Molecular Mapping of Segregation Distortion Loci in *Aegilops tauschii*

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ABSTRACT

Distorted segregation ratios of genetic markers are often observed in progeny of inter- and intraspecific hybrids and may result from competition among gametes or from abortion of the gamete or zygote. In this study, 194 markers mapped in an *Aegilops tauschii* F_2 population were surveyed for distorted segregation ratios. Region(s) with skewed segregation ratios were detected on chromosomes *1D*, *3D*, *4D*, and *7D*. These distorter loci are designated as *QSd.ksu-1D*, *QSd.ksu-3D*, *QSd.ksu-4D*, and *QSd.ksu-7D*. Three regions of segregation distortion identified on chromosome *5D* were analyzed in two sets of reciprocal backcross populations to analyze the effect of sex and cytoplasm on segregation distortion. Extreme distortion of marker segregation ratios was observed in populations in which the F_1 was used as the male parent, and ratios were skewed in favor of TA1691 alleles. There was some evidence of differential transmission caused by nucleo-cytoplasmic interactions. Our results agree with other studies stating that loci affecting gametophyte competition in male gametes are located on *5DL*. The distorter loci on *5DL* are designated as *QSd.ksu-5D.1*, *QSd.ksu-5D.2*, and *QSd.ksu-5D.3*.

DISTORTED segregation ratios in segregating populations may result from competition among gametes for preferential fertilization (Lyttle 1991). Competition among gametes may occur because of gametophyte genes expressed in the haploid gamete resulting in distorted segregation ratios. Many different genes are expressed during postmeiosis of the microspore and pollen development in angiosperms (Mascarenhas 1992). Genetic differences among pollen may lead to gametophyte competition and selection, which result in nonrandom fertilization. Alternatively, hybrid sterility genes that cause the abortion of specific gamete or zygote genotypes can give rise to segregation distortion.

The segregation distorter (*SD*) system of *Drosophila melanogaster* has been studied extensively (for review see Lyttle 1991). Segregation distortion is caused by a group of genetic elements near the centromere of chromosome *2*. Males heterozygous for an *SD* chromosome and a sensitive (SD^+) homologue transmit the *SD* chromosome in excess of the theoretical 50%. Several allelic interactions that cause dysfunction of the sperm that receive the sensitive SD^+ chromosome may occur.

Deviations from expected Mendelian segregation ratios of molecular markers have been reported in maize (Bentolila *et al.* 1992; Murigneux *et al.* 1993; Gardiner *et al.* 1993; Pereira and Lee 1995), barley (Graner *et al.* 1991; Heun *et al.* 1991; Thompson *et al.* 1991; Zivy *et al.* 1992; Kleinhofs *et al.* 1993; Devaux *et al.* 1995), pearl millet (Busso *et al.* 1995; Liu *et al.* 1996), common bean (Paredes and Gepts 1995), and rice (Causse *et al.* 1994; Harushima *et al.* 1996; Yamagishi *et al.* 1996; Xu *et al.* 1997), as well as many other plant species.

Zhang and Dvorák (1990) characterized a set of recombinant chromosomes derived from homoeologous recombination between *Lophopyrum ponticum* (Podp.) Löve chromosome *7Ag* and wheat chromosome *7D*. They concluded that the *7Ag* chromosome possessed a locus that caused segregation distortion resulting from preferential transmission and designated it *Sd-1*. Furthermore, several other Lophopyrum chromosomes of homoeologous group *7* have been found to cause segregation distortion by being preferentially transmitted through male gametes (Dvorák 1980; Scoles and Kibirige-Sebunya 1983).

