Theor Appl Genet (2004) 108:261–273
DOI 10.1007/s00122-003-1432-8

ORIGINAL PAPER

C. Jantasuriyarat · M. I. Vales · C. J. W. Watson · O. Riera-Lizarazu

Identification and mapping of genetic loci affecting the free-threshing habit and spike compactness in wheat (*Triticum aestivum* L.)

Received: 6 February 2003 / Accepted: 13 August 2003 / Published online: 13 September 2003

Abstract Recombinant inbred lines of the International Triticeae Mapping Initiative (ITMI) mapping population were used to localize genetic loci that affect traits related to the free-threshing habit (percent threshability, glume tenacity, and spike fragility) and to spike morphology (spike length, spikelet number, and spike compactness) of wheat (*Triticum aestivum* L.). The ITMI population was planted in three environments during 1999 and 2000, and phenotypic and genotypic data were used for composite interval mapping. Two quantitative trait loci (QTL) that consistently affected threshability-associated traits were localized on chromosomes 2D and 5A. Coincident QTL on the short arm of 2D explained 44% of the variation in threshability, 17% of the variation in glume tenacity, and 42% of the variation in rachis fragility. QTL on chromosomes 2D probably represent the effect of *Tg*, a gene for tenacious glumes. Coincident QTL on the long arm of 5A explained 21% and 10% of the variation in glume tenacity and rachis fragility, respectively. QTL on 5A are believed to represent the effect of *Q*. Overall, free-threshing-related characteristics were predominantly affected by *Tg* and to a lesser extent by *Q*. Other QTL that were significantly associated with threshability-related traits in at least one environment were localized on chromosomes 2A, 2B, 6A, 6D, and 7B. Four QTL on chromosomes 1B, 4A, 6A, and 7A consistently affected spike characteristics. Coincident QTL on the short arm of chromosome 1B explained 18% and 7% of the variation in spike length and spike compactness, respectively. QTL on the long arm of 4A explained 11%, 14%, and 12% of the variation in spike length, spike compactness, and spikelet number, respectively. A QTL on the short arm of 6A explained 27% of the phenotypic variance for spike compactness, while a QTL on the long arm of 7A explained 18% of the variation in spikelet number. QTL on chromosomes 1B and 6A appear to affect spike dimensions by modulating rachis internode length, while QTL on chromosomes 4A and 7A do so by affecting the formation of spikelets. Other QTL that were significantly associated with spike morphology-related traits, in at least one environment, were localized on chromosomes 2B, 3A, 3D, 4D, and 5A.

Communicated by J. Dvorak

C. Jantasuriyarat · M. I. Vales · C. J. W. Watson · O. Riera-Lizarazu (✉)
Department of Crop and Soil Science,
Oregon State University,
107 Crop Science Building, Corvallis, OR 97331, USA
e-mail: Oscar.Riera@oregonstate.edu
Tel.: +1-541-7375879
Fax: +1-541-7371334

Introduction

The shift from the primitive to the cultivated forms of hexaploid wheat includes changes in morphological characters related to seed dispersal. These changes have affected spike dimensions, spike rachis fragility, spikelet disarticulation, awn development, pubescence, grain size, glume tenacity, and threshability. Genotypes with soft glumes that require limited mechanical action during the de-hulling process are considered free-threshing. Based on spike rachis fragility and threshability, hexaploid wheat (*Triticum aestivum* L.; 2*n*=6*x*=42, AABBDD) has historically been divided into six subspecies: *vulgare* (common wheat), *sphaerococcum*, *compactum*, *spelta*, *macha*, and *vavilovii* (Kimber and Sears 1983). Of these subspecies, *sphaerococcum*, *compactum*, and *vulgare* wheats have a tough rachis, and their seeds are easily de-hulled (free-threshing) (Sears 1947; Unrau 1950). On the other hand, subspecies *spelta*, *vavilovii*, and *macha* have a fragile rachis, and they are not free-threshing (Leighty and Boshnakian 1921; Kabarity 1966).

Previous studies revealed that numerous minor and major mutations were involved in the evolution of the free-threshing habit in hexaploid wheat. MacKey (1966) reported a polygenic system scattered throughout all three genomes that counteracted rachis brittleness and glume tenacity. Another system has been described where a major gene or gene complex, *Q*, located on the long arm of chromosome 5A, affects rachis fragility and glume tenacity as well as the speltoid character (MacKey 1954;

Sears 1954; Muramatsu 1963; Kerber and Rowland 1974). In general, the two major effects of the dominant allele of *Q* are speltoid suppression and square-headedness. Wheat plants with the dominant *Q* allele are also characterized by being shorter than wheat plants with the recessive *q* allele, their spikes are more compact, their spike rachises are non-brittle, and their seeds are free-threshing. All non-free-threshing wild wheats are believed to carry the recessive *q* allele, and all free-threshing tetraploid and hexaploid wheats carry dominant *Q* alleles. Variation on the phenotypic effects of *Q* in tetraploid and hexaploid wheats has been attributed to allelic variation (Tsunewaki 1966), to differences in their genetic backgrounds (Muramatsu 1986), and to the interaction of *Q* with other loci controlling glume tenacity and spike rachis fragility (Luo et al. 2000).

Another genetic system governing threshability is associated with the D genome. Kerber and Dyck (1969) found that a synthetic hexaploid (2*n*=6*x*=42, AABBDD) produced by combining a free-threshing tetraploid (2*n*=4*x*=24, AABB) with *Aegilops tauschii* (2*n*=2*x*=14, DD) was non-free-threshing despite the expected homozygosity for the *Q* factor. Using monosomic and telosomic analysis, Kerber and Rowland (1974) showed that the non-free-threshing character of synthetic hexaploid wheat was due to the presence of a partially dominant gene for tenacious glumes, *Tg*, on chromosome 2D. This gene, derived from *Ae. tauschii*, inhibited the expression of *Q*. Thus, a recessive *tg* allele as well as a dominant *Q* allele must be present for the expression of the free-threshing character.

Variations in the gross morphology of wheat spikes (shape, length, and density) are also influenced by *Q* (Sears 1954). In addition, two other major genes—*C* on the long arm of chromosome 2D (Rao 1972) and *S-D1*, on the long arm of chromosome 3D (Rao 1977)—have been described. *Q* determines whether a spike is normal (square-headed) or speltoid, while *C* determines whether a spike is lax or compacted. Sphaerococcoidy (round glumes and spherical grains) is observed in individuals with the recessive *s-D1* allele (Sears 1947). Genes that control photoperiodic response (*Ppd-A1*, *Ppd-B1*, and *Ppd-D1*) on homoeologous group 2 chromosomes have also been found to affect spike development (Sears 1947; Pugsley 1966; Worland 1996). In addition to these major loci, Kuspira and Unrau (1957) showed that a number of other minor genes were also involved in the expression of spike compactness components.

With the development of various DNA-based marker linkage maps and quantitative trait mapping methods, the identification and localization of genetic determinants of a given trait have been facilitated. A limited number of studies have addressed the genetic basis of the free-threshing character and gross spike morphology in hexaploid and tetraploid wheat (Kato et al. 1998, 2000; Simonetti et al. 1999; Sourdille et al. 2000). In some cases, these studies have confirmed the implication of known genes in the regulation of these traits but have also revealed the existence of other factors that are now only described as quantitative trait loci (QTL). The number and location of QTL can be variable and appear to depend on the population that was studied. With the aim of increasing our understanding of the genetic control of threshability and gross spike morphology, we used the International Triticeae Mapping Initiative (ITMI) mapping population to identify and localize genetic loci that affect these traits using composite interval mapping (CIM).

Materials and methods

Mapping population

The ITMI mapping population consists of recombinant inbred lines (RILs) developed from a cross between the hard red spring wheat cultivar Opata 85 (*Triticum aestivum* L.; 2*n*=6*x*=42, AABBDD genomes) and a synthetic hexaploid wheat, W-7984 (Nelson et al. 1995a, 1995b, 1995c; Van Deynze et al. 1995; Marino et al. 1996). For this study, seeds for the ITMI population, the synthetic hexaploid (W-7984), and Opata 85 were provided by Dr. C. Qualset, University of California, Davis.

Experimental design and phenotypic assessment

In April 1999, 110 RILs and their parents (Opata 85 and W-7984) were planted at Hyslop Farm Field Laboratory, Corvallis, Oregon (referred to subsequently as Hyslop farm). Opata 85 and W-7984 were planted in three replications. Due to the limited seed available in 1999, the RILs were planted in non-replicated 15-foot (4.6 m) rows. In April 2000, the mapping population and their parents (Opata 85 and W-7984) were planted at the University East Farm, Corvallis, Oregon (referred to subsequently as East farm). Each line was planted in four-row plots (4 m^2) in a randomized complete block design with two replications. The mapping population and parental lines were also planted in the greenhouse at Oregon State University (referred to subsequently as greenhouse) in February 2000. Each line was represented by one pot containing four plants arranged in a randomized complete block design with two replications.

