Genotype-Environment Interactions at Quantitative Trait Loci Affecting Inflorescence Development in *Arabidopsis thaliana*

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ABSTRACT

Phenotypic plasticity and genotype-environment interactions (GEI) play a prominent role in plant morphological diversity and in the potential functional capacities of plant life-history traits. The genetic basis of plasticity and GEI, however, is poorly understood in most organisms. In this report, inflorescence development patterns in *Arabidopsis thaliana* were examined under different, ecologically relevant photoperiod environments for two recombinant inbred mapping populations (L*er* × Col and Cvi × L*er*) using a combination of quantitative genetics and quantitative trait locus (QTL) mapping. Plasticity and GEI were regularly observed for the majority of 13 inflorescence traits. These observations can be attributable (at least partly) to variable effects of specific QTL. Pooled across traits, 12/44 (27.3%) and 32/62 (51.6%) of QTL exhibited significant QTL × environment interactions in the L*er* × Col and Cvi × L*er* lines, respectively. These interactions were attributable to changes in magnitude of effect of QTL more often than to changes in rank order (sign) of effect. Multiple QTL × environment interactions (in Cvi × L*er*) clustered in two genomic regions on chromosomes 1 and 5, indicating a disproportionate contribution of these regions to the phenotypic patterns observed. High-resolution mapping will be necessary to distinguish between the alternative explanations of pleiotropy and tight linkage among multiple genes.

INFLORESCENCE is a major component of flowering plant morphology (Weberling 1989; Tucker and Grimes 1999). Composed of the flower-bearing shoots and branches, this structure is critically involved in reproduction, and the timing of initiation and developmental progression are important determinants of plant life history and reproductive ecology (Rathcke and Lacey 1985; Fishbein and Venable 1996; Diggle 1999). In the model plant species *Arabidopsis thaliana*, the basic blueprint of inflorescence development is generally understood (discussed in Grbic and Bleecker 1996; Simpson *et al.* 1999). However, the timing of inflorescence developmental events and overall architecture can be influenced to a great extent by environmental factors such as nutrient availability (Zhang and Lechowicz 1994; Van Tienderen *et al.* 1996; Pigliucci 1997; Bonser and Aarssen 2001), light quality (Dorn *et al.* 2000), drought stress (Meyre *et al.* 2001), density (Orbovic and Tarasjev 1999), photoperiod (Clarke *et al.* 1995; Jansen *et al.* 1995; Reeves and Coupland 2000), and vernalization (Clarke *et al.* 1995; Jansen *et al.* 1995; Simpson *et al.* 1999; Reeves and Coupland 2000).

The ability of a genotype to modify phenotypic expression in response to different environmental conditions is referred to as phenotypic plasticity. This phenomenon is typically depicted by the norm of reaction (Schmalhausen 1949), which is simply a plot of measurements for the same trait in different environments. The difference between measurements in different environments is referred to as environmental sensitivity (Falconer 1990). Not all genotypes respond similarly to environmental signals, however, and variation in response (variation in norms of reaction or environmental sensitivities) is manifested as genotype-environment interaction (GEI).

Phenotypic plasticity and GEI are of considerable interest from both ecological and evolutionary genetic perspectives (Via and Lande 1985; Schlichting 1986; West-Eberhard 1989; Scheiner 1993; Via *et al.* 1995; Schlichting and Pigliucci 1998; Sultan 2000; Pigliucci 2001). For populations that regularly experience heterogeneous environments, plasticity may be adaptive because alternative phenotypes can be expressed in different environments. In sessile organisms such as plants, this phenomenon may be of special significance; the inability of plants to escape changing environmental conditions leaves developmental plasticity as the only means of response (Bradshaw 1965). A number of theoretical models have been developed, describing conditions under which adaptive plasticity might evolve (Via and Lande 1985; Lively 1986a; De Jong 1990, 1995; Gomulkiewicz and Kirkpatrick 1992; Van Tienderen 1997), and numerous empirical tests of the adaptive plasticity hypothesis have been conducted (Lively 1986b; Greene 1989; Bronmark and Miner 1992; Schmitt *et al.* 1999). Theoretical models have

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also implicated GEI as a factor that could contribute to the maintenance of genetic variation in natural populations, especially if the genetic basis of GEI is such that alternative alleles at a locus are favored in different environments (Hedrick 1986; Gillespie and Turelli 1989; but see Gimelfarb 1990).

Despite the importance of phenotypic plasticity and GEI in ecological and evolutionary processes, empirical study of the genetic basis of these phenomena has been difficult because most traits of ecological and evolutionary relevance are polygenic and the environment-specific expression of such traits is generally not well understood. Two classes of genetic models have been specified to explain plasticity and GEI (Via *et al.* 1995): (1) the allelic sensitivity model holds that plasticity and GEI arise from differential effects of loci directly contributing to variation in plastic traits (*i.e.*, allele substitutions affect the phenotypic mean, but differently, in different environments), whereas (2) the gene regulation model posits that specific loci may enhance (or suppress) expression of other genes (only the latter affect the phenotypic mean) in an environment-specific fashion. These models are not mutually exclusive, nor do they make restrictions regarding the types of genes expected to be acting under each model.

Quantitative trait locus (QTL) mapping provides an excellent means for exploring the genetic basis of phenotypic plasticity and GEI. Although initial applications had some shortcomings (see Lynch and Walsh 1998 for a review), more recent efforts have proven far more effective, largely due to the incorporation of a QTL × environment interaction component, either by combining QTL mapping results with analysis of variance (ANOVA) models or by integrating this interaction component into actual mapping algorithms (Jiang and Zeng 1995; Wang *et al.* 1999). Additionally, QTL mapping can also be performed on environmental sensitivity scores (standardized differences in trait values measured in different environments). These approaches have provided much better quantitative evaluations of QTL × environment interactions and have been used successfully to investigate plasticity and GEI in animal life span (Shook and Johnson 1999; Leips and Mackay 2000; Vieira *et al.* 2000), Drosophila sensory bristle number (Gurganus *et al.* 1998), reproductive performance (Fry *et al.* 1998; Shook and Johnson 1999), agriculturally relevant crop traits (Jiang *et al.* 1999), flowering time (Clarke *et al.* 1995; Jansen *et al.* 1995; Stratton 1998; Alonso-Blanco *et al.* 1998b), seed dormancy (Van Der Schaar *et al.* 1997), plant secondary metabolite production (Kliebenstein *et al.* 2002), and pollen competitive ability (Sari-Gorla *et al.* 1997).

In a previous report (Ungerer *et al.* 2002), quantitative genetic analyses and QTL mapping of 13 inflorescence development traits were conducted for two sets of recombinant inbred (RI) lines grown under a long-day (14-hr) photoperiod. The current report expands upon those results by examining the same traits and mapping populations in a second, short-day (10-hr) photoperiod and conducting a joint analysis on the combined data (long day plus short day) to determine the extent to which inflorescence development exhibits plasticity and GEI to photoperiod and to explore the genetic basis of these phenomena. Photoperiod is a reliable environmental cue that predicts seasonal change and is thus of ecological relevance for plants; it is known to affect many aspects of plant growth and development (Evans 1975; Thomas and Vince-Prue 1997).

In this report, it is shown that most inflorescence development traits exhibit plasticity and GEI in response to different photoperiod environments and that these phenotypic responses are attributable (at least in part) to variable effects of specific QTL. Further, it is shown that QTL for environmental sensitivity (the standardized difference between traits measured in different photoperiod environments) often co-localize with QTL exhibiting variable effects, although additional QTL for environmental sensitivity map to unique genomic regions. These findings provide insights into how genomes and environmental factors interact to determine phenotypes.

