African Journal of Biotechnology Vol. 10(63), pp. 13719-13723, 17 October, 2011
Available online at http://www.academicjournals.org/AJB
DOI: 10.5897/AJB11.1779
ISSN 1684–5315 © 2011 Academic Journals

Full Length Research Paper

Bayesian analysis of interacting quantitative trait loci (QTL) for yield traits in tomato

Yang Liu[1], Xiaojing Zhou[2], Jianhua Zhang[3], Huaizhi Li[1], Tianming Zhuang[1], Runqing Yang[1]* and Huoying Chen[1]*

[1]School of Agriculture and Biology, Shanghai Jiaotong University, Shanghai 201101, China.
[2]Department of Mathematics, Heilongjiang Bayi Agricultural University, Daqing 163319, China.
[3]Department of Horticulture and Art, Shanghai Commercial College, Shanghai 200235, China.

Accepted 7 September, 2011

An F_2 population derived from the hybrid of Lycopersicon esculentum Mill.XF98-7×Lycopersicon pimpinellifolium LA2184 was used for genome-wide linkage analysis for yield traits in tomato. The genetic map, spanning the tomato genome of 808.4 cM long was constructed with 112 SSR markers distributing on 16 linkage groups. Main and epistatic effect QTLs controlling first flower node, number of flowers per truss, fruit set percentage and fruit weight were located using Bayesian model selection method. A total of 20 significant main effect QTLs and 16 pairs of epistatic QTLs were identified on 16 linkage groups. The proportions of phenotypic variation explained by the detected QTLs ranged from 1.9 to 25.9% and from 0.00 to 17.4% for main-effect and epistatic QTLs, respectively. Most QTL effects were predictable from the parental phenotypes. Additionally, one QTL was found to be pleiotropic, governing simultaneously first flower node and number of flowers per truss.

Key words: Tomato, SSR marker, yield traits, QTL, Bayesian model selection.

INTRODUCTION

Construction of a high density genetic map is the foundation of gene localization, gene cloning and structural and functional genome research. The earliest high density genetic map covering the tomato genome of 1276 cM long has been established by Tanksley et al. (1992) with 1030 restriction fragment length poly-morphism (RFLP) markers in an F_2 population developed from a cross between an elite line of tomato (*L. esculentum*) and the closely related wild species (*Lycopersicon pennellii*). Later, some molecular linkage maps were presented for the purposes of detecting gene loci of interest. Foolad and Chen (1998) have published a linkage map of approximate 600 cM length, which has been constructed with 53 Random amplification of polymorphic deoxyribonucleic (RAPD) markers in F_2 population derived from UCT5 (L. esculentum*)* and LA716 *(L. pennellii*). Using a BC_1 population by hybridization of a *L. esculentum* line (NC84173) and an accession of *L. pimpinellifolium*, a molecular linkage map was drawn, which spanned 1192 cM with an average distance of 7.9 cM between markers (Foolad, 1999). Zhang et al. (2002) have constructed a genetic map by 142 RFLP markers based on a BC_1 population, and the length of the map is 1469 cM. In tomato, the first flower node, the number of flowers per truss, fruit set percentage and fruit weight are the important traits for the production of tomato. These traits are typically controlled by multiple genes, often collectively referred to as

*Corresponding author. E-mail: runqingyang@sjtu.edu.cn, chhy@sjtu.edu.cn Tel: 86213420-6146, 86213420-6934. Fax: 86213420-6146, 86213420-6934.

Abbreviations: SSR, Simple sequence repeat; **QTLs,** quantitative trait loci; **RFLP,** restriction fragment length polymorphism; **RAPD,** random amplification of polymorphic DNA; **FFN,** first flower node; **NFLT,** number of flowers per truss; **FSP,** fruit set percentage; FW, fruit weight.