The advent of molecular markers such as RFLPs has permitted the construction of extensive genetic linkage maps for many crop species. Such linkage maps can be used to survey the entire genome for loci with distorted segregation ratios. *Aegilops tauschii* Coss. (syn. *Ae. squarrosa* L., syn. *Triticum tauschii* (Coss.) Schmal. $2n = 14$, DD) is the D genome progenitor of common bread wheat (*T. aestivum* L. em. Thell., $2n = 6x = 42$, AABBDD). Kam-Morgan *et al.* (1989) began mapping in an *Ae. tauschii* F_2 population derived from the cross of accessions TA1691, var. *meyeri*, and TA1704, var. *typica*. They noted that all five loci of the rudimentary chromo-

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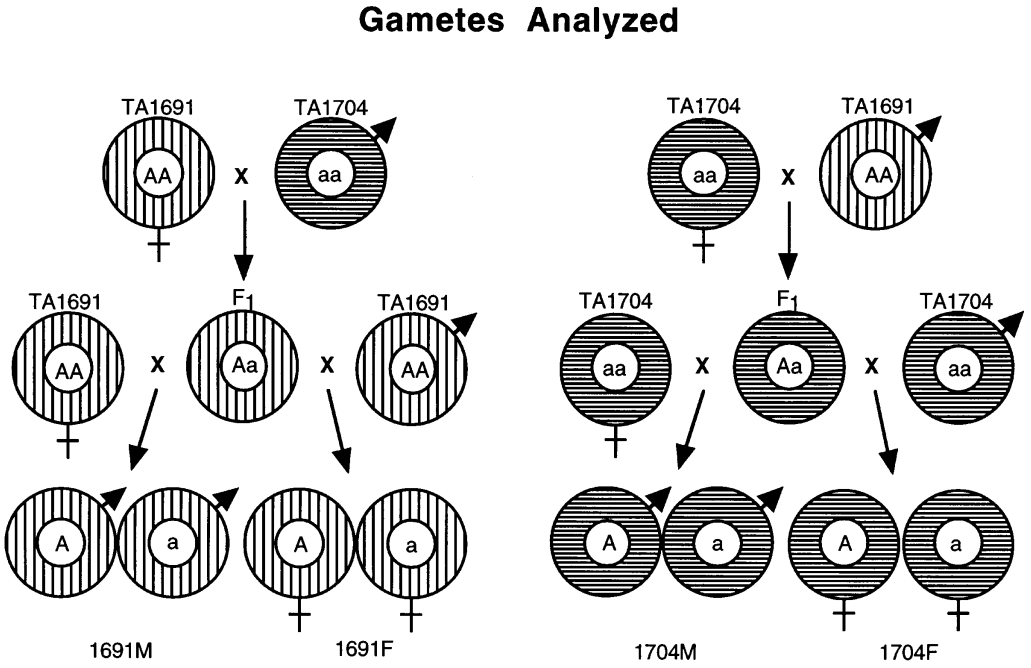

Figure 1.—Crosses made and gametes analyzed in reciprocal BC populations of *Ae. tauschii*.

some *5D* linkage map deviated from the expected 1:2:1 ratio. In 1991, a genetic linkage map of the same population consisting of 178 loci was presented by Gill *et al.* Molecular mapping in this population has continued with the goal of constructing a high-density genetic linkage map of *Ae. tauschii* (E. V. Boyko, K. S. Gill, L. Mickelson-Young, S. Nasuda, W. J. Raupp, D. S. Hassawi, A. K. Fritz, D. Namuth, N. L. V. Lapitan and B. S. Gill, unpublished results).

The objectives of this study were to identify chromosome regions with distorted segregation using the F_2 mapping population and to further analyze regions with distorted segregation on chromosome *5D* in reciprocal backcross (BC) populations. The experimental plan was designed to test the effect of sex (male *vs.* female) and cytoplasm on the segregation distortion phenomenon.