MATERIALS AND METHODS

Mapping populations and plant-growing conditions: Characterization of plasticity and GEI and QTL × environment mapping of inflorescence development traits were conducted in two sets of recombinant inbred (RI) lines. The first set (L*er* × Col, 96 lines) is derived from a cross between ecotypes Landsberg *erecta* and Columbia (Lister and Dean 1993) and the second set (Cvi × L*er*, 158 lines) is derived from a cross between ecotypes Cape Verde Islands and Landsberg *erecta* (Alonso-Blanco *et al.* 1998a). These represent the same lines used in Ungerer *et al.* (2002). Although the L*er* ecotype is a parent in both sets of RI lines, different accessions of this ecotype were used in the construction of the two sets (L*er*-0 [NW20] for L*er* × Col and L*er*-2 [N8581] for Cvi × L*er*; see http://nasc.nott.ac.uk/catalogue.html).

The experimental design and growing conditions followed those of Ungerer *et al.* (2002). The long-day photoperiod treatment consisted of 14-hr days (20°) and 10-hr nights (18°) whereas the short-day treatment consisted of 10-hr days (20°) and 14-hr nights (18°). These photoperiods were chosen to coincide with those experienced by natural plant populations in late fall and late spring as part of a larger experiment comparing inflorescence development under growth chamber and field conditions. All plants were housed in environmentally regulated growth chambers at the North Carolina State University Phytotron Facility. Growth chambers were maintained at near-ambient CO_2 (350–400 ppm) with photosynthetically active radiation (PAR) = 500–540 μmol m^{-2} s^{-1}. A procedural manual for the Phytotron Facility is available at http://www2.ncsu.edu/ncsu/research_outreach_extension/centers/phyto/index.html. Because of the size of this experiment, the different sets of RI lines (and their parental lines) were not grown concurrently but rather were staggered in time.

Inflorescence development traits: Thirteen traits (Table 1

and Ungerer *et al.* 2002) reflecting various aspects of inflorescence development were measured for 15 replicate individuals of each line (for both sets of RI lines) in each photoperiod environment. Because plants were occasionally lost during the experiment and because some seeds failed to germinate, <15 replicate individuals were measured for a small number of lines. No fewer than 11 replicates, however, were scored for any one line. Results for bolting time (in L*er* × Col) are described elsewhere (Weinig *et al.* 2002).

Quantitative genetic analysis of plasticity and GEI: Mixed-model ANOVAs were used to partition variance in inflorescence development traits in the mapping populations into sources attributable to RI line, photoperiod, and their corresponding interaction. For each trait, the model

$$y = \mu + G + P + G \times P + R$$

was evaluated, where G represents genotype (*i.e.*, RI line, random effect), P represents photoperiod (fixed effect), $G \times P$ represents GEI (random effect), and R represents residual error. A significant effect of RI line (G) was interpreted as genetic differences among RI lines for the traits measured, a significant effect of photoperiod (P) was interpreted as the presence of phenotypic plasticity, and a significant interaction ($G \times P$) was interpreted as significant GEI.

Significant GEI can arise from two sources: (1) deviation from unity of the cross-environment genetic correlation ($r_{GE} < 1$; see below) and (2) differences in among-line variance in the separate environments. The contributions of these sources can be determined from the equation

$$V_{G\times E} = [2\sigma_{E1}\sigma_{E2}(1 - r_{GE}) + (\sigma_{E1} - \sigma_{E2})^2]/2$$

(Robertson 1959), where $V_{G\times E}$ is the GEI variance component, σ_{E1} and σ_{E2} are square roots of the among-line variance components in different photoperiods, and r_{GE} is the cross-environment genetic correlation. The first term corresponds to lack of perfect correlation ($r_{GE} < 1$) and the second term corresponds to differences in among-line variance.

The cross-environment genetic correlation (r_{GE}) is the genetic correlation of measurements of the same trait in different environments and here reflects the degree to which the same genes control trait expression across photoperiods. r_{GE} was estimated for each trait as $cov_{E1E2}/\sigma_{E1}\sigma_{E2}$, where cov_{E1E2} is the covariance of RI line means measured in different photoperiod environments and σ_{E1} and σ_{E2} are square roots of the among-line variance components in different environments. All statistical analyses were conducted using software packages SAS (GLM and VARCOMP procedures; SAS Institute 1988) and/or STATVIEW (SAS Institute 1999).

Linkage maps: Genotype data for these lines are publicly available and were obtained on the web at http://nasc.nott.ac.uk/. The L*er* × Col and Cvi × L*er* RI lines have been genotyped for largely different sets of markers and thus different maps were generated for each set of lines. Maps were constructed using Mapmaker/EXP 3.0 (Lander *et al.* 1987). Details of marker selection and map construction are described elsewhere (Ungerer *et al.* 2002). Briefly, the L*er* × Col map spans 576.52 cM and is composed of 222 markers spaced, on average, every 2.61 cM (Figure 1). The Cvi × L*er* map spans 458.45 cM and consists of 138 markers spaced, on average, every 3.35 cM (Figure 1).

QTL analyses: ANOVA results from analyses of phenotypes can indicate whether genetic differences exist among RI lines for inflorescence development traits and whether there are plasticity and GEI for these traits. ANOVA results cannot, however, provide any information regarding the actual genetic factors responsible for these patterns. QTL mapping strategies are an appropriate means of further exploring these statistical observations of phenotypes.

Mapping of QTL associated with plasticity and GEI for inflorescence development was conducted using multiple-trait composite interval mapping (multiple-trait CIM; Jiang and Zeng 1995), which is part of a suite of programs in QTL Cartographer 1.13 (Basten *et al.* 1994, 1999). Measuring the same trait in more than one environment is statistically equivalent to measuring multiple genetically correlated traits in the same environment (Falconer 1952). Multiple-trait CIM allows for the dissection of genetic variation and covariation by estimating the positions and differential effects of QTL for correlated traits (or for the same trait in different environments; Jiang and Zeng 1995). This procedure is similar to conventional CIM in which tests are conducted sequentially along each chromosome to determine whether intervals flanked by molecular markers contain a QTL while statistically accounting for other QTL segregating in the genetic background outside the tested interval. Multiple-trait CIM is different, however, in that QTL mapping is performed jointly on measurements of the same trait in different environments. The hypotheses tested are

$$H_0: a_1 = 0,\ a_2 = 0,$$

$$H_1: \text{at least one of them is not zero,}$$

where a_1 and a_2 represent additive effects of QTL in environments 1 and 2. At test positions where the null hypothesis is rejected, tests of QTL × environment interaction are performed. The hypotheses tested are

$$H_0: a_1 = a_2$$

$$H_1: a_1 \neq a_2.$$

Both sets of hypotheses are tested with the likelihood-ratio (LR) test statistic, $-2\ln(L_0/L_1)$ (where L_0/L_1 is the ratio of likelihoods of hypotheses). Two sets of LR scores (one for the joint analysis and one for the QTL × environment analysis) are thus evaluated. Note that in the current study it was not possible to estimate dominance effects due to the absence of heterozygotes in RI lines.

The identity (and number) of markers selected for genetic background control was determined independently for each trait by forward selection, backward elimination stepwise regression. For each trait, markers were selected separately in each environment and then used jointly in multiple-trait CIM. A 10-cM scan window was used for all analyses and the LR test statistic was calculated every 0.5 cM.

