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Analysis of genotype-environment interactions from a genome-wide survey of quantitative trait loci in a barley population

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ABSTRACT

The presence of genotype-environment interactions (GE) leads to the imperfect genetic correlation between the measurements of the same trait in different environments, thereby limiting the ability of plant breeders to identify superior breeding lines or best cultivars across the environments. We analyzed genetic correlations and distributions of quantitative trait loci (QTL) controlling yields over pairs of 16 environments for a doubled haploid (DH) population of barley (*Hordeum vulgare* L.) derived from the cross between two six-row cultivars, Steptoe and Morex. We examined if the imperfect genetic correlation was associated with the frequency of QTLs that were concurrently detected in pairs of environments. Each of 120 environment pairs would fall into one of the four Scenarios arranged in the 2 × 2 two-way contingency table. These four Scenarios are: high (≥0.6) genetic correlation with QTL concurrence (scenario A), high genetic correlation without QTL concurrence (scenario B), low (<0.6) genetic correlation with QTL concurrence (scenario C) and low genetic correlation without QTL concurrence (scenario D). The numbers of environment pairs under scenarios A, B, C and D were 9, 8, 27 and 76, respectively. Further partitioning of the covariance due to individual concurrent QTLs confirmed the expected occurrence of scenarios A and D and it also enabled us to explain scenarios B and C. Scenario B was more likely due to the cumulative effect of undetectable concurrent QTLs with small effects and/or linked QTLs. Scenario C was likely due to the canceling effect of concurrent QTLs and/or linked QTLs with opposite signs. This study stressed the need to jointly examine contributions of all QTLs to the magnitude of genetic correlation between environments for understanding the nature of GE.

KEYWORDS: genotype-environment interaction, genetic correlation between environments, concurrent quantitative trait loci, *Hordeum vulgare* L.

ABBREVIATIONS

CIM, composite interval mapping; DH, doubled haploid; GE, genotype-environment interaction; MIM, multiple interval mapping; QTL, quantitative trait loci; RFLP, restriction fragment length polymorphisms

INTRODUCTION

Differential performance of genotypes across environments is known as genotype-environment interactions (GE). Quantitative genetic theory [1]

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describes the importance of GE in terms of the genetic correlation between measurements of the same trait in a pair of environments. The complete absence of GE would indicate a perfect genetic correlation between the two environments. In other words, nearly all genes or QTLs controlling the trait are concurrently expressed in the two environments. Conversely, the presence of GE results in an imperfect genetic correlation, suggesting that not all genes or QTLs are concurrently expressed in the two environments. Zero correlation does not necessarily imply a total lack of gene concurrence but could result from the cancellation of cumulative positive and negative genic effects.

This quantitative genetic perspective of GE may be examined from a genome-wide survey of quantitative trait loci (QTLs) in a mapping population that is phenotypically evaluated across different environments as well. The same QTLs that are detected in two or more environments can be used as surrogates for gene concurrence to determine how such QTLs contribute to genetic correlations between the environments. The status of genetic correlation and QTL concurrence can be conveniently arranged in the 2 × 2 two-way contingency table (denoted as Scenarios A, B, C and D). A perfect genetic correlation occurs if all QTLs are concurrent in the environments (Scenario A) or a zero genetic correlation occurs if no QTL is concurrently expressed (Scenario D). However, effects of individual concurrent QTLs can be positive or negative and the cumulative effect of several such QTLs may result in a negligible genetic correlation between the environments (Scenario B). Additionally, a strong genetic correlation may occur without any QTLs concurrence between the environments (Scenario C). The objective of this paper is to examine these expected relationships in detail through the analysis of the distribution of QTLs identified for grain yield measured across 16 environments using a doubled haploid (DH) population of barley (*Hordeum vulgare* L.).

MATERIALS AND METHODS

Description of barley data set

The DH mapping population was developed from a cross between two six-row barley cultivars, Steptoe and Morex by the Oregon State University Barley Breeding Program as part of the North American Barley Genome Mapping Project (NABGMP; *http://wheat.pw.usda.gov*) and has been described in detail by Hayes *et al.* [2] and others [3, 4, 5]. Here we recapture the essential details of population development, agronomic and malting quality evaluation and linkage mapping.

Steptoe is a high yielding, broadly adapted six-row feed barley whereas Morex is a Midwestern six-row cultivar and has been the American malting industry standard. Prior to population development, a single plant from each cultivar was chosen and self-pollinated to provide the plants used for the cross to ensure that the plant material was homozygous and homogeneous. A population of 310 DH lines was originally developed from the F_1 by the *Hordeum bulbosum* technique [6]. The final set of 150 lines was selected at random from this array for genotyping and phenotypic evaluation at a total of 16 environments encompassing different locations with different soil moisture conditions during 1991 and 1992. These test environments were: 1. Crookston, Minnesota (1992), 2. Ithaca, New York (1992), 3.Guelph, Ontario (1992), 4. Pullman, Washington (1992), 5. Brandon, Manitoba (1992), 6. Outlook, Saskatchewan (1992), 7. Goodale, Saskatchewan (1992), 8. Saskatoon, Saskatchewan (1992), 9. Tetonia, Idaho (1992), 10. Bozeman, Montana (irrigated, 1992), 11. Bozeman, Montana (1992), 12. Aberdeen, Idaho (1991), 13. Klamath Falls, Oregon (1991), 14. Pullman, Washington (1991), 15. Bozeman, Montana (irrigated, 1991), and 16. Bozeman, Montana (1991). We focused on the analysis of the yield data because it was measured in all 16 environments.

