RESEARCH

Genetic and Heterosis Analyses using Hayman's Six-Generation Model for Grain Yield and Yield Components in Maize

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ABSTRACT

This study aimed to (i) estimate additive (*a*), dominance (*d*), *aa*, *ad*, and *dd* genetic variances and effects; (ii) investigate the differences for five genetic variances or effects among crosses between a maize (*Zea mays* L.) line from the Suwan-1 heterotic group and five inbred lines from other groups; and (iii) develop a theory to explain the results from Hayman's model and low-, mid-, and high-parent heterosis. Hayman's model was used to evaluate the genetic effects in five six-generation sets between the Suwan-1 cross with five lines from other groups. The magnitude of variance was consistent among all crosses for grain yield (GY) and ear length (EL) but different for ear diameter (ED), row number per ear (RE), kernel number per row (KR), and 100-kernel weight (KW). The *a*, *d*, *aa*, *ad*, and *dd* effects were similar for EL among crosses between lines from different groups, indicating that the effects for EL could be used to differentiate Reid and non-Reid heterotic groups. We postulated a 'linked coexpressed genes' (LCG) model to (i) explain why *a*, *d*, *aa*, *ad*, and *dd* variances and effects might be different when a common line is crossed with other lines and (ii) serve as an improved model explaining heterosis. The LCG model explains what dominance–overdominance theories can explain and explains what dominance–overdominance models do not. For example, it can explain why an inbred line cannot perform like a hybrid and why an F_2 population for a quantitative trait has a normal distribution.

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Abbreviations: *a*, additive variances and effects; *aa*, additive × additive variances and effects; *ad*, additive × dominance variances and effects; *d*, dominance variance and effects; *dd*, dominance × dominance variance and effects; ED, ear diameter; EL, ear length; GMA, generation means analysis; GY, grain yield; HPH, high-parent heterosis; KE, kernel number per ear; KR, kernel number per row; KW, 100-kernel weight; LCG, linked coexpressed genes; LPH, low-parent heterosis; MPH, mid-parent heterosis; RE, row number per ear; SS, sum of squares; YC, yield components.

Generation means analysis (GMA) is widely used to investigate the genetic effects for complex quantitative traits in plant breeding programs (Bernardo et al., 1992; Checa et al., 2006; Azizi et al., 2006; Wilson et al., 2013). The GMA uses the pooled effects of all loci instead of a single locus controlling a quantitative trait. Based on the model established by Hayman (1958), *a*, *d* and various epistatic genetic effects (*aa*, *ad*, and *dd*) can be estimated via GMA using six generations [parents (P_1, P_2), F_1, F_2, BC_1, and BC_2].The information on genetic effects from GMA analyses helps plant breeders devise appropriate strategies and design experiments for inbred line and hybrid development, as well as for population improvement.

Published in Crop Sci. 55:1006–1016 (2015).
doi: 10.2135/cropsci2014.08.0542

The GMA has been used for estimating genetic effects in major crops (White et al., 1982; Kamaluddin et al., 2007; El-Refaey and El-Razek, 2013; Wilson et al., 2013). The GMA has been used in many studies on maize quantitative traits, such as GY, yield components (YC), and other agronomic traits (Thompson et al., 1971; Rahman et al., 1994; Herman et al., 2011). Genetic effects for maize GY and YC have been documented across the world (Gamble, 1962; Yang, 1982; Guo et al., 1986; Yi and Tong, 1987). Yang (1982) studied GY, KW, KR, RE, and EL via GMA. Yang (1982) found that GY, KR, EL, and KW were conditioned by significant *a* and epistatic gene effects; *d* gene effects were important for all studied traits, except KW. Guo et al. (1986) studied more than 10 quantitative traits in maize via GMA and observed significant *d* effects for GY and KW. On average, the variation attributable to *a* gene effects was larger than that attributable to *d* gene effects. In contrast, Yi and Tong (1987) showed that EL, ED, RE, KR, kernel number per ear (KE), and KW were mainly conditioned by *d* gene effects, with *a* and epistatic effects making variable contributions in different crosses.

Different researchers' results have shown that genetic estimates were different for different crosses. Studying leaf and stem resistance to gummy stem blight in cucumber (*Cucumis sativus* L.) with GMA, St. Amand and Wehner (2001) found that *a*, *d*, *aa*, *ad*, and *dd* effects were different among five different crosses in cucumber. In maize, researchers also have documented different results with different crosses. For example, Darrah and Hallauer (1972) demonstrated that an *a*–*d* model sufficiently captured most of the genetic variance; epistatic variance usually accounted for less than 10% of the total genetic variance. Mahto and Ganguli (2001), using completely different maize crosses obtained, via GMA, similar results to those of Darrah and Hallauer (1972) for GY, EL, and KE. Their results showed that both *a* and *d* genetic effects were significant for GY, EL, and KE only for the 'CML85' × 'CML42' cross; the *d* genetic effect was significant and greater than the *a* effect for most of the studied traits in all the crosses. In contrast, Gamble (1962), using different crosses from other researchers, investigated genetic effects for maize GY and major YC, such as ED, EL, RE, KR, and KW and found that epistatic effects were more important than *a* effects but not as important as *d* effects in determining GY and YC. The *aa* and *ad* effects were more important than *dd* effects.

Regression analysis has often been used to calculate *a*, *d*, *aa*, *ad*, and *dd* genetic effects (Bernardo et al., 1992; Cui et al., 2009; Herman et al., 2011). The coefficient of determination (R^2) from regression analysis is frequently used to indicate the relative importance of different genetic effects. Bernardo et al. (1992) used R^2 to determine the relative importance of *a*, *d*, *aa*, *ad*, and *dd* effects and found that different proportions of *a* and *d* effects contributed to resistance to head smut in maize. They reported that *a* genetic effects contributed more to the resistance in both crosses, whereas *d* genetic effects contributed to resistance only in one cross. Cui et al. (2009) calculated the proportion of different genetic effects based on sum of squares (SS) for *a*, *d*, *aa*, *ad*, and *dd* and found that *a* genetic effects accounted for 70.47% of the total SS for RE and 59.99% for ED; for KE, *d* genetic effects contributed 51.51% of the total SS and epistatic genetic effects contributed 37.15%.