Experiment-wide significance thresholds for QTL identification were determined for each trait by permutation analysis (Churchill and Doerge 1994; Doerge and Churchill 1996). The permutation procedure yields different significance thresholds for the joint and QTL × environment LRs. One thousand permutations were performed for each trait.

Because multiple-trait CIM differs slightly from conventional CIM with respect to model evaluation and marker cofactor selection, a two-step procedure was conducted to qualitatively compare the two approaches. First, conventional CIM (Zeng 1994) was performed for each trait separately in each environment. This procedure allowed confirmation of QTL positions in separate environments. Second, the marker nearest each QTL peak (detected in either or both environments) was selected and collectively fitted to the model

$$y = \mu + P + \sum M_i + \sum (M_i \times P) + R,$$

where P and R are defined as above, M_i represents the ith marker detected by conventional CIM in either or both photoperiods, and $M_i \times P$ represents the interaction of the ith marker with photoperiod (*i.e.*, QTL × environment interaction). Significant QTL × environment interactions detected

in these full-ANOVA models were then compared to corresponding results from the multiple-trait CIM analysis (conducted within QTL Cartographer). The two approaches produced near-identical results: QTL exhibiting QTL × environment interaction as determined by multiple-trait CIM almost always had a significant QTL × environment interaction term (or near significant) in full-ANOVA models and QTL not exhibiting QTL × environment interactions as determined by multiple-trait CIM typically did not. Only results from multiple-trait CIM are reported here.

Tests for epistasis among QTL were conducted using ANOVAs to examine interaction effects of QTL with established additive effects (Long *et al.* 1995; Leips and Mackay 2000; Ungerer *et al.* 2002). Tests for epistasis were first performed separately in each environment. The markers selected to conduct these tests, however, were those detected in the multiple-trait CIM joint analysis. It was therefore possible for markers to be tested for epistasis in an environment where main effects of that marker (QTL) were not detected. To determine whether epistatic interactions contribute to plasticity and GEI for inflorescence development traits, the full-ANOVA model

$$y = \mu + P + \sum M_i + (M_j \times M_k) + (M_j \times M_k \times P) + R$$

was evaluated, where P, M_i, and R are defined as above and M_j and M_k are markers involved in significant epistasis. A significant three-way interaction term ($M_j \times M_k \times P$) indicates that the nature of epistasis differs across photoperiods and thus may contribute to observations of plasticity and GEI. Where it was necessary to evaluate multiple models for a given trait, significance thresholds were adjusted using a sequential Bonferroni procedure. All analyses of epistasis were conducted using the GLM procedure of SAS (SAS Institute 1988).

Finally, for each individual RI line and for each trait, an environmental sensitivity score was estimated as $(\bar{E}_{1i} - \bar{E}_{2i})/D$ (Falconer 1990), where D is the difference of the means of all RI lines reared in the two photoperiod environments and $\bar{E}_{1i}$ and $\bar{E}_{2i}$ are the means of replicate individuals of the same RI line in the two different photoperiod environments, where i refers to 1–96 (L*er* × Col) or 1–158 (Cvi × L*er*) RI lines. QTL for sensitivity scores for all traits were then mapped using conventional CIM and significance thresholds determined by permutation.

Comparing this latter analysis to results from the multiple-trait CIM analysis allowed for evaluation of evidence supporting the two classes of genetic models for plasticity and GEI. If the allelic sensitivity model explains most plasticity and GEI, then QTL for environmental sensitivity scores are expected to co-localize with QTL affecting inflorescence development traits directly (QTL detected by multiple-trait CIM), and these QTL are expected to exhibit QTL × environment interactions. Conversely, if the gene regulation model explains most plasticity and GEI, then QTL for environmental sensitivity scores are expected to map to unique genomic regions and there is no expectation of positional overlap with QTL affecting traits directly (Leips and Mackay 2000; Kliebenstein *et al.* 2002).

RESULTS

Quantitative genetic variation, phenotypic plasticity, and GEI for inflorescence development in RI lines: Quantitative genetic statistics for inflorescence traits reared under long days (LD) have been reported previously (Ungerer *et al.* 2002). Corresponding information for the same lines and traits under short days (SD) is provided as supplemental data (http://www.genetics.org/supplemental, Tables 1–3) in combination with the previously reported LD data.

In mixed-model ANOVAs with main effects of RI line and photoperiod and their corresponding interaction, significance of main effects and the interaction term was observed for the majority of traits in both sets of RI lines (Table 1). This result indicates that (1) there are genetic differences among RI lines for the traits specified, (2) there is plasticity in inflorescence development patterns across photoperiods, and (3) there is variation in plastic response among individual RI lines (there is GEI). Only one inflorescence trait (elongated axils in L*er* × Col) failed to exhibit a plastic response to photoperiod (in the full-ANOVA model, $F = 3.29$, $P = 0.07$). This trait did, however, exhibit significant GEI (Table 1). Traits that did not exhibit significant GEI include rosette diameter (in both L*er* × Col and Cvi × L*er*), main inflorescence fruits (L*er* × Col only), and total fruits (L*er* × Col only). Axillary fruits (in L*er* × Col) exhibited marginally significant GEI (Table 1). The failure to detect GEI for some inflorescence development traits may be associated with a lack of statistical power given the low estimated heritability for some traits (supplemental data Table 2).

GEI can arise from the lack of perfect correlation across environments ($r_{GE} < 1$) and from differences in among-line variance for the same trait measured in separate environments. Lack of perfect correlation indicates changes in rank order of reaction norms. On average, the majority of GEI variance in both sets of RI lines was attributable to this source (averaged over all traits, 71.7% in L*er* × Col and 68.8% in Cvi × L*er*). Interestingly, despite these similar averages, the relative partitioning of $V_{G\times E}$ for the same trait often differed substantially between the two sets of RI lines (Table 1). For example, for rosette leaves at bolting the relative contributions of changes in rank order *vs.* changes in variance of reaction norms were 0.97 and 0.03, respectively, in the L*er* × Col lines but 0.16 and 0.84, respectively, in the Cvi × L*er* lines (Table 1).

Variable-effect QTL: Results of multiple-trait CIM are depicted graphically in Figure 1 and are listed in supplemental data Tables 4 and 5 (http://www.genetics.org/supplemental). Overall, 44 and 62 QTL for inflorescence development were identified in the L*er* × Col and Cvi × L*er* mapping populations, respectively. In the L*er* × Col lines, 12 of 44 QTL (27.3%) exhibited significant QTL × environment interaction. In the Cvi × L*er* lines, 32 of 62 QTL (51.6%) exhibited significant QTL × environment interaction. Figure 2 illustrates, for both mapping populations, the number of QTL detected for each of the 13 inflorescence traits and whether they exhibited a significant interaction with photoperiod. QTL exhibiting interaction effects are subclassified into those demonstrating changes in magnitude of effect and those demonstrating changes in rank order of effect (change in sign of the additive

TABLE 1

Quantitative genetic statistics of plasticity and GEI for 13 inflorescence development traits measured in short-day (10-hr) and long-day (14-hr) photoperiods