The procedure for the analysis of restriction fragment length polymorphisms (RFLP) markers including plant DNA isolation, prehybridization and hybridization was given in Kleinhofs *et al.* [3]. A total of 223 RFLP makers were obtained. Linkage analysis mapped these markers onto seven barley chromosomes covering a total genetic distance of 1221.9cM. Of the 223 markers, 37, 37, 31, 33, 29, 22 and 34 makers were mapped on chromosomes 1, 2, 3, 4, 5, 6 and 7 covering a total mapping distance of 169.7, 180.5, 185, 177, 151.2, 156.6 and 201.9cM, respectively.

QTL detection

We conducted QTL mapping for grain yield using Windows QTL Cartographer Version 2.0 that is developed at the North Carolina State University (*http://statgen.ncsu.edu/qtlcart/*). We used composite interval mapping (CIM) to detect additive and dominance effects at individual QTLs and multiple interval mapping (MIM) to detect epistatic effects between QTLs [7, 8]. The threshold for declaring significant QTLs was set at a LOD score of ≥3.0. In this paper, we focused on detection of QTLs for grain yield and their use for elucidating the contributions of QTL concurrence to the genetic correlation between a pair of environments.

Correlation and covariance analysis

To determine the presence of GE, the null hypothesis of perfect genetic correlation between environments ($\rho = 1$) was first tried. GE was absent if the correlation was perfect but present if the correlation was imperfect ($\rho < 1$) [1]. A perfect genetic correlation would imply that all genes or QTLs were concurrently expressed in the two environments [1]. Given that the marker density in the current data set could not cover all possible QTLs controlling grain yield, particularly QTLs with small effects, it was unrealistic to expect the complete QTL concurrence between any two environments. For this reason, we considered a partial QTL concurrence as a null hypothesis for the significance of correlation test. Following Robertson [9], we considered a null hypothesis of correlation $\rho \geq 0.6$ as a partial but high level of QTL concurrence. The alternative hypothesis of $\rho < 0.6$ would imply that the QTL sharing was below the acceptable level. Accordingly, the acceptance of the null hypothesis ($\rho \geq 0.6$) would suggest a high-to-perfect correlation between the environments. A rejection of the null hypothesis would suggest a negligible to no correlation between the environments.

A common QTL was counted if it was concurrently detected in a pair of environments. Thus, the total number of concurrent QTLs was obtained from the counts for all possible pairs of 16 environments (a total number of pairs are 120 = (16×15)/2). Covariances and correlations between the means of DH lines for individual concurrent QTLs were computed using SAS PROC GLM with the MANOVA option [10]. Strictly speaking, these are not genetic covariances and correlations because the mean of a DH line in our test was the sum of genetic effect and residual effect divided by the number of replications. With a sufficiently large number of replications, these covariances and correlations would asymptotically approach the genetic covariances and correlations [11].

The portion of the total genetic covariance between a pair of environments due to a concurrent QTL was calculated as the sum of corrected cross-products between two phenotypic values across four marker classes for two flanking markers. This calculation assumed that there was no other QTL to confound the contribution of the target QTL to the total genetic covariance. In the present study, this assumption would seem reasonable as all the mapped QTLs were fairly spaced. Averages of mapped genetic distances were 4.7, 5.0, 6.2, 5.5, 5.4, 7.5 and 6.1cM per interval between two adjacent markers on chromosomes 1, 2, 3, 4, 5, 6 and 7, respectively.

RESULTS

Distributions of yield QTLs across genome and environments

Of the 39 QTLs detected from all 16 environments, 28 were detected only in a single environment and 11 were concurrently detected in two or more environments. Most QTLs were mainly concentrated on chromosomes 2, 3 and 7, and the remaining QTLs were distributed on the other chromosomes (Fig. 1). There were 2, 12, 8, 3, 4, 3 and 7 QTLs on chromosomes 1, 2, 3, 4, 5, 6 and 7, respectively. The numbers of QTLs varied considerably from zero QTLs in environment 15 to 8 QTLs in environment 7, indicating strong QTL × environment interactions.

