# Overdominant Epistatic Loci Are the Primary Genetic Basis of Inbreeding Depression and Heterosis in Rice. I. Biomass and Grain Yield

**Zhi-Kang Li,*,† L. J. Luo,‡ H. W. Mei,‡ D. L. Wang,§ Q. Y. Shu,§ R. Tabien,* D. B. Zhong,‡ C. S. Ying,‡ J. W. Stansel,* G. S. Khush† and A. H. Paterson*,****

*†Plant Breeding, Genetics, and Biochemistry Division, International Rice Research Institute, Metro Manila, The Philippines, *Department of Soil and Crop Sciences, Texas A&M University, College Station, Texas 77843, ‡China National Rice Research Institute, 310006 Hangzhou, China, §Department of Agronomy, Zhejiang Agricultural University, 310029 Hangzhou, China and **Department of Crop and Soil Sciences, University of Georgia, Athens, Georgia 30602*

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ABSTRACT

To understand the genetic basis of inbreeding depression and heterosis in rice, main-effect and epistatic QTL associated with inbreeding depression and heterosis for grain yield and biomass in five related rice mapping populations were investigated using a complete RFLP linkage map of 182 markers, replicated phenotyping experiments, and the mixed model approach. The mapping populations included 254 $F_{10}$ recombinant inbred lines derived from a cross between Lemont (japonica) and Teqing (indica) and two BC and two testcross hybrid populations derived from crosses between the RILs and their parents plus two testers (Zhong 413 and IR64). For both BY and GY, there was significant inbreeding depression detected in the RI population and a high level of heterosis in each of the BC and testcross hybrid populations. The mean performance of the BC or testcross hybrids was largely determined by their heterosis measurements. The hybrid breakdown (part of inbreeding depression) values of individual RILs were negatively associated with the heterosis measurements of their BC or testcross hybrids, indicating the partial genetic overlap of genes causing hybrid breakdown and heterosis in rice. A large number of epistatic QTL pairs and a few main-effect QTL were identified, which were responsible for >65% of the phenotypic variation of BY and GY in each of the populations with the former explaining a much greater portion of the variation. Two conclusions concerning the loci associated with inbreeding depression and heterosis in rice were reached from our results. First, most QTL associated with inbreeding depression and heterosis in rice appeared to be involved in epistasis. Second, most (~90%) QTL contributing to heterosis appeared to be overdominant. These observations tend to implicate epistasis and overdominance, rather than dominance, as the major genetic basis of heterosis in rice. The implications of our results in rice evolution and improvement are discussed.

INBREEDING depression and heterosis are related phenomena of fundamental importance to evolutionary biology and applied genetics. Inbreeding depression refers to reduced fitness of progenies resulting from inbreeding (Stebbins 1958; Wright 1977). In contrast, heterosis, or hybrid vigor, is defined as the superiority of an $F_1$ hybrid over its parents (Stuber 1994). Both heterosis and inbreeding depression are widely observed in both animal and plant kingdoms. In evolution, inbreeding depression may contribute to formation of reproductive barriers between species and populations, while heterosis may be an important force in maintenance of genetic variation in populations (Crow 1986). In applied genetics, exploitation of heterosis has played a major role in the genetic improvement of many crop plants and animals (Falconer 1981; Stuber 1994).

Heterosis and inbreeding depression are considered two aspects of the same phenomenon (Falconer 1981; Mather and Jinks 1982). Heterosis is clearly related to heterozygosity, but it has long been debated how heterozygosity results in heterosis. Two predominant theories were proposed as the genetic basis of heterosis. The overdominance hypothesis (Shull 1908; East 1936) states that heterozygosity at single loci confers properties that are superior to either homozygote. In contrast, the dominance hypothesis (Bruce 1910; Keeble and Pellew 1910; Jones 1917) proposes that dominant factors from either parent mask deleterious recessive mutations from the other parent in the heterozygous $F_1$. In both cases, inbreeding depression is due to segregation and expression of deleterious recessive mutations in inbred progenies (Allard 1960; Simmonds 1979). A third, less widely embraced hypothesis suggests that heterosis may arise from epistasis between alleles at different loci (Stuber 1994; Goodnight 1999).

Historically, heterosis and inbreeding depression are related to fitness and are influenced by many genes

*Corresponding author:* Zhi-Kang Li, Plant Breeding, Genetics, and Biochemistry Division, International Rice Research Institute, DAPO Box 7777, Metro Manila, The Philippines. E-mail: z.li@cgiar.org

as well as by environments (Mather and Jinks 1982). Recent quantitative trait loci (QTL) mapping studies sought to gain insights into the genetic basis of heterosis and inbreeding depression in crop plants. Using restriction fragment length polymorphism (RFLP) markers, Stuber *et al.* (1992) mapped quantitative trait loci (QTL) contributing to grain yield and its components in a backcross (BC) population derived from crosses between the $F_3$ progeny from a cross (B73 × Mo17) and their parental lines. Main-effect QTL with overdominance or pseudooverdominance effects were found to be largely responsible for heterosis in grain yield and its components in maize. Xiao *et al.* (1994) investigated the genetic basis of heterosis in two rice $BCF_1$ populations between 198 $F_8$ recombinant inbred lines and their parents. Ten QTL influencing grain yield components detected in both BC populations were completely or partially dominant. Recombinant inbred lines (RILs) having phenotypic values superior to the $F_1$ hybrid between the parental lines were found for all traits evaluated. These results led the authors to the conclusion that dominance complementation is the major genetic basis of heterosis in rice.

Results from other studies suggested that epistasis may be an important genetic basis of heterosis. Li *et al.* (1997a,b) reported that hybrid breakdown (part of inbreeding depression) in the $F_4$ progeny from an intersubspecific rice cross may be largely due to disharmonious interactions between alleles at many epistatic loci. Yu *et al.* (1997) reported overdominance at several main-effect QTL and pronounced additive epistasis affecting grain yield and its components in $F_3$ progeny from the most widely grown hybrid cultivar in China, Minhui 63.

The goal of the experiments described here was to study inbreeding depression and heterosis in a diverse sampling of germplasm using an improved statistical methodology to shed light on the relative importance of main-effect QTL and digenic epistatic loci associated with inbreeding depression and heterosis in biomass and grain yield of rice.

## MATERIALS AND METHODS

**Plant materials:** Five related mapping populations were used in this study. These included 254 $F_{10}$ RILs derived by single seed descent from a cross between Lemont (*japonica*) and Teqing (*indica*), two $BC_1F_1$ populations, and two testcross populations. Two $BC_1F_1$ populations included 172 Lemont (LT)$BCF_1$ hybrids (the RILs × Lemont) and 177 Teqing (TQ)$BCF_1$ hybrids (the RILs × Teqing). Two testcross populations were derived from testcrossing the RILs with two testers, Zhong 413 (a widely compatible restorer line developed in China) and IR64 (an *indica* cultivar developed in IRRI), which included 192 $Z413F_1$ hybrids (the RILs × Zhong 413) and 187 $IR64F_1$ hybrids (the RILs × IR64). In addition, the parents (Lemont and Teqing), the $F_1$ (Lemont × Teqing), and a hybrid cultivar, Shanyou63 (the most widely grown commercial hybrid cultivar in China), were used as checks in the phenotyping experiments.

**Phenotyping experiments:** The materials were evaluated in two separate experiments at two locations, Zhejiang Agricultural University (ZAU) and China National Rice Research Institute (CNRRI) in 1996. In the ZAU experiment, the RILs, parents, $F_1$ plants, the two $BC_1F_1$ populations ($LTBCF_1$s and $TQBCF_1$s), and the check hybrid were planted in the seedling nursery on May 25, 1996. The 25-day-old seedlings were transplanted into three-row plots each consisting of a single row of the female RIL and the two $BC_1F_1$ hybrids (the RIL × Lemont and Teqing). The plots were arranged in a randomized complete block design with two replications. Each row within a plot consisted of 15 plants with a spacing of 20 cm between the plants within each row and 35 cm between rows. Four check plots consisting of Lemont, Teqing, $F_1$, and Shanyou63 were randomly arranged in each replication. In the CNRRI experiment, the same three-row plots, each consisting of a single row of a RIL, and two rows of testcross hybrids (the RIL × Z413 and IR64) were used. In addition, the six check plots consisting of Lemont, Teqing, $F_1$, Z413, IR64, and Shanyou63 were also included in each replication. The field arrangement in CNRRI was the same as the ZAU experiment except that three replications were used.

At the maturity stage, three representative plants from the middle of each row plot were sampled and dried in an oven. Each sampled plant was evaluated for grain yield per plant (GY), biomass per plant (BY), and other grain yield components. Data for BY and GY were converted to tons per hectare (t/ha). Both the original data and $\log_e$-transformed data for BY and GY were used in the data analyses.

**Genotyping and construction of the RFLP linkage map:** Genomic DNA of the RILs, parental lines, and testers were extracted from freshly harvested leaves of 25-day-old seedlings grown in the greenhouse at Texas A&M University, College Station, Texas. RFLP mapping was conducted using published procedures (Li *et al.* 1995) and 179 well-distributed RFLP markers from Cornell University and the Japanese Rice Genome Research Program. The RILs were also evaluated for two morphological markers, *C* (apiculus color) and *gl-1* (glabrous leaf), in the field. An additional marker, the reactions of the RILs, parents, and testers to phenol (*Ph*), was evaluated by soaking 10 grains of each test material in 1% phenol solution for 24 hr. Black grain color indicated a positive reaction while unchanged (yellow) grain color indicated a negative reaction. Mapmaker version 3.0 (Lincoln and Lander 1992) was used to construct a complete linkage map with 182 markers covering 12 rice chromosomes. Linkage between markers was determined by the group command with a LOD threshold >6.0 and a recombination fraction of 0.25.

**Data analyses and QTL mapping:** Data of the RI, BC, and testcross populations were analyzed separately. SAS PROC GLM (SAS Institute 1996) was used to test the differences among the RILs and the BC/testcross hybrids. Equations for calculating values of hybrid breakdown (HB, a component of inbreeding depression) of individual RILs and the midparental heterosis for BY and GY of individual BC/testcross hybrids are listed in Table 1. In addition, two other relative heterosis measurements were calculated as follows: the better parental heterosis $H_{BP} = 100 \times (F_1 - BP)/BP$ and the competitive heterosis $H_C = 100 \times (F_1 - \text{Shanyou63})/\text{Shanyou63}$, where BP and Shanyou63 were the better parent and check hybrid. For mapping main-effect and epistatic QTL, data from each of the mapping populations were analyzed separately. Hybrid breakdown values of individual RILs for BY and GY were used as input data to identify QTL associated with hybrid breakdown. The midparental heterosis $H_{MP}$ values and the mean values of individual BC and testcross $F_1$ hybrids for BY and GY were used to identify QTL contributing to heterosis.