quantitative trait loci (QTLs). The genetic architecture of quantitative trait includes the number and locations of QTL, their main effects, epistatic effects and interactions of those QTLs with environments as well. However, the unknown number of QTL and possible huge epistatic effects make the dissection for genetic architecture of quantitative trait extremely complex. Traditional QTL mapping methods based on interval mapping and composite interval mapping had been recommended for detecting epistasis (Carlborg et al., 2005; Fijneman et al,. 1996; Boer et al., 2002; Kao and Zeng, 2002), but the detection became increasingly difficult as the number of detected QTLs increased. In contrast, Bayesian mapping is able to simultaneously estimate all unknown parameters in the genetic architecture of quantitative traits by generating posterior samples from the joint posterior distribution for these unknowns. As a kind of Bayesian mapping methodology, Bayesian model selection approach has been developed (Yi et al., 2005, 2007), which provides an efficient and relatively simple way of identifying epistatic QTL for complex traits. The detection of QTL can not only explain genetic architecture for quantitative traits, but also facilitate marker-assisted selection. Till now, there are few studies about analyzing interactions between QTLs of yield traits in tomato. The objectives of this study were to construct a SSR genetic linkage map in tomato and then to identify main effect and epistatic QTLs for the four traits by Bayesian model selection method in an F_2 population derived from the hybrid of L. esculentum Mill XF98-7×*L.* pimpinellifolium LA2184.

MATERIALS AND METHODS

Plant materials

The hybrids were made between the two inbreeding lines: XF 98-7 of *L. esculentum* Mill developed by Tomato Breeding Laboratory, Shanghai Jiaotong University and *L.* pimpinellifolium accession LA2184 provided by Dr. Robert, Dowman, at department ofHorticulture, University of California. Subsequently, a single F_1 hybrid plant was self-pollinated to produce F_2 progeny. A total of 142 F_2 plants were sampled randomly for construction of the genetic map and location of QTL for yield traits.

Traits evaluation

The node of first flower truss on each plant after blossom was denoted as the trait of first flower node (*FFN*). An average of three trusses on each plant was taken as the trait of number of flowers per truss (*NFLT*). Mean fruit set percentage of three trusses on each plant was defined as the trait of fruit set percentage (*FSP*). At the end of the growing season, ten representative ripened fruit were harvested from each plant for measurement of the average fruit weight (*FW*).

SSR analysis

Following the procedure as described in Fang et al. (1999), total DNA was extracted from fresh leaf tissue of each sampled plant. Amplification reaction mixture contained DNA template of 20 to 40 ng, Mg^{2+} of 2.0 mmol·L^{-1}, dNTP of 0.2 mmol·L^{-1}, primer of 0.4 μmol·L^{-1} and *Taq* DNA polymerse of 0.65 U. The process of PCR reaction was conducted according to the modified procedure proposed by He et al. (2003). The PCR products were isolated by electrophoreses on 6% (wt/vol) denatured polyacrylamide gel. After electrophoresis, the gel was stained with silver nitrate.

QTL mapping

In F_2 population, assume that there are *q* quantitative trait loci responsible for a trait of interest, the genetic mapping model with interacting QTLs can be then constructed on the basis of the Cockerham's genetic model (Kao and Zeng, 2002), denoted by

$$y_i = \mu + \sum_{j=1}^{q} (z_{ij} a_j + w_{ij} d_j) + \sum_{j=1}^{q-1} \sum_{k=j+1}^{q} [x_{1i}(aa)_{jk} + x_{2i}(ad)_{jk} + x_{3i}(da)_{jk} + x_{4i}(dd)_{jk}] + e_i$$

Where, μ is the population mean; α_j and d_j for $j = 1, 2, \mathrm{L}\ , q$ are, respectively the additive and dominant effects of the *j*th QTL, for which variable *z* and *w* are the genotype indicators; *aa*, *ad*, *da* and *dd* are the epistatic effects corresponding to additive×additive, additive×dominant, dominant×additive, and dominant×dominant interactions. $x = zw$ and e_i is the residual error. The genome-wide interacting QTL for the four yeild traits has been analyzed by adopting Bayesian model selection (Yi et al., 2005, 2007) implemented in the freely available package R/qtlbim (www.qtlbim.org) released by Yandell et al. (2007). According to the results from the composite interval mapping, we set the expected number of main-effect QTL at 4, so that upper bound of the number of QTL is 10. The initial values of other parameters are defaulted. For each analysis, the MCMC algorithm ran for 1.2×10^5 iterations after discarding the first 1000 iterations as burn-in to ensure proper mixing of the Markov chain. To reduce serial correlation in the stored samples, the chain was trimmed by keeping one observation in every 40 iterations, yielding 3000 samples from the conditional posterior distribution for inferring the genetic architecture. In posterior analysis, Bayes factors of main effect at per locus and epistatic effect at a pair of loci are individually calculated and compared with a BF threshold of 3, or 2ln (BF) = 2.1, to claim the