With LOD score of >3.0, no epistatic QTL pairs were detected after scanning all 24753 possible pairs for the 223 markers for each of the 16 environments. With a less stringent criterion of LOD score of >2.0, a total of three epistatic QTL pairs were detected for yield, two in environment 5 and the third one in environment 12. The first two epistatic QTL pairs were distributed on three

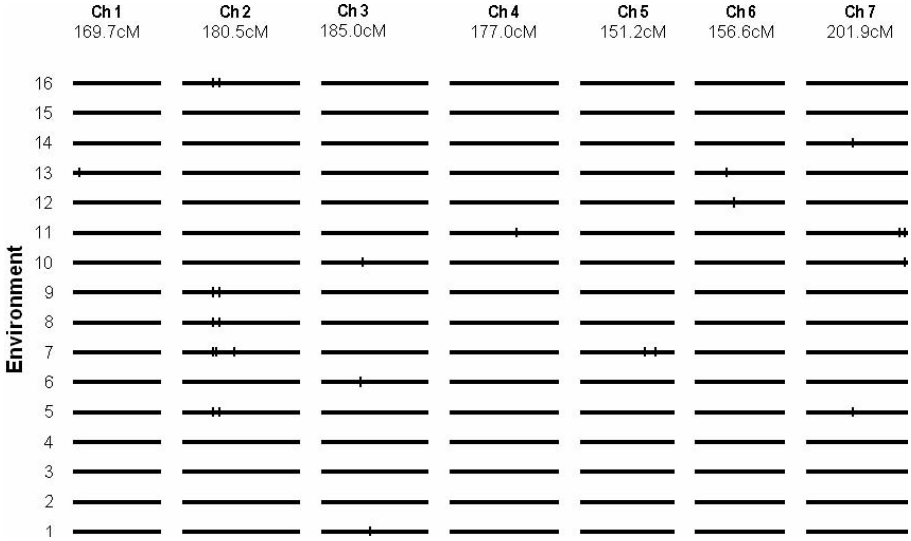

Figure 1. QTL distribution on seven chromosomes of barley for yield of doubled haploid lines evaluated across 16 environments in North America.

different chromosomes: Ch1 (110.6 cM) × Ch3 (53.6cM) and Ch3 (163.1cM) × Ch7 (91.7cM). Interestingly, there were no significant main effects of individual QTLs when these two epistatic pairs were considered separately in some of the 16 environments. In contrast, the two QTLs involved in the third epistatic pair, both on the second chromosome, Ch2 (40.3cM) × Ch2 (59.9cM), had significant main effects with a LOD score of >3.0 in individual environments.

Estimates of genetic correlations between 120 pairs of environments and the distribution of 11 concurrent QTLs (*Q*1 to *Q*11) are given in Table 1. Of 120 environment pairs, 36 pairs had one to four concurrent QTLs and the remaining 84 pairs had zero concurrent QTLs. Seventeen out of the 120 environment pairs had genetic correlation estimates that were not significantly different from the hypothesized value of 0.6 while the remaining 103 pairs had the estimates that were significantly less than 0.6 (Table 2). With joint consideration of genetic correlation and QTL concurrence, the observed counts for the four Scenarios described earlier were 9 for Scenario A (high correlation with QTL concurrence), 8 for Scenario B (high correlation without QTL concurrence), 27 for Scenario C (low correlation with QTL concurrence) and 76 for Scenario D (low correlation without QTL concurrence) (Table 2). To some extent, the status of genetic correlation was associated with the status of QTL concurrence since the Chi-square test of the two-way contingency table given in Table 2 was significant ($\chi^2 = 4.96$; $P = 0.0259$).

The 11 concurrent QTLs identified for the 36 environment pairs (Table 1) were distributed only on three chromosomes: six QTLs on chromosome 2 at the positions of 28.0cM (*Q*1), 39.1cM (*Q*2), 48.0cM (*Q*3), 62.9cM (*Q*4), 64.9cM (*Q*5), and 71.7cM (*Q*6); four QTL on chromosome 3 at 49.3cM (*Q*7), 54.6cM (*Q*8), 56.1cM (*Q*9), and 58.1cM (*Q*10), and one QTL on chromosome 7 at 68.4cM (*Q*11) (Table 3).

Contributions of concurrent QTLs to genetic correlations

The details of the 11 concurrent QTLs that contributed to the genetic covariances and correlations between pairs of environments are given below.

Q1: This QTL was found in five environments (5, 7, 8, 9 and 16) with a positive QTL effect detected in environments 5, 7 and 8, but a negative effect in environments 9 and 16. The covariances due to this QTL among environment pairs varied from –0.07 for environments 5 and 9 to 0.09 for environments 9 and 16. A positive covariance would arise if the effects of a

Table 1. Correlation coefficients (above diagonal) between pairs of environments and QTLs (below diagonal) affecting grain yield in barley with LOD score of ≥3.0 that were commonly detected in various pairs of environments based on a composite interval mapping (CIM) analysis.

