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Gene actions of QTLs affecting several agronomic traits resolved in a recombinant inbred rice population and two backcross populations

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Abstract To understand the types of gene action controlling seven quantitative traits in rice, we carried out quantitative trait locus (QTL) mapping in order to distinguish between the main-effect QTLs (M-QTLs) and digenic epistatic QTLs (E-QTLs) responsible for the trait performance of 254 recombinant inbred lines (RILs) from rice varieties *Lemont*/*Teqing* and two backcross hybrid (BCF_1) populations derived from these RILs. We identified 44 M-QTL and 95 E-QTL pairs in the RI and BCF_1 populations as having significant effects on the mean values and mid-parental heterosis of heading date, plant height, flag leaf length, flag leaf width, panicle length, spikelet number and spikelet fertility. The E-QTLs detected collectively explained a larger portion of the total phenotypic variation than the M-QTLs in both the RI and BCF_1 populations. In both BCF_1 populations, over-dominant (or under-dominant) loci were more important than additive and complete or partially dominant loci for M-QTLs and E-QTL pairs, thereby supporting prior findings that overdominance resulting from epistatic loci are the primary genetic basis of inbreeding depression and heterosis in rice.

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Introduction

Permanent segregating populations, such as recombinant inbred lines (RILs) and doubled haploids (DH), are widely used in quantitative trait loci (QTLs) analyses of self-pollinated plants. However, as the individuals have a homogenous genotype at most of the loci, these kinds of populations are not suitable for genetic analysis involving non-additive genetic effects because of the shortage of heterozygotes. For non-additive gene/QTLs, most genetic analyses and linkage mapping projects have been conducted in early segregating generations, such as the F_2, $F_{2\text{-}3}$ and the BC_1F_2 generations. However, when these populations are used, it is not possible to make repeated observations at the level of the individual or block or to carry out multiple trials. There are two alternative strategies which allow the repeated detection of non-additive effects through the creation of heterozygotes from permanent populations. The first is to create heterozygotes by testcrosses (TC) or backcrosses (BC) from a RI population (Li et al. 2001; Luo et al. 2001; Mei et al. 2003). The second is to develop what is called an "immortalized F_2" population which is generated from intermating between the RILs (Hua et al. 2002, 2003). In the first strategy, the genotype of a hybrid is de-

duced from the genotypes of the parental lines. By comparing QTLs mapped in RILs and their TC hybrid populations, gene actions can be inferred to be mainly additive or non-additive (Mei et al. 2003). In contrast to TC hybrids, BC hybrids have the advantage that the genotype of each hybrid is known at each locus. The homology between alleles from parental RILs and the tester is uncertain in TC populations. The BCF_1 hybrid is homozygous for loci for which the parental RIL carries the same allele as the recurrent parent but is heterozygous for other loci.

The development of hybrid rice plants has greatly increased the yield potential since the 1970s (Yuan 1992; Khush 2001). In recent years many researchers have tried to dissect rice heterosis via QTL approaches, and several publications have emphasized that epistasis plays an important role in heterosis in rice (Yu et al. 1997; Li et al. 2001; Luo et al. 2001). In the study reported here, a RI population derived from the cross between rice varieties *Lemont* and *Teqing* was used as a basic population. BCF_1 populations were developed by backcrossing RILs to both parents. QTL analysis was conducted for both trait performance and heterosis to detect main-effect loci and digenic epistasis. Gene actions were explored again for such aspects as relative importance between additive and non-additive effects and between main-effect loci and epistasis.

Materials and methods

Plant materials and the phenotyping experiment

The rice mapping populations used in this study included a set of 254 RILs (F_{10}) derived by single-seed descent from a cross between vars. *Lemont* (*japonica*) and *Teqing* (*indica*). In addition, two BC populations were developed, the first consisting of 172 BCF_1 ($LTBCF_1$s) hybrids from crosses between the RILs (used as female) and one parent, *Lemont* (LT) and the second one consisting of 177 BCF_1 ($TQBCF_1$s) hybrids from crosses between the RILs and the other parent, *Teqing* (TQ). The parents of the RILs (LT and TQ), the F_1 hybrid (LT × TQ) and a commercial hybrid (Shanyou 63) were used as checks.

The phenotyping experiment was conducted at the experimental farm of the College of Agriculture and Biotechnology, Zhejiang University, Hangzhou, China. All materials were sown in the seedling nursery on May 25, 1996, and 25-day-old seedlings were transplanted into three-row plots with each plot consisting of a single row of a female RIL and two rows of BC hybrids (one with *Lemont* and one with *Teqing*, if available). There were 15 plants in each row, with 20 cm between plants within each row and 35 cm between rows. The plots were arranged in a completely randomized block design with three replications. All of the test plants were measured for the following traits: heading date (HD), which was recorded as days from sowing to the time when panicles emerged from the leaf sheath on 50% of the plants in a row plot; plant height (PH, in centimeters), which was measured as the height from the soil surface to the tips of the tallest panicles of five plants in a row plot; flag-leaf length (FLL, in centimeters) and flag-leaf width (FLW, in millimeters), which were measured as the length and width of the flag leaves of three main tillers of five plants in a row plot before maturity; panicle length (PL, in centimeters), spikelet number per panicle (SN) and filled grain number per panicle (GN), which were measured on five randomly chosen plants in the middle of each plot after maturity. One derived trait, percentage of spikelet fertility (SF = 100×GN/SN), was calculated.

Genotyping and data analysis

The genotyping of the RILs for 179 restriction fragment length polymorphism markers and three morphological markers, including *C* (apiculus color), *gl-1* (glabrous leaves) and *Ph* (grain reaction to phenol), were conducted at Texas A&M University, and the completed linkage map with 182 markers spanned 1,918.7 cM and covered 12 rice chromosomes with an average distance of 11.3 cM between adjacent markers, as described previously by Li et al. (2001).

Square-root transformation was performed for SN to make the trait mean independent from trait variance. SAS procedures PROC GML and CORR (SAS Institute 1996) were used to test the differences among the RILs and BC hybrids and to obtain the basic statistics of the traits. The hybrid breakdown value (HB) is a component of inbreeding depression (Li et al. 1997, 2001) and was calculated as follows: HB = RIL − MP, where MP = (TQ + LT)/2 was the mid-parental value of two parents. Mid-parental heterosis (H_{MP}) of each BC hybrid was calculated as follows: $H_{MP} = F_1 - MP$, where F_1 was the mean trait values of the BC hybrid and MP = (RIL + recurrent parent)/2 was the mid-parental trait values of the corresponding female RIL and the recurrent parent.

QTLs analysis was performed separately for the RI and BC populations. For the RI population, the mean trait values from three replications were used as input data. For each of the BC populations, the mean trait values and mid-parental heterosis (H_{MP}) of the BC hybrids were used as input data. Analysis of main-effect QTLs (M-QTLs) and digenic epistatic QTLs (E-QTLs) were conducted in each mapping population by interval mapping using the mixed linear approach and the computer software QTLMAPPER ver. 1.0 (Wang et al. 1999). A threshold of LOD≥2.0 were used to declare M-QTLs and E-QTLs in the RILs together with LOD≥1.8 in BCF_1 performance and H_{MP}.