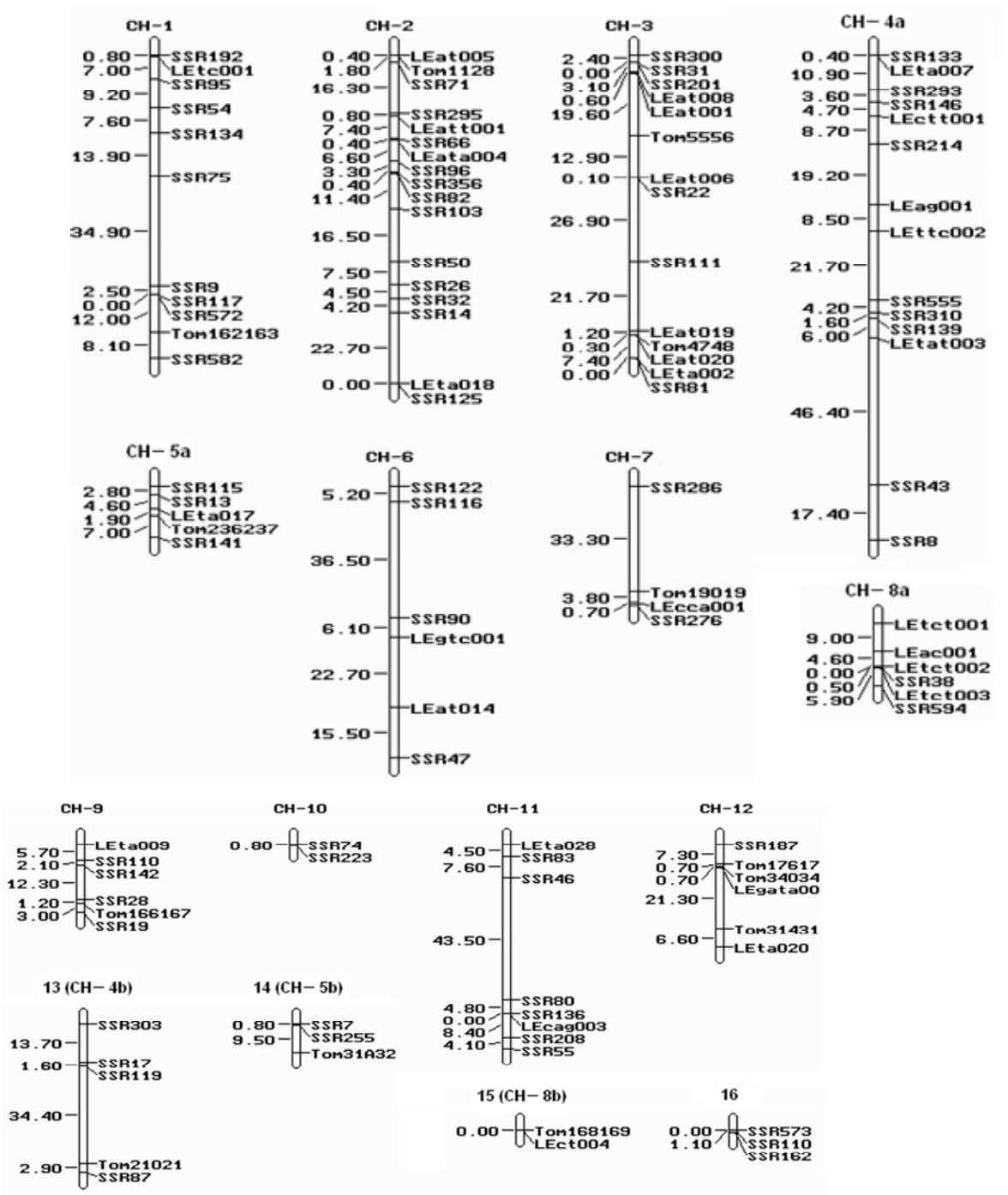

Figure 1. SSR linkage map based on an F_2 population in tomato.

presence of QTL (Kass and Raftery, 1995).

