Theor Appl Genet (1999) 99:978–987

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# Identifying the loci responsible for natural variation in fruit size and shape in tomato

Received: 21 January 1999 / Accepted: 12 March 1999

**Abstract** Fruit size and shape are two major factors determining yield, quality and consumer acceptability for many crops. Like most traits important to agriculture, both are quantitatively inherited. Despite their economic importance none of the genes controlling either of these traits have been cloned, and little is known about the control of the size and shape of domesticated fruit. Tomato represents a model fruit-bearing domesticated species characterized by a wide morphological diversity of fruits. The many genetic and genomic tools available for this crop can be used to unraveal the molecular bases of the developmental stages which presumably influence fruit architecture, size and shape. The goal of this review is to summarize data from the tomato QTL studies conducted over the past 15 years, which together allow the identification of the major QTLs responsible for fruit domestication in tomato. These results provide the starting point for the isolation of the genes involved in fruit-size/shape determination in tomato and potentially other fruit-bearing plants.

**Key word** Domestication · Fruit size · Fruit shape · QTL · Tomato

## Introduction

Fruit is a major and important portion of the human diet, yet the fruit we associate with modern agriculture is not typical of the fruit born by wild plants. For example, wild tomato species produce small, round fruit weighing only a few grams. In contrast, cultivated tomatoes occur in a wide variety of forms and shapes and can weigh as much as 500 g, more than a 100-fold increase in mass over wild-types. This transition from small, more uniform fruit types in the wild, to cultivated types bearing disproportionally large fruit of highly variable shapes is a scenario that has been repeated over and over in the domestication of other fruit-bearing plants such as peppers, eggplants and melons (Nothmann 1986; Simmonds 1995).

While our knowledge of the genes and developmental pathways controlling floral development has advanced rapidly in the past few years, little is known about the control of the size and shape of domesticated fruit. Thus far, none of the genes involved in this process are known. Limiting the progress has been the fact that most variation in fruit size and shape is quantitatively inherited (polygenic), making identification and cloning of individual loci difficult. Second, the current model for plant molecular biology, *Arabidopsis thaliana*, was never domesticated and hence has not developed the level of phenotypic diversity for fruit size and shape seen in agricultural species. Unraveling the genetic pathway by which domesticated plants came to bear fruit of such extreme sizes and shapes will provide insights into recent plant evolution, but may also further reveal how plants control both temporal and spatial growth of organs.

The goal of this review is to summarize what is currently known about the genetic control of fruit development in a model fruit-bearing domesticated species, tomato. Tomato is an excellent system for studying the genetic basis of fruit-size/shape determination, because of the wide morphological diversity of fruits that can be found within the genus *Lycopersicon* and the many genetic and genomic tools that can now be focused on elucidating the control of complex traits.

## Control of fruit size and carpel number

Fruits can be defined anatomically as ‘‘matured carpels with or without accessory structures and/or seeds”

Communicated by F. Salamini

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(Brooks 1957). Tomato fruits, similarly to grapes, bananas and dates, are fleshy fruits classified as berries because the thick pericarp encloses many seeds. When the ovary develops into a fruit, the ovary wall becomes the pericarp. However, in some species of plants, other parts of the total inflorescence structure develop into fruit flesh. For example the edible portion of strawberry fruits is the sweet, fleshy receptable on which many small fruits (the achenes) adhere. In pomes, exemplified by apples and pears, the edible portion is made of an enlarged receptable and floral tube tissues, which imbed the true fruits (ripened ovaries), which are not eaten.

For many tomato varieties differences in fruit size are established by the time of flowering, and the size of the ovary becomes a good predictor of the final size of the fruit (Houghtaling 1935; Yeager 1937). Indeed, clear differences are evident at a much earlier stage, when the ovary primordium is just beginning its development (Houghtaling 1935). Szymkowiak and Sussex (1992) have shown that in tomato the floral meristem size and carpel number is determined by the number of cells of the internal layer (L3) in the shoot apical meristem; thus, L3 can directly determine the eventual sink strength (i.e., activity of nutrient import) of the developing fruit.

A study conducted on semi-isogenic mutants of *Lycopersicon pimpinellifolium* differing in fruit size, indicated that the considerable difference in final fresh weight between the two mutants (approximately 3 vs 9 g) appear to be a function of the number and volume of the cells within the ovary prior to fertilization, the rate and duration of cell division and cell expansion thereafter (Bohner and Bangerth 1988 a).

## Control of fruit shape

The shape of tomato fruit can also vary significantly. Althoug all wild tomato species are characterized by round-shaped fruit, fruit shapes of tomato cultivars can range from round to pear-shaped, to plum, ovate, oblate or stuffer. A developmental analysis of size and shape conducted on seven tomato varieties and the current tomato (*L. pimpinellifolium*) indicated that shape in tomato is essentially determined by flowering, and that at post-anthesis there is only slight added divergence since width increases a little faster than length (Houghtaling 1935). A similar situation is found in various types of Cucurbita (Sinnott and Durham 1929; Sinnott and Kaiser 1934). In contrast, shape differences arising after flowering as the result of unequal growth rates in different directions have been reported in garden pepper (Kaiser 1935) and eggplant (*Solanum melongena*) (Kano et al. 1957).