MATERIALS AND METHODS

Plant materials: The original F_2 mapping population was described in Kam-Morgan *et al.* (1989) and Gill *et al.* (1991), and it consisted of 56 F_2 progeny derived from the cross of *Ae. tauschii* accessions TA1691, var. *meyeri* (female), and TA1704, var. *typica* (male). The international numbers in the wild wheat relatives database (ICARDA/GRU) for TA1691 and TA1704 are 7804 and 8235, respectively. Two sets of reciprocal BC populations were subsequently developed for this study (Figure 1). The F_1 plant derived from the cross TA1691/TA1704 was backcrossed as the male parent [1691M = TA1691 × (TA1691 × TA1704)] and as the female parent [1691F = (TA1691 × TA1704) × TA1691] to TA1691. Additionally, the F_1 derived from the cross TA1704/TA1691 was backcrossed as the male parent [1704M = TA1704 × (TA1704 × TA1691)] and as the female parent [1704F = (TA1704 × TA1691) × TA1704] to TA1704. Forty-three BCF_1 progeny, each from the 1691M and 1704F populations, were used in mapping experiments and for segregation analysis, and 45 and 47 BCF_1 progeny were used from the 1704M and 1691F populations, respectively.

Analysis: The F_2 map consisted of 194 codominant markers. All markers were subjected to a Chi-squared test for fit to a 1:2:1 ratio using the computer program qGene v.2.27n (J. C. Nelson, unpublished results) to identify markers with distorted segregation ratios. We further characterized the direction of distortion by plotting the genotype frequencies of each marker along the genetic linkage map and applying a median skewness curve. The range of expected frequency at the 0.95 probability level for the average number of progeny is 25 ± 12.7% and 50 ± 14.5% for homozygotes and heterozygotes, respectively.

RFLP markers on chromosome *5D* showing distorted segregation were selected as probes for developing maps of the corresponding region of this chromosome in the four BC populations. Markers used to develop maps in the BC populations were subjected to a Chi-squared test for fit to a 1:1 ratio.

Methods for DNA isolation, restriction enzyme digestion, gel electrophoresis, Southern blotting, probe labeling and hybridization, and membrane washing were done as described in Gill *et al.* (1993). Generation of genetic linkage maps for the F_2 population was performed as described in Gill *et al.* (1991), with the exception of 17 markers that have subsequently been added to the maps. Linkage maps were developed using MAPMAKER v.2.0 (Lander *et al.* 1987) with an LOD of 2.0 and the Kosambi mapping function (Kosambi 1944).

RESULTS

F_2 population: The 194 markers spanning the F_2 map covered 1650 cM, resulting in an average of 8.5 cM per marker interval. Markers with the greatest degree of deviation within each region of strong distortion, as well as their Chi-square values, are given in Table 1. To evaluate the direction of segregation distortion, the frequency for individual marker genotypes with data from

TABLE 1

Segregation distortion ratios in the *Aegilops tauschii* F_2 population

Marker	Map position[a]	Chromosome arm[b]	χ^2	P	TA1691 Homozygotes (%)	Heterozygotes (%)	TA1704 homozygotes (%)
Xcmwg706	167.5	*1DL*	14.5	0.0007	47.2	39.6	13.2
Xwg177	68.9	*3DS*	10.7	0.0048	41.8	47.3	10.9
XksuF8	0	*4DS*	8.3	0.0155	40.0	47.3	12.7
Xcdo677	13.0	*5D?-1*	11.8	0.0027	38.0	56.0	6.0
Xtag614	51.9	*5DL-2*	12.3	0.0022	18.0	73.0	9.0
Xwg1026	133.5	*5DL-3*	8.3	0.0158	42.0	38.0	20.0
Xtag439	126.0	*7DS*	7.92	0.0191	9.3	55.6	35.2

Genotype percentages, chromosome locations, and Chi-squared values of marker segregation ratios with the greatest degree of deviation within each region of distortion are given. The theoretical expected genotype frequencies are 25 and 50% for homozygotes and heterozygotes, respectively.

[a] The map position indicates the centimorgan distance from the first marker on the short arm of the genetic linkage map.