Heterosis was discovered more than 100 yr ago (Shull, 1908). The major hypotheses proposed to explain the genetic basis of heterosis are dominance and overdominance (Crow, 1952), pseudo-overdominance (Han and Hallauer, 1989), and epistasis (Jinks and Jones, 1958). After more than 100 yr of breeding practice, these hypotheses have been found to be inadequate to explain some aspects of heterosis. For example, the dominance hypothesis has not been able to explain why we cannot practically obtain inbred lines performing as high as a hybrid and why a trait's distribution is not skewed (Coors and Pandey, 1999). Pseudo-overdominance is related to interactions between two dominant genes closely linked in the repulsion phase. Though this hypothesis has been supported by some evidence (Stuber, 1994; Graham et al., 1997), genomic studies have showed that the genes controlling a trait may not be always linked (Holton and Cornish, 1995; Shinozaki and Shinozaki, 2007); furthermore, genes may not be always linked in the repulsion phase (Fu and Dooner, 2002; Guo et al., 2004). Epistasis hypothesis was not supported in tomato (*Solanum lycopersicum* L.) because heterosis was manifested when epistasis did not exist (Semel et al., 2006).

Heterosis exists for most of the quantitative traits in maize and other cross-pollinated crops. To fully exploit heterosis, US maize breeders generally classify maize germplasm into two heterotic groups, Reid and Lancaster (non-Reid) (Hallauer and Miranda, 1988). Recently, three major heterotic groups have been defined based on a molecular marker study (Romay et al., 2013). Chinese maize breeders, however, classify maize germplasm into four or five heterotic groups (Wang et al., 1997; Peng et al., 1998; Yuan et al., 2001). Fan et al. (2008, 2009) identified Suwan-1 as a new heterotic group and argued that three heterotic groups might be advantageous for maximizing maize-breeding efficiency in China (Fan et al., 2014). The three heterotic groups are Suwan-1, Reid, and non-Reid. Following the identification of Suwan-1, it has been studied intensively and used as a parent in many hybrids released in China (Fan et al., 2009; 2014; Yao et al., 2013).

Cai et al. (2012) suggested that ribulose bisphosphate carboxylase and phospho-enolpyruvate carboxylase might be two physiological or biochemical traits that distinguish the Suwan-1 heterotic group from Reid and non-Reid groups. Are there any genes or quantitative trait loci controlling any quantitative traits that make the three heterotic

groups different? Currently, no information is available on this aspect. The relative importance of *a*, *d*, *aa*, *ad,* and *dd* genetic effects are likely to be useful for plant breeders to determine whether selection should be practiced for a target trait in early generations or late generations in breeding programs. Grain yield, ED, EL, RE, KR, and KW are a few of the key agronomic traits. Though many investigations on the estimation of *a*, *d*, *aa*, *ad*, and *dd* effects have been conducted (Gamble, 1962; Yang, 1982; Guo et al., 1986; Yi and Tong, 1987), the results across studies cannot be easily compared because the crosses used in different studies were usually made from completely different maize inbred lines. To make the results from different crosses comparable, five crosses were made, in which one line from the Suwan-1 heterotic group was used as the female parent and the other five lines from three different maize heterotic groups were used as the male parents. Six generations (P_1, P_2, F_1, F_2, BC_1, and BC_2) were produced from each of the five crosses. In addition, low-parent heterosis (LPH), mid-parent heterosis (MPH), and high-parent heterosis (HPH) were calculated for GY, ED, EL, RE, KR, and KW. Our specific objectives were to: (i) estimate *a*, *d*, *aa*, *ad* and *dd* genetic variances and effects; (ii) investigate the differences, if any, for the five genetic variances or genetic effects among crosses made between a common line (Suwan-1 heterotic group) and five lines from other heterotic groups; and (iii) synthesize a new theory to explain the results from Hayman's model and LPH, MPH, and HPH for GY, ED, EL, RE, KR, and KW.

MATERIALS AND METHODS

Inbred Line Selection and Crosses

Lines used in the study were from three maize heterotic groups (Fan et al., 2008, 2009; 2014): the tropical inbred line 'YML46' (Suwan-1 heterotic group) and five temperate inbred lines: 'Zheng58' and 'Ye107' (Reid) and 'Mo17', 'Chang7–2' and 'Dan598' (non-Reid). In the winter of 2009, YML46 was crossed with each of these five lines: Zheng58, Ye107, Mo17, Chang7–2, and Dan598 in Jinghong, Yunnan Province, to produce F_1 hybrids (Table 1). Seeds from at least 15 F_1 plants were harvested for each of the crosses. More than 100 F_1 plants from each of the five crosses, along with the six parental lines, were grown in 2010. For each F_1 cross, at least 15 F_1 plants were crossed to each parent to produce the BC_1 and BC_2 generations and 10 F_1 plants of each cross were selfed to produce the F_2 generation at Kunming, Yunnan province. Standard cross-pollination and selfing techniques were used. Five sets of P_1, P_2, F_1, F_2, BC_1, and BC_2 generations were thus available for evaluation.

Table 1. Six maize inbred lines, their heterotic groups, and crosses made.

Female line	Male lines	Heterotic group	Crosses
YML46	–	Suwan-1	–
–	Zheng58	Reid	Yes
–	Ye107	Reid	Yes
–	Mo17	Non-Reid (Lancaster)	Yes
–	Chang7–2	Non-Reid (Tangsipingtou)	Yes
–	Dan598	Non-Reid (LvdaRed Cob)	Yes

Field Experiment

In 2011, the seeds of the five sets of the P_1, P_2, F_1, F_2, BC_1, and BC_2 generations were planted at Lincang and Yuxi, Yunnan province. A randomized complete-block design with three replicates was used for the experiment. Standard field management (planting, weed control, application of fertilizer, insecticide, etc.) were applied at both locations. Plots of nonsegregating generations (P_1, P_2 and F_1) consisted of four rows and those of segregating generations (F_2, BC_1, and BC_2) consisted of 10 rows. Each row was 3 m in length and the distance between rows was 0.5 m. In the four-row plots, five plants per row from the middle two rows were chosen (labeled) and in the 10-row plots, five plants per row from the middle eight rows were chosen (labeled) and used for collecting data on GY, ED, EL, RE, KR, and KW. ED, EL, RE, and KR were measured on all ears from selected sample plants. Grain yield was obtained by weighing all kernels on the ear from selected individual plants and KW was estimated by randomly sampling 100 kernels from a pool of kernels from selected sample plants per plot.