Results

Performance of the RILs and mid-parental heterosis of their BC hybrids

The performance of the two parents was significantly different for all of the traits measured (Table 1). *Lemont* had higher values for HD and FLW, while *Teqing* had greater values for the remaining five traits. The F_1 (LT × TQ) plants had significantly earlier HD and higher PH, PL, SN, and SF values than both parents, while their FLL and FLW values were similar to those of the high-value parent. Significant H_{MP} was observed in five traits except FLW (Table 1). The mean values of the RILs were significantly higher than the mid-parental values for PH, FLL and SF, significantly lower for PL, but not different for HD, FLW and SN.

The average levels of heterosis of the BCF_1s showed similar trends along different traits in the $LTBCF_1$s (RIL × LT), $TQBCF_1$s (RIL × TQ) and the hybrid (LT × TQ F_1) (Table 1), with significant negative H_{MP} in HD, significant positive H_{MP} in PH, FLL, PL, SN and SF and little H_{MP} in FLW observed. The average levels of heterosis of the BCF_1s were remarkably lower than the H_{MP} of the LT × TQ F_1s. For example, H_{MP} was 35.7 cm for LT × TQ F_1 compared to a mean H_{MP} of 26.7 cm and 16.4 cm for the $LTBCF_1$s and $TQBCF_1$s respectively.

Wide ranges of variations were observed for every trait in the RILs, BCF_1s and H_{MP}. The RIL population showed transgressive distribution on both sides. Extreme individuals in the RIL and both BCF_1 populations showed trait values exceeding those of the hybrid between two parents. Consequently, many lines showed higher H_{MP} in the BCF_1s than the LT × TQ F_1 (Table 1).

The performance of the hybrid plants was determined by their mid-parental value and the heterosis level. As the recurrent parent was the same in each BCF_1 population, hybrid performance is the cumulative effect of the trait value of the RIL and the heterosis. In the $LTBCF_1$ and $TQBCF_1$ populations, high positive correlations between H_{MP} and hybrid performance (F_1) were found (Table 2). The average R^2 (determination coefficient) was 0.652 (ranging from 0.447 for HD to 0.888 for SN) in the $LTBCF_1$ population and 0.663 (ranging from 0.529 for PL to 0.788 for SN) in the $TQBCF_1$ population. There were positive but low correlations between the trait value of the RILs and that of their BCF_1s. The average R^2 between RILs and F_1s was 0.104 (ranging from zero for SN to 0.166 for FLW) in the $LTBCF_1$ population and 0.123 (ranging from zero for SN to 0.317 for HD) in the $TQBCF_1$ population. There were negative correlations between trait value of RILs and H_{MP} except HD in $TQBCF_1$ population. The average R^2 value was 0.106 (ranging from 0.040 for FLL to 0.193 for HD) in the $LTBCF_1$ population and 0.101 (ranging from zero for HD to 0.237 for PL) in the $TQBCF_1$ population.

M-QTLs detected in the RILs, BCF_1s and mid-parent heterosis

A total of 44 M-QTLs were identified for trait performance of the RILs or BCF_1 hybrids and mid-parental heterosis (Table 3). These M-QTLs were mapped to all of the rice chromosomes except chromosome 10 (Fig. 1).

Seven M-QTLs were identified for HD, explaining 48.0%, 17.0% (7.1% for H_{MP}) and 31.6% (24.2% for H_{MP}) of the total variances in the RILs, $LTBCF_1$s and $TQBCF_1$s, respectively. Two dominant M-QTLs (*QHd3a* and *QHd8*) were detected in RILs and the $TQBCF_1$ population. *QHd3a* had a dominant effect of 2.0 days for early heading, while Q *Hd8* had a dominant effect of 1.5 days for late heading. This result is quite similar to that (*Hd3 and QHd8*) detected in the Zhong 413 testcross F_1 population (Mei et al. 2003). An additional dominant QTL was located on chromosome 3 (*QHd3b*) that had an effect causing delayed heading for 1.2 days in the $LTBCF_1$ population. *QHd1a*, *QHd1b*, *QHd11* appeared to be additive M-QTLs as they were detectable only in RILs or F_1 performance, while *QHd12* seemed to be under-dominant for it was only detectable in H_{MP} ($d = -1.7$) but not in F_1 performance in the $TQBCF_1$ population.

A total of nine M-QTLs were detected for PH, explaining 50.3%, 13.7% (7.9% for H_{MP}) and 31.0% (20.0% for H_{MP}) of the total variance in the RILs, $LTBCF_1$s and $TQBCF_1$s, respectively. *QPh3a* and *QPh8* were located to the same or adjacent intervals as *QHd3a* and *QHd8*. The former had negative effects in the RILs and F_1 performance but a positive effect for H_{MP} in the $LTBCF_1$s. The latter behaved like a dominant M-QTL by increasing PH by 2.8 cm in the $TQBCF_1$s. Five loci (*QPh1a*, *QPh1b*, *QPh3b*, *QPh4* and *QPh6*) appeared to be additive loci, with a significant effect detected in RILs or F_1 performance only. *QPh2* appeared to be an over-dominant QTL, which increased H_{MP} by 2.7 cm in the $TQBCF_1$s, while *QPh12* acted as an under-dominant locus in reducing PH by 1.3 cm in the $TQBCF_1$s.

Six M-QTLs were detected for FLL, explaining 12.0%, 27.3% (8.5% for H_{MP}) and 36.8% (34.6% for H_{MP}) of the total variance in the RILs, $LTBCF_1$s and $TQBCF_1$s, respectively. *QFll1a* had an over-dominant effect of 1.3 cm for H_{MP} detected in the $TQBCF_1$s. *QFll1b* appeared to be additive in the $LTBCF_1$s but partially dominant in the TQBCF1s, reducing FLL by 1.0–2.2 cm. *QFll2* was detectable only in the performance of the $LTBCF_1$s and appeared to be an additive locus. *QFll4* was dominant in the $TQBCF_1$s, increasing FLL by nearly 2.0 cm. *QFll3* and *QFll5* acted as over-dominant QTLs but in opposite directions.