[b] *S* and *L* indicate the short and long arm of the chromosome, respectively. *5D?-1*, *5DL-2*, and *5DL-3* correspond to distorted *regions 1*, *2*, and *3* on chromosome *5D*, respectively.

an average of 54 F_2 plants were plotted for the 194 markers along the genetic linkage map, and median skewness curves were applied (Figure 2). Fifty-seven (29%) markers had segregation ratios that deviated significantly ($P < 0.05$) from the expected 1:2:1 ratio. At the 0.05 level of probability, we would expect 9.7 markers to show distorted segregation ratios caused by random chance. All seven chromosomes had markers with distorted segregation ratios, but large regions of markers showing significant ($P < 0.05$) segregation distortion were detected on chromosomes *1D*, *3D*, *4D*, *5D*, and *7D*.

The marker *Xpsr161* on *1DS* had a segregation ratio that was significantly ($P = 0.03$) distorted because of a deficiency of heterozygous genotypes (32%) and excess TA1691 homozygotes (38%). Ratios of markers adjacent to *Xpsr161* were not distorted; therefore, this marker may be distorted by random chance.

Eight markers spanning 56.2 cM on *1DL* had distorted segregation ratios caused by a deficiency of homozygous TA1704 genotypes and excess TA1691 homozygotes, while the number of heterozygous genotypes was normal. The marker with the greatest degree of segregation distortion in this region was *Xcmwg706* (Table 1), suggesting that it was tightly linked to a segregation distorter locus.

The marker *XksuI24* gave a segregation ratio of 17:34:3 (31.5:63:5.6%) for TA1691 homozygotes/heterozygotes/TA1704 homozygotes, respectively, and was the only significantly ($P = 0.004$) distorted marker on chromosome *2D*. The ratio of *XksuI24* may have been distorted by random chance, but in a sample size of 194 it had the probability of occurring less than once (0.78). Therefore, it is possible that this marker represents a minor distorter locus, but a more thorough investigation of this locus is needed before more accurate conclusions can be drawn.

Of the 32 markers on chromosome *3D*, 17 showed distorted segregation ratios. The markers with the greatest deviation were clustered distally on the short arm, but the distorted region extended through the centromere. This distorted region encompassed 107.1 cM and was the result of excess homozygous TA1691 genotypes and a deficiency of homozygous TA1704 genotypes, while the number of heterozygotes was as expected. The most skewed ratio was given by *Xwg177* (Table 1), suggesting that *Xwg177* is linked to a distorter locus on *3DS*.

XksuE14 mapped distally on *3DL* and had a segregation ratio that was significantly ($P = 0.04$) distorted as a result of a deficiency of TA1704 homozygotes (10.9%) and excess TA1691 homozygotes (34.5%). Adjacent markers were not skewed, suggesting that this marker might have been distorted by random chance. As discussed below, however, *XksuE14* also detected a locus on chromosome *6D* and was the only marker with a distorted segregation ratio on that chromosome.

Four markers clustered on *4DS* showed distorted segregation ratios caused by excess homozygous TA1691 genotypes and a deficiency of homozygous TA1704 genotypes, while the number of heterozygotes was normal. *XksuF8* was the most distal marker on *4DS* and had the greatest degree of distortion in this region (Table 1), indicating that a minor distortion factor may exist on the tip of *4DS*.

The map of chromosome *5D* consisted of 37 markers, 18 of which had distorted segregation ratios. The first nine markers of this chromosome (*region 1*), which spanned 20.6 cM from the short arm through the centromere, had skewed ratios caused by excess TA1691 homozygotes and a lack of TA1704 homozygotes, while the number of heterozygotes was as expected. An adjacent region of distortion (*region 2*), which included six