The following genetic model for each plot mean was used (Bernardo et al., 1992; Lariepe et al., 2012; Checa et al., 2006):

$$Y_{ijklp} = u + L_i + R_j + C_k + G_l + CG_{kl} + LG_{il} + RC_{jk} + LCG_{ikl} + e_{ijklp}; \quad [1]$$

$$G_l = m + \alpha a + \beta d + \alpha^2 aa + 2\alpha\beta ad + \beta^2 dd, \quad [2]$$

where Y_{ijklp} is the observation for the i^{th} location, j^{th} replication, k^{th} cross, l^{th} generation, and p^{th} plot; μ is the general mean; L_i is the i^{th} location effect; R_j is the j^{th} replication effect; C_k is the k^{th} cross effect; G_l is the l^{th} generation effect; CG_{kl} is the interaction effect of the k^{th} cross and l^{th} generation; LG_{il} is the interaction effect of the i^{th} location and l^{th} generation; RC_{jk} is the interaction effect of the j^{th} replication and k^{th} cross; LCG_{ikl} is the interaction effect of the i^{th} location, k^{th} cross, and l^{th} generation; e_{ijklp} is the random error; m is the population (plot) mean; and α and β are the appropriate coefficients for the following pooled genetic effects: *a*, *d*, *aa*, *ad*, and *dd*. The relationships of different generations to the genetic effects are given below (Kang, 1994):

$$\overline{P_1} = m + a - 0.5d + aa - 0.5ad + 0.25dd\,; \quad [3]$$

$$\overline{P_2} = m - a - 0.5d + aa + 0.5ad + 0.25dd\,; \quad [4]$$

$$\overline{F_1} = m + 0.5d + 0.25dd\,; \quad [5]$$

$$\overline{F_2} = m\,; \quad [6]$$

$$\overline{B_1} = m + 0.5a + 0.25aa\,; \quad [7]$$

$$\overline{B_1} = m - 0.5a + 0.25aa\,. \quad [8]$$

Table 2. Analysis of variance for grain yield (GY), ear diameter (ED), ear length (EL), row number per ear (RE), kernel number per row (KR), 100-kernel weight (KW) in maize with the six-generation mean from five crosses at two locations.

Source	df	ED	EL	RE	KR	KW	GY
Location	1	0.693**	0.038	52.552**	58.482**	1.038	7608.001**
Replication	2	0.071	1.182	0.998	4.573	25.788	165.819
Generation	5	0.586**	51.382**	5.412**	305.100**	249.062**	14595.128**
Crosses	4	0.591**	15.980**	18.903**	38.466**	101.332**	890.312
Crosses × generation	20	0.185**	1.450	3.485**	11.342	19.491*	596.585
Location × generation	5	2.773**	25.461**	21.832**	160.377**	144.059**	16502.908**
Crosses × location	4	0.670**	15.791**	22.601**	25.292**	63.949**	642.717
Crosses × location × generation	20	0.164**	3.496**	3.343**	8.868	18.493*	733.425*
Error	118	0.046	1.254	0.586	7.010	10.424	385.186

* Significant at the 5% probability level.

** Significant at the 1% probability level.

The *a, d, aa, ad*, and *dd* effects can be obtained from the following equations (Hayman, 1958; Kang, 1994; Herman et al., 2011):

$$m = \overline{F_2}\,; \qquad [9]$$

$$a = \overline{B_1} - \overline{B_2}\,; \qquad [10]$$

$$d = -0.5\overline{P_1} - 0.5\overline{P_2} + \overline{F_1} + 4\overline{F_2} + \overline{B_1} + \overline{B_2}\,; \qquad [11]$$

$$aa = -4\overline{F_2} + 2\overline{B_1} + \overline{B_2}\,; \qquad [12]$$

$$ad = -0.5\overline{P_1} - 0.5\overline{P_2} + \overline{B_1} + \overline{B_2}\,; \qquad [13]$$

$$dd = \overline{P_1} + \overline{P_2} + 2\overline{F_1} + 4\overline{F_2} - 4\overline{B_1} - \overline{B_2}\,. \qquad [14]$$

LPH, MPH, and HPH for GY, ED, EL, RE, KR, and KW were calculated based on the following formulae:

$$\text{LPH} = \frac{\overline{F_1} - Low\_parent}{Low\_parent}\,; \qquad [15]$$

$$\text{MPH} = \frac{\left[\overline{F_1} - \left(\overline{P_1} + \overline{P_2}\right)\times 0.5\right]}{\left(\overline{P_1} + \overline{P_2}\right)\times 0.5}\,; \qquad [16]$$

$$\text{HPH} = \frac{\overline{F_1} - High\_parent}{High\_parent}\,. \qquad [17]$$

Heterosis, variance, regression, and correlation analyses were conducted using SAS (SAS Institute, 2005). The genetic parameters *a*, *d*, *aa*, *ad*, and *dd* were estimated using the macro SASQuant developed by Gusmini et al. (2007). SASQuant partitions *a*, *d*, and epistatic effects based on the Hayman's mean separation procedure and computes the SE for the estimates as the square root of their variances. SASQuant tests the hypothesis that the estimates are significantly different from zero by performing Fisher's *t*-test. The estimates of *a*, *d*, *aa*, *ad*, and *dd* are obtained by combining the means of generations with different sample sizes. Thus SASQuant can estimate genetic effects such as *a*, *d*, etc. and also provides the SE estimates and allows for statistical test of significance for the corresponding genetic effects. For ANOVAs for individual traits, plot means were used (Table 2 and Table 3), and the variances from location, replication, and family were portioned to reduce the error term for testing significance among generations. For computation of the genetic effects (Table 4), family means for each generation were used.

RESULTS

Analysis of Variance

The ANOVA results for GY, ED, EL, RE, KR, and KW are given in Table 2. Because the mean squares for generations of the studied traits were all significant and the mean squares for crosses for all traits, except GY, were also significant, estimates of *a*, *d*, *aa*, *ad*, and *dd* are reported for all traits by individual crosses.

Sum of Squares and R^2 for *a*, *d*, *aa*, *ad*, and *dd* for GY, and YC in the Five Crosses

The R^2 statistic was used to determine the relative importance of different genetic effects, as has been done in some earlier investigations (Bernardo et al., 1992; Cui et al., 2009). The mean squares for each trait for the five crosses are given in Table 3. To make R^2 comparable across different traits, R^2% was calculated for each trait as the percentage of the SS for each effect (*a*, *d*, *aa*, *ad*, and *dd*) divided by the SS of the generation in each cross. For example, the R^2% for the *a* effect for GY (the first value in the last column in Table 3) is shown in Eq.[18]:

$$R^2\% = \frac{df_a \times MS_a}{df_{\text{Gen}} \times MS_{\text{Gen}}} \times 100 = \frac{1\times 212.395}{5\times 3491.820}\times 100 = 1.217\,, \qquad [18]$$

where df_a is the degrees of freedom of *a*, MS_a is the mean square for *a*, df_{Gen} is the degrees of freedom for the generation and MS_{Gen} is the mean square for the generation.

