha Mutschler, Cornell University. The parental lines had a *L. esculentum* background except for the introgressed *j-2* region of *L. esculentum* VF Calypso 841144, which was originally from *L. cheesmanii* LA166.

For restriction fragment length polymorphism (RFLP) mapping, two mapping populations were used. First, the "Cornell reference mapping population", consisting of 67 F_2 plants from an interspecific cross between *L. esculentum* and *L. pennelli* described by Tanksley et al. (1992), was used to integrate *j-2*-linked RAPD markers into this map. A second F_2 mapping population (151 plants) segregating for *jointless-2* was derived from an interspecific cross between *L. esculentum* Heinz 1706 (*J-2* /*J-2*), and *L. cheesmanii* LA166 (*j-2*/*j-2*). Both *L. esculentum* Heinz 1706 and *L. esculentum* LA166 were obtained from C.M. Rick at the Tomato Genetic Stock Center, University of California at Davis.

DNA analysis

Tomato genomic DNA was extracted following the method of Bernatsky and Tanskley (1986). RAPD analysis was performed with random decamers purchased from Dr. John Carlson, University of British Columbia, Vancouver, Canada as previously described (Giovannoni et al. 1991) except for the 10-µl volume of the polymerase chain reaction (PCR). RAPD markers were amplified using two DNA pools from a segregating population, no. 83–375–8. Wild-type and mutant pools consisted of 10 individual F_2 jointed and jointless phenotypes, respectively. Reproducible polymorphic RAPD markers were hybridized to a survey filter containing restricted DNA of both mapping parents (*L. esculentum* Heinz 1706 *J-2*/*J-2* and *L. cheesmanii* LA166 *j-2*/*j-2*) in order to determine their copy number and convert dominant RAPD markers into codominant RFLP markers.

RFLP analysis was performed as described by Tanksley et al. (1992). Restriction enzymes for mapping included *Bst*NI, *Dra*I, *Hae*III, *Hin*dIII, *Eco*RI, *Eco*RV, and *Sau*3AI. Tomato RFLP markers were obtained from S. Tanksley (Cornell University, Ithaca, N.Y.). DNA probes were labeled using [^{32}P]-dCTP (NEN, USA) by random primer DNA labeling (Feinberg and Vogelstein 1984) and hybridized to the filters (Church and Gilbert 1984).

Linkage analysis

Closely linked RAPD markers to *j-2* were hybridized to Southern blots of the Cornell reference mapping population (Tanksley et al. 1992). Linkage group analysis was performed using the command "group" of MAPMAKER software (Lander et al. 1987). RFLP markers linked to *j-2*-linked RAPD markers were hybridized to Southern filters containing DNA from 151 F_2 plants segregating for *j-2*. The genetic distances in centiMorgans (cM) were calculated using the Kosambi mapping function (Kosambi 1944) with an LOD score of 3.0. All RFLP segregation was scored; linkage analysis and marker order were performed using MAPMAKER software.

Conversion of dominant RFLP marker CT99 into a CAPS marker

One marker, CT99, showed a polymorphic pattern as a single band on restricted genomic DNA of *L. esculentum* Heinz 1706 *J-2*/*J-2* and as two bands on the other parent, *L. cheesmanii* LA166 (*j-2*/*j-2*), with the upper band being the same in size with that of *L. esculentum*. Following the method of cleaved amplified polymorphic sequences (CAPS) (Konieczny and Ausubel 1993), CT99 was partially sequenced from the ends for designing primers. A single DNA fragment was amplified from each parental DNA line in a 25-µl reaction mix (10 m*M* TRIS pH 8.3, 50 m*M* KCl, 1.5 m*M* $MgCl_2$, 0.2 m*M* each dATP, dCTP, dGTP and dTTP, 0.8 µ*M* primers, 40 ng genomic DNA and 0.4 U *Taq* DNA polymerase). Each amplified genomic DNA was digested with a series of restriction endonucleases to find a restriction enzyme that was unique to one parent but not the other, thus providing an RFLP. The primer sequences used to amplify CT99 from both parental genomic DNA samples were: CT99F, 5'-GAGTGAAACGGCTGACAC-'3 and primer CT99R, 5'-TATTGCCCATGGCTCAG-3'. The PCR program used was: 1 cycle of 3 min at 94°C, 1 min 10 s at 60°C, 3 min 15 s at 72°C; then 35 cycles of 30 s at 94°C, 30 s at 60°C, 3 min at 72°C; finally 7 min at 72°C. *Tha*I restriction enzyme was found to give polymorphic bands between an amplified fragment from each parental genomic DNA. Seven microliters of PCR products was digested with *Tha*I in a 20-µl volume according to the manufacturer (NEB, USA), and fragments were separated on a 1.5% (w/v) agarose gel, 0.5 × TBE at 0.7 V/cm for 8 h.