A mixed linear model for simultaneous mapping with back-

**TABLE 1**

**Equations for calculating hybrid breakdown of the recombinant inbred lines and the heterosis of the $BCF_1$ and testcross $F_1$ populations**

| Population[a] | *N* | Equation for measurement[b] |
|---|---|---|
| RILs | 254 | HB = RIL − MP, where MP = (Lemont + Teqing)/2 |
| $LTBCF_1$ | 172 | $H_{MP}$ = $F_1$ − MP, where MP = (RIL + Lemont)/2 |
| $TQBCF_1$ | 177 | $H_{MP}$ = $F_1$ − MP, where MP = (RIL + Teqing)/2 |
| $Z413BCF_1$ | 192 | $H_{MP}$ = $F_1$ − MP, where MP = (RIL + Z413)/2 |
| $IR64BCF_1$ | 187 | $H_{MP}$ = $F_1$ − MP, where MP = (RIL + IR64)/2 |

RIL, recombinant inbred line; $H_{MP}$, heterosis.

[a] $LTBCF_1$ and $TQBCF_1$s are two $BCF_1$ populations obtained by crossing the RILs with the parents, Lemont (LT) and Teqing (TQ); while $Z413F_1$s and $IR64F_1$s are two testcross $F_1$ populations obtained by crossing the RILs with the testers, Zhong 413 (Z413) and IR64.

[b] HB and $H_{MP}$ are estimates of hybrid breakdown and midparental heterosis. $F_1$'s are mean trait values of individual BC or testcross hybrids while RIL is the corresponding female RIL parent for each of the BC or testcross hybrids.

ground genetic variation control was used for interval mapping of both main-effect and digenic epistatic QTL for GY and BY segregating in the RI, BC, and testcross populations (Wang *et al.* 1999). The model can be expressed as

$$y_k = \mu + a_i x_{A_{ik}} + a_j x_{A_{jk}} + aa_{ij} x_{AA_{ijk}} + \sum_f u_{M_{fk}} e_{M_f} + \sum_l u_{MM_{lk}} e_{MM_l} + \varepsilon_k,$$

where $y_k$ is the phenotypic value of a quantitative trait measured on the $k$th individual ($k = 1, 2, \Lambda, n$); $\mu$ is the population mean; $a_i$ and $a_j$ are the main effects (fixed) of the two putative QTL ($Q_i$ and $Q_j$), respectively; $aa_{ij}$ is the epistatic effect (fixed) between $Q_i$ and $Q_j$; $x_{A_{ijk}}$, $x_{A_{jjk}}$, and $x_{AA_{ijk}}$ are coefficients of QTL effects derived according to the observed genotypes of the markers ($M_{i-}$, $M_{i+}$ and $M_{j-}$, $M_{j+}$) and the test positions ($r_{M_i-Q_i}$ and $r_{M_j-Q_j}$); $e_{M_f} \approx N(0, \sigma^2_M)$ is the random effect of marker $f$ with indicator coefficient $u_{M_{fk}}$ (1 for $M_fM_f$ and −1 for $m_fm_f$); $e_{MM_l} \approx N(0, \sigma^2_{MM})$ is the random effect of the $l$th marker interaction (between marker $K_l$ and marker $L_l$) with indicator coefficient $u_{MM_{lk}}$ (1 for $M_KM_KM_LM_L$ or $m_Km_Km_Lm_L$ and −1 for $M_KM_Km_Lm_L$ or $m_Km_KM_BM_B$). $\varepsilon_k \approx N(0, \sigma^2_\varepsilon)$ is the random residual effect. The inclusion of $e_{M_f}$ and $e_{MM_l}$ in the model is intended to absorb additive and epistatic effects of background QTL (additional segregating QTL other than the loci searched) for controlling the noise caused by the background QTL (Wang *et al.* 1999).

A new computer software, QTLMAPPER version 1.0, was developed on the basis of the above model (Wang *et al.* 1999), which allows simultaneous interval mapping of both main effect and digenic epistatic QTL in a RI, doubled haploid (DH), or BC population (with two genotypes at each marker locus). QTL mapping was carried out in three steps using the computer software. First, significant markers were identified across the genome using stepwise regression analyses based on single marker genotypes for putative main-effect QTL and based on all possible pairwise marker pairs for epistatic QTL with a threshold of $P \leq 0.005$. Then, all putative main-effect and epistatic QTL were identified using composite interval mapping in genomic regions centered at the markers (covering two marker intervals in each QTL region) identified in the first step with all QTL fixed in the model to control the background genetic variation. In this way, each of the QTL included in the model were significant at a threshold of $P \leq 0.002$ and $R^2 > 5\%$. This threshold was shown to have a very low probability of false positives (Wang *et al.* 1999). Finally, genetic parameters (effects and test statistics) associated with significant main-effect and epistatic QTL were estimated at the positions of respective LOD peaks in individual putative QTL regions (each putative QTL region covered two marker intervals) using the model and the restricted maximum-likelihood estimation method (Patterson and Thompson 1971, 1974; Wang *et al.* 1999).

The genetic expectations of the parameters estimated in the above model differ according to the nature of the mapping population and the input data. For the RI population, the main effects $a_i$ and $a_j$ are the additive effects of the two putative QTL ($Q_i$ and $Q_j$), and $aa_{ij}$ is the additive epistatic effect between $Q_i$ and $Q_j$ (Wang *et al.* 1999). For the $BCF_1$ populations, however, $a_i$ and $a_j$ are the combined effects of both additive and dominance gene actions ($\frac{1}{2}d - \frac{1}{2}a$) when estimated from the $F_1$ mean values and the QTL dominance effects ($\frac{1}{2}d$) when estimated from the midparental heterosis ($H_{MP}$) values. Similarly, the estimated epistatic effect using $H_{MP}$ measurements is the dominance × dominance ($dd_{ij}$) effect between epistatic QTL, while those from the mean $F_1$ values contained both additive and nonadditive epistatic components (Mather and Jinks 1982). The assumptions underlying the estimation of the epistatic effect are $aa_{ii} = aa_{jj} = -aa_{ij} = -aa_{ji}$ for the RI population and $aa_{ij} = dd_{ij} = -ad_{ij} = -da_{ij}$ for the $BCF_1$ populations, where $aa_{ij}$, $dd_{ij}$, $ad_{ij}$, and $da_{ij}$ are additive × additive, dominance × dominance, additive × dominance, and dominance × additive digenic epistatic effects between $Q_i$ and $Q_j$.

## RESULTS

**RFLP linkage map construction:** The complete linkage map of 182 markers (Figure 2) spanned 1918.7 cM and covered 12 rice chromosomes with an average interval of 11.3 cM between markers. There was a single gap of 54.8 cM on chromosome *9*. The linear orders agreed largely with those obtained from the $F_2$ population of the same cross (Li *et al.* 1995). A total of 46 (25.8%) markers showed segregation distortion, largely clustered in terminal regions of chromosomes *6–11*. On average, Lemont alleles accounted for 47.4 ± 7.7% of the genome, ranging from 16.1 to 62.3%.

**Inbreeding depression in the RILs and heterosis in the BC and testcross hybrids:** The paternal parent of the RILs, Teqing (*indica*), had significantly higher BY and GY than the maternal parent, Lemont (*japonica*),

**TABLE 2**

**Summary statistics on inbreeding depression of the Lemont/Teqing RILs and $H_{MP}$ of their backcross/testcross $F_1$ populations**

| | Biomass (t/ha) | | | Grain yield (t/ha) | | |
|---|---|---|---|---|---|---|
| | Mean | SD | Range | Mean | SD | Range |
| | | | The ZAU experiment | | | |
| Lemont (LT) | 2.74 | 0.64 | — | 1.46 | 0.43 | — |
| Teqing (TQ) | 5.46 | 1.08 | — | 3.47 | 0.64 | — |
| $F_1$ (LT × TQ) | 9.25 | 1.32 | — | 5.42 | 0.71 | — |
| $H_{MP}$ | 5.15 | | | 2.96 | | |
| CK (SY63) | 8.82 | 1.17 | — | 4.62 | 0.86 | — |
| $LTBCF_1$ | 5.96 | 2.00 | ~2.53–16.69 | 3.22 | 1.27 | ~0.87–9.89 |
| ($LTBCF_1$) $H_{MP}$ | 2.36 | 1.90 | ~−2.04–12.19 | 1.23 | 1.21 | ~−0.96–7.02 |
| $TQBCF_1$ | 7.80 | 2.20 | ~2.24–15.20 | 4.40 | 1.31 | ~1.34–7.92 |
| ($TQBCF_1$) $H_{MP}$ | 4.24 | 2.24 | ~−0.76–10.15 | 1.46 | 1.37 | ~−0.50–6.02 |
| RILs | 3.01 | 1.61 | ~1.13–9.63 | 1.47 | 0.78 | ~0.34–4.33 |
| HB (RIL − MP)[a] | −1.13 | 1.61 | ~−3.00–5.52 | −0.99 | 0.78 | ~−2.13–1.86 |
| | | | The CNRRI experiment | | | |
| Lemont (LT) | 3.86 | 1.19 | | 1.76 | 0.50 | |
| Teqing (TQ) | 15.63 | 1.89 | | 8.24 | 0.80 | |
| $F_1$ (LT × TQ) | 13.93 | 1.00 | | 8.09 | 1.76 | |
| $H_{MP}$ | 4.19 | | | 3.09 | | |
| CK (SY63) | 17.44 | 2.96 | | 8.37 | 1.30 | |
| Z413 | 14.07 | 0.34 | | 8.87 | 0.44 | |
| IR64 | 8.61 | 0.50 | | 4.04 | 0.36 | |
| $Z413F_1$ | 13.09 | 3.99 | ~4.37–29.12 | 6.91 | 2.23 | ~2.24–13.05 |
| ($Z413F_1$) $H_{MP}$ | 2.79 | 4.14 | ~−9.38–19.63 | 1.13 | 2.34 | ~−4.22–7.87 |
| $IR64F_1$ | 14.76 | 3.11 | ~4.02–26.35 | 7.50 | 1.79 | ~1.79–13.73 |
| ($IR64F_1$) $H_{MP}$ | 7.81 | 3.26 | ~−1.49–18.13 | 4.27 | 1.84 | ~−0.69–9.60 |
| RILs | 6.09 | 2.27 | ~2.13–13.83 | 2.63 | 1.14 | ~0.46–7.55 |
| HB (RIL − MP)[a] | −3.66 | 2.27 | ~−7.60–4.09 | −2.37 | 1.14 | ~−4.54–2.54 |

[a] HB = RIL − MP, where HB is hybrid breakdown and MP = (Lemont + Teqing)/2.

in both experiments (Table 2). The BY and GY values of the $F_1$ plants in the ZAU experiment were 9.25 and 5.42 t/ha, significantly higher than both parents, but 13.93 and 8.09 t/ha in the CNRRI experiment, similar to the better parent, Teqing. The midparental heterosis of the $F_1$ plants was 4.65 t/ha (101.1%) for BY and 2.96 t/ha (120.0%) for GY in ZAU, and 4.2 t/ha (42.9%) for BY and 3.09 t/ha (61.8%) in CNRRI, respectively.

