Identification of Genetic Factors Affecting Fruit Weight in the Tomato (Solanum lycopersicum L.) Cultivar ‘Micro-Tom’

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‘Micro-Tom’, a dwarf tomato cultivar, has been used as a convenient model system in tomato research. Previous studies have shown that several genes are involved in the phenotype, but to date no study has focused on the fruit weight. In this study, we tried to clarify genetic factors that regulate the fruit weight of ‘Micro-Tom’ using an F2 population derived from ‘Micro-Tom’ and ‘MPK-1’, a mid-size tomato cultivar. The F2 population showed a continuous and transgressive segregation in terms of fruit weight, suggesting that the fruit weight was regulated by multiple loci. To identify these loci, quantitative trait loci (QTL) analysis was performed. Three QTLs located on chromosomes 4, 7, and 9 were found to regulate fruit weight, and were designated as qfw4.1, qfw7.1, and qfw9.1. Of these QTLs, qfw4.1 exhibited the highest logarithm of the odds score. We confirmed the effect of qfw4.1 in the F3 population and showed that it regulates fruit weight without affecting locule number. In addition, being homozygous for the Micro-Tom allele at the marker linked to qfw4.1 reduced vegetative size, suggesting that qfw4.1 regulates not only fruit weight, but also vegetative size in ‘Micro-Tom’.

Key Words: dwarf, QTL analysis, vegetative size.

Introduction

The tomato (Solanum lycopersicum L.) is an important vegetable that is cultivated around the world. Among vegetables, the annual amount of tomato production is second highest: about 182 million tons in 2018 (FAOSTAT, 2018). A two-step domestication process has been proposed as the source of the tomato: A first domestication from S. pimpinellifolium to S. lycopersicum var. cerasiforme occurred in the Andean region of southern Ecuador and northern Peru. Subsequently, S. lycopersicum var. cerasiforme migrated to Mesoamerica, where it evolved into S. lycopersicum var. lycopersicum (Blanca et al., 2012, 2015). During domestication, the fruit morphology, fruit quality, and plant architecture changed. Especially, the fruit size increased dramatically compared with that of its wild relatives (Tanksley, 2004). Fruit size is an important agricultural trait that is controlled by quantitative trait loci (QTL), and many studies have been conducted to identify the relevant loci and genes.

To date, five QTLs controlling fruit size have been cloned or fine mapped: fruit weight (fw) 2.2 (Frary et al., 2000), fw3.2 (Chakrabarti et al., 2013), locule (lc) (Muños et al., 2011), fasciated (fas) (Cong et al., 2008; Xu et al., 2015), and fw11.3 (Mu et al., 2017). Among the genes, fw2.2 and fw3.2 affect cell division rates and/or duration in the developing ovary or fruit, and mutation in these genes increases the number of cell layers in the pericarp (Chakrabarti et al., 2013; Frary et al., 2000). Expression of FW2.2 was observed in the pre-anthesis carpel, and carpels of a large-fruited near-isogenic line (NIL) carrying the mutant fw2.2 allele were larger than those of a small-fruited NIL carrying the wild-type allele at anthesis (Frary et al., 2000). On the other hand, the expression of FW3.2 was very high in the pericarp not of the ovary, but rather the growing fruit, and such expression affects cell layers in the pericarp (Chakrabarti et al., 2013). The functions of FAS and LC control the locule number. Cong et al. (2008) reported that a mutation in the YABBY gene underlies the fas locus, and a 6- to 8-kb insertion in the first intron of YABBY may result in its downregulation. How-
However, Huang and van der Knaap (2011) showed that the mutation at the *fas* locus was not a 6- to 8-kb insertion but a 294-kb inversion in the first intron of *YABBY* and 1 kb upstream of *SICLV3*, participating in the regulation of fruit locule development. Furthermore, transgenic complementation experiments carried out by Xu et al. (2015) provided evidence that disrupting the promoter of *SICLV3* by inversion underlies the *fas* mutant. The *lc* locus is located at a region near *WUSCHEL*, which is a gene controlling the fate of stem cells in the apical meristem (Barrero et al., 2006). Muños et al. (2011) reported that two single-nucleotide polymorphisms are located 1080 bp downstream of *WUSCHEL* and may be involved in regulation of expression. The *Cell Size Regulator* (*CSR*) gene underlies the *fw11.3* (Huang and van der Knaap, 2011; Mu et al., 2017), and it controls pericarp cell size. The expression of *CSR/FW11.3* was highest in the columnella and increased between days 7 and 33 post anthesis. This period coincides with the stage of cell expansion, which in turn suggests that this gene may be involved in cellular differentiation (Mu et al., 2017).