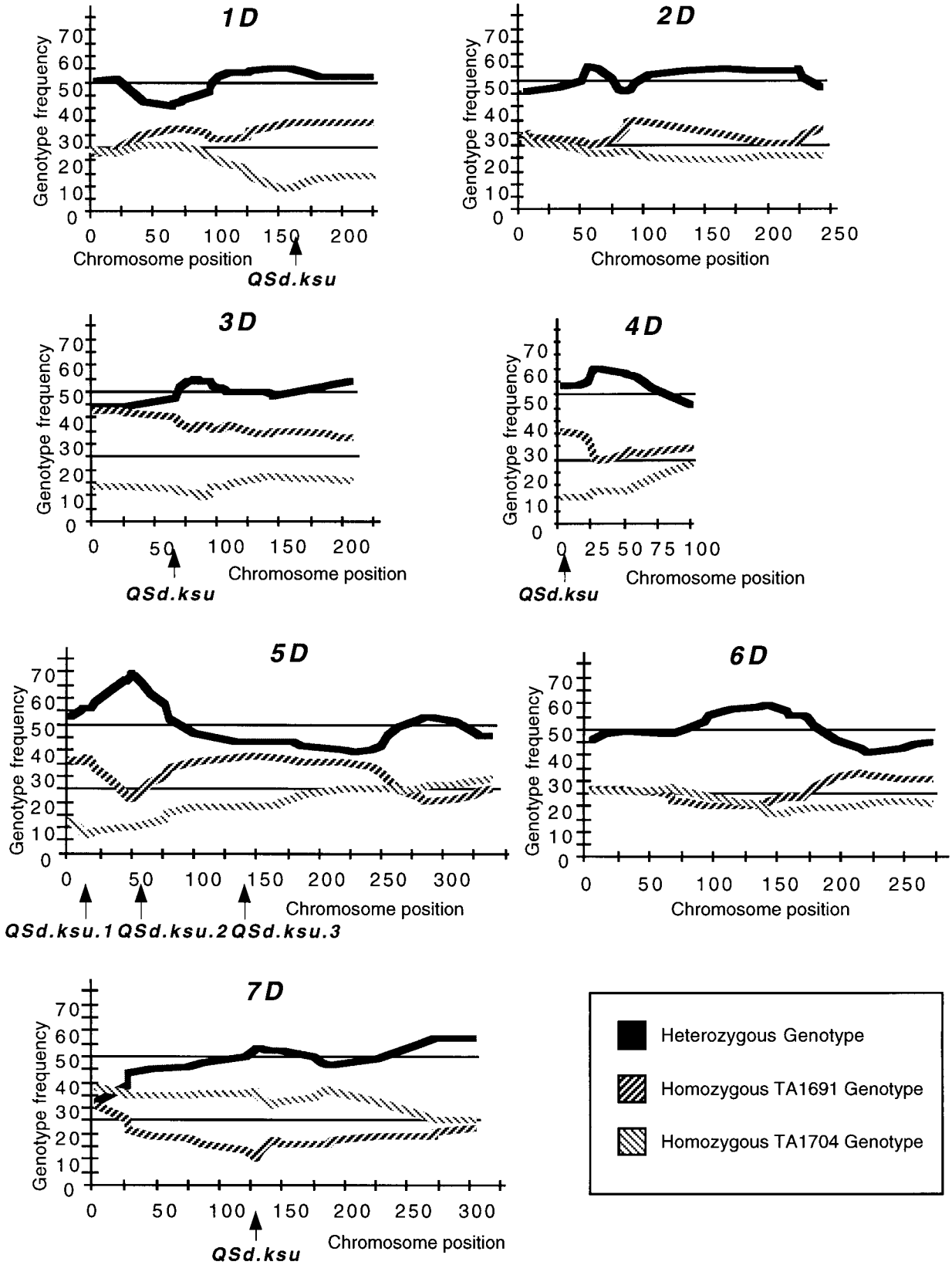

Figure 2.—Genotype frequencies as a function of the genetic linkage map. Frequencies of genotypes in F_2 plants derived from the cross of *Ae. tauschii* accessions TA1691 and TA1704 are plotted along the genetic linkage map with a median skewness curve applied. For each chromosome, the left and right ends of the *x*-axis correspond to the short and long arms of the genetic linkage map, and the *y*-axis indicates the genotype percentages observed for each marker. The legend indicates the genotype that each line represents. In normal segregation, the heterozygous and homozygous genotypes account for 50 and 25%, respectively. For this study, the 0.95 confidence limits are 50 ± 14.5 and 25 ± 12.7% for heterozygous and homozygous genotypes, respectively.