RESULTS

Construction of the genetic map

A total of 530 SSR primers were used to screen in parents and of which, 125 were found to be polymorphic. The 117 polymorphic primers were selected due to their stability and co-dominance to genotype markers on 142 F_2 plants. The results from the chi-square test showed that segregation ratio for each marker all meted the expected 1:2:1 at significant level of 0.05. The linkage map has been drawn using the software package Mapmaker 3.0, whose length is 808.4 cM and average

Table 1. Summary of statistics for main effect QTLs on the yield traits in tomato detected with Bayesion model selection.

Trait	Chr	HPD	Additive	Dominance	Hereditability (%)	2lnBF
FFN	2	[32.5, 37.8]	0.29	-0.02	7.2	2.31
FFN	3	[0.0, 24.4]	0.44*	0.01	10.5	3.53
FFN	11	[0.0,16.1]	0.38*	0.00	12.0	5.32
FFN	5b	[3.2,10.3]	-0.52*	-0.01	15.6	7.21
FFN	8b	[0.0, 0.0]	0.39*	0.02*	12.9	8.33
NFLT	1	[95.2, 96.0]	0.29*	0.01	7.6	3.14
NFLT	2	[5.2, 91.8]	0.38*	0.00	8.7	3.42
NFLT	4	[123.0,125.0]	0.27*	-0.03	6.8	2.49
NFLT	5a	[11.2,16.3]	0.31*	0.00	8.6	2.97
NFLT	9	[11.4, 22.0]	0.38*	0.00	11.7	4.82
NFLT	5b	[3.2,10.3]	0.54*	0.00	16.5	7.23
FSP	3	[46.1, 96.2]	-0.11*	0.00	1.9	5.46
FSP	7	[0.0, 8.5]	0.27*	0.00	5.4	2.49
FSP	12	[0.0, 30.3]	0.01	0.44*	6.2	4.75
FW	1	[70.3, 91.2]	-0.58*	0.00	25.9	5.04
FW	2	[0.0,12.4]	-0.44*	-0.01	15.0	4.75
FW	3	[63.0, 76.7]	-0.44*	0.00	16.9	4.83
FW	5a	[14.9, 16.3]	-0.30*	-0.06	10.9	3.15
FW	9	[8.3, 24.3]	-0.34*	-0.03	12.0	5.26
FW	12	[16.7, 25.9]	-0.35*	-0.01	10.6	4.04

* represents that marked effects are significant.

Table 2. Summary of statistics for epistatic QTLs on the yield traits in tomato detected with Bayesion model selection.

Trait	Position	Hereditability (%)	*aa*	*dd*	*ad*	*da*	2lnBF
FFN	chr2[32.5, 37.8] ×chr5b[3.2, 10.3]	5.2	0.107	0.933	0.458	0.081	5.78
FFN	chr3[0.0, 24.4] ×chr11[0.0, 16.1]	17.1	0.931	0.527	0.935	0.712	6.13
FFN	chr3[0.0, 24.4] ×chr5b[3.2, 10.3]	4.1	0.019	0.390	0.224	0.121	6.69
FFN	chr3[0.0, 24.4] ×chr8b[0.0, 0.0]	2.3	0.003	0.056	0.024	0.040	6.8
FFN	Chr5b[3.2, 10.3]×chr8b[0.0, 0.0]	0.0	0.000	0.003	0.000	0.002	7.48
NFLT	chr1[95.2, 96.0]×chr2[5.2, 91.8]	12.4	0.652	0.773	0.529	0.972	5.66
NFLT	chr1[95.2, 96.0]×chr5b[3.2, 10.3]	10.4	0.115	0.014	0.202	0.179	5.6
NFLT	chr2[5.2, 91.8]×chr9[11.4, 22.0]	6.8	0.353	0.760	0.080	0.720	4.97
NFLT	chr2[5.2, 91.8]×chr5b[3.2,10.3]	9.6	0.225	0.595	0.192	0.904	5.96
NFLT	chr5a[11.2,16.3]×chr5b[3.2,10.3]	11.4	0.001	1.159	0.010	0.002	6.23
FSP	chr3[46.1, 96.2]×chr12[0.0, 30.3]	17.4	0.508	0.639	1.039	1.716	11.35
FSP	chr7[0.0, 8.5]×chr12[0.0, 30.3]	5.3	0.207	0.373	0.717	0.672	4.97
FW	chr1[70.3, 91.2]×chr5a[14.9,16.3]	3.4	0.002	0.203	0.326	0.001	5.78
FW	chr1[70.3, 91.2]×chr9[8.3, 24.3]	11.2	0.413	0.097	0.390	0.728	6.62
FW	chr2[0.0,12.4]×chr12[16.7, 25.9]	10.7	0.190	0.255	0.720	0.423	6.43
FW	chr3[63.0, 76.7]×chr9[8.3, 24.3]	5.3	0.162	0.618	0.095	0.148	5.78