‘Micro-Tom’, a dwarf tomato cultivar, can be grown under fluorescent light at a high density and has a short life cycle. Therefore, it is used as a convenient model system for tomato research. The phenotype of ‘Micro-Tom’ is due to mutations in three major genes: *DWARF* (*Solyc02g089160*), *SELF-PRUNING* (*Solyc06g074350*), and *SIGLK2* (*Solyc10g008160*) (Martí et al., 2006; Pnueli et al., 1998; Powell et al., 2012). The *DWARF* gene encodes a cytochrome P450 protein, which functions in the brassinosteroid biosynthesis pathway. The *DWARF* mutation reduces the internode length and produces rugose and dark green leaves. The *SELF-PRUNING* (*SP*) gene is an ortholog of *TERMINAL FLOWER 1*, a member of the phosphatidyl ethanolamine binding protein group that suppresses flowering in *Arabidopsis thaliana* (Bradley et al., 1997; Pnueli et al., 1998, 2001). The *sp* mutation causes the determinate growth habit in the tomato. *SIGLK2* encodes the Golden 2-like (*GLK*) transcription factor, and its mutation produces uniform ripening of fruits. Previous studies have identified the above factors that determine the characteristic phenotype of ‘Micro-Tom’. However, there has been no study that focused on genetic factors affecting the fruit weight of ‘Micro-Tom’.

In this study, we performed QTL analysis using an F₂ population derived from ‘Micro-Tom’ and ‘MPK-1’, a tomato cultivar that forms mid-size fruits, to identify QTLs that regulate the fruit size of ‘Micro-Tom’. In addition, fruit and vegetative traits in the F₂ and F₄ populations were investigated to explore the effect of the main QTL on vegetative and fruit traits.

**Materials and Methods**

**Plant materials and plant growth conditions**

For QTL analysis of fruit weight we used an F₂ population (n = 89) from a cross between a tomato cultivar, ‘Micro-Tom’ (Tomato Growers Supply Company, Florida, USA), and a parthenocarpic cultivar, ‘MPK-1’, which forms mid-size fruits. Plant growth conditions were those described by Takisawa et al. (2018). For the progeny test, an F₂ population (n = 64) was developed from a single F₁ plant that was heterozygous at the marker linked to *qfw4.1* and homozygous for the MPK-1 allele at the marker linked to other QTLs. Seeds for the F₂ population were sown in April 2018 and the plants were grown in the greenhouse at the Kizu Experimental Farm of Kyoto University at Kizugawa, Japan (Kizu Farm; 34°73′N, 135°84′E). To clarify the effect of *qfw4.1* on vegetative traits, we also used an F₄ population from a single F₁ plant that was used in the progeny test; this F₁ plant was heterozygous at the marker linked to *qfw4.1*. Seeds of the F₂ population were sown in March 2019 and the plants were grown in a greenhouse at the Kizu Farm.

**DNA extraction**

Young leaves were used for extraction of DNA. The DNA for single-nucleotide polymorphism (SNP) analysis was extracted using the DNeasy Plant Mini Kit (Qiagen GmbH, Hilden, Germany) according to the manufacturer’s instructions. The DNA for genotyping of F₁ and F₄ populations was extracted with TPE buffer (100 mM Tris-HCl, pH 9.5, 1 M KCl, 10 mM ethylene-diamine tetraacetic acid, pH 8.0) as described by Kim et al. (2016), with some minor modifications.