Results

Construction of a segregating mapping population for *jointless-2*

jointless-2 has been reported to be a single recessive mutation that completely suppresses the development of the abscission zone structure of tomato flower pedicels (Rick 1956) and located on the distal end of tomato chromosome 11 (reviewed by Rick and Yoder 1988). To verify these reports, we generated an interspecific mapping population of 151 F_2 plants from an F_1 between *L. esculentum* Heinz 1706 (*J-2*/*J-2*) and *L. cheesmanii* (*j-2*/*j-2*). The jointed and jointless phenotypes were easily scored and resulted in 36 jointless and 115 jointed. The segregation ratio corresponds to the expected 3:1 of a single recessive gene with a Chi square value of 0.0406 ($P > 0.05$).

Screening of RAPD markers putatively linked to *J-2*

To obtain tightly linked markers to *J-2*, we performed BSA with bulks derived from 10 jointed (*J-2*/*j-2*) and 10 jointless (*j-2*/*j-2*) F_2 plants from segregating population no. 83–375–8 with an *L. esculentum* background. Of the 600 RAPD markers 13 produced RAPD polymorphisms between the bulks. Each RAPD band was tested to determine if it contained repetitive DNA sequences by hybridization to Southern blots containing restriction enzyme-digested *L. esculentum* DNA. Three RAPD markers were found to contain repetitive elements and were therefore not tested on the RFLP survey filters. RAPD bands that appeared to contain low-copy DNA were then hybridized to RFLP survey filters containing DNA from

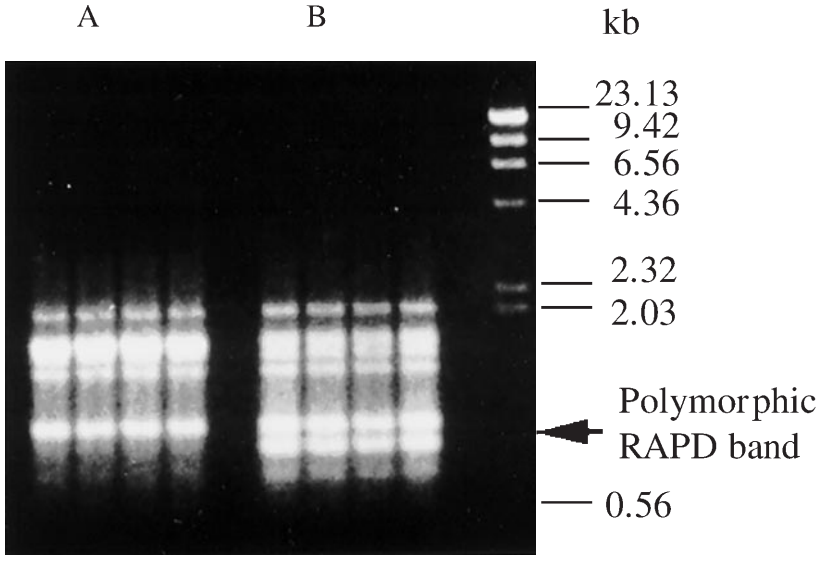

Fig. 2A, B RAPD profile of primer UBC140 on jointless pool *j-2/j-2* (**A**) and wild-type pool *J-2/j-2* (**B**). The polymorphic band (*arrow*) is amplified specifically from the jointless pool