distance between markers is 7.22 cM (Figure 1).

Identification of QTL

The genome-wide Bayes factors comparing the model with and without the locus for the analysis showed evidence of QTL activity on whole chromosomes. Outputs of the analyzed results included genetic effects (additive, dominance and epistatic), 2lnBF, HPD (the region of highest posterior density) and hereditability (that is, proportion of phenotypic variation explained by QTL) for detected QTL, which were summarized in Tables 1 and Table 2.

First flower node (FFN)

Five main effect QTLs for FFN were detected on chromosomes 2, 3, 5b, 8b and 11, and especially, the QTLs on the 8b*th* chromosome was detected on the two markers, that is, Tom168169 and LEct004 (Table 1). All but one QTL (on chromosome 5b) LA2184 alleles contributed to the increased FFN, consistent with the parental phenotypes. Each main effect explained from 7.2 to 15.6% of phenotypic variation. Among five detected QTLs, 5 pairs performed significant epistatic interactions, that is, chromosomes 2 and 5b, 3 and 11, 3 and 5b, 3 and 8b, 5b and 8b, with strong positive effects of *dd*, *ad*, *dd*, *dd* and *dd* on FFN, respectively. Only one interaction between chromosomes 3 and 11 contributed to high proportion of phenotypic variation, whereas the other interactions were at low proportions (0.0 to 5.2%).

Number of flowers per truss (NFLT)

Six main effect QTLs were identified on chromosomes 1, 2, 4, 5 (a, b) and 9, each accounting for between 6.8 and 16.5% of the phenotypic variation for this trait. It was found that the QTL within the HPD [3.2, 10.3] governed simultaneously FFN and NFLT with higher proportion of the phenotypic variation, showing the pleiotropy. For all QTLs, the alleles derived from LA2184 contributed to the increased NFLT. As seen from Table 2, 5 pairs of QTLs, which distributed on chromosome 1 and 2, 1 and 5b, 2 and 9, 2 and 5b, 5a and 5b, showed the strong evidence of epistasis on NFLT. These interactions explained about 10% the phenotypic variation, except for the QTL on chromosome 2 and 9.

Fruit set percentage (FSP)

In four analyzed traits, the number of QTL detected for FSP was the fewest. Only three QTLs for FSP performed significant main effects and two pairs of them showed the strong evidence of epistasis on NFLT. Three QTLs located on chromosome 3, 7 and 12, which explained the phenotypic variance at a low level. The LA2184 alleles contributed to the increased *FSP* at two major QTLs whereas the XF98-7 alleles contributed to the increased FSP only at one major QTL. The interaction between chromosome 3 and 12 strongly impacted FSP with the greatest epistasis of *da* and high heritability.