*Inbreeding depression of the RILs:* Significant reductions for both BY and GY were observed, as a result of hybrid breakdown, in the RI population in both ZAU and CNRRI experiments (Table 2). In the ZAU experiment, hybrid breakdown values were −1.13 (−35.9%) and −0.99 (−40.1%) t/ha for BY and GY, respectively. None of the RILs had higher BY and GY than the $F_1$ plants, but two RILs had significantly higher BY and GY than the better parent, Teqing. Compared to the ZAU experiment, all the materials in the CNRRI experiment had much higher BY and GY and the RILs showed a greater degree of inbreeding depression. Hybrid breakdown values of the RILs were normally distributed (Figure 1) with mean values of −3.66 (−37.5%) and −2.37 (−47.4%) t/ha for BY and GY, respectively. None of the RILs had higher BY or GY than Teqing.

*Heterosis in the BC and testcross hybrid populations:* Highly significant heterosis for both BY and GY were observed in the BC and testcross hybrid populations, and heterosis values of the $BCF_1$ hybrids were distributed normally (Table 2 and Figure 1). On average, the $IR64F_1$ population showed the highest level of heterosis, the $LTBCF_1$ population the second, the $TQBCF_1$ the third, and the $Z413F_1$ the lowest. Within each of the populations, individual $F_1$ hybrids varied considerably in their mean values and heterosis values (Figure 1). Most BC or testcross hybrids showed highly significant positive heterosis. However, hybrids showing significant negative heterosis for BY and GY were observed but were much less frequent in all four $F_1$ populations.

In the ZAU experiment, the mean BY and GY values of the $LTBCF_1$ population were 5.96 and 3.22 t/ha. The heterosis values of individual hybrids were normally distributed with the mean of 2.36 t/ha (107.5%) for BY and 1.23 t/ha (120.1%) for GY. In particular, the top 10 hybrids had mean BY and GY values of 10.49 and 6.25 t/ha, giving a mean heterosis of 6.51 t/ha (265.1%) for BY and 4.55 t/ha (326.7%) for GY, respectively. The mean better parental heterosis and the competitive heterosis of the top 10 hybrids were 249.4 and 18.9%

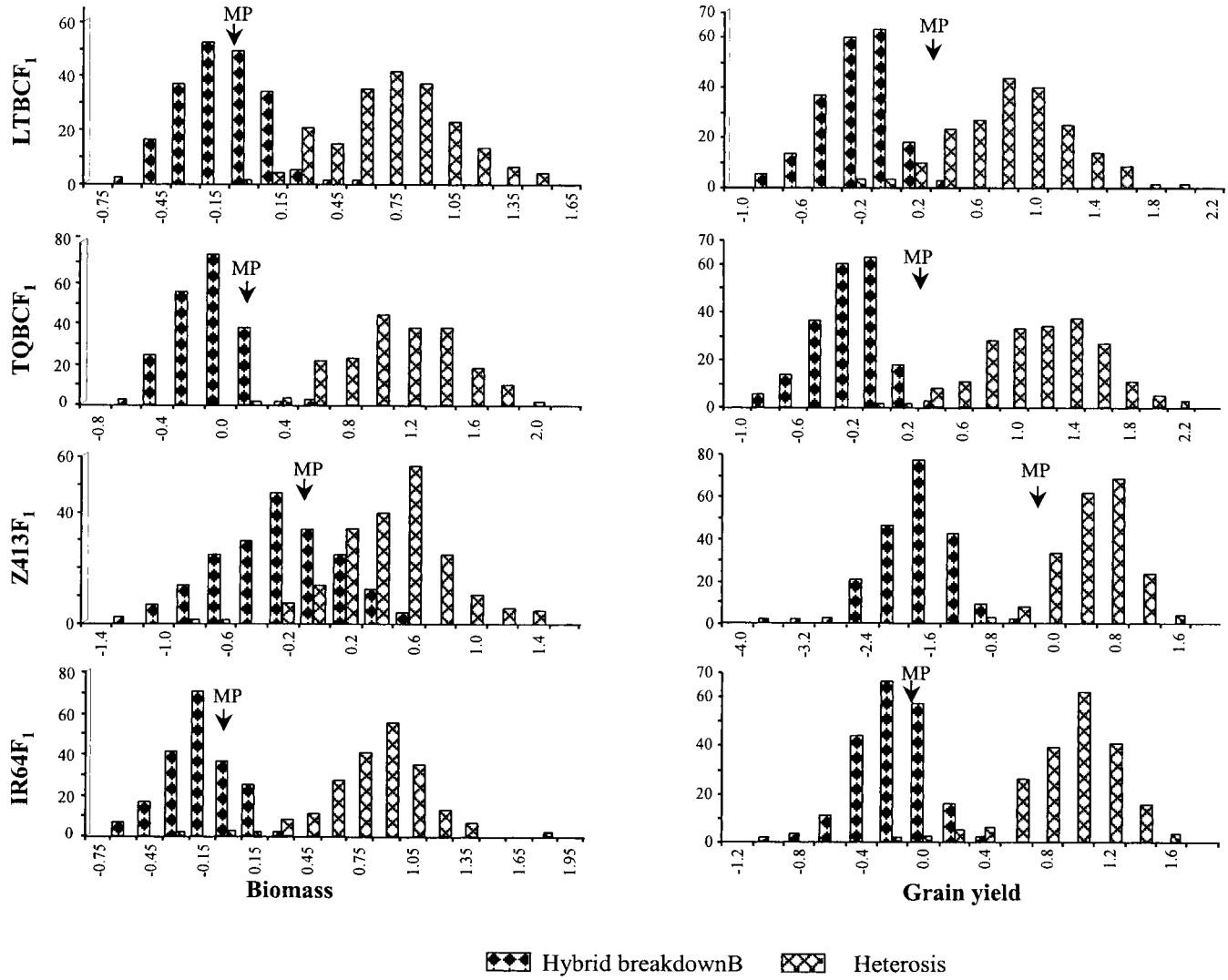



FIGURE 1.—Frequency distribution of hybrid breakdown (HB = RIL − $F_1$) of the Lemont/Teqing recombinant inbred lines and midparental heterosis for biomass and grain yield per plant of their backcross/testcross $F_1$ populations. MP, mean midparental values.

for BY and 324.7 and 35.4% for GY, respectively. The $TQBCF_1$ population had a mean value of 7.80 and 4.40 t/ha for BY and GY. The heterosis values of individual $BCF_1$s were normally distributed with a mean of 4.24 t/ha (84.3%), ranging from −0.76 to 10.15 t/ha for BY and 1.46 t/ha (78.1%) ranging from −0.50 to 6.02 t/ha for GY. The top 10 hybrids had mean BY and GY of 12.07 and 7.17 t/ha, with mean heterosis of 7.89 t/ha (184.3%) and 4.70 t/ha (191.0%), respectively. The better parental heterosis and competitive heterosis of the top 10 hybrids were 119.5 and 36.9% for BY and 106.6 and 55.4% for GY, respectively.

In the CNRRI experiment, the $Z413F_1$ population had very high $F_1$ mean value for both BY (13.09 t/ha) and GY (6.91 t/ha) but the lowest heterosis for BY (2.79 t/ha, or 29.8%) and GY (1.13 t/ha, or 20.3%). The top 10 hybrids in the $Z413F_1$ population had mean BY and GY of 21.58 and 11.47 t/ha and mean heterosis of 12.1 t/ha (112.8%) and 6.12 t/ha (199.4%), respectively. The better parental heterosis and competitive heterosis of the top 10 hybrids were 52.4 and 22.9% for BY and 29.2 and 37.0% for GY, respectively. The $IR64F_1$ population had the highest mean value for both BY (14.76 t/ha) and GY (7.50 t/ha) and the highest heterosis for BY (7.81 t/ha, or 100.8%) and GY (4.27 t/ha, or 124.8%). IR64 performed relatively poorly (with mean BY and GY of 8.61 and 4.04 t/ha). The top 10 hybrids of this population had mean BY and GY of 21.58 and 11.47 t/ha, with the mean heterosis of 12.1 t/ha (176.4%) and 6.12 t/ha (230.2%), respectively. The better parental heterosis and competitive heterosis of the top 10 hybrids were 135.8 and 16.5% for BY and 172.5 and 31.6% for GY, respectively.

**The relationships between hybrid breakdown, heterosis, and $F_1$ performance:** The mean performance of individual BC and testcross hybrids for BY and GY was largely determined by the levels of heterosis of individual hybrids instead of the mean performance of their

maternal RILs (Table 3). The correlations between the $F_1$ mean values and heterosis in the $LTBCF_1$, $TQBCF_1$, $Z413F_1$, and $IR64F_1$ populations were 0.76, 0.82, 0.85, and 0.81 for BY and 0.79, 0.81, 0.86, and, 0.83 for GY, respectively. There was no correlation between the $F_1$ mean values and the mean performances of their maternal RILs (Table 3).