The RILs were evaluated for the free-threshing habit by measuring percent threshability, glume tenacity, and rachis fragility or brittleness. In 1999, five randomly chosen mature spikes of each line were selected for evaluation. In 2000, eight and four randomly chosen mature spikes of each line were selected from plants grown at the East farm and in the greenhouse, respectively. To measure percent threshability, we processed the selected spikes through a gasoline-powered single-plant thresher, collecting both threshed and unthreshed seeds. Threshability was calculated as the percentage of completely threshed seeds from all seeds harvested. Rachis fragility or brittleness, in this study, was measured as the number of spike rachis fragments generated after mechanical threshing. To measure glume tenacity, we used a Hunter force gauge (Model LKG-1; AMETEK, Hatfield, Pa.) to measure the force (N = Newton) necessary to detach glumes at their base from four randomly selected spikelets per spike. Glume tenacity data were not collected for materials grown at Hyslop farm in 1999 because the force gauge was not available. Spike length was evaluated by measuring the length of the spike (in centimeters) from the base of the rachis to the tip of the uppermost spikelet, excluding the awns. The number of spikelets per spike was also counted. Spike compactness was calculated by dividing the number of spikelets in each spike by the length of the spike. In 1999, five randomly selected mature spikes from each line were evaluated. In 2000, eight and four randomly selected mature spikes from each line were evaluated in materials planted at the East farm and in the greenhouse, respectively.

Statistical analyses

Analyses of variance were performed using SAS version 7 (SAS Institute 1996). Heritability or repeatability estimates were calculated as $h^2=\sigma^2_g/(\sigma^2_g+\sigma^2_e/r)$ where σ^2_g is the variance among RILs, σ^2_e is the error variance among RILs, and r is the number of replications. Considering the effect of the genotypes random, we computed the estimates of variance components by equating mean squares to their expectations. Pearson's correlation coefficients were calculated to determine phenotypic correlation among traits.

Our phenotypic data and the genotypic data available in the GrainGenes database (http://wheat.pw.usda.gov) were used for QTL analysis. The linkage map (approx. 500 restriction fragment length polymorphism and simple sequence repeat loci regularly spaced every 10 cM) used has been described previously (Nelson et al. 1995a, 1995b, 1995c; Van Deynze et al. 1995; Marino et al. 1996; Röder et al. 1998; Pestsova et al. 2000). QTL were mapped using the CIM (Zeng 1993; 1994) procedure in the software package QTL Cartographer (Basten et al. 1994, 1999). Least square trait means from each environment (except Hyslop farm) and means across environments were analyzed with QTL Cartographer. A forward-selection backward-elimination stepwise regression procedure was used to identify co-factors for CIM. A 10-cM scan window was used for all analyses, and the likelihood ratio (LR) statistic was computed every 5 cM. Experiment-wide significance ($P<0.05$) thresholds for QTL identification were determined with 1,000 permutations. Tests for epitasis among QTL were performed using analysis of variance. For each test of epitasis, QTL were paired and tested with a model with main effects and a single interaction effect. Marker loci closest to the QTL peaks were used as factors in the analysis of variance. A significant interaction effect was interpreted as evidence for epitasis between QTL.

Results

Analysis of phenotypic data

Percent threshability, glume tenacity, and rachis fragility mean trait values for W-7984, Opata 85, and 110 RILs plus standard deviations, minima, and maxima for three environments are presented in Table 1. The parental lines (Opata 85 and W-7984) differed significantly for glume tenacity, percent threshability, and average number of rachis pieces after threshing in all environments. The estimated heritability or repeatability for glume tenacity, percent threshability, and rachis fragility ranged from 97% to 98%, 85% to 92%, and 86% to 89%, respectively. Percent threshability was negatively correlated ($P<0.001$) with both glume tenacity and rachis fragility (Table 2). Glume tenacity and rachis fragility were positively correlated ($P<0.001$).

Spike length, spikelet number, and spike compactness mean trait values for W-7984, Opata 85, and 110 RILs plus standard deviations, minima, and maxima for three environments are presented in Table 3. W-7984 and Opata 85 did not differ significantly for spike length in Hyslop farm. Opata 85 had significantly shorter spikes than W-7984 when grown at East farm and in the greenhouse, and Opata 85 had more compact spikes and a significantly larger number of spikelets per spike than W-7984 in all three environments. The estimated heritability or repeatability for spike length, spikelet number, and spike compactness ranged from 91% to 93%, 92% to 95%, and 94% to 95%, respectively. The number of spikelets per spike was positively correlated ($P<0.001$)

Table 1 Mean threshability-associated trait values of W-7984, Opata 85, and 110 recombinant inbred lines (RILs) derived from the cross between W-7984 and Opata 85 from three environments[a] (*SD* standard deviation)

Environments	Lines	Glume tenacity[b] (N)	Threshability[c] (%)	Rachis fragility[d]
Hyslop farm, 1999	W-7984	–	26.6c	7.47a
	Opata 85	–	97.7a	1.87b
	RILs-mean	–	70.8	4.90
	RILs-range	–	22.47–100	1.60–10.8
	RILs-SD	–	19.2	1.86
East farm, 2000	W-7984	5.9a	32.7b	4.50a
	Opata 85	0.4b	98.5a	1.59b
	RILs-mean	1.6	72.2	3.88
	RILs-range	0.3–4.9	29.7–97.6	1.48–8.41
	RILs-SD	1.0	19.1	1.60
	h^{2e}	0.97	0.92	0.89
Greenhouse, 2000	W-7984	6.1a	41.6b	6.75a
	Opata 85	0.3b	99.3a	1.25b
	RILs-mean	1.1	90.1	3.51
	RILs-range	0.3–4.2	48.6–100	1.37–7.62
	RILs-SD	0.7	11.4	1.40
	h^{2e}	0.98	0.85	0.86

[a] Mean trait values for the parental lines W-7984 and Opata 85 followed by the same letter are not significantly different at the 0.05 probability level using the F-protected LSD test
[b] Glume tenacity is expressed as the force (N = Newton) necessary to detach a glume at its base in a spikelet
[c] Threshability is expressed as the percentage of threshed seeds (number of threshed seeds/total number of seeds ×100)
[d] Rachis fragility is expressed as the number of spike rachis pieces per spike generated after threshing
[e] Heritability or repeatability estimates were calculated as: $h^2=\sigma^2_g(\sigma^2_g+\sigma^2_e/r)$ where σ^2_g is the variance among recombinant inbred lines, σ^2_e is the error variance among recombinant inbred lines, and r is the number of replications

Table 2 Peason's correlation coefficients between spike length, spikelet number, spike compactness, threshability, glume tenacity, and rachis fragility for 110 RILs derived from the cross between Opata 85 and W-7984

Trait	Spikelet number[a]	Spike compactness[b]	Threshability[c]	Glume tenacity[d]	Rachis fragility[e]
Spike length[f]	0.41***	–0.41***	–0.11*	0.11*	0.31***
Number of spikelets per spike	–	0.65***	0.31***	–0.32***	–0.05ns
Compactness	–	–	0.40***	–0.45***	–0.30***
Threshability	–	–	–	–0.71***	–0.70***
Glume tenacity	–	–	–	–	0.65***

*, ***, Correlation was significant at the 0.05, and <0.0001 probability level, respectively; ns, correlation was not significant
[a] The number of spikelets per spike
[b] The number of spikelets per unit length (centimeter) of spike
[c] The proportion (%) of threshed seeds
[d] The force (N = Newton) necessary to detach a glume at its base
[e] The number of spike rachis pieces per spike after threshing
[f] The length (centimeter) of a spike

Table 3 Mean spike morphology-associated trait values of W-7984, Opata 85, and 110 RILs derived from the cross between W-7984 and Opata 85 from three environments[a] (SD standard deviation)

Environments	Lines	Spike length (cm)	Spikelet number[b]	Spike compactness[c] (spikelets cm^{-1})
Hyslop farm, 1999	W-7984	9.02a	13.1b	1.45b
	Opata 85	9.04a	17.1a	1,89a
	RILs-mean	9.68	15.2	1.58
	RILS-range	7.70–12.6	10.8–19.8	1.23–2.10
	RILs-SD	1.00	1.45	0.16
East farm, 2000	W-7984	8.80a	13.2b	1.49b
	Opata 85	8.15b	18.2a	2.24a
	RILs-mean	8.69	15.6	1.80
	RILs-range	6.38–10.9	12.2–19.6	1.40–2.41
	RILs-SD	0.85	1.51	0.18
	h^{2d}	0.91	0.92	0.94
Greenhouse, 2000	W-7984	10.5a	17.9b	1.70b
	Opata 85	8.70b	21.2a	2.44a
	RILs-mean	9.26	19.2	2.10
	RILs-range	6.34–12.3	14.3–27.1	1.59–3.09
	RILs-SD	1.34	2.72	0.31
	h^{2d}	0.93	0.95	0.95

[a] Mean trait values for the parental lines W-7984, and Opata 85 followed by the same letter are not significantly different at the 0.05 probability level using the F-protected LSD test
[b] Number of spikelets per spike
[c] Spike compactness is expressed as the number of spikelets per unit length (cm) of the spike [number of spikelets / total length of spike (cm)].
[d] Heritability or repeatability estimates were calculated as: $h^2=\sigma^2_g/(\sigma^2_g+\sigma^2_e/r)$ where σ^2_g is the variance among recombinant inbred lines, σ^2_e is the error variance among recombinant inbred lines, and r is the number of replications

with both spike length and spike compactness (Table 2). The number of spikelets per spike and spike compactness were also positively correlated ($P<0.001$).