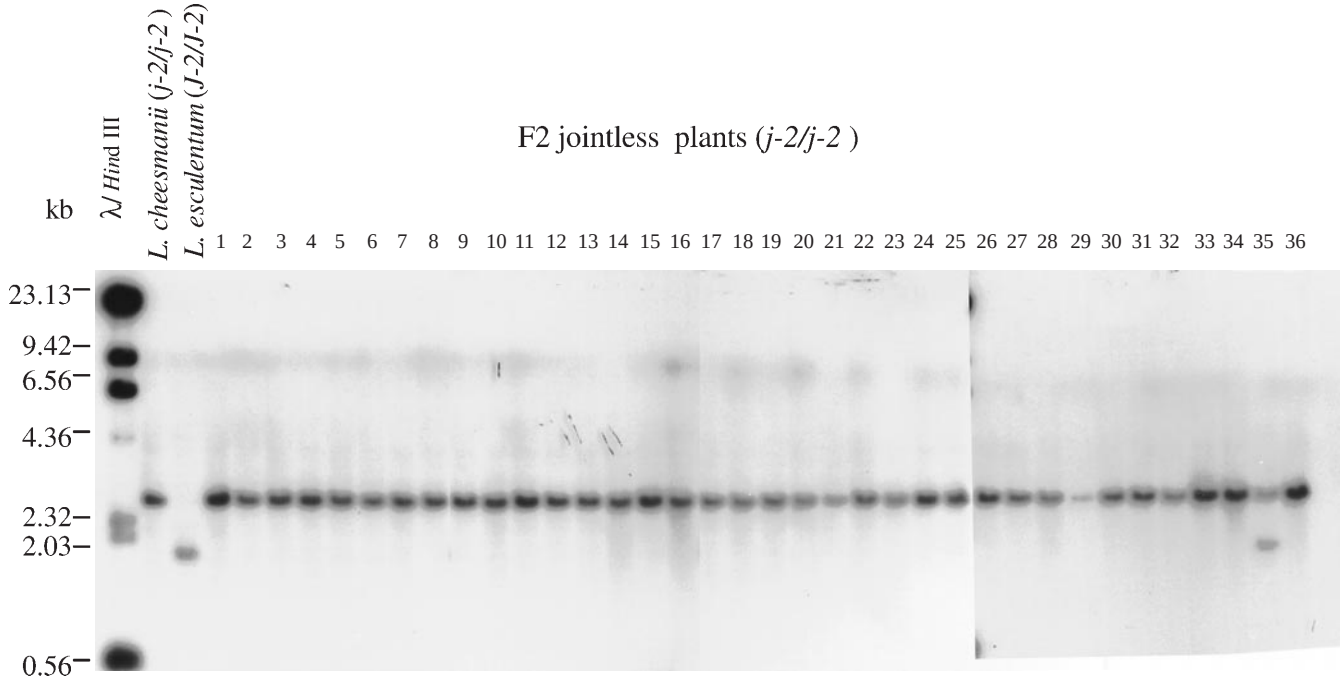

Fig. 3 Autoradiograph of a Southern blot hybridized with radioactively labeled RPD140. DNA (8 μg) from parents and 36 jointless F_2 plants were digested with *Eco*RI and electrophoresed through a 0.8% agarose gel

the parental lines of an interspecific F_2 cross, *L. esculentum* Heinz 1706 *J-2*/*J-2* and *L. cheesmanii* LA166 *j-2/j-2*. Seven RAPD markers did not show any polymorphic patterns. Only 2 of the markers classified as "low copy" appeared to be mappable (A19–2j2 and RPD140). Marker A19–2j2 was a dominant RFLP marker present only in the jointed parent and RPD140, amplified from the jointed pool (Fig. 2), produced a codominant RFLP with the restriction enzymes *Eco*RI and *Eco*RV. These two markers were hybridized to a panel of 36 jointless segregants from the interspecific F_2 cross. The A19–2j2 RFLP band was absent in 6 out of 36 *jointless-2* F_2 plants, suggesting that A19–2j2 was not tightly linked to the *j-2* locus (data not shown). When the RPD140 marker was hybridized to the same panel of jointless plants digested with *Eco*RI, the genotypes of 35 of the 36 plants contained the polymorphic band present specifically in the *jointless-2* parental line, *L. cheesmanii* LA166 but not on the other parent, as shown in Fig. 3, lanes 1–34, and 36. Only a single recombinant plant between RPD140 and the jointless phenotype was detected, as shown in Fig. 3, lane 35. These results suggested that the RPD140 marker was tightly linked to *j-2* and was therefore cloned into the PCR II vector (Invitrogen, USA) to produce a permanent copy of the marker.