Although the size and shape of fruit are largely determined genetically, both these characters can be influenced by environmental effects, such as nutrition, or by internal factors, the most relevant of which is the number and distribution of seeds within the fruit. The size and shape of a fruit can also vary in relation to the position of the fruit within the plant and the sequence of pollination among flowers. In tomato it has been observed that the first fruit on the first truss is generally larger in size than the rest and that it is also multilocular, which further supports the relation of locule number with fruit size (Sawhney and Greyson 1972; Bohner and Bangerth 1988 b). Also, in tomato, fruit growing at the distal position contains fewer cells than fruit at a proximal position of the same truss. The difference in fruit cell-number appears to be determined at the initiation of the floral primordium and is first evident in the carpels. However, the final size differences between proximal and distal fruits can be eliminated when all flowers are pollinated simultaneously (Bohner and Bangerth 1988 b). These observations suggest that actual sink strength is only partially determined by cell number and that it may also be regulated by the temporal and metabolic activities of cells during early fruit development as a function of fruit position and the efficiency of photoassimilate import.

## Role of humans in the creation of diversity in domesticated, fruit-bearing species

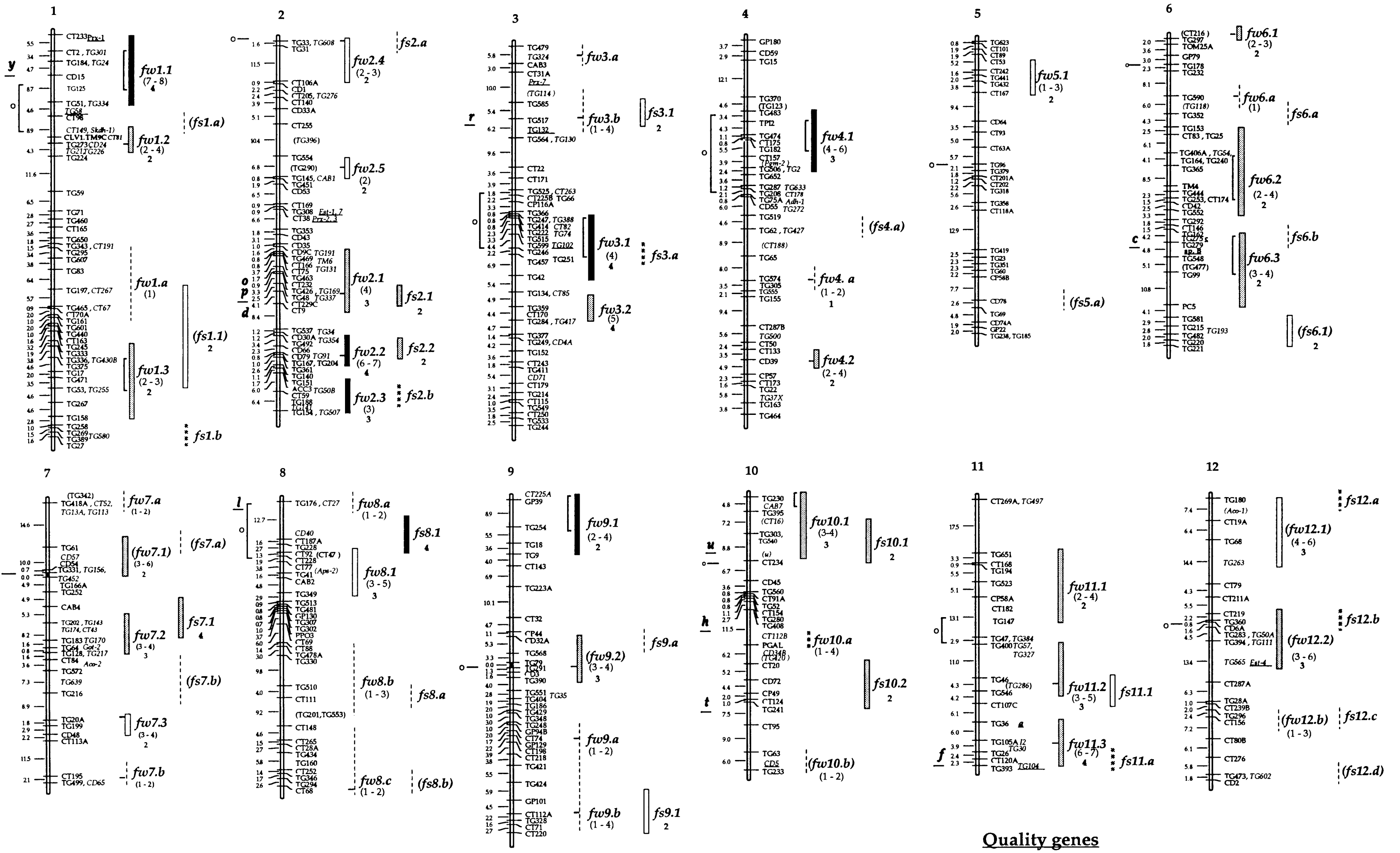
Most fruit-bearing species were domesticated in prehistoric times, so no one knows the actual pathway from which wild species gave rise to the larger-fruited domesticates. The most likely scenario is that early humans selected for mutations associated with larger fruit and variable shapes and, gradually, enough ‘‘large-fruited” and ‘‘non round-shaped” mutations accumulated to give us our present day cultivars. Genetic analysis of crosses between cultivated species and their wild relatives supports this idea. Progeny from these crosses almost always segregate in a continuous fashion with respect to fruit size and fruit shape, indicating that the domestication process involved mutations at a number of different genetic loci (MacArthur and Butler 1938; Benerjee and Kalloo 1989). The quantitative nature of fruit-size variation has severely inhibited the use of classical mendelian techniques to identify and characterize the individual gene mutations which enabled the domestication of fruit bearing species. As a result none of the genes controlling either the size or shape of fruit have been cloned.