markers spanning 25.3 cM on a proximal region of *5DL* (positions 33–58.3 cM), was skewed in favor of heterozygous genotypes and disfavor of homozygous TA1704 genotypes, while the number of homozygous TA1691 genotypes was normal. The markers with the most significantly distorted segregation ratios in *regions 1* and *2* were *Xcdo677* and *Xtag614*, respectively (Table 1).

Xwg1026 and *Xcdo400* mapped to positions 133.5 and 147.8 cM (*region 3*), respectively, and had segregation ratios that deviated significantly ($P < 0.05$) because of a deficiency of heterozygotes and an excess of homozygous TA1691 genotypes. There was no deviation in the number of homozygous TA1704 genotypes. The marker *Xtag754* mapped at position 242.5 cM and showed the same direction of distortion as *Xwg1026* and *Xcdo400*. The distorted ratio of this marker may have been caused by random chance, or it may be possible that it was affected by the distorter locus of *region 3*. From these data, it appears that at least three distorter loci may be present on chromosome *5D*.

The chromosome *6D* map consisted of 32 markers, and only one of these had a segregation ratio that deviated significantly ($P = 0.011$) from the expected ratio. *XksuE14*, located at position 216.6 cM, had a segregation ratio of 23:20:11 (42.6:37:20.4%) for TA1691 homozygotes/heterozygotes/TA1704 homozygotes, respectively. Because adjacent markers as close as 4 cM were not distorted, *XksuE14* may have had a skewed ratio caused by random chance. This marker, however, was also a single distorted marker on the distal region of *3DL*, which may be indicative of a marker-specific effect.

Seven of the 28 markers on the chromosome *7D* linkage map exhibited distorted segregation ratios. *Xpsr160* mapped to the tip of *7DS* and gave a significantly ($P = 0.006$) distorted segregation ratio of 16:15:21 (30.8:28.8:40.4%) for TA1691 homozygotes/heterozygotes/TA1704 homozygotes, respectively. The ratio of the nearest proximal marker was not skewed, but mapped 25 cM from *Xpsr160*. Therefore, a distorter locus may exist on the distal tip of *7DS*, but we cannot discount the possibility that the segregation ratio of this marker was distorted by random chance.

Four markers clustered within 8.5 cM (positions 118.5–127 cM) near the centromere of *7DS* showed significant ($P < 0.05$) deviations from the expected segregation ratios because of a deficiency of TA1691 homozygotes and an excess of homozygous TA1704 genotypes, while the number of heterozygotes was as expected. The strongest deviation ($P = 0.019$) was observed by *Xtag439* (Table 1). This locus may be homologous to the locus reported by Zhang and Dvorák (1990).

The marker *Xwg380* detected a locus on *7DL* at position 189.1 cM. The segregation ratio of this marker was also significant ($P < 0.05$) because of an excess of TA1704 homozygotes and a deficiency of TA1691 homozygotes, while the number of heterozygotes was normal. It is likely, however, that this locus was skewed as a result of random chance.

BC populations: Reciprocal BC populations based on male and female meiosis were used to monitor segregation of loci through male and female gametes and to study cytoplasmic interactions. Genetic clones mapping to the major regions of segregation distortion on chromosome *5D* were selected from the F_2 map and the group *5* physical maps (Gill *et al.* 1996) to be used as probes to develop genetic linkage maps of the corresponding region of *5D* in the reciprocal BC populations. Thirteen and 10 probes were mapped in the 1691M/F and 1704M/F populations, respectively (Figure 3). The order of markers mapped in the BC populations agreed relatively well with the order on chromosome *5D* of the F_2 map, with a few exceptions.