Table 1 Summary statistics of heading date (HD) plant height (PH), flag-leaf length (FLL) and width (FLW), panicle length (PL), spikelet number per panicle (SN, square root transformed) and spikelet fertility (SF) of the 254 *Lemont*/*Teqing* RILs and their two backcross F_1 populations (RILs × two parents, *Lemont* and *Teqing*)

	Trait values[a]						
	HD	PH	FLL	FLW	PL	SN	SF
Lemont (LT)	102.0 ± 2.1	74.2 ± 1.8	20.0 ± 0.7	14.6 ± 0.4	19.0 ± 1.2	8.8 ± 0.9	57.9 ± 1.2
Teqing (TQ)	95.3 ± 2.1	98.2 ± 2.1	27.3 ± 0.6	11.8 ± 0.3	20.8 ± 1.1	12.0 ± 1.2	70.7 ± 0.8
(LT × TQ) F_1	92.0 ± 1.1	121.9 ± 3.0	27.2 ± 0.9	14.3 ± 0.3	23.8 ± 1.2	14.0 ± 1.1	84.2 ± 0.5
(LT × TQ) H_{MP}^{b}	−6.7***	35.7***	3.5***	1.1	3.9**	3.6***	19.9***
Shanyou 63	92.0 ± 1.2	106.1 ± 4.0	28.4 ± 0.6	23.2 ± 0.4	26.0 ± 1.5	9.7 ± 0.9	76.4 ± 1.1
RILs	98.5 ± 8.3	91.4 ± 12.7	25.6 ± 5.7	13.3 ± 2.3	18.4 ± 2.6	9.9 ± 2.0	69.0 ± 11.5
	76.0–113.0	60.2–127.5	10.3–42.8	7.8–19.7	12.3–27.2	5.0 – 15.2	37.1–92.3
RIL-MP	−0.2	5.2***	2.0**	0.1	−1.5*	−0.5	4.7**
$LTBCF_1$	95.7 ± 5.0	109.1 ± 10.2	28.2 ± 4.7	13.8 ± 2.0	22.3 ± 1.8	12.1 ± 2.6	69.6 ± 10.4
	78.0–109.0	87.0–128.8	18.3–39.2	9.7–21.8	18.5–27.4	6.6 – 18.8	35.6–92.0
(LT) H_{MP}	−4.7 ± 5.2	26.7 ± 10.4	6.0 ± 4.4	−0.1 ± 1.8	3.7 ± 1.7	2.2 ± 2.8	3.2 ± 11.0
	−23.0–10.0	−1.1–57.2	−9.9–25.6	−4.2–7.4	−3.3–7.6	−8.7 – 15.8	−30.3–32.7
$TQBCF_1$	92.9 ± 7.3	111.1 ± 10.3	30.4 ± 4.2	13.5 ± 1.9	22.2 ± 1.6	12.9 ± 1.7	76.3 ± 10.1
	75.0–105.0	81.2–131.5	19.5–44.4	9.0–19.3	16.2–26.2	7.5 – 17.5	45.4–94.9
(TQ) H_{MP}	−4.1 ± 6.1	16.4 ± 10.3	4.0 ± 4.4	1.0 ± 1.7	2.7 ± 1.8	1.7 ± 1.9	6.5 ± 9.6
	−19.7–11.9	−7.5–44.0	−9.7–19.7	−3.3–5.4	−1.4–8.8	−7.5 – 13.9	−24.8–28.3

*, **, ***$P \leq 0.05, 0.01, 0.001$, respectively, based on *t* -tests

[a]Trait values are given either as the mean value ± the standard deviation or as a range

[b] The mid-parental heterosis, $H_{MP} = F_1 - MP$, where MP were the mid-parental trait values (LT + TQ)/2 for the LT/TQ F_1, (RIL + LT)/2 for $LTBCF_1$s, (RIL + TQ)/2 for $TQBCF_1$s, respectively

Table 2 Phenotypic correlation (*R*) and determination coefficients (R^2) for seven traits between the *Lemont*/*Teqing* RILs and mid-parent heterosis (H_{MP}) in the two BCF_1 populations (with the parents *Lemont* and *Teqing*)

Trait[a]	Between RILs and F_1				Between H_{MP} and F_1				Between RILs and H_{MP}			
	$LTBCF_1$s		$TQBCF_1$s		$LTBCF_1$s		$TQBCF_1$s		$LTBCF_1$s		$TQBCF_1$s	
	r	R^2	*r*	R^2	*r*	R^2	*r*	R^2	*r*	R^2	*r*	R^2
HD	0.374	0.140	0.563	0.317	0.669	0.447	0.828	0.686	−0.440	0.193	0.002	–
PH	0.269	0.072	0.316	0.100	0.818	0.669	0.823	0.678	−0.334	0.111	−0.278	0.077
FLL	0.393	0.154	0.427	0.182	0.822	0.676	0.802	0.643	−0.200	0.040	−0.197	0.039
FLW	0.407	0.166	0.269	0.072	0.801	0.642	0.775	0.601	−0.221	0.049	−0.400	0.160
PL	0.406	0.165	0.246	0.060	0.733	0.537	0.727	0.529	−0.324	0.105	−0.487	0.237
SN	0.024	–	0.068	–	0.942	0.888	0.888	0.788	−0.312	0.097	−0.399	0.159
SF	0.176	0.031	0.361	0.130	0.842	0.708	0.845	0.714	−0.383	0.147	−0.193	0.037
Mean	0.293	0.104	0.321	0.123	0.804	0.652	0.813	0.663	−0.316	0.106	−0.279	0.101

[a]See Table 1 for abbreviations

Six M-QTLs were detected for FLW, explaining 18.2%, 3.8% (16.2% for H_{MP}) and 38.1% (8.4% for H_{MP}) of the total variance in RILs, $LTBCF_1$s and $TQBCF_1$s, respectively. *QFlw2* had a negative effect for the performance of the $LTBCF_1$s but a positive H_{MP} effect, increasing FLW by 0.55 mm. *QFlw3* and *QFlw4* appeared to be additive M-QTLs for they were detectable only in the RILs or F_1s, while *QFlw6* and *QFlw8b* appeared to be over-dominant as they were only detectable in the H_{MP} of the $LTBCF_1$s. *QFlw8a* acted as a dominant locus; it was detectable for both H_{MP} and $TQBCF_1$ performance.

Seven M-QTLs were detected for PL, explaining 10.3%, 21.5% (9.4% for H_{MP}) and 26.8% (38.3% for H_{MP}) of the total variance in the RILs, $LTBCF_1$s and $TQBCF_1$s, respectively. *QPl1b*, *QPl7* and *QPl8* appeared to be additive loci and decreased PL by 0.5–0.8 cm. *QPl1a* and *QPl6* appeared to have dominant effects of 0.5–0.7 cm. *QPl2* and *QPl5* were detectable only for H_{MP}, implying over-dominant or under-dominant gene action.

Five M-QTLs were detected for SN, explaining 15.9%, 21.6% and 11.9% (25.3% for H_{MP}) of the total variance in the RILs, $LTBCF_1$s and $TQBCF_1$s, respectively. Three M-QTLs (*QSn1*, *QSn4* and *QSn9a*) appeared to be additive loci, while two others (*QSn6* and *QSn9b*) were detected as over-dominant loci.