## Identifying the loci controlling fruit size and shape variation

Tomato was one of the first species in which researchers began mapping quantitative trait loci using molecular markers (Tanksley et al. 1982; Paterson et al. 1988). Over the past 10 years more than 15 molecular mapping studies have been reported for this species, involving the study of more than 50 traits (for a review see Pillen et al. 1996). Two traits common to many of those studies are fruit size and fruit shape.

**Table 1** Number of significant QTLs for fruit size (FW) and fruit shape (FS). &: regions of the genome were identified as putatively containing a QTL if they met one or more of the following criteria: a significant effect was observed for a single marker/trait combination at a single location with $P < 0.001$; significant effects were observed in the same direction (i.e., either all positive effects or all negative effects) for a marker/trait combination at two or more locations with $P < 0.01$; significant effects were observed in the same direction for a marker/trait combination at three or more locations with $P < 0.1$. M = morphological marker; IS = isozyme marker; RF = RFLP; RA = RAPD; FW = fruit size; FS = fruit shape. % NS = % non segregating genome; NA: not available

| Cross | Wild species | Accession/ line | Reference | Population structure | No. of plants or families | No. of markers | Class of markers | % Marker genome coverage | % NS genome | QTL threshold | No. of FW QTLs | No. of FS QTLS |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| CL1 | *L. chmielewskii* | LA1028 | Paterson et al. 1988 | BC1 | 237 | 70 | 2 M (sp, u); 68 RF | 77 | | LOD > 2.4 | 6 | NA |
| CM2 (F2) | *L. cheesmaniii* | LA483 | Paterson et al. 1991 | F2/F3 | 350 | 72 | 2 M (sp, B); 70 RF | 92 | | LOD > 2.4 | 7 | NA |
| CM2 (F3) | *L. cheesmanii* | LA483 | Paterson et al. 1991 | F2/F3 | 350 | 72 | 2 M (sp, B); 70 RF | 92 | | LOD > 2.4 | 9 | NA |
| CMRI | *L. cheesmanii* | LA483 | Goldman et al. 1995 | R1 | 97 | 132 | 2 M (sp, B); 1 IS; 129 RF | 96 | | P < 0.01 | 12 | NA |
| CM3 | *L. cheesmanii* | Line L2 | Monforte et al. 1997 a, b | F2 | 200 | 22 | 3 IS; 19 RF | 38 | | P < 0.05 | 3 | NA |
| HR2 | *L. hirsutum* | LA1777 | Bernacchi et al. 1998 | BC2/BC3 | 200 | 122 | RF | 99 | 3.5 | & | 3 | 9 |
| PF2 | *L. parviflorum* | LA2133 | Fulton et al. (manuscript in preparation) | BC2/BC3 | 170 | 127 | RF | 99 | | & | 8 | 16 |
| PF1 | *L. parviflorum* | NA | Lindhout et al. 1994 | F2 | 292 | 45 | RF | 63 | | LOD > 4 | 1 | NA |
| PV | *L. peruvianum* | LA1708 | Fulton et al. 1997 | BC3/BC4 | 200 | 206 | RF | 99 | 35 | & | 10 | 12 |
| PM1 | *L. pimpinellifolium* | LA1589 | Grandillo and Tanksley 1996 | BC1 | 257 | 210 | 2 M (sp, u); 3 RA; 115 RF | 94 | | P < 0.001 | 7 | 2 |
| PM2 | *L. pimpinellifolium* | LA1589 | Tanksley et al. 1996 | BC2/BC2F1 and BC3 | 170 | 120 | 2 M (sp, u); 2 RA; 115 RF | 94 | | & | 8 | 4 |
| PM3 | *L. pimpinellifolium* | CIAS27 | Weller et al. 1988 | F2 | 1200 | 10 | 6 M; 4 IS; | 18 | | P < 0.05 | 10 | 4 |
| PM4a | *L. pimpinellifolium* | Line L1 | Monforte et al. 1997 a, b | F2 | 200 | 22 | 3 IS; 19 RF | 38 | | P < 0.05 | 4 | NA |
| PM4b | *L. pimpinellifolium* | Line L5 | Monforte et al. 1997 a, b | F2 | 150 | 22 | 3 IS; 19 RF | 38 | | P < 0.05 | 5 | NA |
| PM5 | *L. pimpinellifolium* | NA | Lindhout et al. 1994 | F2 | 292 | 45 | RF | 63 | | LOD > 4 | 1 | NA |
| PN1 | *L. pennellii* | LA716 | Tanksley et al. 1982 | BC1 | 400 | 12 | 12 IS | 25 | | P < 0.05 | 5 | NA |
| PNIL | *L. pennellii* | LA716 | Eshed and Zamir 1995 | IL | 50 | 173 | RF | 97 | | P < 0.05 | 18 | NA |