QTL detection

Glume tenacity

Glume tenacity was evaluated in only two environments (East farm and greenhouse). Four QTL were detected for glume tenacity—one on chromosome 2D, one on 2B, one on 5A, and one on 6A (Table 4, Fig. 1). The QTL on chromosome 2D and chromosome 5A were detected in the two environments tested (East farm and greenhouse). The QTL on chromosome 2D explained 19% and 23% of the phenotypic variance at East farm and in the greenhouse, respectively. The QTL on chromosome 5A explained 22% and 15% of the phenotypic variance at East farm and in the greenhouse, respectively. Two other QTL—one on chromosome 2B and one on chromosome 6A—were only detected in plants grown in the greenhouse. The QTL on chromosome 2B explained 14% of the phenotypic variance, while the QTL on chromosome 6A explained 9% of the phenotypic variance. W-7984 contributed the higher value allele for QTL on chromosomes 2D, 5A, and 6A, while Opata 85 contributed the higher value allele for the QTL on chromosome 2B. The QTL on chromosomes 2D and 5A were also detected in the combined analysis across environments. There were no significant epistatic interactions between QTL.

Table 4 Threshability-associated quantitative trait locus (QTL) location, significance, effect, and proportion of phenotypic variation explained based on composite interval mapping analysis

Trait	QTL symbol	Environment (abbreviation)	Chromosome arm	QTL peak position[a]	2-LOD support limit[b]	LR statistic[c]	R^{2d}	Additive effect[e]
Glume tenacity[f]	*QGt*	East farm, 2000 (EF00)	2DS	49.5 (*Xgwm261*)	45.0–59.5 (*Xbcd102-Xcdo1379*)	30.7	0.19	0.46
			5AL	117.5 (*Xabg391*)	107.5–125.6 (*Xcdo1326-Xgwm126*)	29.8	0.22	0.47
		Greenhouse, 2000 (GH00)	2BS	14.4 (*Xbcd1184*)	4.4–25.0 (*Xfba280-Xfbb121*)	23.6	0.14	–0.28
			2DS	49.5 (*Xgwm261*)	47.5–54.5 (*Xbcd102-Xcdo1379*)	40.0	0.23	0.36
			5AL	120.6 (*Xabg391*)	107.5–134.7 (*Xcdo1326-Xgwm126*)	25.5	0.15	0.29
			6AS	72.0 (*Xcdo270*)	64.0–83.0 (*Xfbb145-Xcdo1428*)	17.0	0.09	0.23
		Combined	2DS	49.5 (*Xgwm261*)	34.4–54.5 (*Xbcd1970-Xcdo1379*)	26.8	0.17	0.36
			5AL	117.5 (*Xabg391*)	107.5–125.6 (*Xcdo1326-Xgwm126*)	27.1	0.21	0.41
Thresha bility[g]	*QFt*	Hyslop farm, 1999 (HY99)	2BL	157.6 (*Xfba385*)	157.6–160.3 (*Xcmwg660-XksuD23*)	25.3	0.13	–6.97
			2DS	49.5 (*Xgwm261*)	49.5–54.5 (*Xgwm455-Xcdo1379*)	68.8	0.34	–11.6
			6DL	180.0 (*XksuD27*)	165.0–187.0 (*Xbcd1510-Xmwg2053*)	19.8	0.10	–6.29
		East farm, 2000 (EF00)	2DS	49.5 (*Xgwm261*)	49.5–54.5 (*Xgwm455-Xcdo1379*)	86.4	0.45	–13.1
			5AL	117.5 (*Xabg391*)	107.5–125.6 (*Xcdo1326-Xgwm126*)	21.6	0.10	–6.10
		Greenhouse, 2000 (GH00)	2AS	54.1 (*XksuD18*)	47.6–64.1 (*Xbcd348-Xfba178*)	17.0	0.09	–3.53
			2DS	49.5 (*Xgwm261*)	49.5–54.5 (*Xgwm455-Xcdo1379*)	53.7	0.38	–7.27
			6AS	75.9 (*Xcdo1315*)	71.0–80.9 (*Xfbb148-Xgwm494*)	24.1	0.15	–4.52
		Combined	2DS	49.5 (*Xgwm261*)	49.5–54.5 (*Xgwm455-Xcdo1379*)	86.2	0.44	–10.5
Rachis fragility[h]	*QRch*	Hyslop farm, 1999 (HY99)	2DS	49.5 (*Xgwm261*)	49.5–54.5 (*Xgwm455-Xcdo1379*)	63.3	0.33	1.09
			5AL	117.5 (*Xabg391*)	107.5–125.6 (*Xcdo1326-Xgwm126*)	29.8	0.16	0.75
		East farm, 2000 (EF00)	2BL	119.0 (*Xfba345*)	109.0–127.0 (*Xbcd1779-Xmwg546*)	16.7	0.07	–0.43
			2DS	49.5 (*Xgwm261*)	49.5–54.5 (*Xgwm455-Xcdo1379*)	81.2	0.43	1.05
			7BL	50.3 (*Xrz476*)	50.3–55.3 (*Xbcd178-Xwg514*)	22.6	0.09	–0.48
		Greenhouse, 2000 (GH00)	2DS	49.5 (*Xgwm261*)	49.5–54.5 (*Xgwm455-Xcdo1379*)	41.3	0.31	0.80
		Combined	2DS	49.5 (*Xgwm261*)	49.5–54.5 (*Xgwm455-Xcdo1379*)	78.9	0.42	1.00
			5AL	117.5 (*Xabg391*)	107.5–125.6 (*Xcdo1326-Xgwm126*)	18.2	0.10	0.46

[a] Position is expressed in centiMorgans. The nearest locus is indicated in brackets
[b] The range is expressed in centiMorgans. The flanking loci are indicated in brackets
[c] LR is the likelihood ratio test statistic 2 $\ln(L_0/L_1)$, where L_0/L_1 is the ratio of likelihoods between the hypothesis that there is no QTL in the tested interval (L_0) and the hypothesis that there is a QTL in the tested interval (L_1) (Basten et al. 1994, 1999)
[d] R^2 is the proportion of the phenotypic variance explained by the QTL after accounting for co-factors
[e] Additive effects indicate an additive main effect of the parent contributing the higher value allele: positive values indicate that higher value alleles are from W-7984 and the negative values indicate that higher value alleles are from Opata 85
[f] The force (N) required to detach a glume at its base
[g] Proportion (%) of threshed seeds
[h] The number of spike rachis pieces after threshing

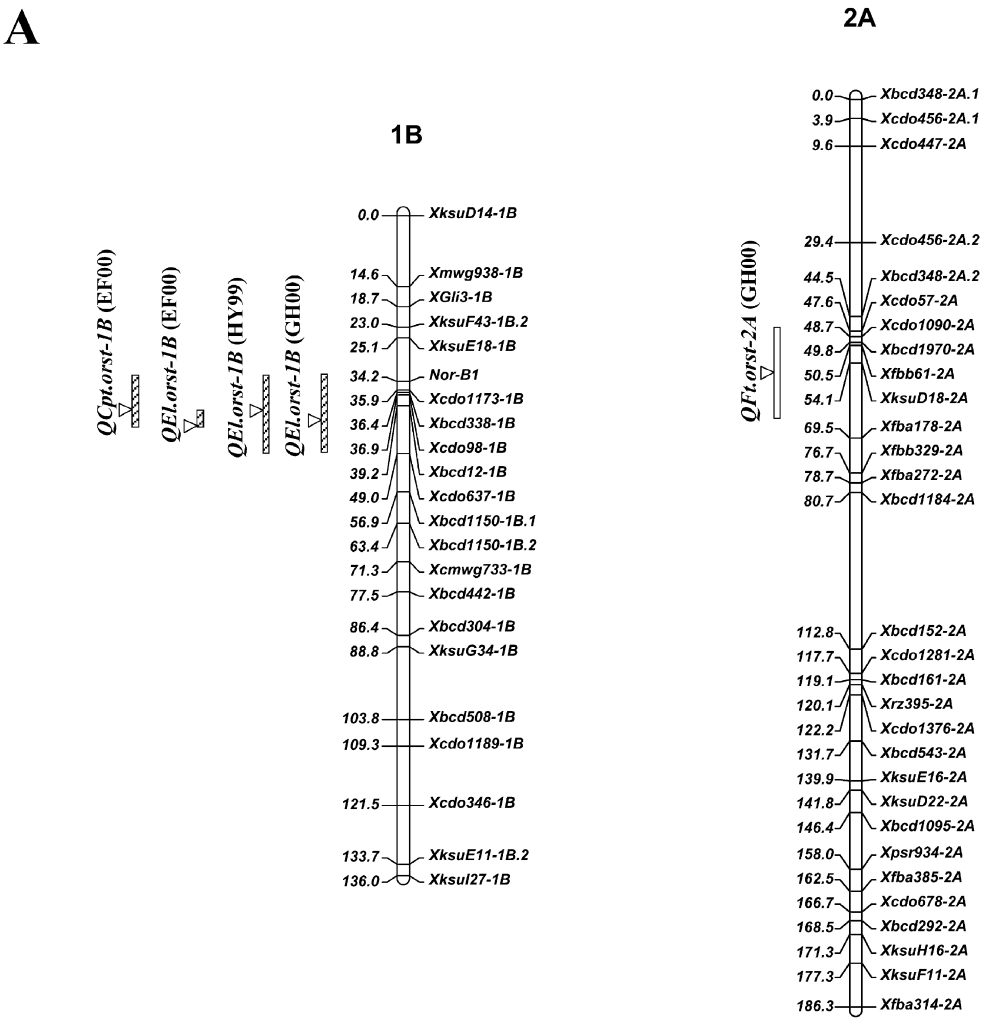


Fig. 1A–F Genetic linkage maps of wheat showing QTL distributed over 13 chromosomes. *Vertical bars* Supported intervals for QTL, *triangles* QTL peaks, *open bars* and *triangles* QTL that affected free-threshing-related traits, *stippled bars* and *triangles* QTL that affected spike morphology-associated traits. The environment and year in which the QTL were detected are given in *brackets*. *Tg* was placed on the map based on this study. *Tg2* and *Q* were placed on the map according to Simonetti et al. (1999) and Kato et al. (1998), respectively