Trait	$[V_G]^a$	Photoperiod[b]	$[V_{G\times E}]^c$	$V_{G\times E}$ partitioned[d]	$[r_{GE}]^e$
			L*er* × Col RI lines		
Length of reproductive phase of main axis (days)	0.584**	**	0.213**	(0.89, 0.11)	0.75 (0.65, 0.83)
Time to maturity of main axis (days)	3.103**	**	2.503**	(0.51, 0.49)	0.71 (0.59, 0.80)
Rosette leaves at bolting	1.428**	**	0.468**	(0.97, 0.03)	0.76 (0.66, 0.83)
Rosette diameter (cm)	0.010**	**	0.001^{NS}	—	0.88 (0.82, 0.92)
Plant height (cm)	9.400**	**	0.555**	(0.30, 0.70)	0.98 (0.97, 0.99)
Main inflorescence fruits	1.421**	**	0.159^{NS}	—	0.93 (0.89, 0.95)
Axillary fruits	1.499**	**	0.891*	(0.92, 0.08)	0.74 (0.63, 0.82)
Nonelongated secondary meristems	0.178**	**	0.075**	(1.00, 0.00)	0.70 (0.58, 0.79)
Elongated axils	0.119**	NS	0.057**	(0.88, 0.12)	0.70 (0.59, 0.79)
Secondary inflorescence meristems on main axis	0.073**	**	0.088**	(0.99, 0.01)	0.45 (0.28, 0.60)
Early flowers	1.179**	**	0.810**	(0.25, 0.75)	0.85 (0.79, 0.90)
Total fruits	4.197**	**	0.569^{NS}	—	0.96 (0.95, 0.98)
			Cvi × L*er* RI lines		
Bolting time (days)	25.624**	**	6.767**	(0, 1.00)	1.02 (undefined)
Length of reproductive phase of main axis (days)	5.280**	**	4.834**	(0.84, 0.16)	0.58 (0.47, 0.67)
Time to maturity of main axis (days)	42.459**	**	18.857**	(0.24, 0.76)	0.93 (0.90, 0.95)
Rosette leaves at bolting	4.177**	**	1.803**	(0.16, 0.84)	0.95 (0.93, 0.96)
Rosette diameter (cm)	0.032**	**	0.003^{NS}	—	0.94 (0.92, 0.96)
Plant height (cm)	12.353**	**	1.159**	(0.99, 0.01)	0.91 (0.88, 0.93)
Main inflorescence fruits	5.106**	**	2.710**	(0.93, 0.07)	0.66 (0.56, 0.74)
Axillary fruits	2.580**	**	10.136**	(0.84, 0.16)	0.25 (0.10, 0.39)
Nonelongated secondary meristems	0.171**	**	0.134**	(0.93, 0.07)	0.61 (0.50, 0.70)
Elongated axils	0.070**	**	0.396**	(0.75, 0.25)	−0.30 (−0.43, −0.15)
Secondary inflorescence meristems on main axis	0.322**	**	0.215**	(0.61, 0.39)	0.73 (0.64, 0.79)
Early flowers	1.511**	**	0.178**	(0.97, 0.03)	0.90 (0.86, 0.92)
Total fruits	14.216**	**	18.793**	(0.99, 0.01)	0.44 (0.30, 0.55)

NS, not significant; $*P \leq 0.05$; $**P \leq 0.0001$.

[a] Among-line variance component from full-ANOVA model; significance indicates genetic differences among RI lines.

[b] Fixed effect of photoperiod from full-ANOVA model; significance indicates plasticity for photoperiod.

[c] RI line by photoperiod interaction variance component from full-ANOVA model; significant interaction indicates genotype × environment interaction (GEI).

[d] Proportion of $V_{G\times E}$ attributable to the departure of the cross-environment genetic correlation from unity (first term) and to changes in among-line variance in different photoperiod environments (second term).

[e] Cross-environment genetic correlation calculated as $cov_{E1E2}/\sigma_{E1}\sigma_{E2}$; parentheses indicate 95% confidence intervals.

effect across the two photoperiods). For QTL exhibiting significant interaction effects, changes in magnitude were substantially more common than changes in rank order in both sets of RI lines (Figure 2).

Multiple QTL clustered near the *erecta* mutation on chromosome II (Figure 1). Clustering of QTL also was observed at the top of chromosome 1 and top, middle, and bottom of chromosome 5. Some of these regions of clustering [*e.g.*, top of chromosome 1 and top and middle of chromosome 5 (Cvi × L*er*)] harbored QTL that consistently exhibited variable effects across photoperiods (QTL × environment interactions), indicating that specific genomic regions may disproportionately contribute to observed plasticity and GEI across multiple traits. Of course, clustering of QTL may represent a far smaller number of actual segregating loci (or a single locus) with pleiotropic effects on multiple traits (see DISCUSSION). QTL positions, support limits, and additive effects in each photoperiod environment are given in supplemental data Tables 4 and 5.

Epistatic interactions contribute to plasticity and GEI: Significant epistasis was detected in both sets of RI lines and in both photoperiods [Figure 3, supplemental data Tables 6 and 7 (http://www.genetics.org/supplemental)]. In the L*er* × Col lines, five interactions were detected, affecting four traits (Figure 3A, supplemental data Table 6). None of the five interactions were found to be significant in both photoperiods when tests were conducted separately (supplemental data Table 6). The more relevant tests of three-way interaction among marker pairs (QTL) and photoperiod (*i.e.*, marker × marker × photoperiod) revealed three of five significant tests (Figure 3A, supplemental data Table 6), indicating that the nature of the marker × marker epistasis