	1[a]	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1		0.24[b]	0.28	0.18	**0.43**	0.28	-0.02	0.11	**0.42**	**0.45**	0.18	**0.52**	0.10	**0.46**	0.39	0.38
2			0.14	0.29	0.24	0.11	0.04	0.11	0.22	**0.47**	0.21	0.12	0.19	0.14	0.26	0.15
3	*Q9*[c]	*Q7*		-0.21	0.12	0.17	-0.22	0.20	0.27	0.18	-0.23	0.38	0.26	0.38	0.23	0.38
4		*Q6*			0.34	0.02	0.27	0.32	0.10	0.17	0.26	-0.06	0.10	0.05	-0.06	-0.18
5		*Q10*				0.25	0.38	**0.47**	0.07	0.30	0.09	0.23	0.10	**0.54**	0.13	0.02
6							0.10	0.15	0.06	0.31	0.05	0.28	0.12	0.28	0.12	0.08
7		*Q6*		*Q4, Q6*	*Q1*			**0.52**	-0.23	-0.05	0.15	-0.14	0.08	0.03	-0.27	-0.32
8					*Q1, Q3*		*Q1*		-0.22	0.13	0.22	0.00	0.09	0.05	-0.23	-0.33
9	*Q9*				*Q1, Q2, Q3*		*Q1*	*Q1, Q3*		**0.46**	0.28	0.35	0.17	0.36	**0.55**	**0.64**
10		*Q10*			*Q10*						**0.42**	0.36	0.12	0.39	**0.47**	0.37
11		*Q6*		*Q6*			*Q6*	*Q5*				0.14	-0.10	0.10	0.25	0.11
12	*Q9*		*Q9*						*Q9*				0.13	0.41	0.38	**0.45**
13														0.04	0.11	0.13
14		*Q6*		*Q6*	*Q8, Q11*		*Q6*				*Q6*				0.40	**0.49**
15																**0.64**
16	*Q9*	*Q6*	*Q9*	*Q6*	*Q1, Q2, Q3*		*Q1, Q3*	*Q1, Q6*	*Q1, Q2, Q3, Q9*		*Q6*	*Q9*		*Q6*		

[a]The 16 environments are; 1. Crookston, Minnesota (1992), 2. Ithaca, New York (1992), 3.Guelph, Ontario (1992), 4. Pullman, Washington (1992), 5. Brandon, Manitoba (1992), 6. Outlook, Saskatchewan (1992), 7. Goodale, Saskatchewan (1992), 8. Saskatoon, Saskatchewan (1992), 9. Tetonia, Idaho (1992), 10. Bozeman, Montana (irrigated, 1992), 11. Bozeman, Montana (1992), 12. Aberdeen, Idaho (1991), 13. Klamath Falls, Oregon (1991), 14. Pullman, Washington (1991), 15. Bozeman, Montana (irrigated, 1991), 16. Bozeman, Montana (1991).

[b]Correlation coefficients between grain yields of pairs of environments; the values in bold are not significantly different from an assumed high genetic correlation of ≥0.6.

[c]QTLs that were detected in pairs of environments with LOD score of ≥3.0; *Q1* is located at 28.0cM on chromosome 2, *Q2* is located at 39.3cM on chromosome 2, *Q3* is located at 48.0cM on chromosome 2, *Q4* is located at 62.9cM on chromosome 2, *Q5* is located at 64.9cM on chromosome 2, *Q6* is located at 71.7cM on chromosome 2, *Q7* is located at 49.3cM on chromosome 3, *Q8* is located at 54.6cM on chromosome 3, *Q9* is located at 56.1cM on chromosome 3, *Q10* is located at 58.1cM on chromosome 3, *Q11* is located at 68.43cM on chromosome 7.

Table 2. Classification of environment pairs based on whether or not the pair has a genetic correlation of ≥0.6 and whether or not the pair has at least one concurrent QTL.

	Correlation ≥0.6	Correlation <0.6	Total
With concurrent QTL	9 (Scenario A) = (1 QTL in 6 pairs; 2 QTLs in 2 pairs; 4 QTLs in 1 pair)	27 (Scenario C) = (1 QTL in 21 pairs; 2 QTLs in 4 pairs; 3 QTLs in 2 pairs)	36 = (1 QTL in 27 pairs; 2 QTLs in 6 pairs; 3 QTLs in 2 pairs; 4 QTLs in 1 pair)
Without concurrent QTL	8 (Scenario B)	76 (Scenario D)	84
Total	17	103	120

concurrent QTL were of the same sign between a pair of environments but a negative covariance would arise if the effects of a concurrent QTL were of opposite signs between a pair of environments, a positive effect in one environment and a negative effect in the other. Consequently, a positive covariance due to the effect of *Q*1 was observed for environment pairs, 5 vs. 7, 5 vs. 8, 7 vs. 8 and 9 vs. 16 whereas a negative covariance was observed for environment pairs, 5 vs. 9, 5 vs. 16, 7 vs. 9, 7 vs. 16, 8 vs. 9, and 8 vs. 16. The estimated genetic correlations for the two environment pairs varied from -0.29 for environments 8 vs. 16 to 0.64 for environments 9 vs. 16.