Table 3 Main-effect QTLs[a] (M-QTLs) affecting seven traits (see Table 1) of *Lemont*/*Teqing* RILs and their two backcross F_1 populations (RILs × *Lemont* and *Teqing*)

Locus	Chromosome	Marker interval	RILs			BCF_1(*Lemont*)			H_{MP} (*Lemont*)			BCF_1 (*Teqing*)			H_{MP}(*Teqing*)		
			LOD	a[a]	R^2	LOD	$a + d$[a]	R^2	LOD	d[a]	R^2	LOD	$a + d$	R^2	LOD	d	R^2
Hd1a	1	C112–RG236	5.17	2.5	9.2												
QHd1b	1	RG532–RG140				2.35	1.3	8.0									
QHd3a	3	C515–RG348a	9.26	−3.5	14.3							8.08	−2.9	13.4	5.94	−2.0	10.2
QHd3b	3	G249–RG418	4.81	2.5	10.4	2.57	1.4	9.0	1.80	1.2	7.1						
QHd8	8	RZ323–C225	7.19	3.4	14.1							4.82	2.3	12.3	4.49	1.5	8.4
QHd11	11	RZ53–RZ781										2.14	1.4	5.9			
QHd12	12	G402–RG20q													2.04	−1.2	5.6
QPh1a	1	RZ14–C944b	5.32	3.8	7.3							2.33	2.7	7.7			
QPh1b	1	RZ776–CDO348	3.77	−3.0	5.8												
QPh2	2	RG520–RZ446b													2.16	2.7	7.6
QPh3a	3	C515–RG348a	4.82	−3.9	7.5	3.33	−3.3	13.7	1.83	1.3	7.0						
QPh3b	3	RZ284–RZ403b										3.29	−2.8	8.1			
QPh4	4	*Ph*–G379										1.83	2.1	4.6			
QPh6	6	RZ682–C236	6.17	−4.8	14.1												
QPh8	8	G104–G2140	4.61	4.1	8.1							4.42	3.2	10.6	2.99	2.8	7.9
QPh12	12	G402–RG20q													2.45	−1.3	4.5
QFll1a	1	RG957–RG462													4.03	1.3	6.4
QFll1b	1	RZ288–C131	5.85	−2.2	12.0	2.12	−1.0	6.5				3.37	−1.3	9.3	2.76	−1.3	7.9
QFll3	3	G249–RG418b										4.43	1.2	8.1	4.45	1.6	10.8
QFll4	4	*Ph*–G379										6.98	2.0	19.4	5.61	1.5	9.5
QFll5	5	RG556–*gl1*				1.95	−1.3	7.5	3.45	−1.6	8.5						
QFll2	2	RG256–RZ260a				3.52	−1.7	13.3									
QFlw2	2	C624x–G45				1.81	−0.40	3.8	4.41	0.55	6.3						
QFlw3	3	RZ474–C746	2.63	−0.60	7.0							3.59	−0.55	10.1			
QFlw4	4	*Ph*–G379	3.66	0.76	11.2							3.57	0.51	6.9			
QFlw6	6	G200a–RZ667							2.06	0.39	3.5						
QFlw8a	8	G2140–RZ323a										7.85	0.80	21.1	2.54	0.48	8.4
QFlw8b	8	L457a–C1073a							2.61	0.55	6.4						
QPl1a	1	RZ14–C944b										5.42	0.61	15.6	4.45	0.70	16.1
QPl1b	1	RG140–RZ288				4.22	−0.71	13.2									
QPl2	2	G45–RZ386a													3.00	0.58	10.9
QPl5	5	RG556–*gl1*							1.95	−0.56	9.4						
QPl6	6	RZ762–C76										3.09	0.52	11.2	3.58	0.59	11.3
QPl7	7	RG678–G20				2.99	−0.56	8.3									
QPl8	8	C424b–RZ143	3.81	−0.80	10.3												
QSn1	1	RZ801–RZ14	5.73	−0.80	15.9												
QSn4	4	*Ph*–G379										3.15	0.47	11.9			
QSn6	6	C235a–G294d													3.88	0.60	15.8
QSn9a	9	G103b–RZ698				3.09	−1.02	21.6									
QSn9b	9	RG451–RZ404													2.55	0.46	9.5
QSf2	2	CDO718–RG437	2.23	2.6	8.9												
QSf6	6	G294a–G1468b	4.83	4.2	10.6	3.81	−4.1	15.7	4.08	−3.9	11.3						
QSf7	7	CDO497–BCD855				3.31	3.5	8.7	2.52	2.8	8.1						
QSf12	12	RG20q–RG91q	6.41	−4.5	13.4												

[a] H_{MP} is the mid-parental heterosis of the BCF_1 hybrids calculated from $H_{MP} = BCF_1 - MP$, where MP = (female RIL + *Lemont* or *Teqing*)/2. In the RILs, QTL effects were associated with the *Lemont* allele (due to replacement of the *Teqing* allele by the *Lemont* allele). In the BC populations, QTL effects for F_1 and H_{MP} were estimated by (the heterozygotes − the homozygotes). The genetic expectation of a QTL effect obtained is the additive gene effect (a) when estimated from the RILs, the additive and dominance effects ($a + d$) from the F_1 mean values, and the dominance effect (d) from the H_{MP} values

Four M-QTLs were detected for SF, explaining 32.9% and 24.4% (19.4% for H_{MP}) of the total variance in the RILs and $LTBCF_1$s. No significant M-QTL was detected in the $TQBCF_1$ population. Two M-QTLs appeared to be additive: the first (*QSf2*) increased SF by 2.6% while the second (*QSf12*) decreased SF by 4.5% in the RILs. *QSf6* appeared to be an under-dominant locus; this locus increased SF by 4.2% in the RILs, while about 4.0% of negative effects were detected for $LTBCF_1$s performance and H_{MP}.

Epistatic QTLs (E-QTLs) detected in the RILs and BC populations

Table 4 shows 29 E-QTL pairs identified in the RILs. These epistatic QTLs included five pairs accounting for 44.8% of the total variation in HD, three pairs explaining 27.9% of the total variation in PH, four pairs explaining 67.3% of the total variation in FLL, one pair explaining 10.9% of the total variation in FLW, six pairs explaining 69.4% of the total variation in PL, five pairs explaining 57.9% of the total variation