**QTL mapped in at least 2 independent studies**

- ▮ PVE or R2 (in%) > 20% in at least 1 study
- ▨ PVE or R2 (in%) 10% -20% in at least 1 study
- ▯ PVE or R2 (in%) < 10% in at least 1 study

**QTL mapped in a single study**

- ⋮ PVE or R2 (in%) > 20%
- ⋮ PVE or R2 (in%) 10% -20%
- ¦ PVE or R2 (in%) < 10%

(QTL name) ( ) = in at least 1 study this QTL was transgressive

QTL name
(n-m)
i

n = number of independent studies in which the QTL was mapped
m = number of independent studies in which the QTL was mapped including subthreshold QTL
i = number of different species in which the QTL was mapped not including subthreshold QTL

**Quality genes**

**Affecting fruit size**

*l* = lutescent (chr. 8)
*o* = ovate (chr . 2)
*p* = peach (chr. 2)
*f* = fasciated (chr. 11)
*t* = tangerine (chr. 10)

**Nearly neutral in fruit size effect**

*d* = dwarf (chr. 2)
*r* = yellow flesh (chr . 3)
*y* = unpigmented fruit epidermis (chr. 1)
*c* = potato leaf (chr. 6)
*h* = hairs absent (chr. 10)
*u* = uniform ripening (chr. 10)

## Fruit-size QTLs

### *Number of QTLs influencing fruit size*

For fruit size/weight (FW) seven wild species of tomato and seven different population structures have been tested, with a marker genome coverage ranging from only 18—25% for the first studies (Tanksley et al. 1982; Weller et al. 1988) to almost 100% for some of the most recent ones (Fulton et al. 1997; Bernacchi et al. 1998) (Table 1). The analytical approaches used to detect QTLs have been variable as have the probability thresholds chosen for declaring a quantitative trait locus (QTL) effect significant. Consequently, the estimated number of QTLs affecting FW varies from 3 (Bernacchi et al. 1998) to more than 18 (Eshed and Zamir 1995), depending on the population, the experimental design, and statistical thresholds. However, by comparing these studies, a common set of 28 QTLs for FW can be identified, that frequently show segregation in at least two independent studies (Fig. 1).

Early studies on the inheritance of tomato fruit size, conducted by means of classical quantitative genetic approaches, suggested that 5—6 gene pairs probably conditioned fruit size (Powers 1941; Fogle and Currence 1950). In a subsequent report, Ibarbia and Lambeth (1969) indicated that more than ten gene pairs, and possibly as many as 20, differentiated the parents for FW in the cross studied. The minimum number of loci affecting FW in an interspecific tomato cross was also estimated, using Wright's method, at between 10 and 11 (Wright 1968). Recently, Zeng et al. (1990) suggested that this estimator is of the order of 1/3 the actual number of genes, which is very close to our estimate of 28 FW QTLs.

However, most of these QTLs have not been fine-mapped yet, and refer to regions of the chromosome of variable extension, which could contain more than one gene influencing FW.

### *Magnitude of effects and gene action of fruit-size QTLs*

The percent of phenotypic variance explained by a given QTL [PVE from interval mapping or $R^2$ (in %) from ANOVA] is used as a measure of its phenotypic effect. Figure 2 shows the PVE or $R^2$ for FW QTLs reported thus far for tomato.