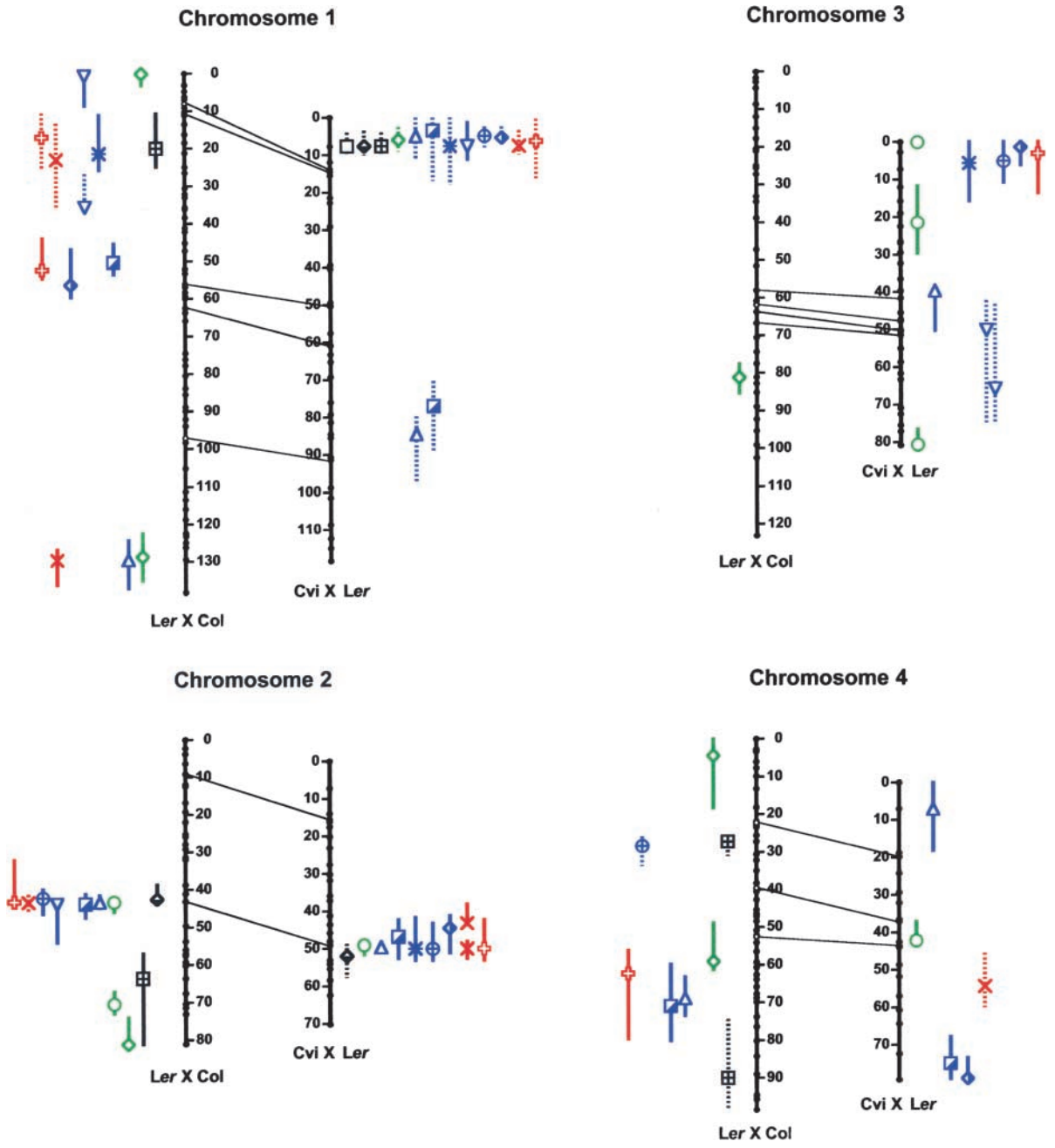

FIGURE 1.—*Arabidopsis thaliana* genetic linkage maps constructed from the L*er* × Col (left chromosomes) and Cvi × L*er* (right chromosomes) RI lines and QTL positions for 13 inflorescence development traits. QTL for different traits are depicted with different symbols and vertical lines associated with each QTL indicate 2 LOD support limits. Dashed vertical lines (support limits) indicate that QTL exhibits GEI. Colors of QTL are associated with trait subcategories as follows (UNGERER *et al.* 2002): black, inflorescence developmental timing; green, basal rosette morphology; blue, inflorescence architecture; and red, fitness. Map positions of genetic markers are depicted as circles on chromosomes. Markers represented as open symbols did not map to unique intervals given the mapping criteria specified and are placed here in the interval of highest likelihood. Markers that did not map to unique intervals were not used in QTL analyses. Genetic markers connected by lines were mapped in both sets of RI lines and represent landmarks for map comparisons. Units of map length are in centimorgans.

differs significantly across photoperiods for some interactions. The majority of markers (QTL) involved in these interactions had larger additive effects in the environment in which the significant epistasis was detected (supplemental data Table 4).

In the Cvi × L*er* lines, 10 significant interactions were detected affecting nine traits (Figure 3B, supplemental data Table 7), with some interactions affecting multiple traits (*e.g.*, the *AXR-1* × *BH.325L* interaction had significant effects on bolting time, length of reproductive phase of main axis, time to maturity of main axis, rosette leaves at bolting, and early flowers). All three-way inter-

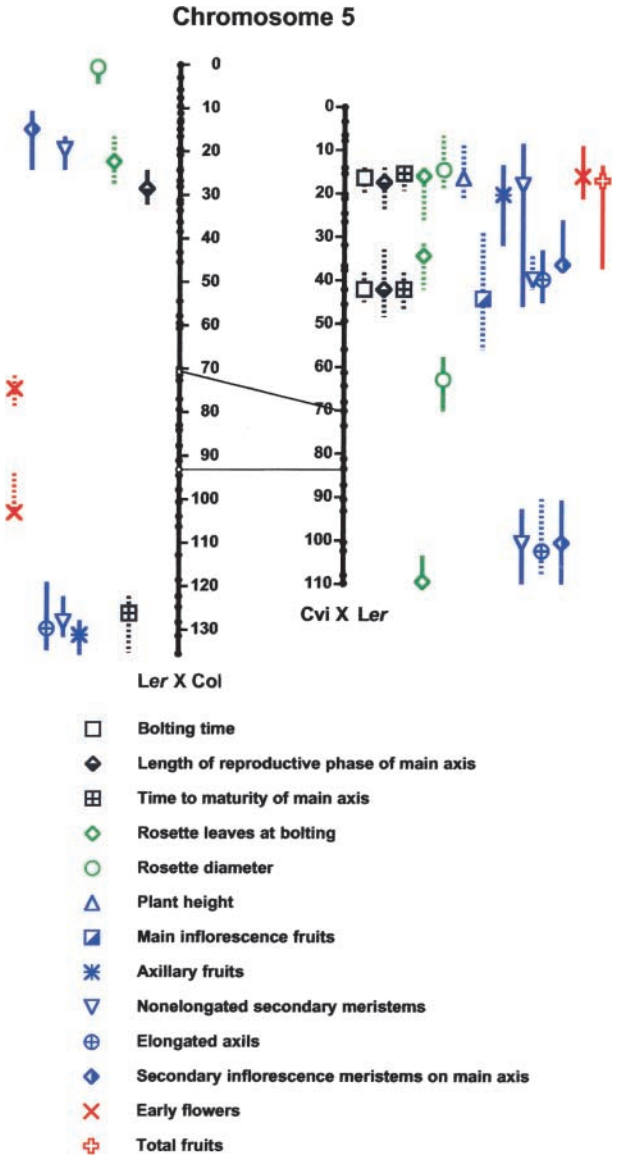

FIGURE 1.—*Continued.*

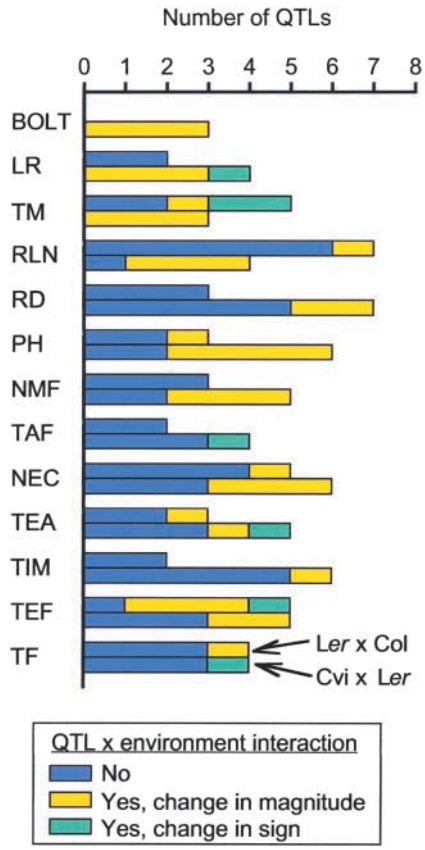

FIGURE 2.—Summary of QTL behavior types for 13 inflorescence development traits in the L*er* × Col (top bars) and Cvi × L*er* (bottom bars) RI lines. Trait abbreviations: BOLT, bolting time; LR, length of reproductive phase of main axis; TM, time to maturity of main axis; RLN, rosette leaves at bolting; RD, rosette diameter; PH, plant height; NMF, main inflorescence fruits; TAF, axillary fruits; NEC, nonelongated secondary meristems; TEA, elongated axils; TIM, secondary meristems on main axis; TEF, early fruits; and TF, total fruits.

actions (marker × marker × photoperiod) were significant, indicating that the nature of epistasis was significantly different across photoperiods for all pairwise marker combinations in the Cvi × L*er* RI lines. In contrast to the L*er* × Col lines, approximately one-half of interactions were significant in both photoperiods when tests were conducted separately in each photoperiod environment. This is noteworthy because even though some of the same interactions were found to be significant under both SD and LD photoperiods, the strength of the interaction differed significantly across photoperiods—the tests of three-way interaction (marker × marker × photoperiod) were significant (supplemental data Table 7). Consistent with interactions detected in the L*er* × Col lines, however, the additive effects of QTL involved in epistasis tended to be larger in the environment in which the epistasis was detected (supplemental data Table 5). In instances where epistasis was found to be significant in both photoperiod environments, additive effects of QTL involved in interactions were larger in the photoperiod in which the epistatic effect was larger.