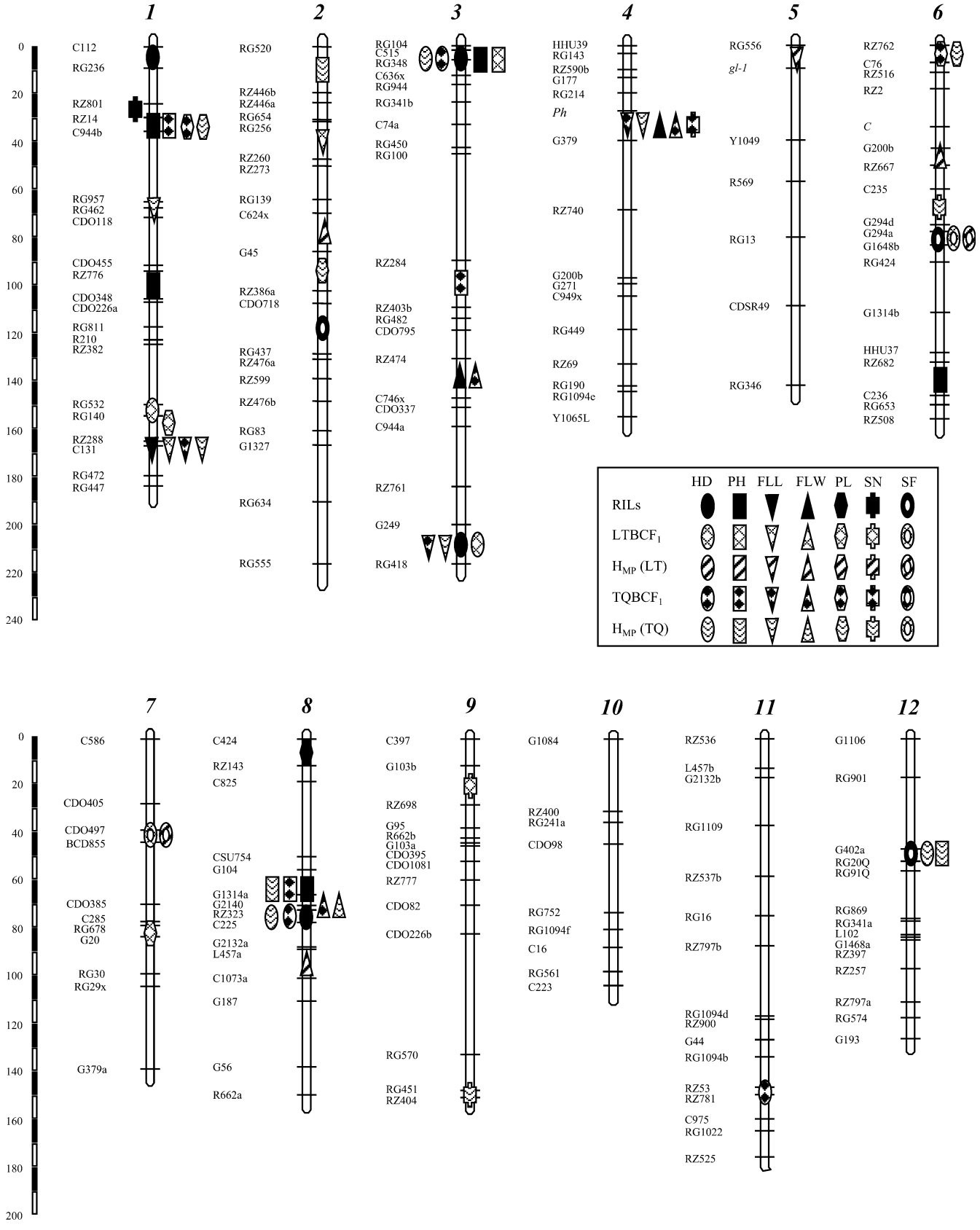

Fig. 1 Main-effect QTLs mapped in *Lemont*/*Teqing* RIL and two relative backcross populations using RILs, F_1s, and mid-parental heterosis values as input data

Table 4 Digenic epistatic QTL pairs affecting seven traits identified in 254 *Lemont*/*Teqing* RILs

Trait[a]	Chromosome	Marker interval i[b]	Chromosome	Marker interval j[b]	LOD	A_i[c]	A_j[c]	AA_{ij}[c]	R^{2d}
HD	1	**C112–RG236**	7	C285–RG678	6.10	2.2***		−1.9	5.1
	1	CDO455–RZ776	12	RZ797a–RG574	6.52			−2.9	9.8
	3	**G249–RG418**	5	RG556–*gl1*	9.90	2.4***		2.2	5.6
	4	G379–RZ740	10	C16–RG561	4.92			3.1	10.6
	8	CSU754–G104	8	**RZ323–C225**	11.17		3.4***	−3.5	13.7
PH	3	RG341b–C74a	6	RZ762–C76	6.26			−4.7	10.7
	4	RG449–RZ69	7	RG30–RG29	5.03	−2.2**		−4.0	8.1
	7	BCD855–CDO385	12	RZ257–RZ797a	4.71			4.3	9.1
FLL	1	C944b–RG957	4	RG449–RZ69	4.20			2.0	10.3
	1	RZ776–CDO348	10	G89–G1084	3.67			−2.1	10.9
	2	C624x–G45	6	RZ516–RZ2	4.46			2.0	9.9
	3	RZ761–G249	8	G104–G1314a	5.15			−1.8	8.3
FLW	3	C515–RG348a	11	C975–RG1022	4.39			0.75	10.9
PL	1	CDO118–CDO455	4	RG1094e–Y1065Lc	3.56			0.95	10.1
	1	CDO226a–RG811	2	C624x–G45	3.22			−0.76	6.6
	2	CDO718–RG437	2	RG83–G1327	4.00			−1.10	13.6
	4	HHU39–RG143	7	BCD855–CDO385	7.32			−1.27	18.3
	4	RG1094e–Y1065Lc	12	RG574-G193	4.52			0.85	8.1
	5	R569a–RG13	11	RZ797b–RG1094d	2.60			1.00	12.7
SN	2	RZ260–RZ273	2	RZ386a–CDO718	6.27		−0.33*	0.66	8.9
	2	G1327–RG634	4	RG190–RG1094e	4.40			0.58	6.7
	4	*Ph*–G379	6	RG653–RZ508	6.08			−0.87	15.1
	5	*gl1*–Y1049	9	CDO1081–RZ777	3.11		−0.38*	0.62	7.8
	6	RZ682–C236	7	C586–CDO405	4.71			0.98	19.4
SF	1	C112–RG236	2	RG634–RG555	2.65			3.4	7.0
	4	RG214–*Ph*	5	R569a–RG13	3.99			4.2	10.4
	4	G200b–G271	8	G187–G56a	2.58			3.2	6.0
	7	C586–CDO405	8	C825–CSU754	4.45			6.1	21.6
	7	CDO385–C285	8	C1073a–G187	4.56			3.0	5.2

*, **, ***M-QTL are significant at $P < 0.05$, 0.001 and 0.0001, respectively

[a]See Table 1 for traits

[b]Markers indicated in bold are those flanking M-QTLs identified in Table 3

[c] A_i and A_j are the main effects of the loci i and j, arising from by the substitution of the *Lemont* allele by the *Teqing* allele. AA_{ij} is the epistatic effect between loci i and j, as defined by Mather and Jinks (1982)

[d] R^2 is the proportion of the total phenotypic variation explained by the AA_{ij}, which were all significant at $P < 0.001$

in SN and five pairs explaining 50.2% of the total variation in SF. Of these E-QTLs, six pairs were detected between an interval having significant additive effect and other loci, while the remaining interactions occurred between two complementary loci.