Percent threshability

Six QTL that affected threshability were detected on chromosomes 2A, 2B, 2D, 5A, 6A, and 6D (Table 4, Fig. 1). The QTL on chromosome 2D was detected in the three environments tested and accounted for 34–45% of the phenotypic variance. At Hyslop farm, two other QTL were detected on chromosomes 2B and 6D. The QTL on chromosome 2B accounted for 13% of the phenotypic variance while that on chromosome 6D accounted for 10% of the phenotypic variance. At East farm, a QTL on chromosome 5A explained 10% of the phenotypic variance. In the greenhouse, QTL on chromosome 2A and chromosome 6A were also detected. The QTL on chromosome 2A explained 9% of the phenotypic variance, while the one on chromosome 6A explained 15% of this variance. Percent threshability increased with the Opata 85 alleles at all these QTL. The QTL on chromosome 2D was significant in the analysis across environments. The location of the QTL on chromosomes 2D, 5A, and 6A coincided with the QTL that affected glume tenacity.

Rachis fragility

The number of spike rachis pieces after threshing was affected by QTL on chromosomes 2B, 2D, 5A, and 7B. The QTL on chromosome 2D was detected in all three environments tested (Table 4, Fig. 1). The proportion of the phenotypic variance explained by this QTL ranged from 31% to 43%. At Hyslop farm, a QTL on chromosome 5A explained 16% of the phenotypic variance. At the East farm, QTL on chromosomes 2B and 7B were also detected. QTL on chromosomes 2B and 7B explained 7% and 9% of the phenotypic variance, respectively. The QTL on chromosomes 2D and 5A were also significant in the analysis across environments. W-7984 contributed the higher value allele at QTL on chromosomes 2D and 5A. Opata 85 alleles at QTL on chromosomes 2B and 7B increased rachis fragility. The location of the QTL on chromosomes 2D and 5A coincided with QTL that also affected glume tenacity and percent threshability.