***Q2*:** This QTL was detected in three environments (5, 9 and 16) with a positive effect in environments 9 and 16 and a negative effect in environment 5. This led to a positive covariance (0.15) between environments 9 and 16 and two negative covariances, one being between environments 5 and 9 (–0.12) and the other being between environments 5 and 16 (–0.09) with the estimated genetic correlations of 0.64, 0.07 and 0.02, respectively.

***Q3*:** This QTL was detected in four environments (5, 8, 9 and16) with a positive effect observed in environments 9 and 16 and a negative effect in environments 5 and 8. The positive covariances due to this QTL were found in two environment pairs, 9 vs. 16 and 5 vs. 8 but negative covariances were found in four pairs, 5 vs. 9, 8 vs. 9, 5 vs. 16 and 8 vs. 16. These covariances ranged from –0.07 for environments 5 and 9 to 0.09 for environments 9 and 16. The estimated genetic correlations for environment pairs varied from –0.33 (between environments 8 and 16) to 0.64 (between environments 9 and 16).

***Q4*:** This QTL was detected in two environments (4 and 7) with positive effects. The covariance due to this QTL was 0.01 with a genetic correlation of 0.27.

***Q5*:** This QTL was also detected in one pair of environments (8 and 11) with positive effects. The covariance due to this QTL was 0.01 with a genetic correlation of 0.22.

***Q6*:** This QTL was found in six environments (2, 4, 7, 11, 14 and 16) with a positive QTL effect in environments 2, 4, 7 and 11, and a negative QTL effect in environments 14 and 16. This resulted in a positive covariance in each of the following environment pairs, 2 vs. 4, 2 vs. 7, 2 vs. 11, 4 vs. 7, 4 vs. 11, 7 vs. 11, and 14 vs. 16, and a negative covariance in each of the following environment pairs, 2 vs. 14, 2 vs. 16, 4 vs. 14, 4 vs. 16, 7 vs. 14, 7 vs. 16, 11 vs. 14, and 11 vs. 16. These covariances ranged from –0.03 for environments 2 and 14 to 0.05 for environments 4 and 11. The estimated genetic correlations for environment pairs varied from –0.32 (between environments 7 and 16) to 0.49 (between environments 14 and 16).

***Q7*:** This QTL was detected in two environments (2 and 3) with positive QTL effects in both environments. The covariance due to this QTL was 0.05, with a genetic correlation of 0.14.

***Q8*:** This QTL was detected in one pair of environments (5 and 14) with positive effects. The covariance due to this QTL was 0.15. The

Table 3. Chromosomal locations of concurrent QTLs in environment pairs, genetic correlation and covariance between paired environments for grain yield in barley.

Shared QTL		d. f	Environment (effect)		Correlation coefficient	Covariance	
QTL	Chromosome(cM)		Environment_*i*	Environment_*j*		QTL	Total
Q1	Ch2(28.0)	133	5(-0.23)	7(-0.28)	0.381	0.0498	0.1643
			5(-0.23)	8(-0.23)	0.482	0.0388	0.1567
			5(-0.23)	9(0.35)	0.036	-0.0713	0.0220
			5(-0.23)	16(0.30)	0.019	-0.0529	0.0088
			7(-0.28)	8(-0.23)	0.524	0.0375	0.1572
			7(-0.28)	9(0.35)	-0.233	-0.0701	-0.1302
			7(-0.28)	16(0.30)	-0.285	-0.0514	-0.1216
			8(-0.23)	9(0.35)	-0.234	-0.0633	-0.0987
			8(-0.23)	16(0.30)	-0.294	-0.0459	-0.0946
			9(0.35)	16(0.30)	0.649	0.0878	0.3885
Q2	Ch2(39.3)	145	5(-0.29)	9(0.46)	0.067	-0.1151	0.0406
			5(-0.29)	16(0.36)	0.016	-0.0906	0.0077
			9(0.46)	16(0.36)	0.641	0.1528	0.3930
Q3	Ch2(48.0)	145	5(-0.26)	8(-0.33)	0.467	0.0542	0.1549
			5(-0.26)	9(0.48)	0.053	-0.0716	0.0311
			5(-0.26)	16(0.34)	0.022	-0.0652	0.0107
			8(-0.33)	9(0.48)	-0.247	-0.0576	-0.0989
			8(-0.33)	16(0.34)	-0.330	-0.0546	-0.1085
			9(0.48)	16(0.34)	0.637	0.0882	0.3695
Q4	Ch2(62.9)	144	4(0.19)	7(0.26)	0.272	0.0070	0.0980
Q5	Ch2(64.9)	144	8(0.20)	11(0.28)	0.221	0.0069	0.0816

Table 3 continued..