The values of R^2% for each trait for the five crosses are also shown in Table 3. The R^2% values for some genetic effects were quite consistent for GY and EL across all five crosses. For GY, R^2% for *d* and *aa* accounted for more than

Table 3. Mean squares (MS) of additive (*a*), dominant (*d*), additive × additive (*aa*), additive × dominance (*ad*), and dominance × dominance (*dd*) for studied traits in five crosses and R^2 percentage (R^2%) of the sum of squares (SS) of *a*, *d*, *aa*, *ad*, and *dd* over the SS for the generation (Gen) in individual crosses.

									R^2%					
Cross†	Source	df	ED‡	EL	RE	KR	KW	GY	ED	EL	RE	KR	KW	GY
1	Gen	5	0.126	14.120**	1.250	66.765**	73.154**	3491.820**	–	–	–	–	–	–
	a	1	0.178	22.891**	4.780	61.732*	101.691*	212.395	28.254	32.424	76.480	18.492	27.802	1.217
	d	1	0.157	11.220*	0.200	46.682	59.367	4862.690*	24.921	15.892	3.200	13.984	16.231	27.852
	aa	1	0.197	30.075**	1.086	184.785**	192.397**	9177.720**	31.270	42.599	17.376	55.354	52.601	52.567
	ad	1	0.000	1.029	0.015	1.913	0.138	207.978	0.00	1.458	0.24	0.573	0.038	1.191
	dd	1	0.098	5.388	0.172	38.714	12.177	2998.300	15.556	7.632	2.752	11.597	3.329	17.173
2	Gen	5	0.017	11.276**	2.028**	30.658**	62.368**	1800.840**	–	–	–	–	–	–
	a	1	0.021	32.844**	0.588	0.159	10.301	387.138	24.706	58.255	5.799	0.104	3.303	4.30
	d	1	0.002	13.489*	3.389	64.738*	94.690*	2398.460	2.353	23.925	33.422	42.232	30.365	26.637
	aa	1	0.011	7.204	5.940**	61.399*	108.543*	2751.900	12.941	12.778	58.58	40.054	34.807	30.562
	ad	1	0.051	0.757	0.007	4.616	80.929	1272.540	60.0	1.343	0.069	3.011	25.952	14.133
	dd	1	0.001	2.086	0.215	22.378	17.374	2194.150	1.176	3.700	2.120	14.598	5.571	24.368
3	Gen	5	0.886**	17.012**	11.708**	136.164**	82.945**	6644.620**	–	–	–	–	–	–
	a	1	3.065**	13.772*	55.824	42.272	72.381	1992.290	69.187	16.191	95.360	6.209	17.453	5.997
	d	1	0.301	21.261*	0.426	258.474**	44.958	12064.300**	6.795	24.995	0.728	37.965	10.840	36.313
	aa	1	0.935**	47.86**	0.773	360.338**	265.525**	17685.200**	21.106	56.266	1.320	52.927	64.024	53.232
	ad	1	0.103	0.009	1.426	1.255	2.311	0.689	2.325	0.011	2.436	0.184	0.557	0.002
	dd	1	0.025	2.160	0.093	18.479	29.550	1480.630	0.564	2.539	0.159	2.714	7.125	4.457
4	Gen	5	0.065	8.472*	2.611**	78.097**	59.593**	2339.350*	–	–	–	–	–	–
	a	1	0.015	18.090*	5.740	57.931	103.817*	97.397	4.615	42.705	43.968	14.836	34.842	0.833
	d	1	0.277	4.069	1.155	105.146*	3.572	3035.690	85.231	9.606	8.847	26.927	1.199	25.953
	aa	1	0.022	13.692	1.585	149.656**	49.086	4224.980	6.769	32.323	12.141	38.326	16.474	36.121
	ad	1	0.012	0.156	2.569	0.337	49.619	537.373	3.692	0.368	19.678	0.086	16.653	4.594
	dd	1	0.001	6.352	2.004	77.414*	91.872*	3801.310	0.308	14.995	15.350	19.825	30.833	32.499
5	Gen	5	0.233**	6.300**	1.757**	38.785**	48.966**	2704.850**	–	–	–	–	–	–
	a	1	0.006	20.937**	1.956	70.169*	0.012	580.770	0.515	66.467	22.265	36.184	0.005	4.294
	d	1	0.873*	4.298	6.117	18.355	71.016*	6949.900**	74.936	13.644	69.630	9.465	29.006	51.388
	aa	1	0.255	3.114	0.096	79.669**	146.217**	5416.460*	21.888	9.886	1.093	41.082	59.722	40.050
	ad	1	0.002	1.456	0.452	4.466	23.818	258.234	0.172	4.622	5.145	2.303	9.728	1.909
	dd	1	0.028	1.697	0.163	21.265	3.767	318.903	2.403	5.387	1.855	10.966	1.539	2.358

* Significant at the 5% probability level

** Significant at the 1% probability level.

† Cross 1, YML46 × Zheng58; Cross 2, YML46 × Ye107; Cross 3, YML46 × Mo17; Cross 4, YML46 × Chang7-2; Cross 5, YML46 × Dan598.

‡ ED, ear diameter; EL, ear length; RE, row number per ear; KR, kernel number per row; KW, 100-kernel weight; GY, grain yield per plant.

20 and 30% of the total genetic variance, respectively, in all five crosses, suggesting that *d* and *aa* in the five crosses played an important role in determining GY. For EL, the sum of R^2% from *a* and *aa* accounted for more than 70% of total variance, suggesting that *a* effect genes, their interactions, or both played a major role in controlling EL. The consistent estimates obtained from five different crosses suggested that the genetic effects estimated from one study might be useable as reference in other studies and also implied that there might be common genes for controlling quantitative traits in these different lines or similar gene interactions in the crosses. The *a*, *d*, *aa*, *ad*, and *dd* genetic effects for ED, RE, KR, and KW differed greatly across the five crosses. For example, R^2% for *d* effects was about 80and 70% for ED for Suwan-1 × Chang7–2 and Suwan-1 × Dan598, respectively. In contrast, in Suwan-1 × Mo17 and Suwan-1 × Ye107, *d* effects only accounted for less than 10% for ED. Differences in R^2% values were also found for *a* effects among crosses for ED. The *a* effect accounted for about 70% of total variation in ED in the Suwan-1 × Mo17 cross, whereas in Suwan-1 × Chang7–2 and Suwan-1 × Dan598, *a* effects accounted for only about 5 and 0% of total variation, respectively. These results implied that R^2% for *a*, *d*, *aa*, *ad*, and *dd* effects in different crosses was clearly different for most of the quantitative traits.