Localization of RPD140 to tomato chromosome 12

To determine the chromosomal location of RPD140, the cloned RPD140 marker was hybridized to an RFLP survey filter containing restricted parental genomic DNAs of the Cornell reference map (Tanksley et al. 1992). An easily scorable RFLP was detected with the enzyme *Eco*RI. RPD140 was then hybridized to a mapping filter containing DNA from 67 F_2 plants restricted with *Eco*RI. The segregation of RPD140 was scored, and the data was compared against all the established linkage groups generated from this cross. RPD140 was surprisingly grouped to a cluster of markers on chromosome 12, and not on chromosome 11 as previously reported on the morphological map (Rick and Yoder 1988).

j-2 is located within a 3-cM interval between TG618 and RPD140 on chromosome 12

The genetic mapping of RPD140 suggested that the *j-2* locus was located on linkage group 12. Unfortunately, the relative position of *j-2* to other markers on chromosome 12 could not be determined because the Cornell reference mapping population did not segregate for the *j-2* phenotype. To determine the genetic relationship of these markers to *j-2* and verify that *j-2* was indeed located in chromosome 12, we screened nine chromosome 12 RFLP markers near RPD140 for RFLPs between Heinz 1706 (*J-2*/*J-2*) and LA166 (*j-2*/*j-2*) and mapped them in our interspecific *j-2* mapping population of 151 F_2 plants.

After identifying the appropriate enzyme-marker combinations that produced codominant polymorphic bands between the two mapping parental lines, we hybridized all nine markers, except for marker CT99, against the 151 F_2 plant mapping population including 36 jointless F_2 plants. Hybridization results were scored and analyzed to produce the map shown on Fig. 4. RPD140 was shown to be tightly linked to *j-2* as it was mapped 0.7 cM from the *j-2* locus. The *j-2* locus was flanked by TG618 on the upper arm and RPD140 on the lower arm in an interval of approximately 3.0 cM. Two of the chromosome 12 markers, CD22 and TG112, cosegregated exactly, with RPD140 which provides additional confirmation that the *j-2* locus resides on chromosome 12.

As for CT99, it was mapped in the same genetic position as TG360 on chromosome 12 of the Cornell reference mapping population. However in our mapping population, TG360 was mapped 7.1 cM away from the *j-2* locus, and the actual position of CT99 relative to the *j-2* locus could not be determined accurately. In order to resolve this discrepancy, we adopted a technique called cleaved amplified polymorphic sequence (CAPS) (Konieczny and Ausubel 1993) in which amplified products from each parental line are digested with a restriction enzyme resulting in a codominant polymorphic marker instead of a dominant marker. Specifically designed primers (CT99F and CT99R) were used to amplify a single 1.35-kb fragment from the *L. esculentum* (Heinz 1706) *J-2*/*J-2* and *L. cheesmanii* (LA166) *j-2*/*j-2* parents. A unique restriction enzyme *Tha*I was found to restrict the 1.35-kb fragments from Heinz 1706 *J-2*/*J-2* and LA166 *j-2*/*j-2* plants into two or three different fragments, respectively (data not shown). The CT99 marker was thus converted into a codominant marker and was mapped using the *Tha*I restriction enzyme on each amplified fragment of the 151 F_2 plant mapping population. The genetic relationship between CT99 and TG360 was resolved and resulted in CT99 being mapped 1.3 cM below TG360 towards *j-2* (Fig. 4).

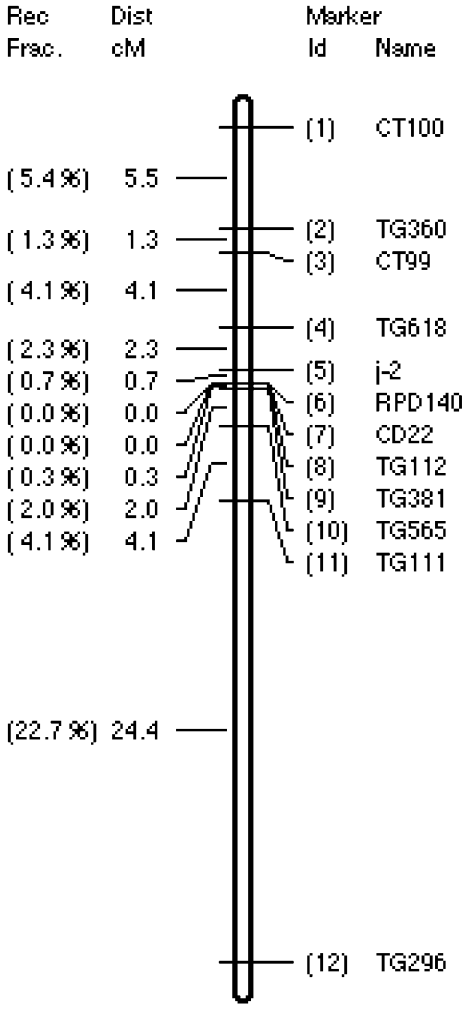

Fig. 4 Map of the *jointless-2* locus on chromosome 12. The *j-2* locus is contained in a 3-cM interval flanked by RPD140 and TG618. The CT99 marker was mapped using the CAPS method and is located 1.3 cM *below* TG360