QTL for environmental sensitivity: The positions and effects of QTL for environmental sensitivity are given in Tables 2 and 3 for the L*er* × Col and Cvi × L*er* mapping populations, respectively. Also provided in these tables is whether 2 LOD support limits for these QTL overlap with support limits for QTL identified by multiple-trait CIM. Eleven of 20 (55%) and 30 of 36 (~83%) sensitivity QTL overlapped in position with declared [or marginally significant ($P < 0.10$)] QTL from multiple-trait CIM in the L*er* × Col and Cvi × L*er* mapping populations, respectively. In these regions of overlap, declared QTL from multiple-trait CIM disproportionately exhibited QTL × environment interactions [6 of 9 QTL (~67%) in L*er* × Col and 26 of 28 QTL (~93%) in Cvi × L*er*]. Again, however, it should be

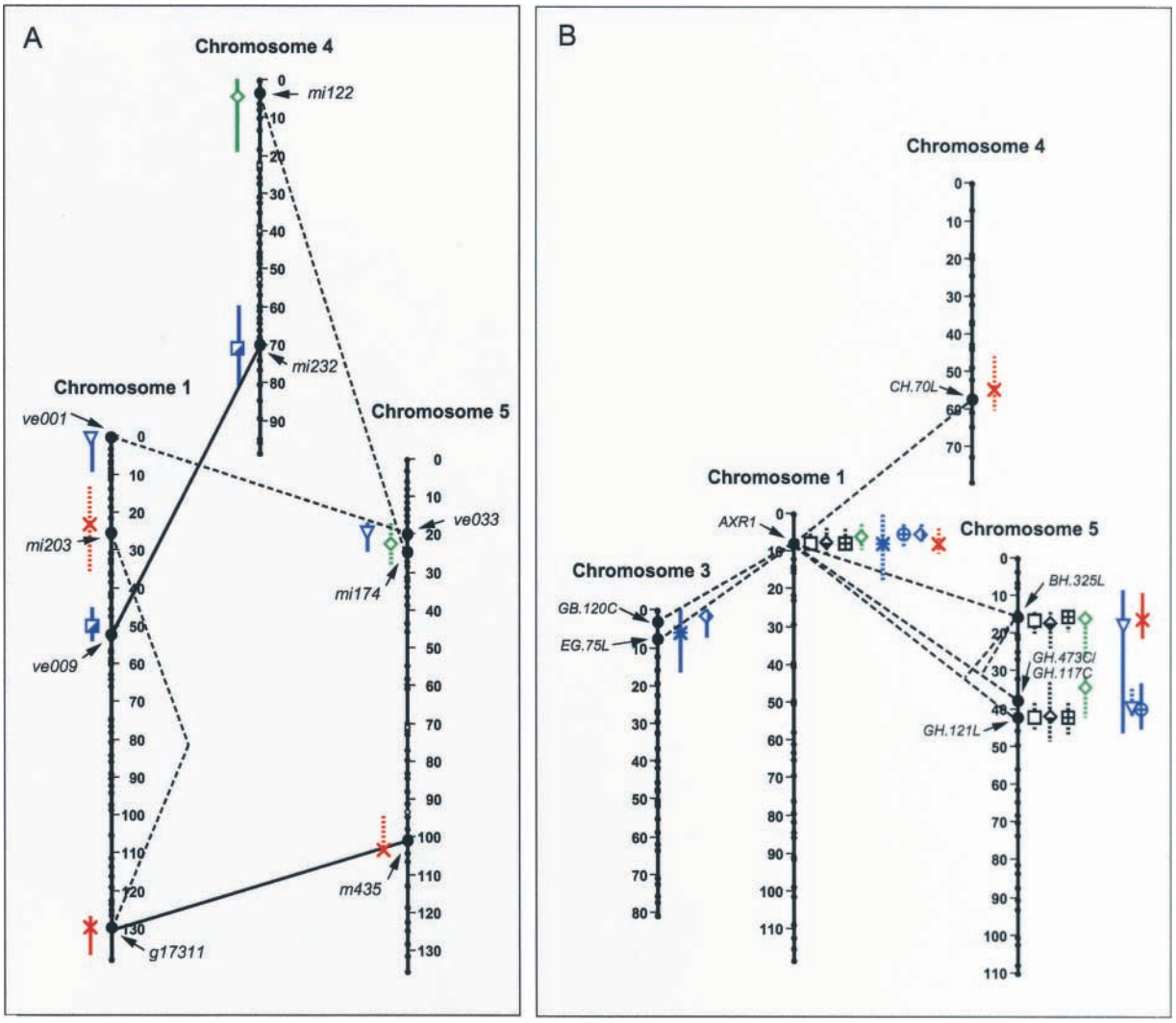

FIGURE 3.—Epistatic interactions detected in the L*er* × Col (A) and Cvi × L*er* (B) RI lines. Lines connect pairs of markers (QTL) with significant epistatic effects. Dashed lines indicate that the magnitude of the interaction was significantly different across photoperiods (significant marker × marker × photoperiod term, see section in MATERIALS AND METHODS for testing epistasis) whereas solid lines indicate that three-way interaction was not significant. Positions of QTL involved in interactions are shown to the left (A) or right (B) of the chromosomes (notation is the same as in Figure 1). Markers *GH.473C* and *GH.117C* on chromosome 5 (B) are 0.97 cM apart and are indicated by the same enlarged symbol.

noted that many QTL for environmental sensitivity mapped to similar genomic regions and may indeed represent the same genetic factor(s).

DISCUSSION

Phenotypic plasticity, GEI, and variable effect QTL: We examined plasticity and GEI in response to variation in photoperiod length for 13 inflorescence development traits in two sets of RI lines using a combination of quantitative genetic and QTL mapping approaches. The majority of inflorescence development traits exhibited strong plasticity and GEI when reared under photoperiods of different length. Most of the GEI variance was found to be attributable to changes in rank order (crossing) of reaction norms with changes in variance across photoperiods being a less common contributor. GEI variance that is attributable to crossing of reaction norms may have important ecological relevance as it suggests that different genotypes may be favored in different environments.