Differences Relative to *a*, *d*, *aa*, *ad*, and *dd* Effects for GY, ED, EL, RE, KR, and KW among Five Crosses

Genetic effects for the six traits in the five crosses were estimated according to the model described in the Materials and Methods. The estimates of the *a*, *d*, *aa*, *ad*, and *dd* effects are given in Table 4. The results revealed several interesting points. First, *a*, *d*, *aa*, *ad*, and *dd* effects were

Table 4. Genetic effects of additive (*a*), dominance (*d*), additive × additive (*aa*), additive × dominance (*ad*), and dominance × dominance (*dd*) for the studied traits in five maize crosses.

Trait†	Parameter	Suwan-1 × Zheng58	Suwan-1 × Ye107	Suwan-1 × Mo17	Suwan-1 × Chang7–2	Suwan-1 × Dan598
ED	*a*	0.16 ± 0.09	−0.10 ± 0.06	0.29 ± 0.07**	−0.11 ± 0.07	−0.31 ± 0.08**
	d	0.42 ± 0.47	0.41 ± 0.37	0.48 ± 0.35	0.40 ± 0.40	0.20 ± 0.43
	aa	0.09 ± 0.35	0.43 ± 0.24	0.05 ± 0.25	0.16 ± 0.27	−0.25 ± 0.31
	ad	0.06 ± 0.16	−0.08 ± 0.13	−0.20 ± 0.13	−0.17 ± 0.14	−0.37 ± 0.14**
	dd	−0.01 ± 0.77	−0.58 ± 0.62	−0.73 ± 0.60	0.02 ± 0.68	0.45 ± 0.72
EL	*a*	1.63 ± 0.33**	1.14 ± 0.34**	0.94 ± 0.36**	1.85 ± 0.57**	1.47 ± 0.54**
	d	4.49 ± 1.89*	3.91 ± 1.82*	2.30 ± 1.92	3.33 ± 2.35	−0.45 ± 2.33
	aa	1.30 ± 1.33	1.55 ± 1.30	−1.60 ± 1.37	1.08 ± 1.84	−1.97 ± 1.82
	ad	0.49 ± 0.57	−0.42 ± 0.62	−0.02 ± 0.60	0.93 ± 0.78	0.36 ± 0.80
	dd	−2.90 ± 3.11	−1.11 ± 3.03	0.58 ± 3.20	−1.65 ± 4.00	4.11 ± 3.92
RE	*a*	0.75 ± 0.29**	−0.13 ± 0.23	1.55 ± 0.25**	−0.61 ± 0.25*	−1.42 ± 0.31**
	d	0.21 ± 1.57	−0.63 ± 1.28	1.91 ± 1.38	2.07 ± 1.36	1.87 ± 1.66
	aa	0.55 ± 1.15	0.81 ± 0.88	1.53 ± 1.95	2.33 ± 0.90*	1.14 ± 1.18
	ad	0.20 ± 0.56	0.08 ± 0.46	−0.48 ± 0.49	−1.54 ± 0.54**	−1.31 ± 0.54*
	dd	0.15 ± 2.58	−0.67 ± 2.13	−3.12 ± 2.32	−2.92 ± 2.33	0.10 ± 2.76
KR	*a*	4.18 ± 0.80**	0.15 ± 0.72	0.63 ± 0.86	3.67 ± 1.23**	3.93 ± 0.80**
	d	4.31 ± 4.50	10.20 ± 4.52*	9.50 ± 4.75*	11.51 ± 5.42*	1.00 ± 4.67
	aa	−3.27 ± 3.18	4.16 ± 2.82	−2.59 ± 3.37	2.63 ± 4.22	−3.98 ± 3.49
	ad	2.63 ± 1.46	0.06 ± 1.89	−1.31 ± 1.56	2.12 ± 1.88	2.22 ± 1.43
	dd	2.55 ± 7.41	−4.16 ± 7.67	1.74 ± 7.85	−0.80 ± 9.08	4.83 ± 7.46
KW	*a*	−2.00 ± 1.32	−2.15 ± 1.74	−0.01 ± 1.85	2.29 ± 0.96*	−1.18 ± 0.92
	d	4.92 ± 6.30	22.12 ± 6.79**	2.67 ± 6.94	−3.65 ± 4.83	−9.22 ± 6.92
	aa	−2.44 ± 4.77	15.00 ± 5.33**	−5.40 ± 5.42	−8.26 ± 3.53*	−15.40 ± 5.72**
	ad	0.75 ± 1.95	−1.65 ± 2.41	2.73 ± 2.53	6.15 ± 1.50**	−1.47 ± 1.58
	dd	−0.72 ± 10.47	−23.22 ± 11.72*	2.57 ± 12.16	15.99 ± 8.05*	17.60 ± 9.96
GY	*a*	17.21 ± 5.95**	−0.42 ± 6.28	9.03 ± 6.33	20.73 ± 5.84**	−1.00 ± 6.06
	d	39.14 ± 37.04	119.13 ± 32.92**	59.99 ± 33.96	28.97 ± 34.72	9.09 ± 34.80
	aa	−23.95 ± 25.89	78.12 ± 22.67**	−24.81 ± 24.39	−21.65 ± 24.54	−45.76 ± 25.31
	ad	16.81 ± 10.72	5.82 ± 11.66	−3.12 ± 10.26	22.71 ± 10.69*	−8.98 ± 10.53
	dd	44.25 ± 60.11	−99.27 ± 55.72	25.78 ± 56.18	65.05 ± 56.58	78.96 ± 56.41
Heterotic group‡		SR	SR	SNR	SNR	SNR

* Significant at the 5% probability level

** Significant at the 1% probability level

† ED, ear diameter; EL, ear length; RE, row number per ear; KR, kernel number per row; KW, 100-kernel weight; GY, grain yield per plant.