In both ZAU and CNRRI experiments, hybrid breakdown and heterosis for GY and BY were distributed on both sides of the midparental value (at the zero point) with little overlapping (Figure 1). The hybrid breakdown values of individual RILs were negatively correlated with their heterosis values across all four $F_1$ populations. The correlation was highly significant ($P < 0.0001$) but moderate in magnitude ($r = -0.39$, $-0.59$, $-0.50$, and $-0.52$ for BY and $-0.47$, $-0.63$, $-0.57$, and $-0.49$ for GY; Table 3), suggesting that hybrid breakdown of the RILs and heterosis of their $F_1$ hybrids indeed shared a partially overlapping genetic basis. The mean performance of the paternal parents (Lemont, Teqing, Z413, and IR64) was also negatively associated with the midparental heterosis ($r = -0.84$ for BY and $-0.97$ for GY) but positively associated with the relative competitive heterosis of their $F_1$ hybrids ($r = 0.66$ for BY and 0.63 for GY).

**Main-effect and epistatic QTL associated with hybrid breakdown in the RILs:** The segregation of the RILs for BY and GY could be largely explained by many main-effect and epistatic QTL (Figure 2, Tables 4–6). In the ZAU experiment, two main-effect QTL affecting GY and BY were identified on chromosomes *2* and *11*. However, a total of eight pairs of epistatic QTL were identified. Among these loci, two (on chromosomes *7* and *10*) had significant additive effects (Table 6). The allele increasing GY and BY at all main-effect QTL but one was from Teqing. Five of the eight significant epistatic effects on GY and/or BY were positive and the remaining three were negative. In the CNRRI experiment, four main-effect QTL were mapped to chromosomes *3*, *4*, *6*, and *9* (Table 5). Ten pairs of epistatic QTL affecting GY and/or BY were identified. Of these, four loci located on chromosomes *3*, *5*, *6*, and *12* had significant main effects (Table 6). The Teqing allele at all main-effect QTL increased GY and BY. All significant epistatic effects but one were positive. The observation that most epistatic effects were positive indicated that hybrid breakdown for GY and BY in the RILs was largely due to the disharmonious interactions between alleles from different parents at these loci (Li *et al.* 1997a).

**Main-effect and epistatic QTL associated with the $F_1$ performance and heterosis:** Several main-effect QTL and many epistatic QTL were largely responsible for the phenotypic variation for BY and GY in the $BCF_1$ and testcross populations (Tables 4, 5, 7, Figure 2). In the $LTBCF_1$ population, a single main-effect QTL and seven pairs of epistatic loci affecting the $F_1$ performance and/or heterosis were identified (Tables 4 and 7). Of these

**TABLE 3**

**Phenotypic correlation for BY and GY between the performance of the Lemont/Teqing RILs and $H_{MP}$ in their $BCF_1$ and testcross $F_1$ populations**

| | | Between RIL values and $F_1$ values | | | | Between $H_{MP}$ and $F_1$ values | | | | Between RIL values and $H_{MP}$ | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Lemont | Teqing | Z413 | IR64 | Lemont | Teqing | Z413 | IR64 | Lemont | Teqing | Z413 | IR64 |
| GY | $r$ | 0.173 | −0.060 | −0.062 | 0.075 | 0.791 | 0.811 | 0.857 | 0.825 | −0.466 | −0.633 | −0.567 | −0.490 |
| | $R^2$ | 0.030 | 0.004 | 0.004 | 0.006 | 0.626 | 0.658 | 0.734 | 0.681 | 0.217 | 0.401 | 0.321 | 0.240 |
| BY | $r$ | 0.306 | −0.017 | 0.030 | 0.067 | 0.760 | 0.819 | 0.851 | 0.814 | −0.386 | −0.587 | −0.500 | −0.519 |
| | $R^2$ | 0.094 | 0.000 | 0.001 | 0.004 | 0.578 | 0.671 | 0.724 | 0.663 | 0.149 | 0.345 | 0.250 | 0.269 |

BY, biomass; GY, grain yield.

epistatic QTL, eight loci (on chromosomes *1–3*, *5*, *7*, and *8*) had significant main effects on BY and/or GY (Table 7). The heterozygote at all main-effect QTL except one (near RG30 of chromosome *7*) increased BY and/or GY. Four of the epistatic effects were positive, and the remaining three were negative.

In the $TQBCF_1$ population, two main-effect QTL (chromosomes *6* and *9*) and eight pairs of epistatic loci affecting the $F_1$ mean and heterosis of BY and GY were identified and mapped to chromosomes *1*, *4*, *6–9*, *11*, and *12* (Tables 4 and 7, Figure 2). Six of these epistatic loci had significant main effects on the $F_1$ mean and/or heterosis of BY and GY. At all main-effect QTL except one (near RZ382 of chromosome *1*), the heterozygote had greater BY and/or GY than the homozygous Teqing genotype. Five of the epistatic effects were positive, and the remaining three were negative.

For the $Z413F_1$ population, two main-effect QTL and nine pairs of digenic epistatic loci affecting the $F_1$ mean and/or heterosis were identified and mapped to nine of the rice chromosomes (*1–8* and *12*; Tables 5 and 7, Figure 2). Of the epistatic QTL, seven had significant main effects. The Lemont/Z413 (japonica/indica) heterozygote at all main-effect QTL except one (near RG653 on chromosome *6*), had greater BY and/or GY than the Teqing/Z413 (indica/indica) heterozygote. Of the significant epistatic effects, five were positive and the other four were negative.

For the $IR64F_1$ population, two main-effect QTL and eight pairs of digenic epistatic loci affecting the $F_1$ mean and/or $H_{MP}$ were identified and mapped to nine of the rice chromosomes (*2–8*, *11*, and *12*; Tables 5 and 7, Figure 2). Of these epistatic QTL, four had significant main effects on BY and/or GY. The Teqing/IR64 (indica/indica) heterozygote at all main-effect QTL had greater BY and/or GY than the Lemont/Z413 (japonica/indica) heterozygote. Of the significant epistatic effects, three were positive and the other five were negative.

## CONCLUSIONS AND DISCUSSION

**Decomposition of inbreeding depression in rice:** Prevailing in outcrossing species, inbreeding depression has been intensively investigated. However, inbreeding depression in self-pollinated plant species like rice has received little attention. The 60–70% reduction of the Lemont/Teqing RI population from the $F_1$ was highly significant but underestimated the overall degree of inbreeding depression in the Lemont/Teqing cross. This was attributable to purging of deleterious alleles and/or less fit multilocus genotypes by natural selection during the development of the RILs (Li *et al.* 1995, 1997a,b). Genetically, the inbreeding depression values of individual RILs and the mean values of BC or testcross hybrids for BY and GY have two components. One is the deviation of the RILs from the midparental value,

**TABLE 4**

**Main-effect QTL affecting GY (in t/ha) and BY (in t/ha) of the Lemont/Teqing RILs and $H_{MP}$ of their $BCF_1$ hybrids**

| | | | RILs | | | $BCF_1$ (Lemont) | | | $H_{MP}$ (Lemont) | | | $BCF_1$ (Teqing) | | | $H_{MP}$ (Teqing) | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Trait | Chromosome | Marker interval | LOD | $a$ | $R^2$ | LOD | $a + d$ | $R^2$ | LOD | $d$ | $R^2$ | LOD | $a + d$ | $R^2$ | LOD | $d$ | $R^2$ |
| BY | *2* | RG437–RZ476a | 5.98 | −0.53 | 15.8 | 4.28 | 0.72 | 10.2 | 3.30 | 0.69 | 9.6 | | | | | | |
| GY | *2* | RG437–RZ476a | 5.23 | −0.25 | 12.9 | 4.11 | 0.51 | 9.6 | 3.82 | 0.52 | 10.2 | | | | | | |
| BY | *11* | G44–RG1094b | 2.45 | −0.20 | 9.0 | | | | | | | | | | | | |
| GY | *11* | G44–RG1094b | 2.28 | −0.35 | 7.9 | | | | | | | | | | | | |
| BY | *6* | G294a–G1468b | | | | | | | | | | 7.48 | 1.04 | 19.5 | 8.51 | 1.08 | 22.3 |
| GY | *6* | G294a–G1468b | | | | | | | | | | 10.81 | 0.60 | 12.5 | 8.81 | 0.62 | 11.5 |
| BY | *9* | RG451–RZ404 | | | | | | | | | | 2.46 | 0.74 | 6.2 | | | |
| GY | *9* | RG451–RZ404 | | | | | | | | | | 3.37 | 0.42 | 5.4 | | | |

[a] In the RI population, 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 heterozygote minus the homozygote. The genetic expectation of a QTL effect obtained is the additive gene effect ($a$) when estimated from the RI, the additive and dominance effects ($a + d$) from the $F_1$ mean values, and the dominance effect ($d$) from $H_{MP}$ values.