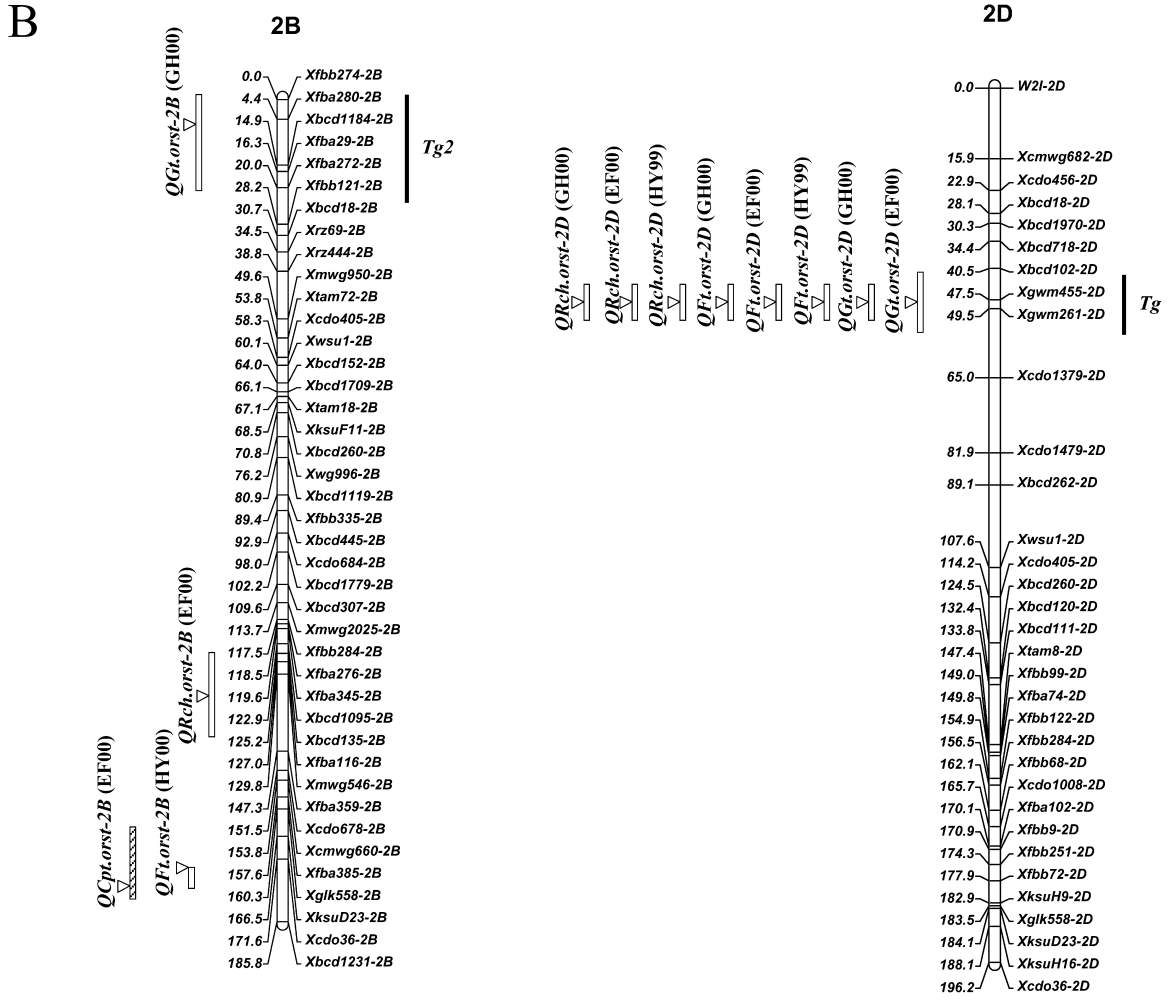

B
2B
QGt.orst-2B (GH00)
QRch.orst-2B (EF00)
QCpt.orst-2B (EF00)
QFt.orst-2B (HY00)
Tg2
0.0 Xfbb274-2B
4.4 Xfba280-2B
14.9 Xbcd1184-2B
16.3 Xfba29-2B
20.0 Xfba272-2B
28.2 Xfbb121-2B
30.7 Xbcd18-2B
34.5 Xrz69-2B
38.8 Xrz444-2B
49.6 Xmwg950-2B
53.8 Xtam72-2B
58.3 Xcdo405-2B
60.1 Xwsu1-2B
64.0 Xbcd152-2B
66.1 Xbcd1709-2B
67.1 Xtam18-2B
68.5 XksuF11-2B
70.8 Xbcd260-2B
76.2 Xwg996-2B
80.9 Xbcd1119-2B
89.4 Xfbb335-2B
92.9 Xbcd445-2B
98.0 Xcdo684-2B
102.2 Xbcd1779-2B
109.6 Xbcd307-2B
113.7 Xmwg2025-2B
117.5 Xfbb284-2B
118.5 Xfba276-2B
119.6 Xfba345-2B
122.9 Xbcd1095-2B
125.2 Xbcd135-2B
127.0 Xfba116-2B
129.8 Xmwg546-2B
147.3 Xfba359-2B
151.5 Xcdo678-2B
153.8 Xcmwg660-2B
157.6 Xfba385-2B
160.3 Xglk558-2B
166.5 XksuD23-2B
171.6 Xcdo36-2B
185.8 Xbcd1231-2B
2D
QRch.orst-2D (GH00)
QRch.orst-2D (EF00)
QRch.orst-2D (HY99)
QFt.orst-2D (GH00)
QFt.orst-2D (EF00)
QFt.orst-2D (HY99)
QGt.orst-2D (GH00)
QGt.orst-2D (EF00)
Tg
0.0 W2I-2D
15.9 Xcmwg682-2D
22.9 Xcdo456-2D
28.1 Xbcd18-2D
30.3 Xbcd1970-2D
34.4 Xbcd718-2D
40.5 Xbcd102-2D
47.5 Xgwm455-2D
49.5 Xgwm261-2D
65.0 Xcdo1379-2D
81.9 Xcdo1479-2D
89.1 Xbcd262-2D
107.6 Xwsu1-2D
114.2 Xcdo405-2D
124.5 Xbcd260-2D
132.4 Xbcd120-2D
133.8 Xbcd111-2D
147.4 Xtam8-2D
149.0 Xfbb99-2D
149.8 Xfba74-2D
154.9 Xfbb122-2D
156.5 Xfbb284-2D
162.1 Xfbb68-2D
165.7 Xcdo1008-2D
170.1 Xfba102-2D
170.9 Xfbb9-2D
174.3 Xfbb251-2D
177.9 Xfbb72-2D
182.9 XksuH9-2D
183.5 Xglk558-2D
184.1 XksuD23-2D
188.1 XksuH16-2D
196.2 Xcdo36-2D
C
3A
QSpn.orst-3A (GH00)
0.0 Xglk683-3A
10.1 Xtam61-3A
16.5 Xcdo460-3A
23.4 Xbcd1428-3A
24.9 Xfba91-3A
28.6 Xcdo395-3A
38.7 Xbcd1823-3A
41.5 Xbcd706-3A
42.9 Xmwg22-3A
44.3 Xmwg14-3A
49.1 XksuA6-3A
51.2 Xcdo638-3A
56.5 Xpsr903-3A
63.4 XATPase-3A
65.2 Xcdo1174-3A
65.6 Xbcd828-3A
67.4 Xcdo118-3A
73.3 Xwg177-3A
74.7 XksuH2-3A
78.8 Xbcd115-3A
80.9 Xfbb271-3A
82.1 Xmwg961-3A
88.4 Xfbb353-3A
97.7 Xfba175-3A
115.7 Xmwg30-3A
149.9 Xfbb293-3A.1
154.7 Xbcd1773-3A
161.7 Xtam63-3A
171.3 Xabc172-3A.1
195.1 Xfbb293-3A.2
203.9 Xmwg11-3A
209.8 Xcdo482-3A
231.1 Xbcd451-3A
3D
QSpn.orst-3D (GH00)
0.0 Xcdo549-3D
11.6 Xfbb370-3D
33.8 Xfba91-3D
44.6 Xfba241-3D
47.8 Xbcd1532-3D
50.1 Xcdo1435-3D
53.4 Xmwg22-3D
56.9 Xcdo407-3D
62.6 XksuA6-3D
77.5 Xmwg688-3D
82.6 Xbcd134-3D
88.9 Xabc176-3D
96.3 Xbcd288-3D
102.2 Xfbb237-3D
113.0 Xbcd22-3D
115.6 Xbcd1555-3D
118.4 Xwg110-3D
121.1 Xbcd515-3D
126.8 XksuH15-3D
130.8 Xbcd361-3D
134.7 Xfba310-3D
139.7 Xcnl2-3D
140.6 Xfbb269-3D
142.5 Xfbb316-3D
171.3 XksuG59-3D
4A
QCpt.orst-4A (GH00)
QCpt.orst-4A (EF00)
QSpn.orst-4A (EF00)
QCpt.orst-4A (HY99)
QSpn.orst-4A (HY99)
QEl.orst-4A (HY99)
0.0 Xfbb1-4A
8.6 Xfba40-4A
9.7 Xfba78-4A
14.8 Xfbb227-4A
17.3 Xcdo1387-4A
18.2 Xcdo959-4A
20.7 XksuF8-4A
24.7 Xfba211-4A
30.1 Xbcd8-4A
36.3 XksuG12-4A
39.4 Xwg622-4A
66.5 Xmwg549-4A
69.0 Xabg390-4A
70.9 Xbcd1670-4A
77.1 Xcdo475-4A
81.9 XksuD9-4A
85.4 Xbcd130-4A
88.1 Xfba231-4A
93.2 Xfbb154-4A
99.5 Xcdo545-4A
111.6 Xfbb114-4A
144.7 Xbcd129-4A

Fig. 1 (coninued)

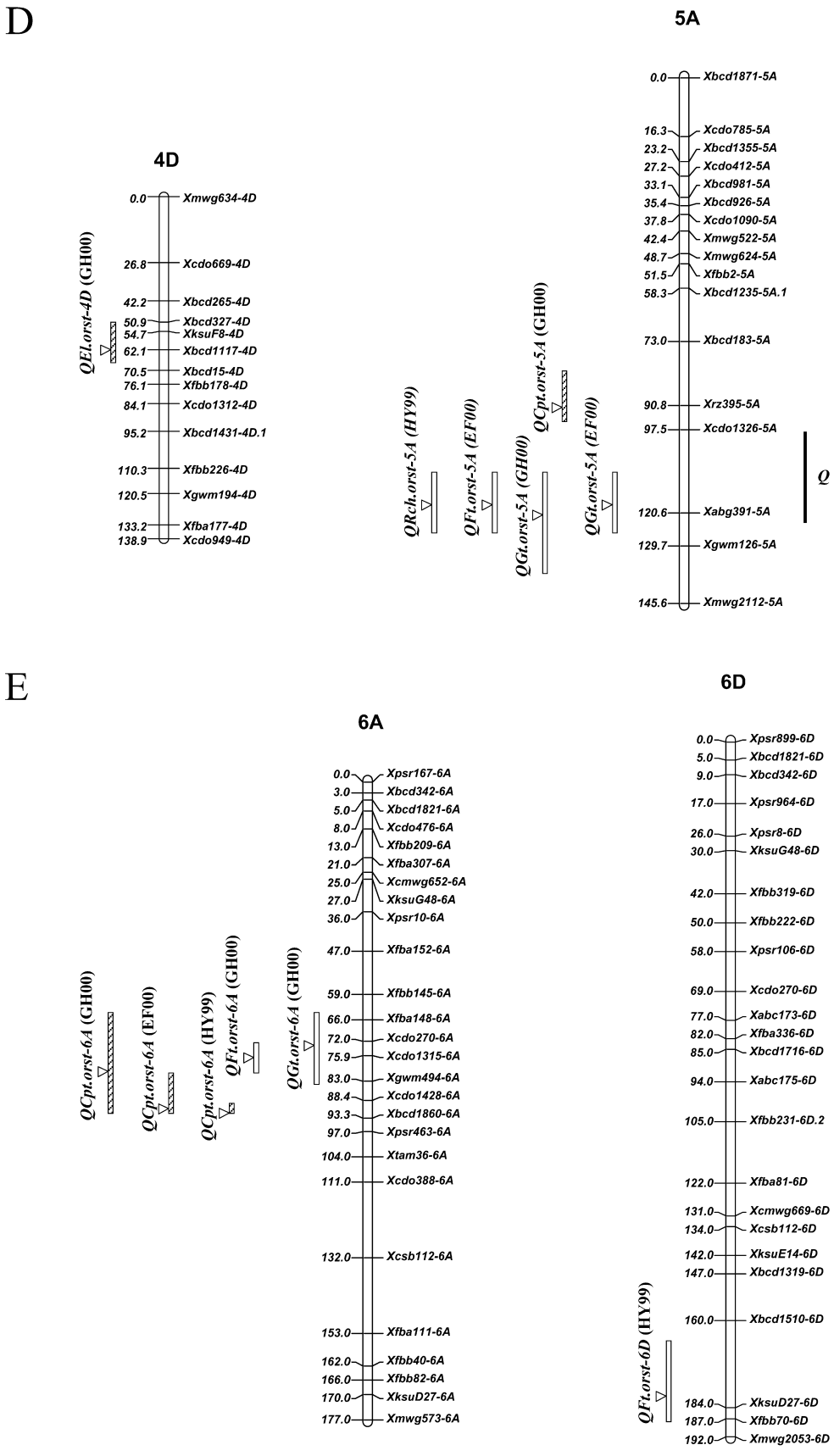
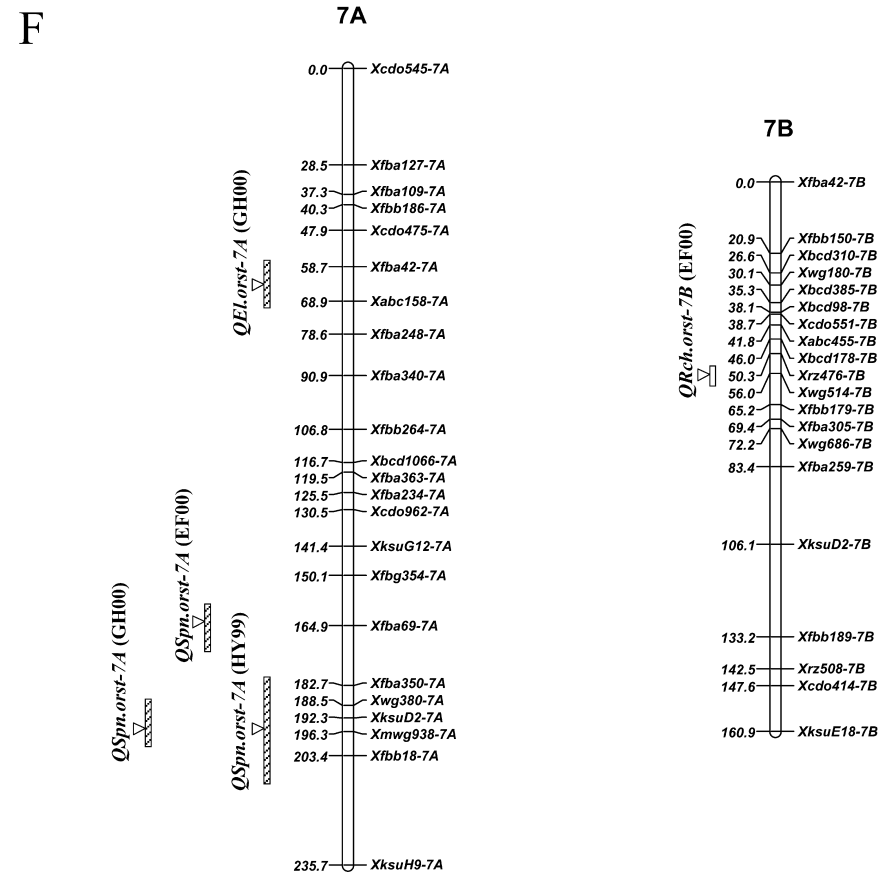