Table 5 shows 29 E-QTL pairs detected in the LTBC population. There were four QTL pairs detected for HD, totally explaining 23.3% and 33.8% of the total variation in F_1 and H_{MP}, respectively; four QTL pairs for PH, explaining 48.7% and 46.7% of the total variation for F_1 and H_{MP}; five QTL pairs for FLL, explaining 40.5% and 46.2% of the total variation for F_1 and H_{MP}; five QTL pairs for FLW, explaining 56.7% and 39.4% of the total variation for F_1 and H_{MP}; only two QTL pairs for PL, explaining 17.3% and 12.6% of the total variation for F_1 and H_{MP}; five QTL pairs for SN, explaining 58.6% and 67.0% of the total variation for F_1 and H_{MP}; four QTL pairs for SF, explaining 44.2% and 31.7% of the total variation for F_1 and H_{MP}.

Table 6 shows 37 E-QTL pairs detected in the TQBC population. There were five QTL pairs detected for HD, totally explaining 41.0% and 49.1% of the total variation for F_1 and H_{MP}, respectively; three QTL pairs for PH, explaining 28.4% and 11.3% of the total variation for F_1 and H_{MP}; six QTL pairs for FLL, explaining 31.7% and 49.1% of the total variation for F_1 and H_{MP}; seven QTL pairs for FLW, explaining 42.8% and 49.1% of the total variation for F_1 and H_{MP}; six QTL pairs for PL, explaining 38.1% and 48.6% of the total variation for F_1 and H_{MP}; seven QTL pairs for SN, explaining 54.2% and 56.0% of the total variation for F_1 and H_{MP}; three QTL pairs for SF, explaining 18.0% and 32.4% of the total variation for F_1 and H_{MP}.

Discussion

The unique use of a set of RILs and their testcross hybrids for QTL mapping allowed us to measure directly heterosis and resolve gene actions (Liu et al. 1996). However, the testcross hybrid (TCF_1) retains an allele from the parental RIL and another one from the tester for each locus and, consequently, some uncertainty remains with respect to the reliability of the QTL

Table 5 Digenic epistatic QTL (E-QTL) pairs affecting the mean performance and heterosis of seven traits identified in the *Lemont* BC population (RILs × *Lemont*)

Trait[a]	Chromosome	Marker interval *i*	Chromosome	Marker interval *j*	BCF_1 (*Lemont*)					BC H_{MP} (*Lemont*)				
					LOD	A_i[b]	A_j[b]	AA_{ij}[b]	R^2[c]	LOD	A_i[b]	A_j[b]	AA_{ij}[b]	R^2[c]
HD	3	RG100–RZ284	3	CDO337–C944a	3.56			1.8	15.5					
	4	RG1094e–Y1065Lc	6	C235a–G294d						4.93			−1.9	13.1
	4	*Ph*–G379	8	G187–G56a						4.87			1.9	12.7
	11	G2132b–RG1109	11	RG16–RZ797b	2.68			1.2	7.8	2.68			1.5	8.0
PH	3	C944a–RZ761	12	RZ257–RZ797a	3.94			3.6	9.0	2.94			3.1	8.6
	4	RG143–G177	8	C225c–G2132a	3.30			−3.7	9.7	1.86		−1.9**	−2.2	4.9
	4	RZ590b–RG214	11	RZ781–C975	2.30			2.7	5.0	3.22			3.9	13.1
	5	*gl1*–Y1049	7	C586–CDO405	3.65			6.0	25.0	2.01			4.6	20.1
FLL	2	G1327–RG634	4	HHU39–RG143						3.85			−1.6	9.2
	3	RG348a–C636x	3	G249–RG418b	2.02			1.3	6.6					
	3	C944a–RZ761	9	CDO226b–RG570a	3.71			2.1	18.8	4.89	−0.9*		2.2	17.1
	4	RG143–G177	4	RG190–RG1094e	3.21			1.5	9.5	4.86			1.6	9.5
	4	RZ590b–RG214	6	C236–RG653	3.43			−1.2	5.6	3.63			−1.7	10.4
FLW	2	RG520–RZ446b	4	C949–RG449	3.48			0.63	7.5					
	3	C515–RG348a	3	RZ761–G249	3.17			0.47	4.5					
	4	HHU39–RG143	9	RG451–RZ404	5.78			−0.77	11.4	5.49			−0.71	13.3
	6	RZ762–C76	11	RZ781–C975	5.10			0.78	11.8	4.95			0.70	12.7
	10	G89–G1084	12	RZ797a–RG574	5.38			−1.06	21.5	3.28			−0.71	13.4
PL	1	CDO455–RZ776a	12	G1468a–RZ397	3.17			0.50	6.5					
	4	RG1094e–Y1065Lc	10	C16-RG561	3.50			0.64	10.8	2.77			0.63	12.6
SN	1	RZ382–RG532	2	RG83–G1327	4.04	−0.52*		1.09	18.9	2.29			0.86	11.3
	1	C112–RG236	11	RZ53–RZ781	2.58			−0.91	13.2	2.89			−1.25	23.0
	5	RG556–*gl1*	8	C424b–RZ143						2.59	−0.72**		−1.08	17.2
	6	RZ2–*C*	6	RG424–G1314b	3.88			0.98	15.4					
	6	C236–RG653	9	G103b1–RZ698	4.44		−0.47*	0.72	11.1	2.56			1.02	15.5
SF	1	RG140–RZ288	12	RZ257–RZ797a	3.97			−3.6	9.2	2.34			−3.1	7.1
	6	G200a–RZ667	9	G95–R662b	3.46			−3.4	8.6	2.05			−3.1	7.1
	8	RZ143–C825a	11	RG1094d–RZ900	6.07			4.5	14.9	3.39			4.2	13.0
	12	RG901a–G402	12	RZ257–RZ797a	4.54			4.0	11.5	1.89			2.2	4.5

*, **, ***$P < 0.05$, $P < 0.001$, and $P < 0.0001$, respectively

[a] See Table 1 for traits

[b] A_i and A_j are the main effects of the loci *i* and *j*, estimated by (the heterozygotes—the homozygotes) using the mean F_1 and H_{MP} values. AA_{ij} were the epistatic effects between loci *i* and *j* defined by Mather and Jinks (1982), which were all significant at $P < 0.001$

[c] R^2 is the proportion of the total phenotypic variation explained by the AA_{ij}.

analysis and gene action dissection mainly as a result of the unknown homology and dominant/recessive relationship between alleles from the RIL and tester (Mei et al. 2003). The genotypes of backcross hybrids (BCF_1s) can be clearly deduced from marker information of parental RILs—i.e. homozygotes of alleles from the recurrent parent or heterozygotes. Therefore, the genetic effects in BCF_1s can be more precisely defined in contrast to those in the TC F_1s; i.e. $a = (P_1P_1 - P_2P_2)/2$; $H_{MP} = d = (BCF_1 - (P_1P_1 + P_2P_2)/2)$ and $BCF_1 = (a + d)$ (when P_1 is the recurrent parent) or $(a + d + P_2P_2)$ (when P_2 is the recurrent parent, where P_2P_2 was constant for all BCF_1s). Then QTL actions can be inferred by comparison between mapping results in RIL and BCF_1 performance and mid-parental heterosis. Additive QTLs were detectable only in RIL or BCF_1 trait values without a significant dominant effect. QTLs with $d/a \leq 1$ were referred to as being complete or partial loci and were expected to have an estimate in BCF_1 performance $(a + d)$ equal to or higher than twice the H_{MP} (d). QTLs with $d/a > 1$—i.e. $2d$ $(2 \times H_{MP}) > a + d$ (BCF_1)—or only detectable for H_{MP} were referred as over-dominant loci (Melchinger et al. 1998; Li et al. 2001).