The 28 conserved FW QTLs can be classified on the basis of the magnitude of their effects (Fig. 1). For six (22%) of the QTLs the phenotypic variance explained in at least one independent study was greater than 20%. The FW QTL data collected so far, suggest that those QTLs that have been found to exert a major effect in at least one independent study, are also more likely identified in the largest number of different species. This is the case for *fw1.1, fw2.1, fw2.2, fw3.1, fw3.2,* and *fw11.3* which have been mapped in at least four independent interspecific studies. We refer to these as major QTLs and propose that they account for the majority of FW variation in tomato evolution. QTLs of major effect, and detected in a wide range of species, represent good candidates for future map-based cloning. However, only when the QTLs are cloned and complementation studies are conducted in all the species, will it be possible to determine whether each of the consensus QTLs identified really corresponded to a single gene or to more than one gene.

Gene action refers to the interaction of the alleles at a genetic locus. For tomato FW QTLs gene action could be determined in only two cases, CM2 and PNIL. In both the inheritance mode is intermediate between additivity and dominance (of the small-fruited allele). For the major FW QTL, *fw2.2*, partial dominance (d/a of 0.44) was observed for the *Lycopersicon pennellii* allele as compared with the *Lycopersicon esculentum* allele (Alpert et al. 1995). Partial dominance of small fruit size was suggested also in earlier studies conducted on several populations of tomato analyzed by means of classical quantitative genetic approaches (MacArthur and Butler 1938; Khalf-Allah and Mousa 1972; Banerjee and Kalloo 1989).

◀ **Fig. 1** Molecular linkage map of the tomato genome (based on Tanksley et al. 1992 and Pillen et al. 1996). Displayed to the right of each chromosome are *bars* indicating the positions for QTLs known to control fruit size (*fw*) and fruit shape (*fs*). QTLs are named according to trait abbreviations and are followed by the chromosome number. For QTLs mapped in at least two independent studies, a *second number* is used to distinguish QTLs mapping to the same chromosome and affecting the same trait (e.g., *fw1.1*). For QTLs mapped in a single study, *lower-case letters* are used (e.g., *fw1.a*)

## Fruit-shape QTLs

### *Number of QTLs influencing fruit shape*

Genetic studies of tomato fruit shape (FS) trace back to the beginning of this century (Hedrick and Booth 1907; Price and Drinkard 1908). Many reports have confirmed the presence of a major gene regulating the relative length of fruit – that is the length /diameter ratio – located at the locus *o* or *pr* on chromosome 2 (Jones 1917; Lindstrom 1929; MacArthur 1931; Currence 1934). Although several studies have suggested that other genes also affect FS (Groth 1912; Crane 1915; Warren 1924; MacArthur 1928; Yeager 1937; Butler 1941; Young and MacArthur 1947), the genetic basis of FS appears to be less complex than for FW. A major component of FS is locule number, which in tomato has been shown to be controlled by the genes *f* (fasciated) on chromosome 11 and *lc* (locule number) on chromosome 2 (MacArthur 1935; Yeager 1937; Zielinsky 1945; Young and MacArthur 1947; Butler 1951). In the homozygous recessive condition these genes induce fruit with a larger number of locules as compared to the wild-type.

QTLs influencing FS in tomato have been mapped by means of morphological and/or molecular markers in six