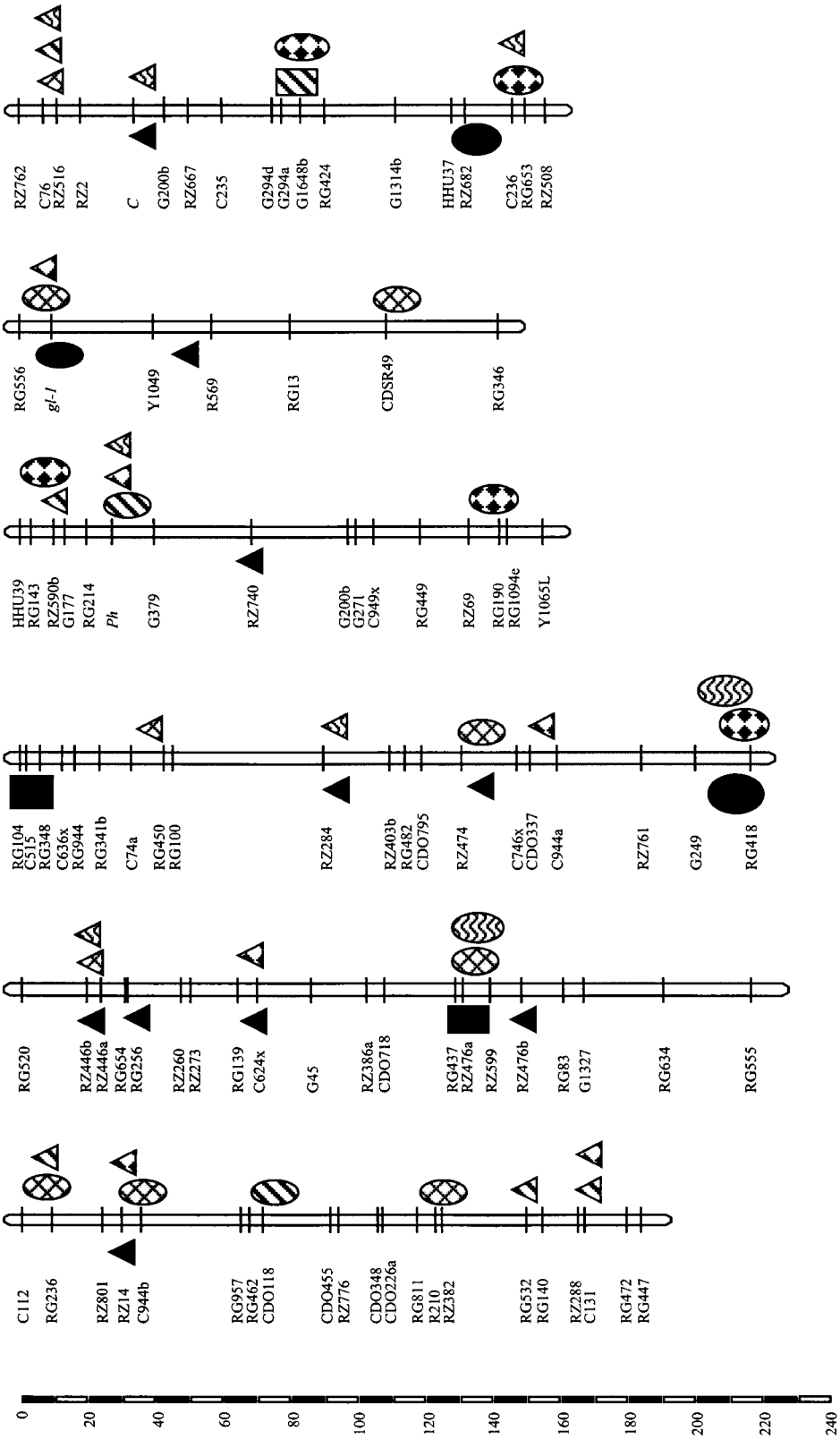
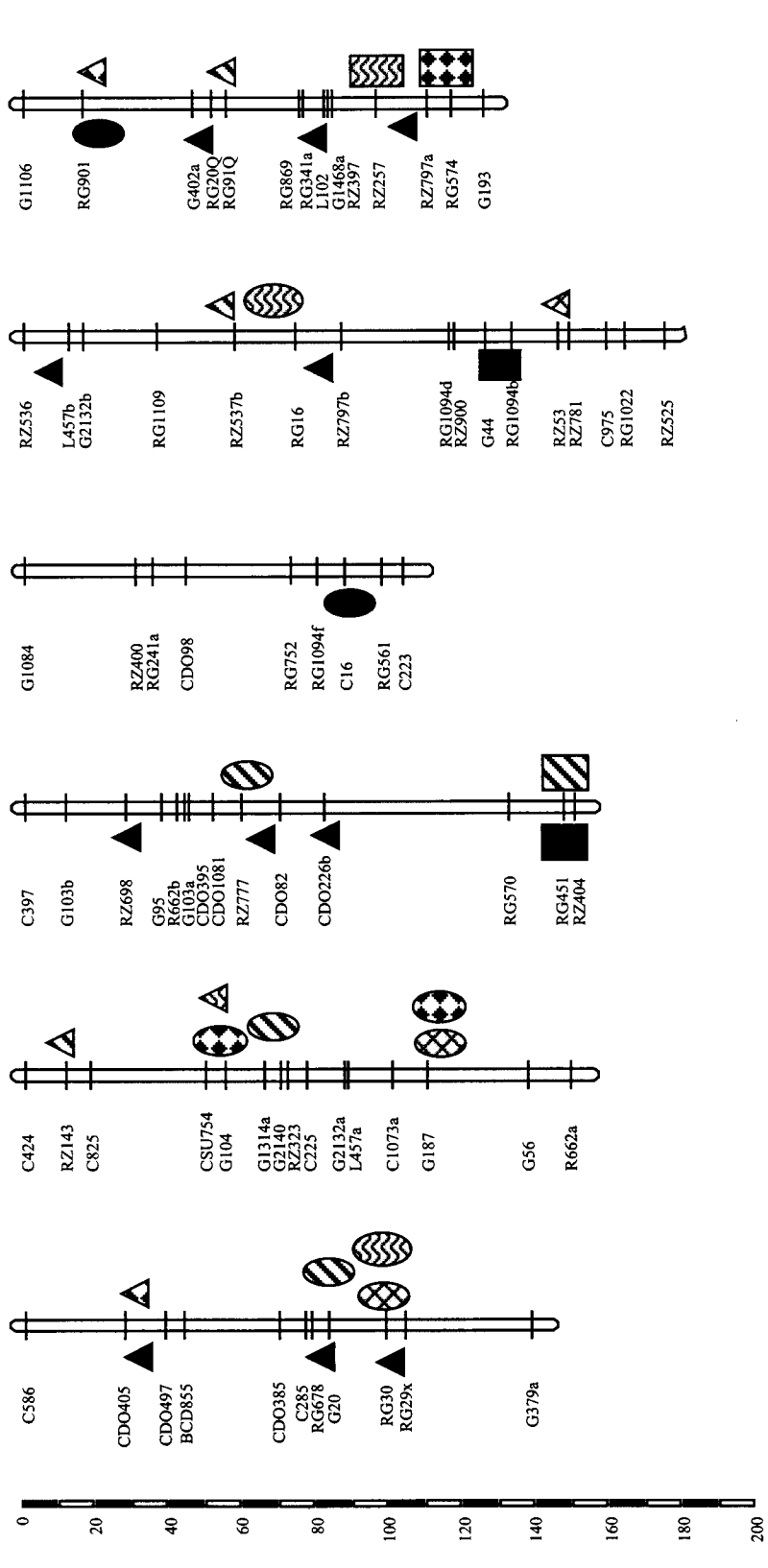



FIGURE 2.—Genomic locations of main-effect QTL and epistatic loci affecting grain yield and biomass detected in the Lemont/Teqing recombinant inbred lines (RILs) and their backcross/testcross $F_1$ populations. $LTBCF_1$, $TQBCF_1$, $Z413F_1$, and $IR64F_1$ represent the two backcross and two testcross $F_1$ populations, respectively.




Main-efect QTLs detected in the RILs, $LTBCF_1$, $TQBCF_1$, $Z413F_1$, and $IR64F_1$ populations, respectively.

Epistatic loci with significant main effects detected in the RILs, $LTBCF_1$, $TQBCF_1$, $Z413F_1$, and $IR64F_1$ populations, respectively.

Epistatic loci detected in the RILs, $LTBCF_1$, $TQBCF_1$, $Z413F_1$, and $IR64F_1$ populations, respectively.

FIGURE 2.—*Continued.*

**TABLE 5**

**Main-effect QTL affecting GY (in t/ha) and BY (in t/ha) of the Lemont/Teqing RILs and $H_{MP}$ of their testcross $F_1$ hybrids**

| | | | RILs | | | $F_1$ (Z413) | | | $H_{MP}$ (Z413) | | | $F_1$ (IR64) | | | $H_{MP}$ (IR64) | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Trait | Chromosome | Interval | LOD | Effect | $R^2$ | LOD | Effect | $R^2$ | LOD | Effect | $R^2$ | LOD | Effect | $R^2$ | LOD | Effect | $R^2$ |
| BY | *3* | C515–RG348a | 10.96 | −0.94 | 14.8 | | | | | | | | | | | | |
| GY | *3* | C515–RG348 | 11.05 | −0.48 | 14.3 | | | | | | | | | | | | |
| BY | *3* | G249–RG418 | 3.81 | −0.58 | 5.6 | 2.92 | 1.19 | 9.4 | 4.04 | 1.29 | 11.2 | | | | | | |
| GY | *3* | G249–RG418 | 8.34 | −0.46 | 12.2 | 3.95 | 0.57 | 10.8 | 4.07 | 0.65 | 12.7 | | | | | | |
| BY | *6* | HHU37–RZ682 | 4.79 | −0.66 | 8.8 | | | | | | | | | | | | |
| GY | *6* | HHU37–RZ682 | 2.79 | −0.30 | 5.3 | | | | | | | | | | | | |
| BY | *9* | RG451–RZ404 | 3.94 | −0.54 | 5.8 | | | | | | | | | | | | |
| GY | *9* | RG451–RZ404 | 3.58 | −0.34 | 7.2 | | | | | | | | | | | | |
| BY | *12* | RG574–G193 | | | | 3.26 | 0.93 | 9.3 | 4.43 | 1.17 | 11.1 | | | | | | |
| GY | *12* | RG574–G193 | | | | 4.25 | 0.63 | 11.0 | 4.10 | 0.65 | 11.6 | | | | | | |
| BY | *11* | RG16–RZ797b | | | | | | | | | | 2.45 | −0.68 | 7.9 | 3.18 | −0.73 | 8.8 |
| GY | *11* | RG16–RZ797b | | | | | | | | | | 3.75 | −0.43 | 11.1 | 4.86 | −0.50 | 12.6 |
| BY | *12* | RZ397–RZ257 | | | | | | | | | | 2.17 | −0.58 | 5.2 | 2.75 | −0.65 | 6.2 |
| GY | *12* | RZ397–RZ257 | | | | | | | | | | 3.30 | −0.37 | 6.3 | 3.01 | −0.42 | 6.6 |

In the RI population, QTL effects were associated with the Lemont allele (the effects due to replacement of the Teqing allele by the Lemont allele). In the testcross populations, QTL effects for $F_1$ and $H_{MP}$ (midparental heterosis) were estimated by the Lemont/tester genotype minus the Teqing/tester genotype.