**Genotyping of F₂, F₃, and F₄ populations**

For 89 plants of the F₂ population, SNP analysis was conducted according to the method of Takisawa et al. (2018). To develop an insertion-deletion (Indel) marker, *fw8* (forward primer sequence: 5′-AAGCAGCTCATGTGATTATG-3′, reverse primer sequence: 5′-GCTGGGTTCCATGGG-3′), which is flanked by *qfw4.1*, we used the sequence data for the ‘Micro-Tom’ genome from TOMATOMICS (Kudo et al., 2017) and the ‘MPK-1’ genome obtained by Takisawa et al. (2018). The distance between *fw8* and the nearest marker (Affx-107976622) of *qfw4.1* is 357 kb. To genotype the F₁ and F₄ population, *fw8* was subjected to polymerase chain reaction (PCR)-based marker analysis. PCR reactions were performed in a total volume of 8 μL using BIOTAQTM DNA polymerase (Bioline, London, UK) according to the manufacturer’s instructions, with some minor modifications. Amplification was carried out under the following conditions: 94°C for 5 min; 30 cycles of 94°C for 30 s, 60°C for 30 s, and 72°C for 30 s; and a final extension step at 72°C for 7 min. The PCR products were run on 1.0% agarose gels and stained with ethidium bromide.

**Linkage map construction and QTL analysis**

We applied 1428 SNPs to construct a linkage map as
shown in Takisawa et al. (2018), except for three erroneous SNPs (Affx-107982944, Affx-107976009, and Affx-108000340) on chromosome 4 (Table S1). A genetic map was constructed using JoinMap4.1 mapping software with default settings of a maximum-likelihood mapping algorithm. With the linkage map and fruit weight from the F₂ population, QTL analysis was conducted. Composite interval mapping was performed with Windows QTL Cartographer software, v.2.5 (Wang et al., 2012). The logarithm of odds (LOD) thresholds of fruit weight were determined by one thousand permutation tests at the 5% level. In order to evaluate the effect of detected QTLs, the F₂ population was classified in terms of the genotypes of the nearest markers linked to the QTLs. Multiple comparisons were conducted with Tukey’s test.

**Investigation of fruit and vegetative traits**

We pollinated flowers at anthesis, and 5–10 fruits per plant were weighed from the 89 plants of the F₂ population and 6–10 mature fruits per plant were weighed from the 64 plants of the F₂ population. After weights had been measured, fruits were cut at the equatorial plane and the locule number in the F₂ population was determined. In addition, stem length and leaf and stem weight were investigated at 61 days after seeding in 10 plants of the F₂ population that were homozygous for the Micro-Tom allele and MPK-1 allele and heterozygous at the marker linked to qfw4.1.

**Results**

**Frequency distribution of the fruit weight in the F₂ population**

The average fruit weights for the ‘Micro-Tom’ and ‘MPK-1’ were 5.6 and 27.1 g, respectively. The F₂ population showed a continuous and transgressive segregation in terms of average fruit weight, which ranged from 6.8 to 75.1 g (Fig. 1). This transgressive segregation was likely to be observed because ‘MPK-1’ was not genetically fixed, and the continuous segregation suggests that the fruit weight for the F₂ population was regulated by multiple loci.

**QTL analysis of fruit weight**

The linkage map covered a total of 2058 cM and contained 12 chromosomes. The average marker interval of this map was 1.4 cM. We detected three QTLs for fruit weight on chromosomes 4, 7, and 9, which were designated as qfw4.1, qfw7.1, and qfw9.1, respectively (Table 1; Fig. S1). The QTLs qfw4.1, qfw7.1, and qfw9.1 were positioned at 269.4 cM on chromosome 4 (LOD 19.3, \(R² = 12.4\)), at 5.5 cM on chromosome 7 (LOD 4.1, \(R² = 8.3\)), and at 159.5 cM on chromosome 9 (LOD 6.5, \(R² = 22.2\)), respectively (Table 1). The additive effects of all QTLs were positive, indicating that the Micro-Tom allele reduced the fruit weight on all QTLs. The fruit weights of F₂ plants heterozygous and homozygous for the MPK-1 allele at the marker nearest to qfw4.1 (Affx-107976622) were higher than for plants homozygous for the Micro-Tom allele (Table 2). On the other hand, the fruit weights for F₂ plants heterozygous and homozygous for the Micro-Tom allele at the marker nearest to qfw9.1 (Affx-107997069) were significantly lower than for plants homozygous for the MPK-1 allele. In addition, there was no significant difference among the fruit weights of F₂ plants with each genotype at the marker nearest to qfw7.1 (Affx-107981829).