Fig. 1 (coninued)



Spike length

Four QTL that affected spike length were detected on chromosomes 1B, 4A, 4D, and 7A (Table 5, Fig. 1). The QTL on chromosome 1B was significant in all three environments, explaining between 15% and 23% of the phenotypic variance. The QTL on chromosome 4A accounted for 22% of the phenotypic variance at Hyslop farm. QTL on chromosomes 4D and 7A were significant in the greenhouse only. The QTL on chromosomes 4D and 7A accounted for 11% and 18% of the phenotypic variance, respectively. QTL on chromosomes 1B and 4A were also significant in the analysis across environments. Spike length was increased by the alleles from Opata 85 at QTL on chromosomes 1B and 4A. W-7984 contributed the higher value allele for QTL on chromosomes 4D and 7A. There were no significant interactions between QTL.

Spikelet number

Five QTL on chromosomes 3A, 3D, 4A, and 7A affected the number of spikelets per spike (Table 5, Fig. 1). The QTL on chromosome 4A was significant in two environments (Hyslop farm and East farm), explaining between 14% and 23% of the phenotypic variance. There were two QTL on chromosome 7A that affected spikelet number. One QTL on chromosome 7A accounted for 8% and 27% of the phenotypic variance at Hyslop farm and in the greenhouse, respectively. Another QTL on chromosome 7A was significant at East farm only, explaining 18% of the phenotypic variance. QTL on chromosomes 3A and 3D were significant in the greenhouse. The QTL on chromosomes 3A and 3D explained 11% and 16% of the phenotypic variance, respectively. QTL on chromosomes 4A and 7A were also significant in the analysis across environments. Spikelet number was increased by alleles of Opata 85 in all cases.

Spike compactness

Five QTL on chromosomes 1B, 2B, 4A, 5A, and 6A affected spike compactness (Table 5, Fig. 1). The QTL on chromosomes 4A and 6A were significant in all environments. The QTL on chromosome 4A accounted for 9–31% of the phenotypic variance, while the QTL on chromosome 6A accounted for 25–34% of the phenotypic variance. A QTL on chromosome 1B accounted for 13% of the phenotypic variance at East farm. A QTL on chromosome 2B was only significant at East farm, explaining 7% of the phenotypic variance. A QTL on chromosome 5A explained 14% of the phenotypic variance in the greenhouse. The QTL on chromosomes

Table 5 Spike morphology-associated trait locus location, significance, effect, and percentage of phenotypic variation accounted for based on composite interval mapping analysis

Trait	QTL Symbol	Environment (Abbreviation)	Chromosome arm	QTL peak position[a]	2-LOD support limit[b]	LR statistic[c]	R^{2d}	Additive effect[e]
Spike length[f]	*QEl*	Hyslop farm, 1999 (HY99)	1BS	36.4 (*Xbcd338*)	30.1–44.2 (*XksuE18-Xcdo637*)	29.0	0.15	−0.40
			4AL	93.2 (*Xfbb154*)	88.1–99.5 (*Xbcd130-Xfbb114*)	34.3	0.22	−0.48
		East farm, 2000 (EF00)	1BS	39.2 (*Xbcd12*)	36.4–39.2 (*Xcdo1173-Xcdo637*)	41.8	0.23	−0.42
		Greenhouse, 2000 (GH00)	1BS	39.2 (*Xbcd12*)	30.1–54.0 (*XksuE18-Xbcd1150.1*)	25.1	0.16	−0.60
			4DL	62.1 (*Xbcd1117*)	50.9–67.1 (*Xbcd265-Xbcd15*)	17.7	0.11	0.47
			7AS	63.7 (*Xabc158*)	52.9–68.7 (*Xcdo475-Xfba248)*	22.7	0.18	0.61
		Combined	1BS	39.2 (*Xbcd12*)	36.4–44.2 (*Xcdo1173-Xcdo637*)	34.2	0.18	−0.42
			4AL	98.2 (*Xcdo545*)	88.1–109.5 (*Xbcd130-Xfbb114*)	20.9	0.11	−0.33
Spikelet number[g]	*QSpn*	Hyslop farm, 1999 (HY99)	4AL	70.9 (*Xbcd1670*)	59.4–77.1 (*Xwg622-XksuD9*)	40.2	0.23	−0.71
			7AL	196.3 (*Xmwg938*)	182.7–213.4 (*Xfba69-XksuH9*)	16.0	0.08	−0.42
		East farm, 2000 (EF00)	4AL	70.9 (*Xbcd1670*)	59.4–75.9 (*Xwg622-Xcdo475*)	25.9	0.14	−0.60
			7AL	164.9 (*Xfba69*)	160.1–174.9 (*Xfbg354-Xfba350*)	24.1	0.18	−0.64
		Greenhouse, 2000 (GH00)	3AS	63.4 (*XATPase*)	61.5–67.4 (*Xpsr903-Xwg177*)	18.1	0.11	−0.90
			3DL	140.6 (*Xfbb269*)	130.8–147.5 (*XksuH15-XksuG59*)	22.5	0.16	−1.11
			7AL	196.3 (*Xmwg938*)	188.5–201.3 (*Xfba350-Xfbb18*)	38.7	0.27	−1.47
		Combined	4AL	70.9 (*Xbcd1670*)	54.4–77.1 (*Xwg622-XksuD9*)	21.5	0.12	−0.57
			7AL	201.3 (*Xfbb18*)	192.3–213.4 (*Xwg380-XksuH9*)	24.8	0.18	−0.68
Spike compactness[h]	*QCpt*	Hyslop farm, 1999 (HY99)	4AL	116.6 (*Xfbb114*)	104.5–131.6 (*Xcdo545-Xbcd129*)	18.3	0.17	0.07
			6AS	88.4 (*Xcdo1428*)	88.0–88.4 (*Xgwm494-Xbcd1860*)	43.5	0.25	−0.08
		East farm, 2000 (EF00)	1BS	36.4 (*Xbcd338*)	30.1–39.2 (*XksuE18-Xcdo637*)	26.7	0.13	0.07
			2BL	160.3 (*Xglk558*)	147.3–165.3 (*Xmwg546-XksuD23*)	16.0	0.07	−0.05
			4AL	111.6 (*Xfbb114*)	104.5–126.6 (*Xcdo545-Xbcd129*)	16.9	0.09	0.06
			6AS	88.0 (*Xcdo1428*)	80.9–88.4 (*Xcdo1315-Xbcd1860*)	46.0	0.25	−0.09
		Greenhouse, 2000 (GH00)	4AL	116.6 (*Xfbb114*)	104.5–131.6 (*Xcdo545-Xbcd129*)	27.9	0.31	0.18
			5AL	90.8 (*Xrz395*)	83.0–95.8 (*Xbcd183-Xcdo1326*)	23.6	0.14	−0.12
			6AS	80.9 (*Xgwm494*)	64.0–88.4 (*Xfbb145-Xbcd1860*)	30.8	0.34	−0.19
		Combined	1BS	36.4 (*Xbcd338*)	30.1–44.2 (*XksuE18-Xcdo637*)	16.3	0.07	0.05
			4AL	111.6 (*Xfbb114*)	104.5–136.6 (*Xcdo545-Xbcd129*)	18.3	0.14	0.07
			6AS	88.4 (*Xcdo1428*)	88.0–88.4 (*Xgwm494-Xbcd1860*)	52.0	0.27	−0.10

[a] Position is expressed in centiMorgans. The nearest locus is indicated in brackets
[b] The range is expressed in centiMorgans. The flanking loci are indicated in brackets
[c] LR is the likelihood ratio test statistic 2 ln(L_0/L_1), whereL_0/L_1 is the ratio of likelihoods between the hypothesis that there is no QTL in the tested interval (L_0) and the hypothesis that there is a QTL in the tested interval (L_1) (Basten et al. 1994, 1999)
[d] R^2 is the proportion of the phenotypic variance explained by the QTL after accounting for co-factors
[e] Additive effects indicates an additive main effect of the parent contributing the higher value allele: positive values indicate that higher value all eles are from W-7984 and the negative values indicate that higher value alleles are from Opata 85
[f] The length (centimeters) of a spike
[g] The number of spikelets per spike
[h] The number of spikelets per unit length (centimeters) of a spike

1B, 4A, and 6A were also significant in the analysis across environments. A W-7984 allele at QTL on chromosomes 4A and 1B increased spike compactness. Opata 85 contributed higher value alleles at QTL on chromosomes 6A, 2B, and 5A. The location of QTL on chromosome 1B and 4A coincided with the location of QTL that also affected spike length.