The declaration of QTLs is based on significant levels of the hypothesis test. The significant level for markers to be selected into the model and the threshold value at which to declare a QTL with significant effect will severely influence the final mapping results. A small difference of LOD score near the threshold value can alter the conclusion of whether a locus will be declared or not. So there is a risk in referring to QTL actions as being detectable or not in the BCF_1 or H_{MP}—i.e. type-I error and type-II error (Belknap et al. 1996). We obtained relatively higher LOD scores in the RIL population than in BCF_1 population (Table 3, 4, 5, 6). In the testcross (TC) population, a reduced detecting power was observed for TC performance than line performance; this was caused by "the mask effect of the tester" (Gallais and Rives 1993). Consequently, the probability of type-II error should be higher in the BCF_1 and H_{MP} than in the RILs. This is the reason for a lower LOD threshold being adopted in this study, especially for BCF_1 and H_{MP}. Thus, the proportion of

Table 6 Digenic E-QTL pairs affecting the mean performance and heterosis of the seven traits identified in the *Teqing* BC population (RILs × *Teqing*)

Trait[a]	Chromosome	Marker interval *i*	Chromosome	Marker interval *j*	BCF_1 (*Teqing*)					BC H_{MP} (*Teqing*)				
					LOD	A_i[b]	A_j[b]	AA_{ij}[b]	R^2[c]	LOD	A_i[b]	A_j[b]	AA_{ij}[b]	R^2[c]
HD	1	RG140–RZ288	11	RZ797b–RG1094d	3.03			1.8	4.8	3.25			1.7	7.9
	2	CDO718–RG437	5	Y1049–R569a	4.70			2.6	10.0	3.62			2.0	11.0
	4	RG214–*Ph*	11	RG1022–RZ525a	4.53			−2.4	8.5	2.14		−0.9*	−1.1	5.1
	6	G1314b–HHU37	7	CDO405–CDO497	4.43	−1.4**		−2.8	11.6	1.99			−2.3	13.4
	7	C285–RG678	12	G1106–RG901a	4.53			−2.1	6.1	4.40			−2.0	11.7
PH	1	C944b–RG957	7	C586–CDO405	3.28	4.3***		2.4	6.2					
	2	RZ476a–RZ599	4	RG214–*Ph*	5.92		2.0**	3.0	7.8	4.63		1.7**	3.4	11.3
	3	C944a–RZ761	4	RG1094e–Y1065Lc	4.90	1.5*		4.1	14.4					
FLL	2	RG520–RZ446b	10	G89–G1084	3.13			−1.3	7.0					
	3	G249–RG418b	4	C949–RG449	4.43	1.2***		−1.3	7.9	4.45	1.6***		−1.5	9.3
	2	RG520–RZ446b	3	C746–CDO337						4.36			1.5	9.1
	2	G45–RZ386a	4	G177–RZ590b	3.77			−1.6	12.0	6.78			−1.8	13.4
	3	G249–RG418b	10	RG752–RG1094f						2.97			−1.4	8.3
	4	*Ph*–G379	10	RZ400–RG241a	6.98	−2.0***		1.1**	4.8	5.14	1.4***		1.2	5.1
FLW	10	RZ400–RG241a	12	G1106–RG901a						2.86			0.51	8.4
	2	G1327–RG634	5	CDSR49–RG346						3.15			0.61	12.7
	3	C515–RG348a	3	RZ474–C746	7.69		−0.46***	−0.57	7.8					
	4	RZ590b–RG214	7	RG29–G370b	5.10	0.33**	0.35**	0.56	7.0	3.35		−0.38**	0.40	3.8
	4	G379–RZ740	6	RG653–RZ508	3.82			−0.60	8.0					
	4	G379–RZ740	12	RG20q–RG91q	4.15			0.56	7.1	4.26			0.65	14.5
	6	RZ667–C235a	9	R662b–G103a	5.76			−0.70	12.9	3.53			−0.55	10.2
PL	2	G1327–RG634	4	RZ69–RG190	5.54		0.42***	−0.45	5.6	1.99		0.33**	−0.29	4.5
	3	G249–RG418b	11	G2132b–RG1109	2.07	0.31**		0.43	5.3	4.13	0.31**		0.64	10.0
	5	RG556–*gl1*	8	C424b–RZ143	4.57			0.68	13.0	2.32			0.42	6.6
	6	*C*–G200a	9	G103b1–RZ698	5.38			0.71	14.2	2.07		0.34**	0.37	5.8
	1	CDO118–CDO455	1	RZ382–RG532a						2.69			−0.66	10.7
	4	RZ740–G200b	6	RG653–RZ508						2.70			0.60	9.0
SN	1	RG462–CDO118	6	G200a–RZ667	6.71			0.68	13.5	4.42			0.72	15.6
	1	CDO455–RZ776a	1	RG472–RG447	3.97			0.46	6.0					
	4	HHU39a–RG143a	11	RG16–RZ797b	6.16			0.69	13.8	2.51			0.47	4.4
	4	G177–RZ590b	6	RG424–G1314b	3.97			−0.54	8.4	3.39		0.28**	−0.59	6.9
	4	RG214–*Ph*	11	RZ781–C975	5.20			−0.66	12.5	7.45			−0.85	14.5
	3	C746–CDO337	6	G1314b–HHU37						2.62			0.54	5.8
	5	RG556–*gl1*	11	RG1109–RZ537b						3.61			−0.66	8.8
SF	1	CDO348–CDO226a	6	RZ516–RZ2	1.90			−2.1	5.4	4.90			−3.8	12.9
	2	RG139–C624x	3	RZ474–C746	2.59			−2.7	6.9	3.18			−3.0	8.2
	2	RG437–RZ476a	2	G1327–RG634	3.12	3.1***		−2.2	5.7	3.64	2.1**		−3.6	11.3

*, **, ***$P < 0.05$, $P < 0.001$, and $P < 0.0001$, respectively

[a] See Table 1 for traits

[b] A_i and A_j are the main effects of the loci *i* and *j*, estimated by (the heterozygotes—the homozygotes) using the mean F_1 and H_{MP} values. AA_{ij} were the epistatic effects between loci *i* and *j* defined by Mather and Jinks (1982), which were all significant at $P < 0.001$

[c] R^2 is the proportion of the total phenotypic variation explained by the AA_{ij}.

additive QTLs may be highly estimated in this study. Considering the precision limitation of the QTL approach itself, the results reported here should be applied with some wariness and require further confirmation by using near-isogenic lines covering target QTLs and epistatic pairs.