Q6	Ch2(71.7)	144	2(0.19)	4(0.22)	0.285	0.0497	0.1184
			2(0.19)	7(0.24)	0.035	0.0267	0.0164
			2(0.19)	11(0.25)	0.209	0.0497	0.1176
			2(0.19)	14(-0.23)	0.139	-0.0306	0.1171
			2(0.19)	16(-0.16)	0.154	-0.0023	0.0765
			4(0.22)	7(0.24)	0.288	0.0240	0.1034
			4(0.22)	11(0.25)	0.264	0.0480	0.1157
			4(0.22)	14(-0.23)	0.052	-0.0363	0.0345
			4(0.22)	16(-0.16)	-0.177	-0.0031	-0.0686
			7(0.24)	11(0.25)	0.147	0.0248	0.0715
			7(0.24)	14(-0.23)	0.031	-0.0113	0.0224
			7(0.24)	16(-0.16)	-0.315	-0.0033	-0.1358
			11(0.25)	14(-0.23)	0.098	-0.0279	0.0875
			11(0.25)	16(-0.16)	0.112	-0.0001	0.0588
			14(-0.23)	16(-0.16)	0.488	0.0046	0.3848
Q7	Ch3(49.3)	134	2(0.27)	3(0.22)	0.135	0.0497	0.0594
Q8	Ch3(54.6)	140	5(0.20)	14(0.69)	0.558	0.1534	0.4576
Q9	Ch3(56.1)	143	1(0.45)	3(0.22)	0.277	0.0556	0.1258
			1(0.45)	9(0.36)	0.419	0.1092	0.2726
			1(0.45)	12(0.84)	0.525	0.1708	0.4942
			1(0.45)	16(0.40)	0.383	0.1165	0.1955
			9(0.36)	12(0.84)	0.345	0.2368	0.3891
			9(0.36)	16(0.40)	0.642	0.1712	0.3925
			12(0.84)	16(0.40)	0.447	0.2497	0.3947
Q10	Ch3(58.1)	143	2(0.24)	5(0.19)	0.237	0.0423	0.1196
			2(0.24)	10(0.36)	0.466	0.0868	0.2576
			5(0.19)	10(0.36)	0.300	0.0498	0.1605
Q11	Ch7(68.4)	137	5(-0.27)	14(-0.53)	0.537	0.1361	0.4198

estimated genetic correlation between these two environments was 0.56.

***Q9*:** This QTL was detected in five environments (1, 3, 9, 12 and 16) with positive QTL effects in all environments. Thus, the covariances due to this QTL were all positive but ranged from 0.06 for environments 1 and 3 to 0.25 for environments 12 and 16. The estimated genetic correlations ranged from 0.28 for environments 1 and 3 to 0.64 for environments 9 and 16.

***Q10*:** This QTL was detected in three environments (2, 5 and 10) with positive QTL effects in all environments. The covariances due to this QTL were positive in all environment pairs: 0.04 for environments 2 and 5 with genetic correlation of 0.24, 0.09 for environments 2 and 10 with genetic correlations of 0.47, and 0.05 for environments 5 and 10 with genetic correlation of 0.30.

***Q11*:** This QTL was detected in two environments (5 and 14) with negative QTL effects in both environments. The covariance due to this QTL was 0.14 for this environment pair with a genetic correlation of 0.54.

DISCUSSION

In this study we demonstrated, through the analysis of a QTL mapping data set of barley ([2]; *http://wheat.pw.usda.gov*), how individual QTLs detected over two or more environments contributed to the genetic covariances and correlations between pairs of environments. We confirmed the majority of the same individual QTLs as found in several earlier studies using the same or similar datasets [e.g., 2, 3, 4, 5]. For example, Hayes *et al.* [2] reported seven QTLs affecting yield in five environments and we were able to confirm five out of the seven yield QTLs in the same five environments. Slight discrepancy in the number of detected QTLs between our study and the earlier studies might be due to the use of different statistical models and/or different numbers of test environments. We will now focus on discussing our main results regarding the four Scenarios describing the statuses of genetic correlations and concurrent QTLs between environments. There has been little relevant work done in the past on the topic.

The occurrence of Scenario A would be expected theoretically and thus its explanation is straightforward. In each of the environment pairs of Scenario A, there was at least one concurrent QTL which contributed appreciably to the total covariance. Two specific examples are shown in Fig. 2. First, the covariance between environments 12 and 16 due to the occurrence of *Q*9 was 0.25, accounting for 63.2% of the total genetic covariance (0.40) with a significant correlation coefficient of 0.45. Clearly, the correlation of this environment pair is largely due to the presence of *Q*9. Second, environments 9 and 16 were significantly correlated (r = 0.64) and four QTLs (*Q*1, *Q*2, *Q*3 and *Q*9) were detected in both environments (Table 3). The cumulative effect of these four QTLs accounted for 77.8% (19.8%, 15.4%, 1.2% and 41.4%, respectively) of the total genetic covariance (Fig. 2).