Between two and seven QTL were detected by multiple-trait CIM for each inflorescence development trait. For a substantial percentage of QTL, expression was highly sensitive to photoperiod environment—there was QTL × environment interaction. Combined over all traits, 27.3 and 51.6% of QTL exhibited significant QTL × environment interactions in the L*er* × Col and Cvi × L*er* lines, respectively. These percentages are similar to those found in other plant studies that have assessed QTL × environment interactions across distinct environments (JANSEN *et al.* 1995; SARI-GORLA *et al.*

TABLE 2

QTL for photoperiod sensitivity in L*er* × Col RI lines

Trait	QTL map position: chromosome-cM (2 LOD support limit)	Additive effect[a]	Overlap with QTL for trait[b]
Length of reproductive phase of main axis (days)	III-70.58 (58.59–76.39)	−0.172	N
Time to maturity of main axis (days)	I-45.22 (42.18–56.15)	−0.067	N
	IV-28.74 (22.91–38.32)	0.061	Y
	V-130.22 (113.78–135.32)	0.056	Y
Rosette leaves at bolting	II-81.05 (74.05–81.05)	0.109	Y
	IV-14.71 (0.01–45.94)	0.103	Y
	V-76.99 (73.15–79.37)	−0.107	N
Rosette diameter (cm)	I-45.22 (45.18–53.78)	−0.577	N
	IV-63.91 (54.59–69.27)	0.493	M
Plant height (cm)	II-44.62 (41.54–48.17)	0.273	Y
Main inflorescence fruits	IV-3.32 (0.01–6.08)	−0.122	N
	IV-42.68 (42.00–45.44)	−0.149	N
	IV-56.48 (53.64–67.70)	0.116	Y
Nonelongated secondary meristems	I-31.12 (22.22–41.12)	−0.075	Y
	IV-25.62 (19.20–38.32)	0.089	M
Early flowers	I-32.16 (13.02–35.64)	0.089	Y
	II-43.17 (41.04–46.67)	−0.182	Y
	IV-9.87 (0.01–21.85)	−0.078	N
	V-126.71 (112.32–131.22)	−0.100	N
Total fruits	IV-0.01 (0.01–13.21)	−0.114	N

[a] The additive effect is defined as $(Q_1Q_1 - Q_2Q_2)/2$, where Q_1Q_1 and Q_2Q_2 represent the mean environmental sensitivity of RI lines homozygous for alternative genotypes at a QTL position. The sign of the additive effect corresponds to the direction of effect of the Columbia allele on the phenotype.

[b] Whether sensitivity QTL overlap in position with QTL detected by multiple-trait CIM: Y, yes; N, no; M, QTL detected by multiple-trait CIM is marginally significant ($P < 0.10$). Underlining indicates that QTL exhibit QTL × environment interaction.

1997; Van Der Schaar *et al.* 1997; Alonso-Blanco *et al.* 1998b; Borevitz *et al.* 2002; Kliebenstein *et al.* 2002). Further, the finding that QTL × environment interactions demonstrate changes in magnitude of effects more often than changes in rank order is also consistent with previous studies distinguishing between these QTL behavior types (Sari-Gorla *et al.* 1997; Fry *et al.* 1998; Stratton 1998; Jiang *et al.* 1999). It is interesting to note that whereas changes in rank order were common among reaction norms, they were rare among QTL effects. Although this might appear contradictory, changes in rank order of reaction norms need not require congruent patterns of QTL effects. Rather, changes in rank order of reaction norms can be explained by changes in magnitude of QTL effects alone (Fry 1993; Fry *et al.* 1998).

QTL × environment interactions were not found for all traits, and the distribution of these interactions across traits was generally consistent with the corresponding quantitative genetic analyses: inflorescence traits that did not exhibit GEI at the phenotypic level (or that did so only marginally) harbored fewer QTL × environment interactions. For instance, in the L*er* × Col lines, three traits failed to exhibit GEI at the phenotypic level and one did so only marginally (Table 1). Twelve QTL were detected for these four traits but only one (8.3%) exhibited QTL × environment interaction. In contrast, the eight remaining traits all displayed highly significant GEI at the phenotypic level, and 11/32 [34.4%] of corresponding QTL exhibited QTL × environment interaction (Figure 2, supplemental data Table 4). In the Cvi × L*er* lines, only one trait (rosette diameter) failed to exhibit significant GEI at the phenotypic level (Table 1). Although 2 of 7 QTL for this trait exhibited interaction with the environment, their effects were of similar magnitude but opposite in sign (in both photoperiods). Given that phenotypes are determined by the summation of effects of all relevant loci, the combined effects of these two QTL may have canceled, resulting in no GEI detected at the phenotypic level.

In addition to differences in individual QTL effects across photoperiod environments, differences in interaction effects of QTL also were observed in the form of significant three-way interactions (marker × marker × photoperiod; Figure 3, supplemental data Tables 6 and 7). This was true even when the same interaction was found to be significant separately in each photoperiod environment (supplemental data Table 7). Significant marker × marker × photoperiod interactions were not observed, however, for traits that failed to exhibit sig-

TABLE 3

QTL for photoperiod sensitivity in Cvi × L*er* RI lines

Trait	QTL map position: chromosome-cM (2 LOD support limit)	Effect[a]	Overlap with QTL for trait[b]
Bolting time (days)	I-7.01 (3.51–9.71)	1.120	Y
	V-10.81 (0.01–20.20)	−0.473	Y
	V-32.31 (20.70–42.15)	−0.463	Y
Length of reproductive phase of main axis (days)	I-7.71 (3.01–10.21)	0.299	Y
	II-38.14 (36.86–41.20)	−0.159	N
	V-16.90 (14.52–23.17)	−0.226	Y
	V-40.16 (31.81–45.49)	−0.192	Y
Time to maturity of main axis (days)	I-7.71 (4.01–9.71)	0.498	Y
	II-37.14 (30.18–42.70)	−0.178	N
	III-32.32 (25.46–41.60)	0.143	N
	V-21.17 (15.02–24.56)	−0.262	Y
	V-40.16 (31.54–42.15)	−0.251	Y
	V-97.65 (92.00–109.40)	0.166	M
Rosette leaves at bolting	I-6.01 (3.01–9.21)	0.658	Y
	V-15.40 (11.31–19.90)	−0.274	Y
	V-41.66 (31.81–49.33)	−0.234	Y
	V-99.65 (89.16–109.40)	0.169	Y
Rosette diameter (cm)	I-76.02 (67.73–88.01)	0.225	N
Plant height (cm)	I-5.51 (0.01–11.94)	0.594	Y
	I-83.80 (70.82–90.67)	0.456	Y
	V-16.40 (8.81–22.67)	−0.492	Y
Main inflorescence fruits	I-5.51 (0.01–13.44)	0.325	Y
	I-81.80 (72.32–90.01)	0.268	Y
	V-34.31 (26.54–42.15)	−0.253	Y
Axillary fruits	I-9.71 (0.51–18.00)	0.123	Y
	III-65.25 (56.46–74.97)	0.133	M
Nonelongated secondary meristems	III-52.45 (50.95–58.66)	0.235	Y
	V-44.15 (34.81–50.83)	−0.244	Y
	V-96.15 (87.16–109.40)	0.163	Y
Elongated axils	I-5.51 (2.01–9.21)	0.354	Y
	III-46.45 (42.15–51.45)	0.123	N
	V-102.32 (90.16–108.95)	0.115	Y
Secondary inflorescence meristems on main axis	I-5.01 (2.01–7.51)	0.702	Y
Early flowers	IV-55.25 (49.12–60.65)	0.228	Y
Total fruits	I-6.51 (1.01–14.40)	0.173	Y
	III-62.64 (50.95–75.46)	0.106	N

[a] The additive effect is defined as $(Q_1Q_1 - Q_2Q_2)/2$, where Q_1Q_1 and Q_2Q_2 represent the mean environmental sensitivity of RI lines homozygous for alternative genotypes at a QTL position. The sign of the additive effect corresponds to the direction of effect of alleles of the Landsberg *erecta* ecotype.

[b] Whether sensitivity QTL overlap in position with QTL detected by multiple-trait CIM: Y, yes; N, no; M, QTL detected by multiple-trait CIM is marginally significant ($P < 0.10$). Underline indicates that QTL exhibit QTL × environment interaction.

nificant GEI at the phenotypic level, a result consistent with the distribution of QTL × environment interactions.