Eleven of the 13 markers making up the 1691M map showed segregation ratios that deviated significantly ($P < 0.05$) from the expected 1:1 ratio (Figure 3). All 11 markers with distorted ratios were skewed in favor of TA1691 homozygotes, while there was a deficiency of heterozygous genotypes. The segregation ratios of five of these markers, clustered within 4.6 cM, were distorted at the 0.00005 level of probability and gave maximum segregation distortion ratios of 37:6 (86:14%) for TA1691 homozygotes/heterozygotes, respectively. Four of these markers corresponded to distorted *region 1* of the chromosome *5D* F_2 linkage map.

The fifth marker in this cluster, *XksuH1*, along with *Xwg530* and *Xcdo1168.1*, represented distorted *region 2* of the chromosome *5D* F_2 linkage map. *XksuH1* had a ratio of 37:6 (86:14%) for TA1691 homozygotes/heterozygotes, respectively. *Xcdo400* was distorted in *region 3* of the *5D* F_2 map and was also significantly ($P < 0.05$) distorted in the 1691M population. It gave a ratio of 28:15 (65.1:34.9%) for TA1691 homozygotes/heterozygotes, respectively.

The 13 markers that were mapped in the 1691M population were also mapped in the 1691F population. In the 1691F population, none of the 13 markers showed deviation from the expected 1:1 ratio. The relative order of markers between the 1691M and 1691F populations was fairly consistent, with only a few exceptions (Figure 3). Because population sizes were quite small, we would expect some discrepancy in the order of markers that are tightly linked. Indeed, one rearrangement in the order of closely linked markers was observed between the 1691M and 1691F maps. Lorieux *et al.* (1995) have shown that the order of markers in linkage groups may be affected by segregation distortion.

We also observed differences in the magnitude of recombination between markers on the two linkage maps. It appeared that there may have been more recombination in female gametes. But the population sizes may not be large enough to draw accurate conclusions regarding the comparison of recombination frequencies between markers.