The occurrence of Scenario B (i.e., the presence of significant correlation without concurrent QTLs) is unexpected. It may be due to the presence of tightly linked QTLs, each affecting the same trait in a different environment, and/or by undetected QTLs with small effects. Considering two tightly linked QTLs, one being expressed in one environment while the other in the second environment, the contributions of these two QTLs to the genetic correlation would not be easily separated. For example, environments 9 and 10 had no concurrent QTLs (Fig. 3) but had a significant genetic correlation of r = 0.46 (i.e., not significantly different from the hypothetical value $\geq$0.6 for high level of QTL concurrence). Two QTLs, *Q*9 and *Q*10, were estimated to be 3.5cM apart from each other based on interval mapping of three flanking markers, *ABG399* (at 52.6cM), *BCD828* (at 56.1cM) and *MWG680* (at 58.9cM) on chromosome 3. However, *Q*9 was detected only in environment 9 whereas *Q*10 was detected only in the paired environment 10. In the covariance analyses, the covariances of these two QTLs were very similar with *Q*9 (at 54.6cM) and *Q*10 (at 58.1cM) accounting for 42.9% and 49.8% of total cross-products, respectively. Should the middle marker, *BCD828*, be ignored, the two tightly linked QTLs would behave as if they are one QTL and thus this new 'ghost' QTL would be detected in both environments. The covariance

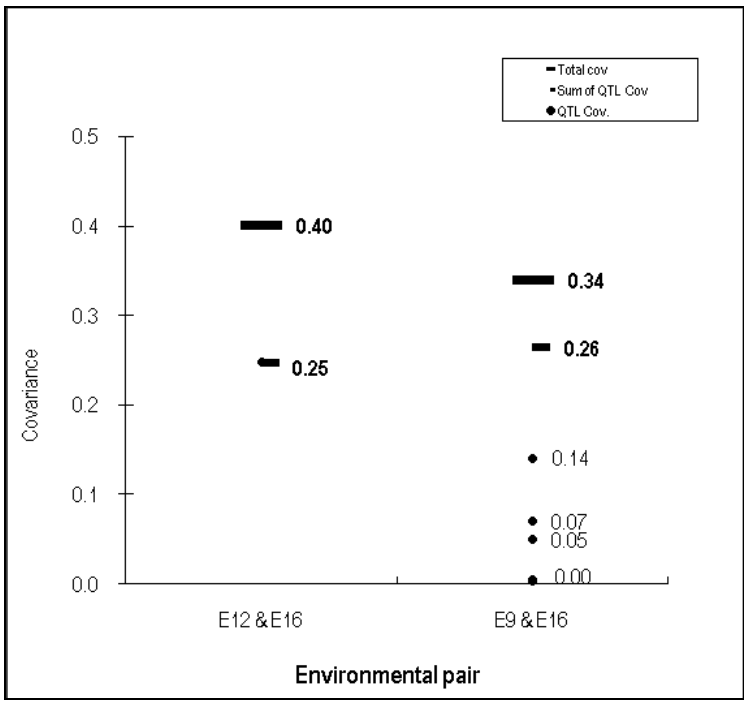

Figure 2. Two cases of Scenario A (high genetic correlation between a pair of environments with concurrent QTLs): Covariance of one concurrent QTL between environments 12 and 16 (left) and covariance of cumulative effects of four concurrent QTLs between environments 9 and 16 (right).

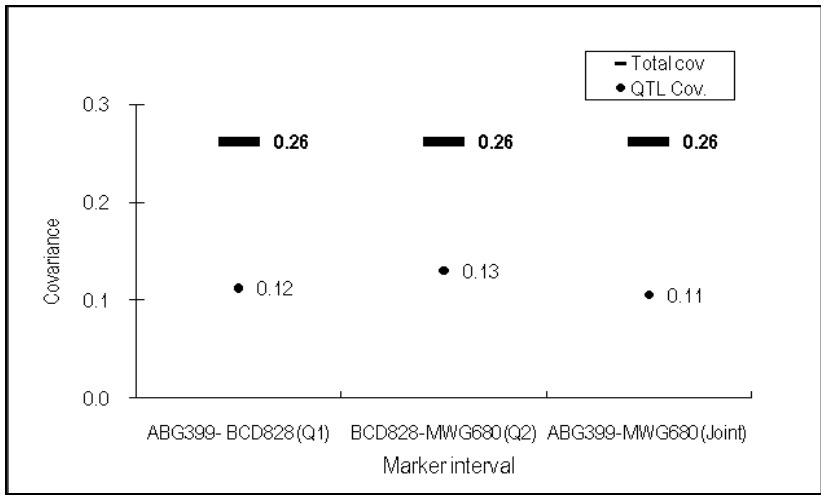

Figure 3. An example of Scenario B (high genetic correlation between a pair of environments without concurrent QTLs): Covariances of two tightly linked QTLs considered separately and jointly.

due to this 'ghost' QTL detected using the wider marker interval *ABG399-MWG680*, would account for 40.3% of total covariance between the environments 9 and 10. Thus, the joint contribution of the two tightly linked QTLs turns out to be very close to the contribution of the single QTL, thereby explaining the Scenario B (the effect of linked QTLs).