**Progeny test and investigation of the relationship between fruit weight and locule number**

Among the three QTLs, the LOD score was highest at the qfw4.1 locus. Therefore, to confirm the effect of qfw4.1 on fruit weight and to clarify the relationship between fruit weight and locule number, we developed an F₂ population from a single F₁ plant with a heterozygous allele at the marker nearest to qfw4.1 and homozygous for MPK-1 at the markers nearest to qfw7.1 and qfw9.1. The fruit weights of plants heterozygous and

![Fig. 1. Frequency distribution of fruit weights in the F₂ population (n = 89).](image-url)
homzygous for the MPK-1 allele at fw8, a marker linked to qfw4.1, were significantly greater than plants homozygous for the Micro-Tom allele (Table 3). On the other hand, there was no significant difference in the locule number among plants with each genotype.

**The effect of qfw4.1 on vegetative traits**

Using the F4 population, the effects of qfw4.1 on vegetative traits were examined. The stem length and the fresh weights of leaves and stems of plants heterozygous and homozygous for the MPK-1 allele at fw8 were significantly greater than those of plants homozygous for the Micro-Tom allele at 61 days after seeding (Table 4; Fig. S2).

### Discussion

Our study revealed that three QTLs, qfw4.1, qfw7.1, and qfw9.1, affected the fruit weight of ‘Micro-Tom’. Grandillo et al. (1999) showed that there were three QTLs, fw4.1, fw4.a, and fw4.2, on chromosome 4; five QTLs, fw7.a, fw7.1, fw7.2, fw7.3, and fw7.b, on chromosome 7; and four QTLs, fw9.1, fw9.2, fw9.a, and fw9.b, on chromosome 9. In addition, Zhou et al. (2016) reported that one QTL for fruit weight was detected on chromosome 4 in a BC2S2 population derived from the tomato inbred line 1052 and S. pennelli LA0716, and the QTL was designated as fw4a. According to Grandillo et al. (1999), fw4.2 is positioned between RFLP markers CT50 and CT173, which are at 61007810–61008303 and 62612040–62612361 on chromosome 4 in SL2.40 (Sol Genomics Network database), and fw9.a and fw9.b are positioned between RFLP markers TG248 and CT220, which are at 63196768–63197263 bp and 67545779–67546054 bp on chromosome 9 in SL2.40 (Sol Genomics Network database). In addition, the physical position of fw4a was 31651604–56807847 on chromosome 4 in SL2.40.

In this study, qfw4.1 was found between SNP markers Affx-93198177 and Affx-107973206, which are at 57315080–62674212 on chromosome 4 in SL2.40, and qfw9.1 was positioned between SNP markers Affx-107980432 and Affx-107992489, which are at 62395763–63741353 on chromosome 9 in SL2.40. The position of qfw4.1 overlapped not with fw4.a, but rather fw4.2, and the position of qfw9.1 overlapped with fw9.a and fw9.b, suggesting that qfw4.1 and qfw9.1 correspond to fw4.2 and fw9.a and fw9.b, respectively. On the other hand, there is no reported QTL in the region of qfw7.1, which indicates that this is a new QTL that regulates fruit size.

In the tomato, cell number, cell size, and locule number determine the fruit size. There are five cloned genes that are involved in fruit size: fw2.2 (Frary et al., 2000),

### Table 2. Average fruit weights of each genotype for the markers linked to qfw4.1, qfw7.1, and qfw9.1 in the F2 population.

| Locus | Genotype | Fruit weight | n |
|-------|----------|--------------|---|
|       |          | Mean         | SE |
| qfw4.1| M        | 14.0         | 1.0 |
|       | H        | 37.1         | 1.7 |
|       | K        | 38.7         | 3.4 |
| qfw7.1| M        | 27.0         | 2.0 |
|       | H        | 32.4         | 2.7 |
|       | K        | 36.5         | 3.4 |
| qfw9.1| M        | 28.1         | 2.5 |
|       | H        | 27.1         | 2.3 |
|       | K        | 42.8         | 3.4 |

* Genotypes of the nearest marker. M = Micro-Tom homozygous, H = Heterozygous, K = MPK-1 homozygous.
* Values followed by the same letter are not significantly different at P < 0.05 using Tukey’s test.
* SE: standard error.
* n: number of plants for each genotype.