**TABLE 6**

**Digenic epistatic QTL affecting hybrid breakdown of GY (in t/ha) and BY (in t/ha) in the Teqing/Lemont recombinant inbred population**

| Environment | Trait | Chromosome | Marker interval *i* | Chromosome | Marker interval *j* | LOD | $a_i$ | $a_j$ | $aa_{ij}$ | $R^2$ (%) |
|---|---|---|---|---|---|---|---|---|---|---|
| ZAU | BY | *2* | RZ476b–RG83 | *7* | RG678–G20 | 3.09 | | | −0.37*** | 7.9 |
| ZAU | GY | | | | | 2.58 | | | −0.19*** | 8.1 |
| ZAU | BY | *5* | Y1049–R569a | *10* | RG1094f–C16 | 3.82 | | | 0.51*** | 13.9 |
| ZAU | GY | | | | | 4.37 | | | 0.30*** | 12.9 |
| ZAU | BY | *7* | RG30–RG29 | *10* | RG1094f–C16 | 3.94 | | | −0.39*** | 8.8 |
| ZAU | GY | | | | | 4.61 | 0.16** | | −0.24*** | 11.2 |
| ZAU | BY | *9* | RZ698–G95 | *12* | RG901–G402 | 3.61 | | | −0.53*** | 14.8 |
| ZAU | GY | | | | | 4.24 | | | −0.33*** | 14.1 |
| ZAU | BY | *1* | RZ14–C944b | *4* | RZ740–G200b | 2.78 | | | 0.37*** | 7.2 |
| ZAU | BY | *2* | RZ446b–RZ446a | *10* | C16–RG561 | 3.24 | | | 0.37*** | 9.0 |
| ZAU | GY | *2* | RZ599–RZ476b | *3* | RG100–RZ284 | 3.80 | | | 0.19*** | 8.5 |
| ZAU | GY | *6* | RZ682–C236 | *11* | L457b–G2132b | 2.59 | −0.13* | | 0.31*** | 6.9 |
| CNRRI | BY | *2* | RG256–RZ260 | *12* | RZ257–RZ797a | 2.59 | | | 0.51*** | 6.2 |
| CNRRI | GY | | | | | 3.79 | | | 0.32*** | 8.5 |
| CNRRI | BY | *4* | RZ740–G200b | *11* | RZ797b–RG1094d | 3.03 | | | 0.44*** | 5.2 |
| CNRRI | GY | | | | | 3.95 | | | 0.30*** | 7.6 |
| CNRRI | BY | *5* | *gl1*–Y1049 | *11* | RZ536a–L457b | 4.05 | −0.52*** | | 0.65*** | 8.5 |
| CNRRI | GY | | | | | 5.20 | −0.30*** | | 0.20** | 5.2 |
| CNRRI | BY | *7* | CDO405–CDO497 | *9* | G103a–CDO395 | 3.30 | | | 0.47*** | 5.6 |
| CNRRI | GY | | | | | 3.52 | | | 0.22*** | 5.6 |
| CNRRI | BY | *3* | RZ474–C746 | *6* | *C*–G200a | 2.86 | | | 0.47*** | 5.6 |
| CNRRI | BY | *3* | RZ761–G249 | *12* | L102–G1468a | 4.76 | −0.49*** | | 0.36** | 4.3 |
| CNRRI | GY | *2* | RG139–C624x | *3* | RZ284–RZ403b | 4.90 | | | 0.29*** | 7.4 |
| CNRRI | GY | *2* | C624x–G45 | *9* | RZ777–CDO82 | 3.73 | | | −0.30*** | 8.0 |
| CNRRI | GY | *6* | RZ667–C235a | *12* | RG901a–G402 | 3.47 | −0.20** | | 0.26*** | 6.8 |
| CNRRI | GY | *11* | RG16–RZ797b | *12* | G1106–RG901 | 3.99 | | −0.17* | 0.24*** | 6.3 |

$a_i$ and $a_j$ were the main effects of the loci *i* and *j*, arising from the substitution of the Lemont allele by the Teqing allele, and $aa_{ij}$ was the epistatic effect between loci *i* and *j*, as defined by MATHER and JINKS (1982). *, significance levels of $P < 0.05$. **, significance levels of $P < 0.001$. ***, significance levels of $P < 0.0001$.

**TABLE 7**

**Digenic epistatic QTL affecting $F_1$ performance and $H_{MP}$ of GY (in t/ha) and BY (t/ha) detected in the LT and TQ $BCF_1$ and two testcross $F_1$ populations**

| | | | | | | $F_1$ | | | | | $H_{MP}$ | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Trait | Chromosome | Interval *i* | Chromosome | Interval *j* | LOD | $a_I$ | $a_j$ | $aa_{ij}$ | $R^2$ (%) | LOD | $a_I$ | $a_j$ | $aa_{ij}$ | $R^2$ (%) |
| LT | BY | *1* | R210–RZ382 | *2* | RZ599–RZ476b | 4.96 | 0.31* | 0.68*** | 0.31* | 5.1 | 2.52 | 0.30* | 0.40** | 0.39** | 6.7 |
| LT | GY | | | | | 4.09 | 0.17* | 0.39*** | 0.23* | 6.4 | 3.15 | 0.38** | 0.25* | 0.30** | 7.9 |
| LT | BY | *1* | C112–RG236 | *11* | RZ53–RZ781 | 2.11 | 0.39* | | −0.48** | 7.5 | | | | | |
| LT | GY | | | | | 2.74 | 0.18* | | −0.26** | 7.5 | | | | | |
| LT | BY | *2* | RZ446b–RZ446a | *7* | G20–RG30 | 5.10 | | 0.54** | 0.77*** | 12.0 | 4.13 | | 0.59** | 0.80*** | 13.4 |
| LT | GY | | | | | 3.36 | | | 0.44*** | 11.7 | 3.46 | | | 0.44*** | 10.9 |
| LT | BY | *3* | C74a–RG450 | *5* | CDSR49–RG346 | 2.43 | | 0.55*** | −0.55*** | 8.6 | 2.80 | | 0.47** | −0.68*** | 11.8 |
| LT | GY | | | | | 3.45 | | 0.38** | −0.41** | 11.2 | 3.01 | | 0.20* | −0.45*** | 11.6 |
| LT | BY | *5* | RG556–*gl1* | *8* | C424b–RZ143 | | | | | | 3.30 | 0.70** | | −0.70*** | 12.1 |
| LT | GY | | | | | | | | | | 3.12 | 0.23** | | −0.48*** | 12.3 |
| LT | GY | *1* | C944b–RG957 | *3* | RZ474–C746 | 3.51 | 0.24* | 0.24* | 0.25** | 7.1 | 2.34 | | | 0.34** | 8.9 |
| LT | GY | *6* | C76–RZ516 | *8* | G187–G56a | 3.40 | | 0.25* | 0.34** | 9.6 | 2.10 | | 0.18* | 0.36** | 9.4 |
| TQ | BY | *1* | CDO118–CDO455 | *1* | RZ382–RG532a | 4.04 | 0.68** | | −0.65*** | 8.5 | 3.97 | 0.70** | | −0.74*** | 10.9 |
| TQ | GY | | | | | 5.28 | 0.39*** | | −0.44*** | 8.9 | 5.39 | 0.43*** | | −0.44*** | 5.9 |
| TQ | BY | *1* | RG236–RZ801 | *6* | RZ762–C76 | 2.52 | | | 0.54** | 5.5 | 2.98 | | | 0.59*** | 8.5 |
| TQ | GY | | | | | 5.09 | | | 0.40*** | 7.4 | 5.01 | | | 0.45*** | 6.6 |
| TQ | BY | *1* | RZ288–C131 | *12* | RG20q–RG91q | 3.49 | | | 0.57** | 5.8 | 2.53 | | | 0.54** | 7.7 |
| TQ | GY | | | | | 5.72 | | | 0.57*** | 8.1 | 3.64 | | | 0.51*** | 7.5 |
| TQ | BY | *4* | G177–RZ590b | *9* | CDO1081–RZ777 | | | | | | 2.59 | | | −0.54** | 7.7 |
| TQ | GY | | | | | 5.74 | | | −0.48*** | 10.4 | 4.50 | | 0.23* | −0.40*** | 4.9 |
| TQ | BY | *4* | *Ph*–G379 | *11* | RG1109–RZ537b | 2.71 | 0.36* | | 0.56** | 6.8 | 2.11 | | | 0.53** | 7.3 |
| TQ | GY | | | | | 3.93 | 0.22* | | 0.28** | 5.8 | 3.21 | | 0.24* | 0.36** | 5.4 |
| TQ | BY | *7* | RG678–G20 | *8* | RZ143–C825a | 3.75 | 0.32* | | −0.59** | 6.9 | 3.42 | 0.40** | | −0.52** | 6.8 |
| TQ | GY | | | | | 5.69 | | | −0.52*** | 12.5 | 4.41 | | | −0.48*** | 7.1 |
| TQ | BY | *1* | RG472–RG447 | *12* | RG91q–RG869 | 2.74 | | | 0.71** | 9.3 | 3.20 | | | 0.79*** | 11.4 |
| TQ | BY | *8* | G2140–RZ323a | *9* | CDO1081–RZ777 | 3.29 | 0.51** | | 0.57** | 7.9 | | | | | |
| Z413 | BY | *1* | RZ14–C944b | *6* | G1468b–RG424 | 3.39 | | 0.60* | 0.94*** | 7.0 | 4.25 | | 1.03*** | 0.93*** | 7.6 |
| Z413 | GY | | | | | 5.52 | | 0.38** | 0.68*** | 8.9 | 5.04 | | 0.50** | 0.67*** | 8.3 |
| Z413 | BY | *2* | RG139–C624x | *8* | CSU754–G104 | 3.77 | | 1.21*** | −0.62* | 4.6 | 4.18 | | 1.36*** | −0.63* | 5.1 |
| Z413 | GY | | | | | 3.24 | | 0.49** | −0.38* | 5.0 | 3.62 | | 0.59*** | −0.41* | 5.1 |
| Z413 | BY | *3* | CDO337–C944a | *4* | HHU39–RG143 | 2.72 | | | −0.96*** | 7.1 | 3.48 | | 0.51* | −1.16*** | 9.5 |
| Z413 | GY | | | | | 2.99 | | | −0.59*** | 6.5 | 4.10 | | | −0.71*** | 8.8 |
| Z413 | BY | *4* | RZ69–RG190 | *6* | C236–RG653 | 4.58 | −1.62*** | 1.60*** | −1.47*** | 10.9 | 4.19 | −1.47*** | 1.55*** | −1.47*** | 12.0 |
| Z413 | GY | | | | | 3.22 | −0.91** | 0.67* | −0.72*** | 9.4 | 3.94 | −0.96** | 0.78* | −0.76*** | 9.4 |
| Z413 | BY | *4* | *Ph*–G379 | *8* | G187–G56a | 4.42 | | | −1.16*** | 8.6 | 4.91 | | 0.52* | −1.38*** | 11.3 |
| Z413 | GY | | | | | 3.24 | | | −0.67*** | 8.5 | 4.82 | | | −0.80*** | 10.0 |
| Z413 | BY | *1* | C131–RG472 | *4* | RG1094e-Y1065Lc | 2.49 | | | 0.86** | 6.2 | | | | | |
| Z413 | BY | *3* | G249–RG418 | *5* | RG556–*gl1* | 3.94 | 0.86*** | | 0.86** | 6.4 | | | | | |