## FW QTL

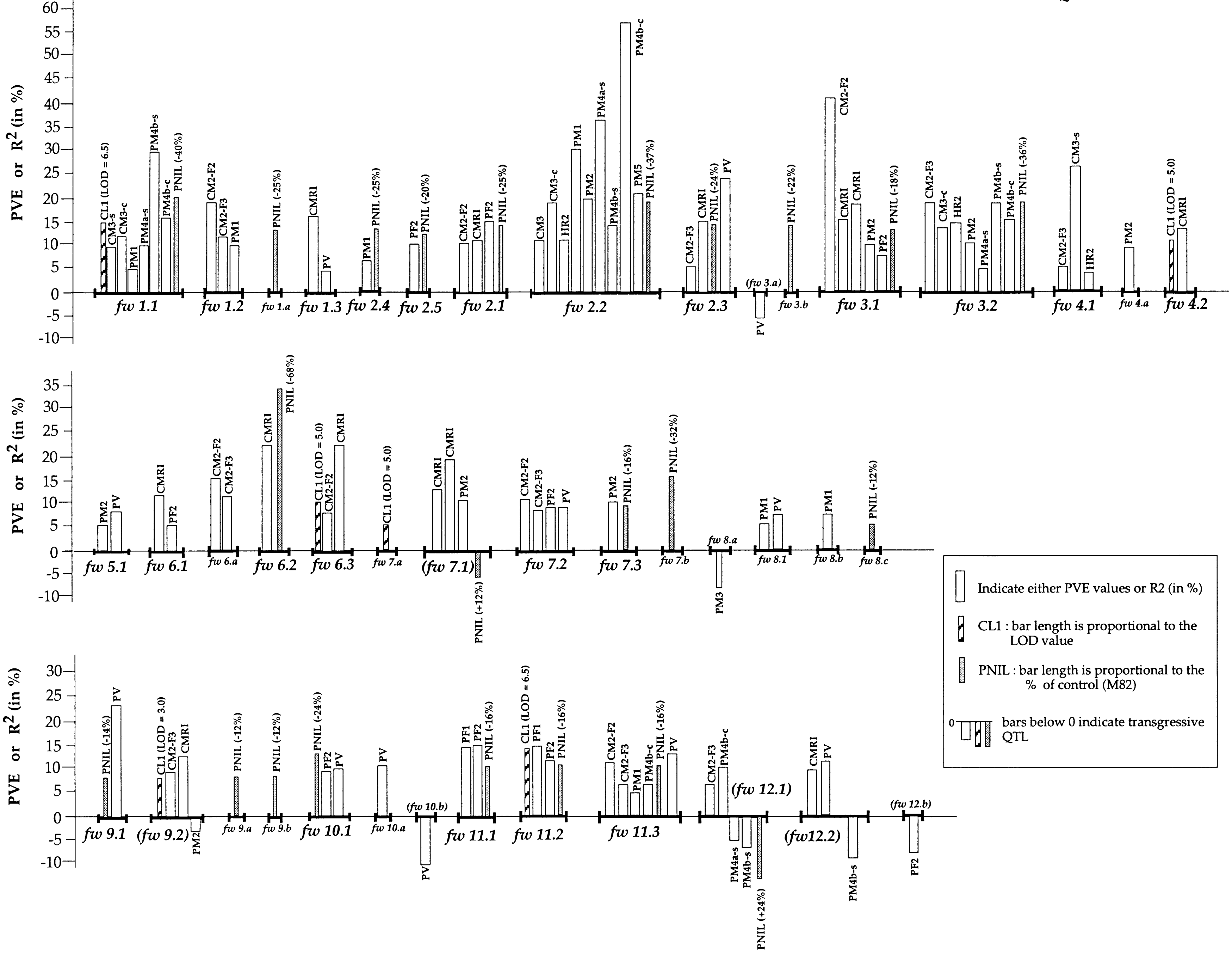
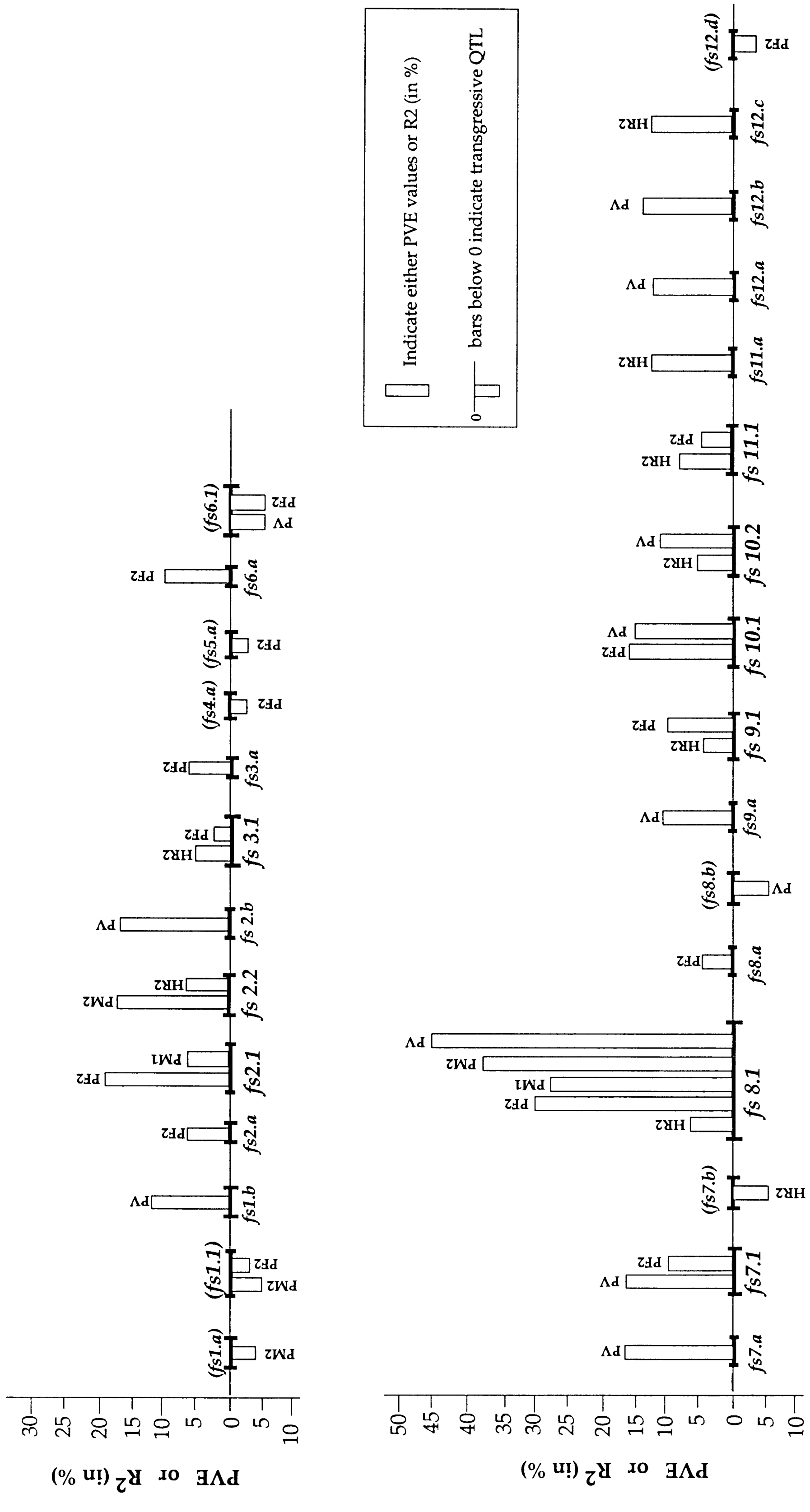