‡ SR, Suwan-1 heterotic group × Reid heterotic group; SNR, Suwan-1 heterotic group × non-Reid heterotic group.

different among crosses. For example, for ED, *a* effects were significant in Suwan-1 × Mo17 and Suwan-1 × Dan598 but were not significant in the other three crosses. For RE, *a* effects were significant in all crosses, except in Suwan-1 × Ye107. For GY, EL, KR, and KW, different genetic effects were also observed among all five crosses. These results confirmed what we found with R^2%, namely that the genetic effects were different when they were estimated from different crosses. Second, *a* effects were significant in all five crosses for EL but the *d* effects were significant only in Suwan-1 × Zheng58 and Suwan-1 × Ye107 and not significant in the other three crosses. Because Suwan-1 × Zheng58 and Suwan-1 × Ye107 represented crosses between the Suwan-1 heterotic group and the Reid heterotic group and the other three crosses were between the Suwan-1 heterotic group and the non-Reid heterotic group, the magnitude of the significant *d* effects might be determined by their genetic background and thus might be used as a basis for grouping maize lines into heterotic groups. This result suggested that differences in genetic effects among crosses from different maize heterotic groups might be another method for distinguishing between maize heterotic groups in addition to using the specific combining ability method (Fan et al., 2009, 2014) and enzymatic activities (Cai et. al., 2012). Third, when we examine significant genetic effects only in Table 4, we find that the signs for *d* and *dd* for KW in Suwan-1 × Ye107 and the signs for *a* and *aa* for KW in Suwan-1 × Chang7–2 were different. These results suggested that the genes controlling KW are likely to be complementary and dispersed in the two parents in these two crosses.

Correlation between Three Types of Heterosis (LPH, MPH, and HPH) and the Five Genetic Effects for the Six Traits in Five Crosses

To discover if any genetic effect (i.e., *a*, *d*, *aa*, *ad*, and *dd*) contributed significantly to heterosis (i.e., LPH, MPH, and HPH), correlation coefficients between the three types of heterosis and *a*, *d*, *aa*, *ad*, and *dd* effects for each of the six traits were calculated. No significant relationships (data not given) were found between the three types of heterosis (i.e., LPH, MPH, and HPH) and the five genetic parameters (i.e., *a*, *d*, *aa*, *ad*, and *dd*) for all the studied traits. This result suggested that heterosis (i.e., LPH, MPH, and HPH) is not contributed by a specific genetic effect and that heterosis in different crosses may be contributed by a major genetic effect (i.e., the effect with the highest R^2%) and several other minor genetic effects (i.e., effects with a relatively low R^2%) (Table 3).

Heterosis Analysis of the Six Traits in the Five Crosses

When we counted the number of crosses where LPH was negative (LPH < 0) and the number of crosses where HPH was positive (HPH > 0) in the five crosses at two locations, some interesting results were observed. First, negative LPH existed for some traits in some crosses, suggesting that 'underdominance heterosis' existed for these traits, since LPH reflects the difference between F_1 and the low parent. Second, positive HPH was also observed for some traits in some crosses. These results represented 'overdominance heterosis', as HPH is calculated from the difference between F_1 and the high parent. Third, the number of crosses with negative LPH and HPH variants and those with positive LPH and HPH variants were different for the same crosses in different locations or environments. These results implied that the environment influenced whether heterosis was attributable to dominance, underdominance or overdominance. Fourth, the lowest LPH was −22.2 and the highest HPH was 211.7. These results suggested greater potential use of overdominance than underdominance for certain traits.

DISCUSSION

Genetic Effects may Differentiate Among Maize Heterotic Groups

Specific combining ability is a widely accepted criterion for classifying maize heterotic groups (Hallauer and Miranda, 1988; Fan et al., 2008, 2014). Two enzymes, phosphorenolpyruvate carboxylase and ribulosebisphosphate carboxylase (Cai et al., 2012), could possibly be responsible for differentiating the Suwan-1 heterotic group from the Reid and non-Reid groups. The current study showed that the *d* effect was significant in the Suwan-1 × Reid crosses but nonsignificant in the Suwan-1 × non-Reid crosses. These results suggested that besides specific combining ability (Fan et al., 2008, 2009, 2014) and enzyme activity (Cai et al., 2012), genetic effects estimated from the Hayman model may be used as another basis for differentiating between or among maize heterotic groups. However, because only the *d* effect was different among heterotic groups for EL only in the five crosses, research with additional crosses and additional traits would be needed to confirm this interesting finding.

A Genetic Model to Explain the Differences in *a*, *d*, *aa*, *ad*, and *dd* Variances and Effects among Crosses

Different consistent trends or patterns of R^2% values for genetic effects were not found among crosses between Suwan-1 and Reid (i.e., Suwan-1 × Zheng58 and Suwan-1 × Ye107) and between Suwan-1 and non-Reid lines (i.e., Suwan-1 × Mo17, Suwan-1 × Chang7–2, and Suwan-1 × Dan598) for the studied traits (Table 3). These results suggested that the differences in the variances of the five genetic effects among crosses were cross-dependent. Because YML46 was used as the female parent in all five crosses, the differences in variances relative to the five genetic effects would seem to be dependent on how the genes in YML46 interacted with genes in the other five lines. Then the question arises as to why do *a*, *d*, *aa*, *ad*, and *dd* effects change when YML46 is crossed with different lines. Our answer to this question is that there must be some different genes in the other lines that interact with the genes in YML46 differently in different crosses.

We know that a quantitative trait is usually controlled by multiple genes, that these multiple genes could be either present or absent in any given line, and that the expression of the gene present in a cross may be activated or suppressed by interaction between genes or gene products (i.e., enzymes, inhibitors, and other bioproducts) from the other line in the cross (different crosses will have different internal environments). Thus the expression level (or hybrid performance) for the trait would be determined by whether the absent gene controlling a trait in one line can be compensated for by the corresponding present gene in the other line and how the corresponding present genes controlling the same trait in two lines interact with each other in different crosses. These interactions between genes from two lines may further be influenced by the plant's internal environment and external environment (i.e., weather, nutrients, various stresses, etc.). The external environment usually changes a plant's internal environment. The presence or absence of genes has been commonly reported in maize (Fu and Dooner, 2002; Guo et al., 2004; Springer and Stupar, 2007). Enzymatic activities can be suppressed or activated by gene products in a plant through feedback inhibition (van der Linde et al., 2012),