Discussion

Traits associated with the free-threshing habit

A quantitative trait mapping approach was used to identify loci that affected threshability, glume tenacity, and spike rachis fragility. Coincident QTL on the short arm of chromosome 2D (2DS) (near *Xgwm261*) controlled glume tenacity, threshability, and rachis fragility (Table 4, Fig. 1). A W-7984 allele at these QTL decreased threshability and increased glume tenacity and rachis fragility. Since *Ae. tauschii* was the donor of chromosome 2D in W-7984, the QTL near *Xgwm261* probably correspond to *Tg*, a gene for tenacious glumes. This interpretation is consistent with the report of Simonetti and co-workers (1999), who found a major QTL on chromosome 2B that affected threshability in a RIL population derived from a cross between durum wheat and *T. dicoccoides*. The QTL on chromosome 2B (named *Tg2*) was believed to represent a homoeolog of *Tg*. Based on map comparisons (see Röder et al. 1998 and Korzun et al. 1998), the location of our QTL on chromosome 2D and Simonetti et al.'s QTL on chromosome 2B are homoeologous. Thus, our study also represents the first report where *Tg* has been regionally localized on a linkage map of chromosome 2D.

A major QTL on the long arm of chromosome 5A (5AL) (near *Xabg391*) also affected rachis fragility and glume tenacity. An Opata 85 allele at this QTL reduced glume tenacity and rachis fragility. Based on map comparisons (Kato et al. 1998, 2000), this QTL probably correspond to *Q*. In contrast to the report of Simonetti et al. (1999), the effect of *Q* on threshability was minor (significant in only one environment) compared to *Tg* (Table 4, Fig. 1). This is probably due to different alleles of *Q* in the populations studied. In our case, W-7984, the synthetic hexaploid parent of the ITMI population, possesses a dominant *Q* allele from its free-threshing durum parent. Likewise, Opata 85, the common wheat parent of the ITMI population, also possesses a dominant *Q* allele. In theory, the ITMI RILs were homozygous for *Q*. Thus, variation in the effect of *Q* on threshability and threshability-associated traits was not expected. The minor effect of *Q* on threshability and rachis fragility, in this study, probably reflects minor allelic differences between hexaploid and tetraploid forms of *Q* (Muramatsu 1963, 1986; Tsunewaki 1966). The overriding effect of *Tg* on threshability and rachis fragility in our study is also consistent with the suggestion that *Tg* is a semi-dominant gene that interferes with the expression of *Q* (Kerber and Rowland 1974).

QTL that affected threshability-associated traits were also detected on chromosomes 2A, 2B, 6A, 6D, and 7B (Table 4, Fig. 1). The QTL on the short arms of 2B (2BS) and 2A (2AS) affected glume adherence and percent threshability, respectively. The location of the QTL on 2BS coincides with the location of *Tg2* (Simonetti et al. 1999). In addition, the location of the QTL on 2AS suggests the existence of another locus that may be homoeologous to *Tg* and *Tg2*. Coincident QTL on the short arm of chromosome 6A (6AS) that affected both glume tenacity and threshability were localized in the same region where *QFt.mgb-6A* was reported by Simonetti et al. (1999). QTL on the long arms of chromosomes 2B, 6D, and 7B affected threshability and rachis fragility. These QTL have not been reported previously. Since QTL for rachis fragility were not detected on homoeologous group 3 chromosomes, allelic variation at brittle rachis loci, *Br1* (Chen et al. 1998), *Br2*, and *Br3* (Watanabe and Ikebata 2000), appears to be non-existent in the ITMI population. From an agronomic perspective, it may be important that future studies address the relationship between QTL known to affect threshability and variation in the threshability of free-threshing wheat cultivars.

Overall, our study is consistent with reports suggesting that a minimum of two genes control the free-threshing character in crosses involving synthetic wheats (Kerber and Dyck 1969; Kerber and Rowland 1974; Villareal et al. 1996). Furthermore, our study suggests that free-threshing-related characteristics are predominantly affected by *Tg* and to a lesser extent by *Q*. Recently, Faris et al. (2003) proposed that an *APETALA2* (*AP2*)-like gene in wheat is a candidate for *Q*. In Arabidopsis, the *AP2* transcription factor acts upstream of major flower-specific homeotic genes and has been implicated in various aspects of flower and seed development (Jofuku et al. 1994; Okamuro et al. 1997). Thus, the effect of *Q* on various floral-related characters in wheat and the known functions of *AP2* in Arabidopsis appear to be consistent. The identity of *Tg* is unknown, but our genetic analysis suggests that its action during flower development should directly or indirectly intersect with that of *Q*.

Spike-associated traits

In this study, major QTL that affected spike length, spikelet number, and spike compactness were identified on chromosomes 1B, 4A, 6A, and 7A. A QTL that affected spike length was consistently detected on the short arm of chromosome 1B (1BS) (Table 5, Fig. 1). This QTL has also been reported in other studies that used the ITMI population (Börner et al. 2002; Li et al. 2002). In our study, this region on 1BS also affected spike compactness. An Opata 85 allele increased spike length and simultaneously decreased spike compactness. Our interpretation of this antagonistic relationship is that the QTL on 1BS represents a locus that alters spike length by

modulating spike rachis internode length. Since spike compactness is the ratio between the number of spikelets and spike length, increases in spike length without an increase in the number of spikelets would result in a simultaneous decrease in spike compactness.

A QTL on the long arm of 4A (4AL) also controlled spike length (Table 5. Fig. 1). The location of this QTL coincided with QTL reported by Börner et al. (2002) and Li et al. (2002). In our study, linked QTL that affected spikelet number and spike compactness were also detected on 4AL. Opata 85 alleles increased spike length and spikelet number but decreased spike compactness. Thus, one QTL affected spike length by altering the number of spikelets (or spike rachis internodes) that developed on a spike, while the other QTL affected spike length by changing rachis internode length.

A QTL in the short arm of chromosome 6A consistently affected spike compactness. A W-7984 allele at this locus reduced spike compactness. A minor QTL that affected spike length, in this region, was also reported by Börner et al. (2002), with a W-7984 QTL allele contributing to increased spike length. Thus, the effect of this QTL on spike length, like the QTL on 1BS, probably results from changes in spike rachis internode elongation.

QTL on the long arm of chromosome 7A (7AL) affected spikelet number (Table 5, Fig. 1) with Opata 85 contributing the higher value allele. Li et al. (2002) also detected a minor QTL for spike length and major QTL for spikelet number in this region of chromosome 7A. Thus, the effect of QTL on 7AL on spike length appears to be mediated through the formation of spikelets (or spike rachis internodes). A QTL on the short arm of 7A (7AS) was also detected in one environment. Börner et al. (2002) also detected a QTL in this region. W-7984 contributed the higher value allele in both studies. We could not determine whether spike length changes due to the QTL on 7AS were due to changes in rachis internode number, internode length, or both.

Other QTL that affected spike characteristics in one of the three environments studied include those on chromosomes 2B (spike compactness), 3A (spikelet number), 3D (spikelet number), 4D (spike length), and 5A (spike compactness) (Table 5, Fig. 1). Due to its location, the QTL on the long arm of 5A (5AL) does not appear to represent the effect of *Q*. Overall, the absence of linkage between major QTL and genes known to affect spike morphology such as *C*, *S-D1, Ppd-D1, Ppd-B1*, or *Ppd-A1* in this study suggests the absence of allelic variation at these loci. This is in contrast to the studies of Sourdille et al. (2000) and Li et al. (2002) where QTL on the short arms of chromosomes 2B (2BS) and 2D (2DS) were found to affect spikelet number. The location of the QTL on 2BS and 2DS corresponded to the location of two genes for photoperiodic response (*Ppd-B1* and *Ppd-D1,* respectively). Since photoperiod response genes are known to affect plant height, tillering, and spikelet number (Worland 1996), it is possible that the QTL on 2BS and 2DS may represent the effect of these genes. Börner et al. (2002) also detected a minor QTL on 2DS in 1 of the 11 environments studied. So, our inability to detect this QTL on 2DS also suggests that its expressivity will vary in different environments.

The identification of numerous loci controlling spike characteristics is not entirely unexpected since Ausemus et al. (1967) suggested that all chromosomes except 1B, 5A, and 6D were involved in the regulation of these traits. Our study of spike morphological characteristics in RILs of the ITMI population and a comparison of other studies that used the same population (Börner et al. 2002; Li et al. 2002) suggest that spike length was predominantly affected by QTL that either modulated rachis internode elongation (QTL on chromosomes 1B and 6A) or the formation of spikelets (QTL on chromosomes 4A and 7A). This is in contrast to Sourdille et al.'s (2000) study where the majority of QTL (on chromosomes 1A, 2D, 4A, and 5A) affected spike length by altering rachis internode length. These differences suggest that the genetic basis for variation in spike characteristics in these populations is quite different. Perhaps these differences could be combined and exploited in an attempt to manipulate spike dimensions in a crop improvement context.