PVE or R2 (in %)
60
55
50
45
40
35
30
25
20
15
10
5
0
-5
-10
fw 1.1
CL1 (LOD = 6.5)
CM3-s
CM3-c
PM1
PM4a-s
PM4b-s
PM4b-c
PNIL (-40%)
fw 1.2
CM2-F2
CM2-F3
PM1
fw 1.a
PNIL (-25%)
fw 1.3
CMRI
PV
fw 2.4
PM1
PNIL (-25%)
fw 2.5
PF2
PNIL (-20%)
fw 2.1
CM2-F2
CMRI
PF2
PNIL (-25%)
fw 2.2
CM3
CM3-c
HR2
PM1
PM2
PM4a-s
PM4b-s
PM4b-c
PM5
PNIL (-37%)
fw 2.3
CM2-F3
CMRI
PNIL (-24%)
PV
(fw 3.a)
PV
fw 3.b
PNIL (-22%)
fw 3.1
CM2-F2
CMRI
CMRI
PM2
PF2
PNIL (-18%)
fw 3.2
CM2-F3
CM3-c
HR2
PM2
PM4a-s
PM4b-s
PM4b-c
PNIL (-36%)
fw 4.1
CM2-F3
CM3-s
HR2
fw 4.a
PM2
fw 4.2
CL1 (LOD = 5.0)
CMRI
PVE or R2 (in %)
35
30
25
20
15
10
5
0
-5
-10
fw 5.1
PM2
PV
fw 6.1
CMRI
PF2
fw 6.a
CM2-F2
CM2-F3
fw 6.2
CMRI
PNIL (-68%)
fw 6.3
CL1 (LOD = 5.0)
CM2-F2
CMRI
fw 7.a
CL1 (LOD = 5.0)
(fw 7.1)
CMRI
CMRI
PM2
PNIL (+12%)
fw 7.2
CM2-F2
CM2-F3
PF2
PV
fw 7.3
PM2
PNIL (-16%)
fw 7.b
PNIL (-32%)
fw 8.a
PM3
fw 8.1
PM1
PV
fw 8.b
PM1
fw 8.c
PNIL (-12%)
PVE or R2 (in %)
30
25
20
15
10
5
0
-5
-10
fw 9.1
PNIL (-14%)
PV
(fw 9.2)
CL1 (LOD = 3.0)
CM2-F3
CMRI
PM2
fw 9.a
PNIL (-12%)
fw 9.b
PNIL (-12%)
fw 10.1
PNIL (-24%)
PF2
PV
fw 10.a
PV
(fw 10.b)
PV
fw 11.1
PF1
PF2
PNIL (-16%)
fw 11.2
CL1 (LOD = 6.5)
PF1
PF2
PNIL (-16%)
fw 11.3
CM2-F2
CM2-F3
PM1
PM4b-c
PNIL (-16%)
PV
(fw 12.1)
CM2-F3
PM4b-c
PM4a-s
PM4b-s
PNIL (+24%)
(fw12.2)
CMRI
PV
PM4b-s
(fw 12.b)
PF2
Indicate either PVE values or R2 (in %)
CL1 : bar length is proportional to the LOD value
PNIL : bar length is proportional to the % of control (M82)
0
bars below 0 indicate transgressive QTL

**Fig. 3** Phenotypic effects of fruit-shape QTLs detected in interspecific crosses of tomato. Figure legend same as for Fig. 2



◀ **Fig. 2** Phenotypic effects of fruit-size QTLs detected in interspecific crosses of tomato. *White bars* indicate either PVE values or R$^2$ (in %). For the CL1 and PNIL studies different scales were employed. For CL1-data *striped bars* are used and the length is proportional to the LOD values. For the PNIL-study *gray bars* are used and the length is proportional to the control % (M82). *Bars below 0* indicate transgressive QTLs. Data from the PN1 and PM3 studies are not reported in this figure. For the PM4a, PM4b and CM3 studies, "*c*" and "*s*" indicate the results obtained under control and salinity conditions, respectively

QTL mapping studies (Table 1). Overall four wild species of tomato and four different population structures have been tested, with a marker genome coverage averaging from 18% (Weller et al. 1988) to almost 100% (Fulton et al 1997; Bernacchi et al. 1998). The estimated number of FS QTLs detected in each study ranged from 2 (Grandillo and Tanksley 1996) to 16 (Fulton et al., manuscript in preparation). A common set of 11 FS QTLs could be identified which was segregating in at least two independent studies (Fig. 1).