It is also possible that the presence of Scenario B was due to one or more undetected QTLs whose effects were too small to be detected in either or both environments for the size of mapping population (N = 150). For example, environments 15 and 16 were significantly correlated (r = 0.64) but without concurrent QTLs at the criterion of LOD score of ≥3.0. If the criterion was relaxed at a smaller LOD score (LOD score of <3.0), a QTL on chromosome 3 at 56.1cM would have been detected in the two environments and its effect would account for 46% of the total covariance.

The occurrence of Scenario C (i.e., non-significant correlation with the presence of concurrent QTLs) is likely due to cumulative effects of several concurrent or linked QTLs of opposite signs. For example, environments 11 and 14 were not significantly correlated (r = 0.09*)* even though in both environments, one QTL at LOD score of ≥3.0 with a negative covariance of -0.05, and two other QTLs detected at a less stringent criterion (LOD score of <3.0), one with a negative covariance (–0.05) and the other with a positive covariance (0.02) were detected. Additionally, a linked QTL pair on chromosome 2 at 52.6cM (environment 11) detected at a LOD score of <3.0 and at 54.6cM (environment 14) detected at a LOD score of ≥3.0) had a large positive covariance (0.14) (Fig. 4). Summing all these positive and negative covariances would lead to a negligible contribution to the total covariance.

The occurrence of Scenario D (i.e., non-significant correlation without concurrent QTL) is expected because it indicates that of the QTLs detected in the two environments, none was common in both environments, and therefore would result in no or weak genetic correlation. However, this does not preclude the possibility that the present study was unable to detect many QTLs with small effects of opposite signs.

This study has some limitations. First, the use of the mean yields of individual DH lines over replications did not allow us to estimate genetic variances and covariances entirely free from residual errors. As argued earlier, the use of phenotypic variances and covariances as surrogates of genetic counterparts would be reasonable if the number of replications is large. With the limited number of replications (3-4 in this study), there may be some residual errors left in the estimated phenotypic variances and covariances. The amount of left-over errors depends on the magnitudes of original residual errors in individual environments. Second, the heterogeneity of error variances over the environments causes unequal opportunities to detect QTLs across individual environments. In other words, it is more likely to detect QTLs in the environments with smaller error variances than in those with larger error variances. For this reason, the number of environment pairs with concurrent QTLs (36 out of 120 in this study) was probably underestimated. Third, the population size of N = 150 DH lines in this study is typical of what has been used (N = 100 – 150) for QTL mapping studies [12]. Such a population is considered small and leads to the Beavis effect [13, 14] including (i) fewer major QTLs being detected and (ii) overestimated effects of the detected QTLs. In order to minimize the Beavis effect and to increase the chance of detecting minor QTLs, a population size of N = 500 – 1000 has been recommended. Unfortunately, evaluating 500 to 1000 progenies for a cross of interest is generally impractical in most plant breeding programs [12].

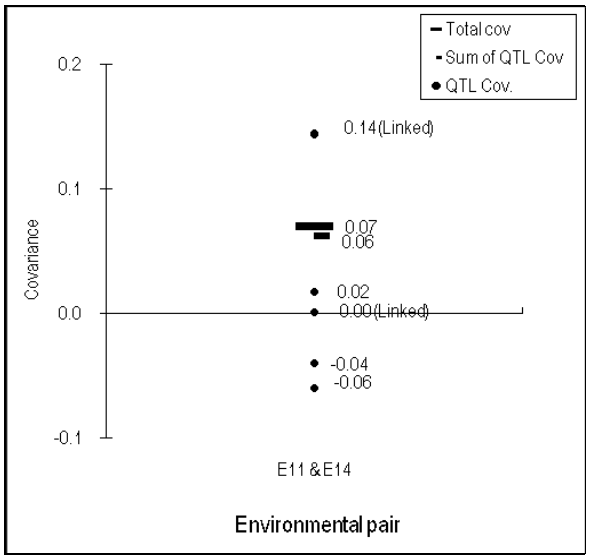

Figure 4. An example of Scenario C (no genetic correlation between a pair of environments but with concurrent QTLs): cumulative effect of covariances between environments 11 and 14 due to individual QTLs with effects of opposite signs.

In conclusion, our analysis of genome-wide QTL distributions across environments provides a new opportunity to dissect the complex GE structure. We demonstrate that genetic correlation between measurements of the same trait in different environments may or may not be explained by the presence of QTLs that are concurrently expressed in these environments. High genetic correlation could be due to the existence of concurrent QTLs, but lack of high genetic correlation may actually be due to the presence of many concurrent QTLs with effects of opposite signs. The further covariance analysis of concurrent QTL and linked QTL helped to explain how and which concurrent QTLs contribute to the total covariance. This study suggests that the genetic correlation between a pair of environments does not always reflect the existence of common QTLs expressed in the two environments or linked QTL because of the canceling of positive and negative effects of concurrent and linked QTLs. It seems more important to determine how concurrent QTLs and linked QTLs contribute to the strength of overall genetic correlation rather than to simply determine how many concurrent QTLs are uncovered in the test environments.

ACKNOWLEDGMENTS

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