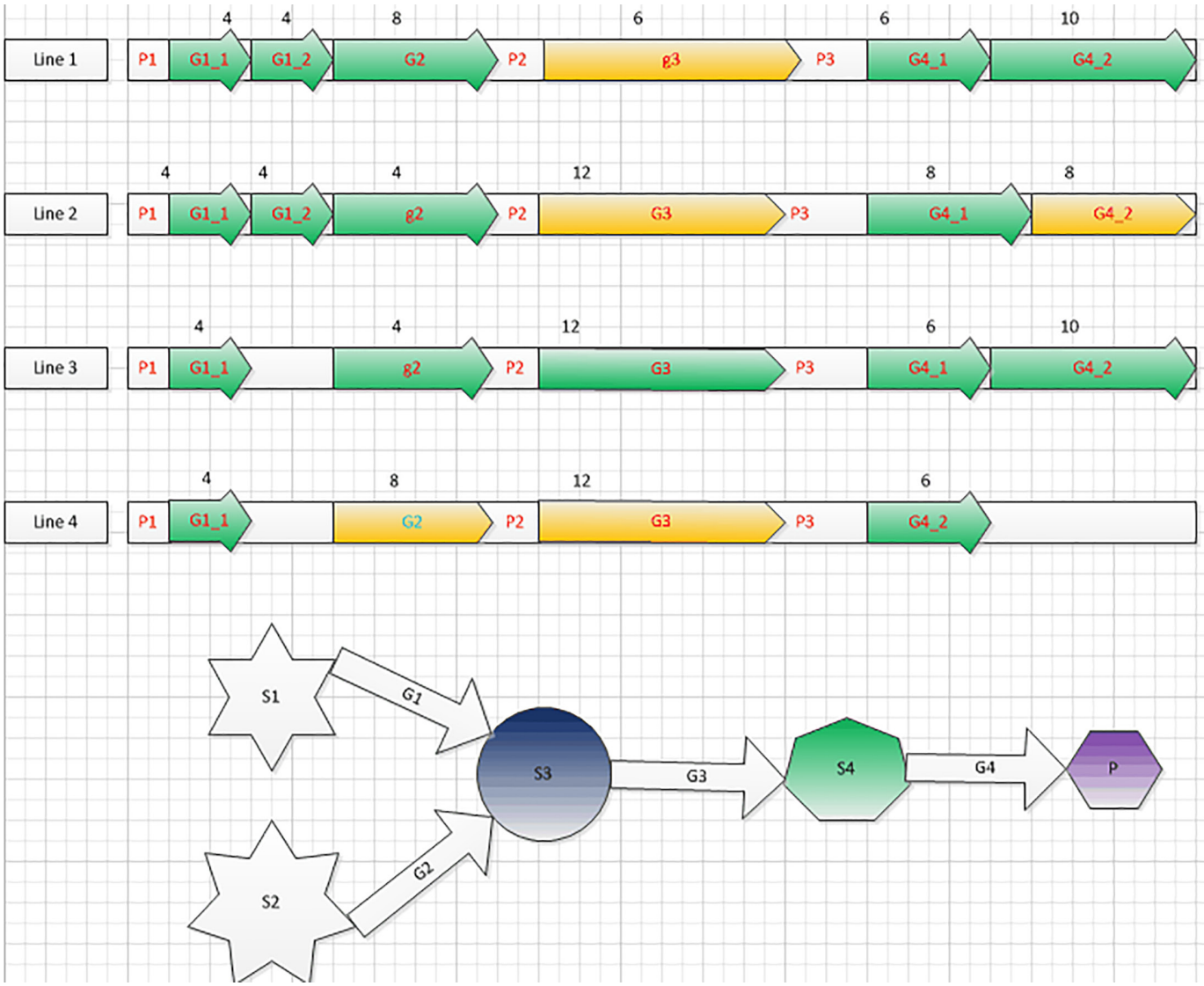

Figure 1. Four fictitious lines (Line 1 to Line 4) with genes, substrates, or final product (P). P1, P2, and P3 represent three promoters and G1, G2, G3, and G4 denote different genes controlling a trait. G1_1 and G1_2 are similar copies of the G1 gene that produces same substrate, Substrate 3 (S3) from either Substrate 1 (S1) or Substrate 2 (S2). Similarly, G4_1 and G4_2 are copies of G4 that produced P from the same substrate, Substrate 4 (S4). S4 was produced from S3 by enzyme coded by G3. The numbers above each gene copy represent the units of the outputs obtained via the enzymes coded by the corresponding genes.

by the influence of environmental factors (Bardzik et al., 1971), or by both combined.

A genetic model called LCG is postulated to explain our experimental results. Figure 1 represents an attempt at how we can use LCG model to illustrate the differential attainment of *a*, *d*, *aa*, *ad*, and *dd* effects in different crosses via complementary functions between the present and absent alleles of a gene and coexpression of both present alleles of a gene. Four fictitious lines are used for this illustration. Three promoters and four genes for controlling a trait are assumed in all four lines (Fig. 1) and the output quantities of the products from the four genes are assigned different values (placed above the gene; Fig. 1). Furthermore, the gene, highlighted in yellow, produces an enzyme inhibitor or any bioproduct that can suppress the expression of the corresponding gene, highlighted in green. This effect is similar to how a dominance or pseudo-overdominance effect gene works. The final product attainable by a line will be based on the smallest output (limiting factor or bottleneck) in the bioprocess produced by the enzymes coded from the four genes and how the enzymes interact with each other within the plant's environment. Based on the LCG model, the alleles of multiple linked genes are coexpressed. Different genes can code for either enzymes or inhibitors and either carbohydrate or lipids, etc. The interaction of all related bioproducts in a plant will determine the expression level of a trait. Because alleles are coexpressed, the units of the products of a cross are calculated by averaging the gene outputs of its two parent lines. The lowest number of units of a product from a gene will determine the output of the cross. Detailed calculations with the LCG model can

Table 5. Units of products† produced by enzymes coded by different genes with four assumed lines and their crosses.