The RILs mostly carried homologous alleles for varied ranges from two parents, theoretically near 50% from each parent without skewness. So the BCF_1s had fewer heterozygous loci than the hybrid between the two parents (LT × TQ F_1). The reduction in the proportion of heterozygous loci in the BCF_1 population probably caused the reduced average level of heterosis in the BCF_1s compared to the LT × TQ F_1. However, wide variations were observed in trait value and H_{MP} of the BCF_1s (Table 1). The H_{MP} of some BCF_1s was stronger than that of the LT × TQ F_1, while some other lines expressed an H_{MP} in the opposite direction. It can be concluded that heterosis was generally related to the average level of heterozygosity in a hybrid population but had poor correlation with heterozygosity at the individual level. In other words, high heterosis came from heterozygosity at certain loci and not from genome-wide heterozygosity (Zhang et al 1995; Yu et al 1997). The fact that heterosis of the BCF_1s derived from the RIL exceeded that of the parental F_1 can be explained by the elimination of heterozygous loci with under-dominant effects and additive × dominant epistasis. The contribution of locus-specific heterozygosity or homozygosity to heterosis should be the genetic basis of special combining ability.

More M-QTL and E-QTL pairs were detected using the TC population together with RILs because of the increased ability to dissect QTLs with different types of gene action (Mei et al. 2003). The increased power in detecting more QTLs was confirmed while using BCF_1s

Table 7 Summarized results on gene action of QTLs affecting seven traits detected in the *Lemont*/*Teqing* RILs and two BC populations. Refer to the footnotes of previous tables for definitions

Trait	RILs		*Lemont* BC population								*Teqing* BC population							
			Additive		Complete or partial dominance			Over- or under-dominance			Additive		Complete or partial dominance			Over- or under-dominance		
	Number	R^2	Number	R^2 (F_1)	Number	R^2 (F_1)	R^2 (H_{MP})	Number	R^2 (F_1)	R^2 (H_{MP})	Number	R^2 (F_1)	Number	R^2 (F_1)	R^2 (H_{MP})	Number	R^2 (F_1)	R^2 (H_{MP})
M-QTLs																		
HD	4	48.0	1	8.0	1	9.0	7.1				2	19.3	1	12.3	8.4	1		5.6
PH	6	50.3			1	13.7	7.0				3	20.4				3	10.6	20.0
FLL	1	12.5	2	19.8				1	7.5	8.5	2	27.1	1	19.4	9.5	3	17.4	37.2
FLW	2	18.2						3	3.8	16.2	2	17.0	1	21.1	8.4			
PL	1	10.3	2	21.5				1		9.4						3	26.8	38.3
SN	1	15.9	2	41.2							1	11.9				2		25.3
SF	3	32.9						3	24.4	19.4								
Mean	2.6	26.9	1.0	12.9	0.3	3.2	2.0	1.1	5.1	7.6	1.3	11.7	0.6	9.5	5.2	1.7	7.8	18.0
E-QTLs																		
HD	5	44.8	1	15.5				3	7.8	33.8			1	8.5	5.1	4	32.5	44.0
PH	3	27.9			1	9.7	4.9	3	39.0	41.8	2	20.6				1	7.8	11.3
FLL	4	39.4	1	6.6				4	33.9	50.2	1	7.0				5	24.7	45.2
FLW	1	10.9	2	12.0	1	21.5	13.4	2	23.2	26.0	2	15.8	1	7.0	3.8	4	20.0	46.8
PL	6	69.3	1	6.5				1	10.8	12.6			3	32.8	16.9	3	5.3	29.7
SN	5	57.9	1	15.4				4	43.2	67.0	1	6.0	1	13.8	4.4	5	34.4	51.6
SF	5	50.2			1	11.5	4.5	3	32.7	27.2						3	18.0	32.4
Mean	4.1	42.9	0.9	8.0	0.4	6.1	3.3	2.9	27.2	36.9	0.9	7.1	0.9	8.9	4.3	3.6	20.4	37.3

together with RILs in this study. In total, 44 M-QTL (or 6.2 per trait) and 95 E-QTL pairs (or 13.6 per trait) were identified, most of which were detected in BCF_1 populations.

By comparing the genetic effects detected in the RILs, F_1 performance and mid-parental heterosis (H_{MP}), we were able to summarize the relative importance of M-QTLs and E-QTLs and additive and non-additive gene actions (Table 7). In the RI population, the numbers of M-QTL pairs varied from one to six, with an average of 2.6, and the contribution rate (R^2) varied from 10.3% to 50.3%, with an average $R^2 = 26.9\%$; The numbers of E-QTL pairs varied from one to six, with an average of 4.1, and the R^2 varied from 10.9% to 69.3%, with an average $R^2 = 42.9\%$. For main-effect QTLs, there was on average 1.0 additive locus in the LTBC population, with an $R^2 = 12.9\%$, while there 0.3 dominant QTLs, with an $R^2 = 3.2\%$ for the F_1 and an $R^2 = 2.0\%$ for H_{MP}, and 1.1 over- or under-dominant loci (denoted as over-dominant loci hereafter), with an $R^2 = 5.1\%$ for the F_1 and an $R^2 = 7.6\%$ for H_{MP}. In the TQBC population, 1.3 additive M-QTL pairs were detected having an $R^2 = 11.7\%$, while 0.6 dominant loci were detected with an $R^2 = 9.5\%$ for the F_1 and 5.2% for H_{MP}, and 1.7 over-dominant QTLs with an $R^2 = 7.8\%$ for the F_1 and with $R^2 = 18.0\%$ for H_{MP}.

For epistatic QTLs, only an average of 0.9 additive QTL pairs were detected in both BC populations for each trait, making about 8% contribution rates. On average, 0.4 QTL pairs was detected in the LTBC population with dominance behavior, having a 6.1% and 3.3% contribution rate for F_1 and H_{MP}, respectively, while there were 0.9 QTL pairs in TQBC, with a 8.9% and 4.3% contribution rate for F_1 and H_{MP}, respectively. Most QTL pairs behaved like over-dominant loci, with an average number of 2.9 and 3.6 for the LTBC and TQBC populations, respectively. The total contribution rates were 27.2% and 20.4% for the F_1, and 36.9% and 37.3% for H_{MP} in the LTBC and TQBC population, respectively.

In total, 11 M-QTLs were detected as common loci (or very near loci) in this study (Table 3) and in the (RI + TCF_1) populations (Mei et al. 2003): two loci for HD (*QHd3a, QHd8*), five loci for PH (*QPh3a*, *QPh3b*, *QPh4*, *QPh6*, *QPh8*), two loci for FLL (*QFll3*, *QFll4*), one locus for FLW (*QFlw4*) and one locus for SN (*QSn1*). If we consider that the phenotyping trials were carried out at two different locations, the results imply a good reliability of QTL mapping since 25–33.3% of the loci overlapped. A large number of unique QTLs were detectable only in the TC or BC design, a result probably caused by the increased mapping power gained by using TCF_1s and BCF_1s, together with QTL × E (environment) interaction in this study.

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