(*continued*)

**TABLE 7**

**(Continued)**

| | Trait | Chromosome | Interval *i* | Chromosome | Interval *j* | $F_1$ | | | | | $H_{MP}$ | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | LOD | $a_I$ | $a_j$ | $aa_{ij}$ | $R^2$ (%) | LOD | $a_I$ | $a_j$ | $aa_{ij}$ | $R^2$ (%) |
| Z413 | BY | *7* | C586–CDO405 | *12* | RG901–G402 | 3.19 | | | 1.38*** | 10.2 | 3.52 | | | 1.64*** | 13.4 |
| Z413 | GY | *4* | RG214–*Ph* | *12* | RG901–G402 | 3.98 | | | 0.81*** | 10.6 | 3.75 | | | 0.75*** | 9.3 |
| IR64 | BY | *2* | RZ476a–RZ599 | *5* | RG13–CDSR49 | 3.65 | −0.45* | | −0.84*** | 6.4 | 3.73 | | | −0.96*** | 8.2 |
| IR64 | GY | | | | | 3.93 | −0.32** | | −0.44*** | 5.3 | 3.88 | | | −0.48*** | 6.0 |
| IR64 | BY | *2* | RG520–RZ446b | *8* | C825a-CSU754 | | | | | | 3.35 | | | 0.56*** | 6.9 |
| IR64 | GY | | | | | 2.17 | | | 0.35** | 4.9 | 3.13 | | | 0.50*** | 6.4 |
| IR64 | BY | *3* | RZ761–G249 | *8* | C825a–CSU754 | 2.66 | | | 0.85*** | 6.6 | 2.73 | | | 0.77*** | 5.8 |
| IR64 | GY | | | | | 3.31 | | | 0.51*** | 7.0 | 3.41 | | | 0.48*** | 5.8 |
| IR64 | BY | *3* | G249–RG418 | *11* | RG1109–RZ537b | 3.66 | | −0.59** | −1.00*** | 9.0 | 3.13 | | −0.72** | −0.86** | 6.5 |
| IR64 | GY | | | | | 3.83 | −0.26* | −0.32* | −0.62*** | 9.1 | 3.65 | −0.31* | −0.33* | −0.61*** | 7.7 |
| IR64 | BY | *4* | *Ph*–G379 | *6* | *C*–G200a | 2.57 | | | −0.75*** | 5.1 | 4.09 | | | −0.92*** | 7.4 |
| IR64 | GY | | | | | 3.15 | | | −0.49*** | 6.5 | 3.94 | | | −0.51*** | 6.6 |
| IR64 | BY | *6* | C236–RG653 | *11* | RG16–RZ797b | 5.21 | | | 1.27*** | 14.6 | 5.03 | | | 1.19*** | 12.6 |
| IR64 | GY | | | | | 5.36 | | | 0.70*** | 13.3 | 5.15 | | | 0.65*** | 10.8 |
| IR64 | BY | *2* | RG520–RZ446b | *3* | RZ284–RZ403b | 3.06 | | | −0.95*** | 10.9 | 3.25 | | | −1.19*** | 12.1 |
| IR64 | GY | *6* | RZ762–C76 | *7* | RG30–RG29 | 3.15 | | 0.56** | −0.59*** | 8.6 | 3.22 | | 0.66** | −0.57** | 7.2 |

$a_i$ and $a_j$ were the main effects of the loci *i* and *j*, arising from the substitution of the Lemont allele by the Teqing allele, and $aa_{ij}$ was the epistatic effect between loci *i* and *j*, as defined by Mather and Jinks (1982). *, significance levels of $P < 0.05$. **, significance levels of $P < 0.001$. ***, significance levels of $P < 0.0001$.

which is due to additive gene action, and causes hybrid breakdown (Stebbins 1958; Oka 1988; Li *et al.* 1997a,b). Genes of this type are directly detectable in the RILs but confounded in the BC and testcross populations. The other is the deviation of the $F_1$ value from the midparental value or heterosis due to genes of nonadditive action, which are segregating and contributing to heterosis in the BC and testcross populations but are not directly detectable in the RILs.

The decomposition of inbreeding depression into its additive (hybrid breakdown) and nonadditive (heterosis) components is very important in understanding the genetic basis of heterosis. The presence of hybrid breakdown in self-pollinated plant species such as rice has long been observed (Stebbins 1958; Oka 1988; Li *et al.* 1997a,b) but not expressed mathematically as part of inbreeding depression and heterosis in quantitative genetic theory (Falconer 1981). In our mapping populations, the genetic overlap between hybrid breakdown and heterosis was ~30% for GY and 25% for BY. This group of overlapping genes is of particular importance since they contributed positively to heterosis when in heterozygous status and negatively to the mean performance of the inbred RILs (resulting in hybrid breakdown) when in homozygous status. This also provided an explanation for the observation that the mean performance of the female RILs was not correlated with the mean performance of their BC/testcross hybrids. This is consistent with the observed heterosis in rice reported by Zeng, who found no correlation between $F_1$ mean and the midparental values for grain yield and biomass in 34 commercial rice hybrids (Zeng *et al.* 1979). In numerous classic quantitative genetic or breeding studies using diallel and/or test crosses, additive gene action was shown to be important to the mean performance of $F_1$ hybrids in rice and other crop species (*cf.* Simmonds 1979; Virmani 1994). This is not surprising since the materials used in most classical studies of test or diallel crosses had been more or less subjected to selection for improved performance (additive gene action). Thus, selection for improved performance of those inbred lines in breeding might have eliminated most hybrid breakdown genes or gene combinations observed in our base RI mapping population.

**Genetic basis of heterosis in rice:** Two unique features of this research are its experimental design and statistical methods used. Our crossing schemes and experiments using related RI, BC, and testcross populations were specifically designed to allow simultaneous mapping and characterization of loci contributing to inbreeding depression and heterosis. Data from the parents, RILs, $BCF_1$ hybrids, testcross hybrids, and testers in the same experiments provided direct measurements of hybrid breakdown and heterosis. In this way, both additive and nonadditive gene actions at the detected loci were more accurately resolved. For instance, the QTL main effects obtained using the heterosis values of the $BCF_1$ populations were estimates of the QTL dominance effects ($\frac{1}{2}d$) while those obtained from the $F_1$ mean values contained both additive and dominance effects ($\frac{1}{2}d - \frac{1}{2}a$; Mather and Jinks 1982). Similarly, for the epistatic loci, the estimated epistatic effects using heterosis measurements should be the dominance × dominance effects of the epistatic QTL, while those from the mean $F_1$ values contained both additive and nonadditive epistatic components (Mather and Jinks 1982). Use of two testcross populations offered additional advantages in understanding the genetic basis of heterosis since test crosses are the most common way to identify superior hybrids in animal and plant breeding programs. The main and epistatic effects of QTL obtained from the $F_1$ mean and heterosis values of the testcross populations also reflected the relative importance of different types of gene action in heterosis.

Second, the mixed model approach used in this study is an extension of the composite interval QTL mapping method (Zeng 1993, 1994) with inclusion of digenic epistasis and the appropriate background genetic variation control of all significant main-effect and epistatic QTL in the model (Wang *et al.* 1999). Computer simulation demonstrated that in a DH, RI, or BC population of 200 individuals, QTL with main and/or epistatic effects >5% in $R^2$ can be reliably detected and estimated (Wang *et al.* 1999). This was the basis of the threshold $P \leq 0.002$ and $R^2 \geq 5\%$ used in this study. Using this method, we were able to identify many main-effect and epistatic QTL responsible for >70% of the total phenotypic variation for BY and GY in each of the mapping populations. If we had used methods such as MapMaker/QTL or regression, we would have reached similar results as Xiao *et al.* (1994) with one to three mapped main-effect QTL explaining <30% of the phenotypic variation in each of the mapping populations.

*Epistasis is a common feature of most loci associated with inbreeding depression and heterosis:* This conclusion was supported by the following three observations. First, in each of the mapping populations, the majority (43.7% for BY and 58.6% for GY in the RILs, 42.5 and 58.3% in $LTBCF_1$s, 42.5 and 58.3% in $TQBCF_1$s, 59.0 and 49.4% in $Z413F_1$s, and 56.1 and 48.3% in $IR64F_1$s) of the phenotypic variation in the $F_1$ mean and heterosis values was due to epistatic QTL, while a much smaller portion (28.5% for BY and 30.4% for GY in the RILs, 9.6 and 10.2% in $LTBCF_1$, 14.7 and 24.0% in $TQBCF_1$, 20.5 and 23.1% in $Z413F_1$, and 14.1 and 18.3% in $IR64F_1$) of the variation was due to main-effect QTL. Second, 25 (86%) of the 29 QTL with significant main-effects were involved in epistasis detected in one or more populations (Figure 2). Together, of at least 54 QTL identified in this study, only 4 were not involved in epistasis in any of the mapping populations (Figure 2). The pronounced epistasis detected in this study was not due to multiplicative gene action since all identified epistatic loci were detectable using both original and $\log_e$-transformed

data. In a similar experimental design, Xiao *et al.* (1994) identified a single main-effect QTL in each of the two rice $BCF_1$ populations, which had $R^2$ of ~6–7% for GY with the majority of the phenotypic variation unexplained. Apparently, their failure to detect epistasis was largely attributed to the unavailability of appropriate mapping methodology. With a similar experimental design, Stuber *et al.* (1992) reported 6 and 8 main-effect QTL responsible for ~60% of the phenotypic variation of GY in two maize $BCF_1$ populations, though epistasis was not adequately evaluated. These and other data suggest that main-effect QTL tend to explain a greater portion of phenotypic variation for GY in maize than in rice (Stuber *et al.* 1992; Lin *et al.* 1996; Veldboom and Lee 1996; Li *et al.* 1997a,b; Yu *et al.* 1997). Epistasis for complex traits appears to be more pronounced in self-pollinated crop species than in outcrossing species. This is not surprising since coadapted gene complexes generated by epistasis between or among unlinked loci can be more easily maintained in the former than in the latter (Allard 1988).