Fruit weight (FW)

On chromosome 1, 2, 4, 5a, 9 and 12, the main effects of six QTLs were identified to be significant and among these chromosomes, the interactions of four pairs were significant (Table 2). It could be found that a large QTL on chromosome 1 was responsible for *FW*, accounting for 25.9% of the phenotypic variance. For all QTLs, the alleles derived from the small-fruited parent (LA2184) contributed to the reduced *FW* in the F_2 progeny, as expected. Of four interactions, two pairs on chromosome 1 and 9, chromosome 2 and 12 explained above 10% phenotype variation.

DISCUSSION

Many studies about detection of quantitative trait loci for tomato have been reported. For example, Paterson et al. (1998) and Eshed and Zamir (1995) have individually found 6 and 18 QTLs responsible for fruit weight in tomato. Fulton et al. (1997) have detected 8 QTLs for fruit weight, whose reliability has been verified in the BC_2, BC_3 and BC_4 progenies in tomato. Alpert and Tanksley (1996) and Grandillo and Tanksley (1999) have detected 28 QTLs, of which the largest QTL was able to account for 30% of the phenotypic variance for fruit weight in tomato. Currently, Frary et al. (2003) have finely mapped quantitative trait loci for improved fruit characteristics from Lycopersicon chmielewskii chromosome 1. Rousseaux et al. (2005) have used *L. pennellii* introgression lines to analyze QTLs for fruit antioxidants in tomato. However, these QTLs mentioned above have been identified by adopting either least square or maximum likelihood method. It had been demonstrated on both theory and practice that the Beyesian model selection is superior to least square or maximum likelihood method, in terms of detecting power of QTL (Yi et al., 2005, 2007; Yandell et al., 2007, Yang et al., 2010, 2011). We had employed Bayesian model selection to dissect the genetic architecture of four yield traits on the genetic map for tomato constructed *by* self, and identified a total of 18 significant additive and 2 dominant QTLs (shown in Table 1), uncovered all the QTLs detected with maximum likelihood method (Liu et al., 2005). Some QTLs were located finely, such as the ones on chromosome 2 for FFN, chromosome 5a for NFLP and FW, and chromosome 8b for FFN. The main effects of detected QTLs on analyzed traits explained from 1.9 to 25.9% of phenotypic variation. Many QTLs with hereditability of above 10% effect could be considerably interesting for breeding purposes and scientific reasons. When main effects of two QTLs were individually or both non-significant, no interactions between them were found. Although, some QTLs had lower main effects, such as QTLs for NFLT on chromo-some 1, 2 and QTL for FSP on chromosome 3, they generated higher interactions with other QTLs (Table 2). The presence of epistasis allows us to consider simultaneously markers associated with QTLs in marker-assisted selection. One pleiotropic QTL was found in this study, but Bayesian model selection for multiple traits was required to develop for precisely mapping the kind of QTLs. Mapping interacting QTLs will increase the genetic markers' information associated with yield traits and promote the process of identifying

causative genes. Beneficial genetic variation knowledge can be incorporated in breeding programs to enhance genetic improvement through marker-assisted selection for tomato.

ACKNOWLEDGEMENTS

The research was supported by Shanghai Municipal Agricultural Commission (2007#5-1) and the Shanghai Leading Academic Discipline Project (B209) to HY, and the Chinese National Natural Science Foundation Grant 30972077 to RY.

REFERENCES

Alpert KB, Tanksley SD (1996). High-resolution mapping and isolation of yeast artificial chromosome contig containing FW2.2: a major fruit weight quantitative trait locus in tomato. Proc. Natl. Acad. Sci. USA, 93(26): 1503-1550.

Boer MP, Ter Braak CJF, Jansen RC (2002). A penalized likelihood method for mapping epistatic quantitative trait loci with one dimensional genome searches. Genetics, 162: 951-960.

Carlborg O, Brockmann GA, Haley CS (2005). Simultaneous mapping of epistatic QTL in DU6i x DBA/2 mice. Mamm. Genome, 16: 481-494.

Fang XJ, Huang YM, Chen QF, Sun M (1999). Genetic analysis of some elite rice cultivars and hybrid rice combinations by using allozyme and RAPD markers. Sci. Agric. Sinica. 32(2): 1-8.

Fijneman RJ, De Vries SS, Jansen RC, Dermant P (1996). Complex interactions of new quantitative trait loci, Sluc1, luc2, Sluc3 and Sluc4, that influence the susceptibility to lung cancer in the mouse. Nat. Genet. 14: 465-467.