### *Magnitude of effects and gene action of fruit-shape QTLs*

Figure 3 shows the PVE or $R^2$ (in %) for the FS QTLs reported thus far for tomato. For the studies conducted on the three wild species *L. pimpinellifolium*, *Lycopersicon peruvianum*, und *Lycopersicon parviflorum*, *fs8.1* is the FS QTL explaining the largest portion of the total phenotypic variation. The $R^2$ values range from 27% for PM1 to 45% for PV. Other FS QTLs of large effect, detected in more than one independent study, are *fs2.1*, *fs2.2*, *fs7.1*, *fs10.1* and *fs10.2*.

The two major FS QTLs, *fs2.1* and *fs8.1*, have been further characterized, and for both round shape was partially dominant over the more elongate shape type (Grandillo et al. 1996; Ku et al., manuscript in preparation).

## Effects of qualitative genes on tomato fruit size and fruit shape

Several early studies of fruit in tomato have shown that qualitative genes, whether with or without shape effects, often also exert size effects or are linked to genes affecting size and shape (Lindstrom 1928; MacArthur 1934, 1935; Yeager 1937; MacArthur and Butler 1938). Some of these genes can affect fruit size by changing the shape of the fruit or the locule number, or both. For example, ovate (*o*), mapping on chromosome 2, elongates and narrows the fruit and reduces ist weight by half; peach (*p*), on chromosome 2, is associated with slightly increased fruit size and fasciated (*f*), mapping at the bottom of chromosome 11, increases locule and seed number and fruit width, and can therefore increase weight by up to 60% (MacArthur and Butler 1938). Other recessive genes, characterized by very distinguished qualitative phenotypes, can also influence fruit size. For example lutescent foliage (*l*), a chlorophyll deficiency, mapping at the top of chromosome 8, slows down both plant and fruit growth, retarding maturity by 2 weeks and reducing fruit weight by 30%. Tangerine (*t*), a mutant characterized by orange colored fruit flesh and stamens (mapping on chromosome 10), and possibly peach (*p*), characterized by fruits with a dull surface and increased hairiness (mapping on chromosome 2), appear to enlarge the fruit perhaps by acting on cell-expansion mechanism (MacArthur and Butler 1938).

Many other qualitative genes including dwarf (*d*), on chromosome 2, yellow flesh (*r*), on chromosome 3, unpigmented fruit epicarp (*y*), on chromosome 1, potato leaf (*c*), on chromosome 6, hairs absent (*h*) and uniform (*u*), on chromosome 10, have been shown to be nearly neutral in size effect but to exhibit linkage with ‘‘size genes’’ (MacArthur and Butler 1938).

The approximate map position of 10 of the 11 qualitative genes discussed above has been reported on the molecular tomato map (Tanksley et al. 1992) (Fig. 2). By means of transposon tagging, the tomato dwarf gene was more precisely placed at 8.4 cM from TG48 towards the distal end of the long arm of chromosome 2 (Thomas et al. 1994; Bishop et al. 1996). All these 11 qualitative genes, with the exception of *t*, fall in a region of the genome where significant QTLs for tomato fruit size have been reported (Fig. 1).

## Map-based cloning of fruit-size/shape QTLs: prospects and implications

From the aspect of developmental biology, the cloning of fruit-size/shape QTLs will provide a starting point for understanding a critical, but largely unexplored, aspect of plant development – how ovaries are transformed from small reproductive organs into the large and conspicuous fruit with an array of shapes and sizes that we all associate with edible fruit. Although considerable scientific investigation and resulting knowledge has been acquired in recent years regarding the genetic regulation of floral development (primarily via identification of *Arabidopsis* homeotic mutations and their counterparts in different species; Coen and Meyerowitz 1991; Pnueli et al. 1994) and the terminal ripening stage of fruit development (Gray et al 1994), very little is currently known regarding the genetic mechanisms operating during the intervening developmental stages which presumably influence fruit (ovary) architecture, size, and shape (Gillaspy et al. 1993). The cloning of fruit-size/shape QTLs will eventually allow us to understand the molecular basis of that process and may help to reconstruct the events involved in the domestication of tomato and other fruit-bearing crop species.

**Acknowledgements** In dedication and memory of Dr. Kevin Alpert, a great colleague and inspiration for this work. We thank Theresa Fulton and Dr. Anne Frary for their critical reading of and helpful comments on the manuscript.

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