Entry†	Female	Male	Cross	G1 + G2‡	G3	G4	Theories
Line1 × Line2	6	12	9	(16 + 12)/2 = 14	(6 + 12)/2 = 9	(6 + 16)/2 = 11	Incomplete dominance
Line1 × Line3	6	8	3	(16 + 8)/2 = 12	(6 + 0)/2 = 3	(16 + 16)/2 = 16	Underdominance
Line1 × Line4	6	6	9	(8 + 12)/2 = 10	(6 + 12)/2 = 9	(16 + 6)/2 = 11	Overdominance
Line2 × Line3	12	8	6	(12 + 8)/2 = 10	(12 + 0)/2 = 6	(16 + 6)/2 = 11	Underdominance
Line2 × Line4	12	6	10	(8 + 12)/2 = 10	(12 + 12)/2 = 12	(16 + 6)/2 = 11	Incomplete dominance
Line3 × Line4	8	6	6	(4 + 12)/2 = 8	(12 + 0)/2 = 6	(16 + 6)/2 = 11	Dominance

† Line 1, Line2, Line 3, and Line 4 can produce 6, 12, 8, and 6 units of the final product, respectively, according to the least enzyme capability assumption. For example, for Line 1, G1_1, G1_2, and G2 can produce 16 units of Substrate 3 (S3), G3 can produce 6 units of Substrate 4 (S4), and G4_1 and G4_2 can produce 16 units of the final product. Because G3 only produces 6 units of S4 (the lowest amount of units), final product will only be 6 units regardless of how many more units can be generated by other genes. The values under G1_G2, G3, and G4 are the units of substrate or final products the genes can produce in the cross. For example, in the Line2 × Line3 cross, G1 + G2 can produce 12/2 (from Line 2) + 8/2 (from Line 3) = (12 + 8)/2 = 10 units of S3. Because the product of G3 from Line 2 suppresses or inhibits the enzyme coded by G3 from Line 3, only 12/2 = 6 units of S4 from Line 2 are produced. Again, because G4_2 from Line2 suppresses G4_2 from Line 3, only (18 + 6)/2 = 11 units of the final product can be produced. In this cross, the lowest amount of S4 is 6 units. That number is the final product that can be produced by the cross. The same procedure and logic are used for computing the final product each cross can produce. These are the main theories for explaining the various types of heterosis observed in each cross.

‡ G1, G2, G3, and G4 are four genes that produce enzymes. G1_1 and G1_2 are two copies of the G1 gene. Similarly, G4_1 and G4_2 are two copies of the G4 gene.

be found in Table 5. The results in Table 5 show that the trait values are greatly different among different crosses and explain quite well why different *a*, *d*, *aa*, *ad*, and *dd* variances and effects can be obtained in different crosses.

A Genetic Model to Explain All Types of Heterosis

A negative LPH tells us that a trait value in a hybrid is lower than that of the low parent in a cross; in genetics, it indicates negative dominance (i.e., underdominance) or a pseudo-overdominance gene action. A positive HPH indicates that the hybrid trait value is higher than that of the high parent in a cross; in genetics, it indicates positive dominance (overdominance) or a pseudo-overdominance gene action. Negative LPH and positive HPH were observed for several traits in several crosses and were different for different traits at different locations (Table 6), suggesting that heterosis might be different because of individual genes' expression or differential gene interactions in different crosses in different environments. If we calculate LPH and HPH with fictitious data from Table 5, dominance, incomplete dominance, underdominance and overdominance can be determined based on the LPH and HPH values. As the trait values of the male parent, the female parent and the cross are based on the LCG model and the dominance, incomplete dominance, underdominance, and overdominance genetic phenomena are all covered under the heterosis calculated from these trait values, the LCG can be a single improved genetic model for explaining all types of heterosis (i.e., dominance, incomplete dominance, underdominance, and overdominance). With the LCG model, the alleles in a gene are coexpressed and multiple genes determine the expression of a quantitative trait, overdominance is definitely possible and the F_2 generation for a quantitative trait will be normally distributed. Because gene interactions (suppression or complementary) in a cross may not be the same in a single line, no inbred line will have a performance as high as that of a hybrid.

Table 6. Statistics on the numbers of crosses with low-parent heterosis (LPH) and high-parent heterosis (HPH).

Location	Trait†	No. of crosses with LPH < 0	Min. LPH in the five crosses	No. of crosses with HPH > 0	Max. HPH in the five crosses
Linchang	ED	3	–17.7	0	–11
	EL	0	4.6	5	11.2
	RE	3	–22.2	0	–15
	KR	1	–0.7	4	13.3
	KW	0	7.1	3	16.2
	GY	3	–9.8	1	13.1
Yuxi	ED	0	21.9	5	28.4
	EL	0	46.6	3	28
	RE	0	9.8	4	7.3
	KR	0	49	5	67.5
	KW	0	70.2	5	59.3
	GY	0	233.4	5	211.7

† ED, ear diameter; EL, ear length; RE, row number per ear; KR, kernel number per row; KW, 100-kernel weight; GY, grain yield per plant.

Multiple present and absent genes are assumed for controlling qualitative traits in the new theory. This assumption is supported by results from previous studies (Holton and Cornish, 1995; Selinger and Chandler, 1999). Holton and Cornish (1995) summarized that there were eight regulatory genes involved in anthocyanin biosynthesis and one new gene (*pac1* or *pale aleuronic color1*) was added to the list by Selinger and Chandler (1999). All these genes either determine the timing and distribution or amount of anthocyanin pigmentation in maize. A study of anthocyanin variation in grapes (*Vitis vinifera* L.)(Fournier-Level et al., 2009) also found that genes controlling anthocyanin were clustered together and various combinations of isogenes in the cluster determined the complex quantitative trait for anthocyanin content in grapes.

CONCLUSIONS

The consistency of the magnitudes of the genetic variance for *a*, *d*, *aa*, *ad*, and *dd* among all crosses for GY and EL suggested that the results from this study might serve as a useful

reference for these two quantitative traits. However, the magnitude of the genetic variances for ED, RE, KR, and KW were different among the five crosses. The LCG model not only explains why *a*, *d*, *aa*, *ad*, and *dd* variances and effects might be different when a common line is crossed with other lines but it may also be a suitable genetic model for explaining heterosis. The *d* effect for EL was similar among the crosses between the Suwan-1 and Reid maize heterotic groups and was different in those between the Suwan-1 and non-Reid maize heterotic group, indicating that the *d* genetic effect could be another way of differentiating between Reid and non-Reid heterotic groups. The LCG model explains what the dominance and overdominance theories of heterosis can explain; in addition, it explains what dominance and overdominace models cannot explain. For example, the LCG model can explain why an inbred line cannot perform like a hybrid and why an F_2 population for a quantitative trait has a normal distribution.

Acknowledgments

The study was supported by the Yunnan Advanced Talent Introduction Project Foundation (2011HA001) and the Yunnan Scientific and Technological Project (2012BB012), China. We also thank Yunnan Science and Technology Leader Foundation (2014HA002) and National 863 Programs (2011AA10A103-2). The authors thank Prof. Li Xuezhi (Lincang Institute of Agricultural Science of Yunnan province) for his help in data collection for this study.

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