Environmental sensitivity QTL: In the Cvi × L*er* RI lines, the positions of QTL for environmental sensitivity were in general agreement with those for QTL affecting inflorescence development traits directly. Thirty of 36 sensitivity QTL (~83%) overlapped in position with either declared QTL or regions in which QTL signal was detected but significance thresholds were not quite exceeded (from multiple-trait CIM, Table 3). Moreover, most (~93%) of these QTL (detected by multiple-trait CIM) exhibited interaction effects with photoperiod environment, indicating that environmental sensitivity QTL disproportionately map to regions with differential effects across photoperiods. These observations are largely consistent with expectations under the allelic sensitivity model of phenotypic plasticity and GEI, but suggest that other genetic mechanisms may be acting as well. The same comparison in the L*er* × Col RI lines was less clear, however, as only 55% of sensitivity QTL overlapped with those detected by multiple-trait CIM. The differences observed among sets of RI lines could be attributable to differences in power to detect these

QTL (96 and 158 RI lines in L*er* × Col and Cvi × L*er*, respectively) or could reflect real biological differences and underlying genetic mechanisms of GEI between the two mapping populations.

Comparisons to previous reports: In a previous report (Ungerer *et al.* 2002) the same sets of RI lines and traits were evaluated under a long-day photoperiod only. Although mapping methods differed between these two studies (CIM *vs.* multiple-trait CIM), a large degree of overlap of QTL positions was expected and was also observed. The majority of QTL detected in Ungerer *et al.* (2002) also were detected in this study. Discrepancies (presence/absence of QTL) between the previous and this study result almost exclusively from likelihood-ratio tests being near, but not exceeding significance thresholds in one or the other study. Differences may also be attributable to the selection of marker cofactors in CIM *vs.* multiple-trait CIM. In the latter, markers are selected separately in each environment and then used collectively in the joint analysis.

Similarly, many of the significant epistatic interactions previously detected in Ungerer *et al.* (2002) also were detected in this study. Additional interactions depicted in Figure 3 that were not detected in Ungerer *et al.* (2002) were (1) found only under the SD photoperiod, (2) not tested in Ungerer *et al.* (2002) because one or both markers (QTL) previously were not significant, or (3) indeed tested but were not found to be significant in the previous report. This last category is most likely attributable to slightly different ANOVA models (numbers and identities of main-effect markers used) and differences in significance thresholds set by the sequential Bonferroni correction, which is based on the number of tests necessary to examine all pairwise combinations of main-effect markers (QTL) in the model.

Results from this study can also be compared with other previous studies examining these same lines (some of which also examined QTL × environment interactions). Jansen *et al.* (1995) mapped QTL (and tested for QTL × environment interactions) for rosette + cauline leaf number (as a measure of flowering time) using the L*er* × Col RI lines. These authors conducted their study under short days (10 hr light), long days (16 hr light), and continuous light, all with and without vernalization treatment. Of the 12 QTL detected in that study, 7 appear to have been detected in our study although direct comparisons of QTL positions are made difficult by large differences in map resolution (average of ~7 markers/chromosome in Jansen *et al.* 1995 *vs.* ~44 markers/chromosome in our study). In addition, whereas Jansen *et al.* (1995) measured combined rosette and cauline leaf number, we measured rosette leaves only.

A greater degree of similarity was observed between our study and that of Alonso-Blanco *et al.* (1998b), which mapped QTL for flowering time and leaf number at flowering under short-day (8 hr light), long-day (16 hr light), and long-day + vernalization treatments in the Cvi × L*er* lines. For relevant trait comparisons in relevant environments, all of the same QTL were detected in the two studies with the exception of one small-effect QTL for rosette leaf number (on chromosome 1 at ~40 cM) detected in Alonso-Blanco *et al.* (1998b) but not in our study. Furthermore, all of the same QTL × environment interactions detected in Alonso-Blanco *et al.* (1998b) were also found in our study. Congruence in QTL positions was also observed between our study and that of Alonso-Blanco *et al.* (1999), in which a different set of partially overlapping inflorescence development traits were mapped, although in Alonso-Blanco *et al.* (1999) mapping populations were grown under long days only.

Molecular mechanisms of plasticity and GEI: Characterizing how QTL effects differ across environments is an important first step in elucidating how genetic and environmental factors interact to determine phenotypes. To understand the molecular basis of plasticity and GEI for inflorescence development, however, it is necessary to identify genes underlying natural variation in inflorescence development traits and determine how expression/protein activity differs across ecologically relevant environments.

The genes underlying two flowering-time QTL described in this study (both exhibiting QTL × environment interaction) have recently been identified. The flowering-time QTL at the top of chromosome 1 (in Cvi × L*er*) is attributable to a single-amino-acid substitution (Valine → Methionine) in the blue-light photoreceptor *CRY2* (in the Cape Verde ecotype; El-Assal *et al.* 2001). It is particularly interesting (and perhaps not surprising) that a light sensing photoreceptor underlies differential phenotypic responses across photoperiod environments. The hypothesis that this same molecular polymorphism corresponds to the multiple additional QTL in this region (via pleiotropic effects) seems plausible, but will require high-resolution mapping to test.

The flowering-time QTL at the top of chromosome 5 (detected in Cvi × L*er*) could correspond to the MADS-box transcription factor *FLC* (Michaels and Amasino 1999; Sheldon *et al.* 1999). The Landsberg *erecta* (L*er*) ecotype possesses a loss-of-function allele at *FLC* (Koornneef *et al.* 1994; Lee *et al.* 1994; Michaels and Amasino 1999; Sheldon *et al.* 1999), which segregates in both sets of RI lines analyzed here. Analysis of the *FLC* coding sequence among the Landsberg *erecta*, Columbia, and Cape Verde Island ecotypes revealed a single-amino-acid substitution in the first exon in Landsberg *erecta* (data not shown). This substitution (Arginine → Lysine) is a conservative amino acid replacement, however, and is not likely to result in loss of function. Another differentiating genetic feature among these ecotypes is the presence of a 1.2-kb insertion in the first intron of the Landsberg *erecta* ecotype (data not shown). Whether this

insertion results in loss of function has not been determined.

As with *CRY2* at the top of chromosome 1, high-resolution mapping is required to address the extent to which *FLC* may have pleiotropic effects on multiple additional inflorescence traits and the extent to which multiple linked genes with independent effects on each trait segregate in these mapping populations. This will have clear relevance to the underlying genetics of plasticity and GEI, as a great deal of the positional overlap between sensitivity QTL and those detected by multiple-trait CIM occurs in the genomic regions where these two genes are located.

In conclusion, it should be noted that this study has considered only two distinct environments differing by a single factor. This factor (photoperiod length) has ecological relevance in that it is a principal determinant of seasonal change and can be a cue to initiate (or delay) reproduction (Evans 1975; Thomas and Vince-Prue 1997). Natural environments are assuredly much more complex, however, with far more environmental factors varying both spatially and temporally. It is likely that natural environments will amplify the complexity of GEI and its genetic underpinnings. QTL mapping studies that have examined multiple environments and/or included sex-specific effects (Fry *et al.* 1998; Jiang *et al.* 1999; Leips and Mackay 2000; Vieira *et al.* 2000) have found higher proportions of QTL exhibiting interactions with the environment. Nevertheless, it is clear that genetic and environmental factors are inextricably linked and phenotypic expression is determined by their joint effects.

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