Foolad MR (1999). Comparison of salt tolerance during seed germination and vegetative growth in tomato by QTL mapping. Genome, 42: 727–734.

Foolad MR, Chen FQ (1998). RAPD markers associated with salt tolerance in an interspecific cross of tomato (*Lycopersicon esculentum*×*L. pennellii*), Plant Cell Rep. 17(4): 306-312.

Frary A, Doganlar S, Frampton A, Fulton TM, Uhlig J, Yates H, Tanksley SD (2003). Fine mapping of quantitative trait loci for improved fruit characteristics from Lycopersicon chmielewskii chromosome 1. Genome, 46: 235-243.

Fulton TM, Beck-Bunn T, Emmatty D, Eshed Y, Lopez J, Petiard V, Uhlig J, Zamir D, Tanksley SD (1997). QTL analysis of an advanced backcross of *Lycopersicon peruvianm* to the cultivated tomato and comparisons with QTLs found in other wild species. Theor. Appl. Genet. 95: 881-894.

Grandillo S, Ku HM, Tanksley SD (1999). Identifying the loci responsible for natural variation in fruit size and shape in tomato. Theor. Appl. Genet. 99: 978-987.

He C, Poysa V, Yu K (2003). Development and characterization of simple sequence repeat (SSR) markers and their use in determining relationships among *Lycopersicon esculentum* cultivars. Theor. Appl. Genet. 106: 363-373.

Kao CH, Zeng ZB (2002). Modeling Epistasis of Quantitative Trait Loci Using Cockerham's Model. Genetics, 160: 1243-1261.

Kass RE, Raftery AE (1995). Bayes factors. J. Am. Stat. Assoc. 90: 773-795.

Liu Y, Chen HY, Wei YT, Zhuang TM (2005). Construction of a genetic map and localization of QTLs for yield component traits in tomato by SSR markers. Prog. Nat. Sci. 15(9): 793-797.

Paterson AH, Lander ES, Hewitt JD, Peterson S, Lincoln SE, Tanksley SD (1988). Resolution of quantitative traits into Mendelian factors by using a complete linkage map of restriction fragment length polymorphisms. Nature, 335: 721-726.

Rousseaux MC, Jones CM, Adams D, Chetelat R, Benett A, Powell A (2005). QTL analysis of fruit antioxidants in tomato using Lycopersicon pennellii introgression lines, Theor. Appl. Genet. 111: 1396-1408.

Tanksley SD, Ganal MW, Prince JP, De Vicente MC, Bonierbale MW, Broun P, Fulton TM, Giovannoni JJ, Grandillo S, Martin GB, Messeguer R, Miller JC, Miller L, Paterson AH, Pineda O, Roder MS, Wing RA, Wu W, Young ND (1992). High density molecular linkage maps of the tomato and potato genomes. Genetics, 132: 1141-1160.

Yandell BS, Mehta T, Banerjee S, Shriner D, Venkataraman R, Moon JY, Neely WW, Wu H, von Smith R, Yi N (2007). R/qtlbim: QTL with Bayesian interval mapping in experimental crosses. Bioinformatics, 23: 641-634.

Yang R, Wang X, Wu Z, Prows DR, Lin M (2010). Bayesian model selection for characterizing genomic imprinting effects and patterns. Bioinformatics, 26: 235-241.

Yi N, Banerjee S, Pomp D, Yandell BS (2007). Bayesian Mapping genome-wide interacting quantitative trait loci for ordinal trait. Genetics, 176: 1855–1864.

Yi N, Yandell BS, Churchill GA, Allison DB, Eisen EJ, Pomp D (2005). Bayesian model selection for genome-wide epistatic quantitative trait loci analysis. Genetics, 170: 1333–1344.

Zhang LP, Khan A, Nino-Liu D, Foolad MR (2002). A molecular linkage map of tomato displaying chromosomal locations resistance gene ananlogs based on a *Lycopersicon esculentum* × *Lycopersicon hirsutum* cross. Genome, 45(1): 133-146.