Discussion

Scoring the F_2 mapping population

The concept of mapping a gene with RFLP markers relies on mapping parental lines that are genetically diver-

gent enough to reveal allelic differences between parents so that markers can segregate in the progenies and be informative. However, crosses of distantly related species with a different trait will not necessarily express the desired phenotype if it is affected by some unknown modifiers, as was shown for *j-2* in a previous interspecific cross between *L. esculentum J-2*/*J-2* and *L. pennellii j-2*/*j-2* (Wing et al. 1994). In our cross, 157 F_2 plants were scored for the presence or absence of flower pedicel abscission zone structures, and not a single progeny could be identified unambiguously as being jointless. Miller and Tanksley (1990) studied the extent of polymorphism between the standard tomato cultivar, *L. esculentum* VF36, and other *Lycopersicon* accessions and showed that *L. esculentum* is more closely related *to L. pennellii* than to *L. cheesmanii*. For a single probe-enzyme combination in a cross between *L. esculentum* and *L. pennellii*, 85% of the probes were, on aerage, polymorphic. When five restriction enzymes were applied up to 99% of the probes were polymorphic. Even though the interspecific cross between *L. esculentum J-2*/*J-2* and *L. pennellii j-2*/*j-2* was supposed to have informative segregating markers, they did not show distinct phenotypes of jointless, making it difficult to score (Wing et al. 1994). The percentage of polymorphism between *L. esculentum* and *L. cheesmanii* in a single probe-enzyme combination was only 27%; with five restriction enzymes it was 67%, lower than that of polymorphism between previously studied parents. However, the progeny phenotypes segregating for *j-2* could be scored easily, suggesting 100% penetrance of *j-2* in this cross. In the first mapping population, 36 of 151 F_2 progenies (derived from *L. esculentum J-2*/*J-2* and *L. cheesmanii j-2*/*j-2*) were phenotypically distinct as being jointless, accounting for approximately 25% of the total F_2 plants and indicating that the jointless phenotype corresponds to a single Mendelian inheritance pattern.

Identification and genetic mapping of a tightly linked marker to *jointless-2*

Molecular markers for the *j-2* locus have not been reported. In order to apply a map-based cloning strategy to isolate the *j-2* gene, we required a molecular marker. In this study we identified a single low-copy RAPD marker linked to the *j-2* locus using BSA and converted it into a codominant RFLP marker (RPD140) for genetic mapping that surprisingly mapped to tomato chromosome 12 using the Cornell reference tomato mapping population. This chromosome placement disagrees with the finding of C. Rick (1956) that the *j-2* locus is located on chromosome 11. In fact, there has always been a question as to whether the *j-2* locus was ever precisely mapped. Lachman and Tristan (1973) examined the linkage among *j-2*, *hl*, and *a* loci on chromosome 11 and placed *j-2* at the distal end of chromosome 11, 45.4 cM away from the *hl* locus. Butler (1973) questioned the placement of *j-2* on chromosome 11 with such a genetic distance so close to 50 cM. Moreover, a re-evaluation of the chromosome 11 classical genetic map suggested that *j-2* locus could not be accurately placed on chromosome 11 (Van Tuinen et al. 1998).

To confirm that the *j-2* locus is indeed located on tomato chromosome 12, we demonstrated that ten chromosome 12 RFLP markers, all showing linkage with RPD140 in the Cornell reference map, showed linkage to the *j-2* gene in our *L. esculentum* × *L. cheesmanii* cross. Our work here confirms the suspicions that *j-2* is not on chromosome 11 and clearly demonstrates that the *j-2* locus derived from the original *L. cheesmanii* accession (LA166) is located on tomato chromosome 12 flanked by two closely linked markers RPD140 and TG618 in a 3.0-cM interval. The positioning of the *j-2* locus on chromosome 12, between TG618 and RPD140, now allows us to proceed to the next steps in its isolation through map-based cloning by physical mapping and chromosome walking.

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