Acknowledgements We thank Dr. C. J. Peterson for use of field facilities and Elizabeth Yoon, Ryan Millimaki, and Jumnit Hong for their technical assistance. A graduate scholarship to C. Jantasuriyarat from the Royal Thai Government and funding from the Oregon Wheat Commission are gratefully acknowledged.

References

Ausemus ER, McNeal FH, Schmidt JW (1967) Genetics and inheritance. In: Quisenbeery KS, Reitz LP (eds) Wheat and wheat improvement. ASA CSSA, SSSA Publ, Madison, pp225–267

Basten CJ, Weir BS, Zeng ZB (1994) ZMAP–a QTL cartographer. In: Smith C, Gavora JS, Chesnais BBJ, Fairfull W, Gibson JP, Kennedy BW, Burnside EB (eds) Proc 5th World Cong Genet Appl Livestock Prod: Comput Strategies Software, vol 22. University of Guelph, Guelph, Ontario, Canada, pp 65–66

Basten CJ, Weir BS, Zeng ZB (1999) QTL CARTOGRAPHER version 1.13. A reference manual and tutorial for QTL mapping. http://statgen.ncsu.edu/qtlcart/index.php

Börner A, Schumann E, Furste A, Coster H, Leithold B, Röder MS, Weber WE (2002) Mapping of quantitative trait loci determining agronomic important characters in hexaploid wheat (*Triticum aestivum* L.). Theor Appl Genet 105:921–936

Chen Q-F, Yen C, Yang J-L (1998) Chromosome location of the gene for brittle rachis in the Tibetan weedrace of common wheat. Genet Resour Crop Evol 45:407–410

Faris JD, Fellers JP, Brooks SA, Gill BS (2003) A bacterial artificial chromosome contig spanning the major domestication locus *Q* in wheat and identification of a candidate gene. Genetics 164:311–321

Jofuku KD, den Boer BGW, Van Montagu M, Okamuro JK (1994) Control of Arabidopsis flower and seed development by the homeotic gene *APETALA2*. Plant Cell 6:1211–1225

Kabarity A (1966) On the origin of the new cultivated wheat. II. Cytogenetical studies on the karyotypes of some *Triticum macha* varieties. Beitr Biol Pflanzen 42:339–346

Kato K, Miura H, Akiyama M, Kuroshima M, Sawada S (1998) RFLP mapping of the three major genes, *Vrn1, Q,* and *Bl*, on

the long arm of chromosome 5A of wheat. Euphytica 101:91–95
Kato K, Miura H, Sawada S (2000) Mapping QTLs controlling grain yield and its components on chromosome 5A of wheat. Theor Appl Genet 101:1114–1121
Kerber RE, Dyck PL (1969) Inheritance in hexaploid wheat of leaf rust resistance and other characters derived from *Aegilops squarrosa*. Can J Genet Cytol 11:639–647
Kerber RE, Rowland GG (1974) Origin of the threshing character in hexaploid wheat. Can J Genet Cytol 16:145–154
Kimber G, Sears ER (1983) Assignment of genome symbols in *Triticeae*. In: Sakamoto S (ed) Proc 6th Int Wheat Genet Symp. Maruzen, Kyoto, pp 1195–1196
Korzun V, Röder MS, Ganal MW, Worland AJ, Law CN (1998) Genetic analysis of dwarfing gene (*Rht8*) in wheat. Part I. Molecular mapping of *Rht8* on the short arm of chromosome 2D of bread wheat (*Triticum aestivum* L.) Theor Appl Genet 96:1104–1109
Kuspira J, Unrau J (1957) Genetic analyses of certain characters in common wheat using whole chromosome substitution lines. Can J Plant Sci 37:300–326
Leighty CE, Boshnakian S (1921) Genetic behaviour of the spelt form in crosses between *Triticum spelta* and *Triticum aestivum*. J Agric Res 7:335–364
Li WL, Nelson JC, Chu CY, Shi LH, Huang SH, Liu DJ (2002) Chromosomal locations and genetic relationships of tiller and spike characters in wheat. Euphytica 125:357–366
Luo MC, Yang ZL, Dvořák J (2000) The *Q* locus of Iranian and European spelt wheat. Theor Appl Genet 100:602–606
MacKey J (1954) Neutron and X-ray experiments in wheat and a revision of the speltoid problem. Hereditas 40:65–180
MacKey J (1966) Species relationships in *Triticum*. Hereditas 2:237–276
Marino CL, Nelson JC, Lu YH, Sorrels ME, Leroy P, Lopes CR, Hart GE (1996) RFLP-based linkage maps of the homoeologous group 6 chromosomes of hexaploid wheat (*Triticum aestivum* L. em. Thell). Genome 39:359–366
Muramatsu M (1963) Dosage effect of the spelt gene *q* of hexaploid wheat. Genetics 48:469–482
Muramatsu M (1986) The *vulgare* super gene, *Q*: its universality in *durum* wheat and its phenotypic effects in tetraploid and hexaploid wheats. Can J Genet Cytol 28:30–41
Nelson JC, Sorrels ME, Van Deynze AE, Lu YH, Atkinson M, Bernard M, Leroy P, Farris JD, Anderson JA (1995a) Molecular mapping of wheat: major genes and rearrangements in homoeologous groups 4, 5, and 7. Genetics 141:721–731
Nelson JC, Van Deynze AE, Autrique E, Sorrels ME, Lu YH, Merlino M, Atkinson M, Leroy P (1995b) Molecular mapping of wheat. Homoeologous group 2. Genome 38:516–524
Nelson JC, Van Deynze AE, Autrique E, Sorrels ME, Lu YH, Negre S, Bernard M, Leroy P (1995c) Molecular mapping of wheat. Homoeologous group 3. Genome 38:525–533
Okamuro JK, Caster B, Villaroel R, Van Montagu M, Jofuku KD (1997) The AP2 domain of *APETALA2* defines a large new family of DNA binding proteins in *Arabidopsis*. Proc Natl Acad Sci USA 94:7076–7081
Pestsova E, Ganal MW, Röder MS (2000) Isolation and mapping of microsatellite markers specific for the D genome of bread wheat. Genome 43:689–697
Pugsley AT (1966) The photoperiodic sensitivity of some spring wheats with special reference to the variety Thatcher. Aust J Agric Res 17:591–599
Rao MVP (1972) Mapping of the compactum gene C on chromosome 2D of wheat. Wheat Inf Serv 35:9
Rao MVP (1977) Mapping of the sphaerococcum gene 's' on chromosome 3D of wheat. Cereal Res Commun 5:15–17
Röder MS, Korzun V, Wendehake K, Plaschke J, Tixier MH, Leroy P, Ganal MW (1998) A microsatellite map of wheat. Genetics 149:2007–2023
SAS Institute (1996) SAS/STAT user's guide. Version 6.12. SAS Institute, Cary, N.C.
Sears ER (1947) The sphaerococcum gene in wheat. Genetics 32:102–103
Sears ER (1954) The aneuploids of common wheat. Mo Agric Exp Stn Res Bull 572
Simonetti MC, Bellomo MP, Laghetti G, Perrino P, Simeone R, Blanco A (1999) Quantitative trait loci influencing free-threshing habit in tetraploid wheats. Genet Resour Crop Evol 46:267–271
Sourdille P, Tixier MH, Charmet G, Gay G, Cadalen T, Bernard S, Bernard M (2000) Location of genes involving in ear compactness in wheat (*Triticum Aestivum*) by means of molecular markers. Mol Breed 6:247–255
Tsunewaki K (1966) Comparative gene analysis of common wheat and its ancestral species. II. Waxiness, growth habit and awnedness. Jpn J Bot 19:175–229
Unrau J (1950) The use of monosomes and nullisomes in cytogenetic studies in wheat. Sci Agric 30:66–89
Van Deynze AE, Dubcovsky J, Gill KS, Nelson JC, Sorrels ME, Dvorak J, Gill BS, Lagudah ES, McCouch SR, Appels R (1995) Molecular-genetic maps for group 1 chromosomes of *Triticeae* species and their relation to chromosomes in rice and oat. Genome 38:45–59
Villareal RL, Mujeeb-Kazi A, Rajaram S (1996) Inheritance of threshability in synthetic hexaploid (*Triticum turgidum* and *T. tauschii*) by *T. aestivum* crosses. Plant Breed 115:407–409
Watanabe N, Ikebata N (2000) The effects of homoeologous group 3 chromosomes on grain colour dependent seed dormancy and brittle rachis in tetraploid wheat. Euphytica 115:215–220
Worland AJ (1996) The influence of flowering time genes on environmental adaptability in European wheats. Euphytica 89:49–57
Zeng Z (1993) Theoretical basis for separation of multiple linked gene effects in mapping quantitative trait loci. Proc Natl Acad Sci USA 90:10972–10976
Zeng Z (1994) Precision mapping of quantitative trait loci. Genetics 136:1457–1468