Our results revealed several interesting properties of epistasis in rice. First, epistasis does not necessarily occur between main-effect QTL. For instance, of the 50 epistatic QTL pairs contributing to heterosis, 3 occurred between alleles at two main-effect QTL (type I), 20 between alleles at a main-effect QTL and a "background" locus (type II), and 27 between alleles at two complementary loci (type III). Of the 18 epistatic QTL pairs associated with hybrid breakdown, 6 were of type II and the rest (66.7%) were of type III. Second, all detected epistasis occurred between alleles at two unlinked QTL. These were consistent with the results on grain yield components observed in the $F_4$ progeny of the same cross (Li *et al.* 1997a). Third, the overall magnitude of the QTL epistatic effects detected in the present study was slightly greater than the mean main-effect QTL by 5.2% for BY and 16.3% for GY in the BC and testcross populations, but smaller by 19.1% for BY and 11.5% for GY in the RILs. It should be pointed out that the estimated epistatic effects obtained in this study almost certainly underestimated the true QTL epistatic effects. This is because the assumptions that $aa_{ii} = aa_{jj} = -aa_{ij} = -aa_{ji}$ for the RI population and $aa_{ij} = dd_{ij} = -ad_{ij} = -da_{ij}$ for the $BCF_1$ populations generally do not hold true (Li *et al.* 1997a). Our results that both positive and negative heterosis resulted from interactions between alleles at many epistatic loci suggest an explanation for the complexity of heterosis in rice and many other species.

*Epistasis plays an important role in the evolution of rice:* It is a long-debated issue tracked back to Wright's shifting balance theory and Fisher's large population size theory regarding the relative importance of epistasis as a genetic basis underlying evolutionary changes (*cf.* Wade and Goodnight 1998). As discussed above, the pronounced epistasis for fitness traits in rice appeared to be reflected in two aspects, the large portion of the total phenotypic variance in fitness (GY) and its components contributed by epistatic loci (Luo *et al.* 2001) and the predominant pattern by which most interactions occurred between alleles of complementary loci. In the former case, the contribution of epistasis to population variance was far greater than most theoreticians had assumed (Goodnight 1995; Wade and Goodnight 1998). In the latter case, our results lend strong support to Wright's (1969) statement that "Evolution depends on the fitting together of favorable complexes that can not be described in themselves as either favorable or unfavorable."

Epistasis as the genetic basis for hybrid breakdown, outbreeding depression, and recombination load is demonstrated in almost all experimental studies involving interspecific crosses (*cf.* Wade and Goodnight 1998). Similarly, breakdown of coadapted indica and japonica gene complexes by recombination appeared responsible for the significant level of hybrid breakdown observed in the RILs. This conclusion is supported by the observation that most epistatic effects, $aa_{ij}$, detected in the RI population were positive (>0). According to Mather and Jinks (1982), this indicated that most recombinant type interactions between alleles from different parents at the epistatic QTL resulted in reduced BY and GY. In other words, hybrid breakdown of the RILs is due largely to incompatible interactions between *indica* (Teqing) and *japonica* (Lemont) alleles at unlinked epistatic QTL, as reported previously (Li *et al.* 1997a,b). It would be expected that main-effect QTL tend to increase the mean fitness of the RI population as a result of selection favoring the alleles for increased GY and/or BY.

The suggestion from the correlation analysis that a common group of genes contribute to both hybrid breakdown of the RILs and heterosis of the BC/testcross hybrids was supported by the close correspondence in genomic locations of detected QTL in the related populations (Figure 2). For instance, 7 of the 14 main-effect QTL and 10 of the 14 epistatic QTL associated with hybrid breakdown were also detected as main-effect and/or epistatic QTL affecting heterosis in at least one of the BC/testcross populations. We further noted a significant portion of QTL mapped in similar locations in different BC/testcross populations, even though no phenotypic correlation exists between the mean values of different hybrid populations (data not shown). For example, of the 29 main-effect QTL detected, 7 (24.1%) were detectable in more than one mapping population. When the epistatic loci were included, 25 (50%) of the 50 loci were detected in more than one mapping population.

As a predominantly selfing plant species, subdivision of populations into subspecies and different local ecotypes of rice was correlated with ecological variability and environmental heterogeneity. For instance, the

maximum diversity at both phenotypic (including isozymes) and molecular levels (RFLPs, randomly amplified polymorphic DNA and simple sequence repeats) of rice is along the Himalayas where the environments were most heterogeneous (our unpublished data; Chang 1976; Oka 1988; Li and Rutger 2000). In other words, predominantly selfing plant species present the most extreme form of the shifting balance process where there is more pronounced epistasis at multiple loci and subdivision of populations by inbreeding. It can be conceived that with strong epistasis and genotype-by-environment interactions for fitness traits, local adaptation can be readily achieved by rare multilocus genotypes arising from recombination of occasional outcrossing between subpopulations, leading to multiple fitness peaks in the diverse environments. In these cases, epistasis and genotype-by-environment interactions act as evolutionarily diverging forces (Wade and Goodnight 1998), while recombination produces novel multilocus genotypes on which selection and inbreeding (or genetic drift) can operate. Thus, our results lend strong support to Wright's shifting balance theory and suggest that epistasis combined with genotype-by-environment interactions may have played a key role in the evolution of rice and other predominantly selfing plant species.

*"Overdominance" is associated with most loci contributing to heterosis in rice:* This conclusion comes from the following two results. First, 14 (58.3%) of the 24 main-effect QTL detected in the BC and testcross populations appeared to be overdominant as they were either only detectable using heterosis values or had a $d/a$ ratio $>2.0$. Two were dominant with $d/a$ ratios of 1.43 and 1.11. The remaining 8 were additive as they were detected only by the $F_1$ mean values. Second, the dominance effects at all main-effect QTL detected in the two $BCF_1$ and $Z413F_1$ populations were positive ($>0$), resulting in increased GY and/or BY. Interestingly, at all 6 main-effect QTL detected in the $IR64F_1$ population, the indica heterozygotes (Teqing/IR64) had positive effects, resulting in increased GY and/or BY while all the japonica/indica heterozygotes (Lemont/IR64) had negative effects, resulting in reduced GY and/or BY. Similarly, 28 (87.5%) of the 32 detected epistatic QTL pairs appeared to be "overdominant" as the epistatic effects estimated from heterosis values were equal to or greater than those estimated from the $F_1$ mean values. In other words, most epistatic QTL contributing to heterosis showed only the dominance × dominance gene action. There were only 4 pairs of additive epistatic loci that were detectable by only the $F_1$ mean values of the testcross hybrids.

These data strongly support the notion that heterosis for rice yield derives largely from epistatic interactions between loci that result in apparent overdominance. Pronounced overdominance at main-effect QTL for GY is reported also in maize and rice (Stuber *et al.* 1992; Yu *et al.* 1997). Heterosis at single loci may result from true overdominance at single loci or from pseudooverdominance generated by repulsion-phase linkage between partially dominant genes (Crow 1952). It is conceivable that overdominance at the main-effect QTL in maize is more likely true since outcrossing and selection do not favor a high frequency of repulsion linkage or a high level of epistasis between unlinked genes in maize populations. Our results on epistasis were certainly different from the situations described by Simmonds (1979), in which single-locus pseudooverdominance could arise from interactions between homozygous alleles at two loci. Heterosis can be generated by dominance × dominance epistasis (Goodnight 1999), but our results indicated that epistatic overdominance effects did not generally occur between loci having significant main effects (either additive or dominance effects). The overdominance at most main-effect and epistatic QTL observed in this study was unlikely due to repulsive linkage of completely or partially dominant genes; otherwise, one would have to explain why selection should favor such a high level of genetic load maintained by repulsion linkage in the rice genome.

Genetically, complete or partial dominance should be more likely for loci where there is a null allele (nonfunctional allele). However, studies on isozymes have indicated a high frequency of codominance and a very low frequency of null alleles at most isozyme loci in rice (Li and Rutger 2000). Then, the long-debated issue on the genetic basis of heterosis would become the question of how codominance at the genic level in hybrids could lead to overdominance at the phenotypic level. Biochemical or physiological evidence and interpretation for the phenotypic overdominance resulting from the codominant heterotic genes/QTL should shed light on this important issue.

**Implications for genetic improvement and marker-aided breeding for improved productivity in rice:** Our results indicated that the genetic basis of hybrid breakdown and heterosis in rice is very complex, reflected by the large number of loci involved, their wide genomic distribution, and complex epistatic relationships. These results have important implications for genetic improvement of rice. Our observation that the top 10 hybrids in the BC and testcross populations out-yielded the best commercial hybrid cultivar, Shan you63, by 23.8% (BY) and 39.9% (GY) indicate that there is tremendous genetic variation and potential for heterosis in rice productivity. Thus, development of hybrid cultivars should be more efficient and promising than breeding for inbred varieties with regard to further increasing the productivity of rice through exploitation of intersubspecific heterosis for both increased biomass and its partitioning. To do so, however, backcross breeding should be more effective to introgress rare desirable alleles or allele combinations from distantly related donor parents and to overcome the genetic drag arising from incompatible epistasis. Marker-aided transfer of desirable QTL identi-

fied in this study to improve productivity is expected to be difficult because of epistasis, possible genotype-by-environment interactions, and few main-effect candidate QTL. Nevertheless, a main-effect QTL mapped between G249 and RG418 on chromosome *3* is of particular interest. This QTL was detected in three of the five mapping populations in this study and was mapped to the same genomic location in the $F_4$ progeny of the Lemont/Teqing cross and several other japonica/indica mapping populations (Lin *et al.* 1996; Wu *et al.* 1996; Li *et al.* 1998). This QTL is associated with changes of the source size (leaf length and size) and sink capacity (Li *et al.* 1998). The large additive and dominance effects of this QTL justify its potential use in genetic improvement of both inbred and hybrid cultivars through marker-aided transfer in breeding programs.

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