### Table 3. Average fruit weights and locule numbers of each genotype for the markers linked to qfw4.1 in the F2 population.

| Locus | Genotype | Fruit weight | Locule number | n *
|-------|----------|--------------|---------------|
|       |          | Mean         | Mean          | |
|       |          | SE           | SE            | |
| qfw4.1| M        | 16.3         | 3.4           | 12 |
|       | H        | 45.4         | 3.1           | 32 |
|       | K        | 52.1         | 3.2           | 20 |

* Genotypes of the nearest marker. M = Micro-Tom homozygous, H = Heterozygous, K = MPK-1 homozygous.
* Values followed by the same letter are not significantly different at P < 0.05 using Tukey’s test.
* SE: standard error.
* n: number of plants for each genotype.

### Table 4. Average stem length, leaf and stem weight of each genotype for the marker linked to qfw4.1 in the F2 population.

| Locus | Genotype | Stem length | Leaf weight | Stem weight | n *
|-------|----------|-------------|-------------|-------------|
|       |          | Mean        | Mean        | Mean        | |
|       |          | SE          | SE          | SE          | |
| qfw4.1| M        | 19.5        | 8.7         | 5.4         | 10 |
|       | H        | 29.2        | 18.8        | 10.9        | 10 |
|       | K        | 28.4        | 17.7        | 9.7         | 10 |

* Genotypes of the nearest marker. M = Micro-Tom homozygous, H = Heterozygous, K = MPK-1 homozygous.
* Values followed by the same letter are not significantly different at P < 0.05 using Tukey’s test.
* SE: standard error.
* n: number of plants for each genotype.
differences were found in locule number for plants (Chakrabarti et al., 2013, lc (Muños et al., 2011), fas (Cong et al., 2008; Xu et al., 2015), and fw11.3 (Mu et al., 2017). It has been reported that fw2.2 and fw3.2 are related to regulating cell division, fw11.3 is involved in regulating cell size, and fas and lc change fruit size through the locule number. In this study, no significant differences were found in locule number for plants homozygous for the MPK-1 allele and for the Micro-Tom allele at the marker linked to qfw4.1, which indicates that it regulates fruit size not via locule number, but through cell size or cell number, as with fw11.3 or fw2.2 and fw3.2.

We found that qfw4.1 reduced not only the fruit weight, but also the vegetative size. It has been reported that three genes, DWARF, SP, and SIGLK2, determine the characteristic phenotype of ‘Micro-Tom’ (Martí et al., 2006; Pnueli et al., 1998; Powell et al., 2012). Of the three genes, only DWARF affects vegetative size, and the dwarf mutation reduces internode length and produces rugose and dark green leaves. However, DWARF is positioned not on chromosome 4, but on chromosome 2 (Martí et al., 2006). Meissner et al. (1997) reported that dwarf and miniature are the two genes involved in the ‘Micro-Tom’ phenotype and suggested that miniature is a recessive gene. Martí et al. (2006) also suggested that ‘Micro-Tom’ carries at least one mutation, in addition to dwarf, that reduces internode length. Although these reports suggest the existence of miniature which regulates the ‘Micro-Tom’ phenotype, its chromosome position remains unknown. In this study, qfw4.1 significantly reduced stem length and vegetative weight and the effect was recessive. These results suggest that qfw4.1 may correspond to miniature. However, the population in the present study was different from those used in the studies of Meissner et al. (1997) or Martí et al. (2006). Further research will be needed to clarify the relationship between qfw4.1 and miniature.

In conclusion, we identified three QTLs regulating fruit weight in ‘Micro-Tom’. Of the three QTLs, the LOD score of qfw4.1 was the highest and its effect was confirmed in an F3 population. The QTL qfw4.1 regulates fruit size not by locule number, but by cell size or cell number. In addition, it regulates vegetative size and may be a candidate QTL for miniature, which determines the ‘Micro-Tom’ phenotype. ‘Micro-Tom’ is considered a good model for the study of fruits. Research results on the characterization of genes regulating the fruit and vegetative size of ‘Micro-Tom’ are making an important contribution to understanding the characteristics of the ‘Micro-Tom’ phenotype.

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