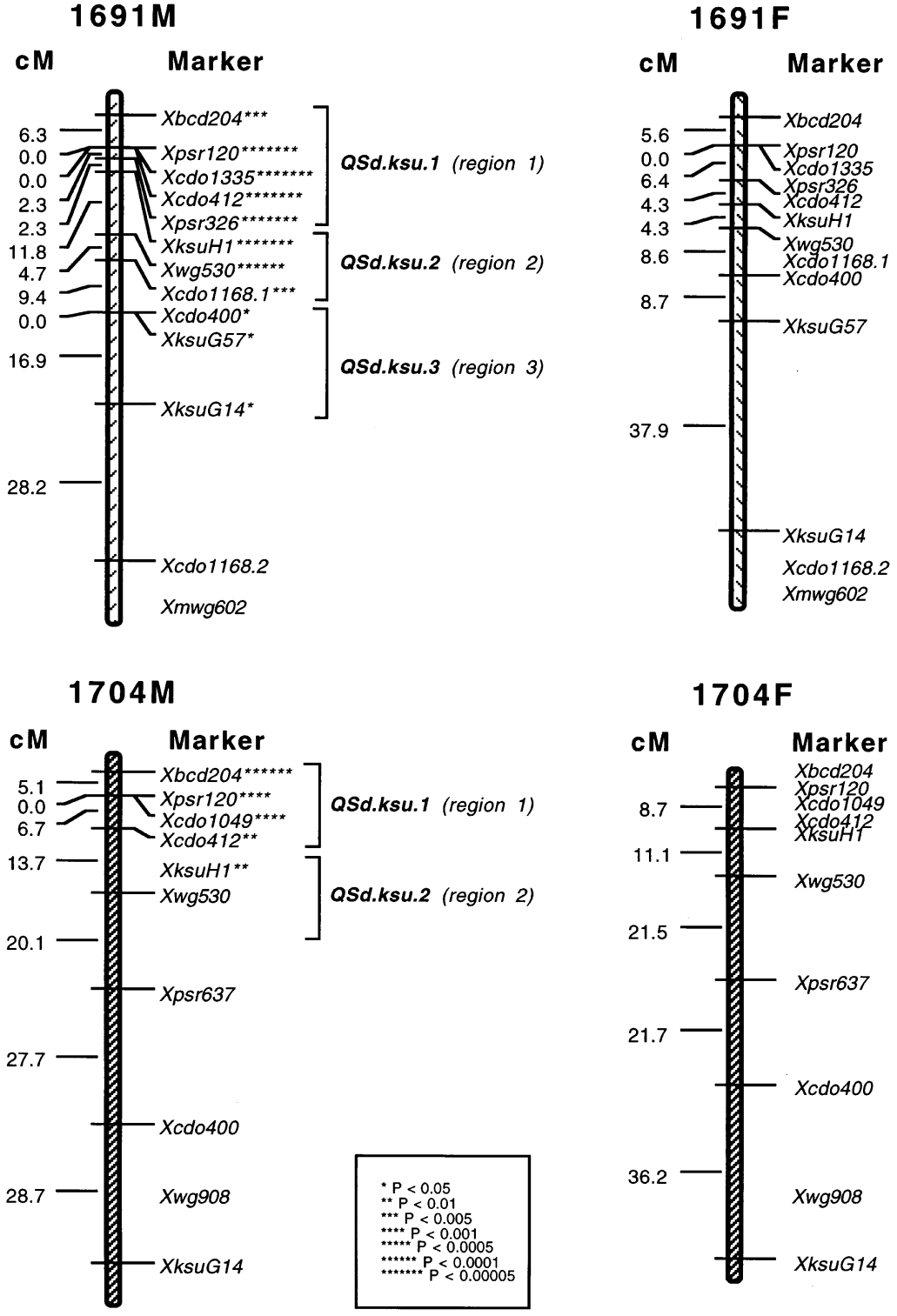

Figure 3.—Genetic linkage maps of chromosome *5D* constructed in the two sets of reciprocal BC populations. The maps represent the corresponding regions of segregation distortion observed in chromosome *5D* of the F_2 population. Markers mapping at LOD < 2.0 were placed in intervals. The legend indicates the degree of marker segregation ratio distortion.

Ten probes were mapped in the 1704M/F populations. In the 1704M population, five of these markers gave ratios that deviated from the expected 1:1 ratio because of a deficiency of TA1704 homozygotes and excess heterozygotes. Four of these distorted markers spanned a region of 11.8 cM that corresponded to distorted *region 1* of the *5D* F_2 map. The most distorted ratio in this region was given by *Xbcd204*, which segregated 5:34 (12.8:87.2%, $P < 0.0001$) for TA1704 homozygotes/heterozygotes, respectively. Markers *XksuH1* and *Xwg530* correspond to distorted *region 2* of the *5D* F_2 map. *Xwg530* segregated in a Mendelian fashion, but *XksuH1* gave a segregation ratio that was significantly ($P = 0.008$) distorted. In a BC population, it was impossi-

ble to determine if *XksuH1* was distorted in the same direction as in the F_2 population (excess heterozygotes with a lack of TA1704 homozygotes and the expected number of TA1691 homozygotes). It is possible that *region 2* was not distorted in this population and that *XksuH1* was distorted because of linkage to the distorter locus in *region 1*. Alternatively, *region 2* may have been distorted in this population, but the preferential selection of gametes may have been less intense, resulting in *Xwg530* segregating in a normal fashion. Markers corresponding to distorted *region 3* of the *5D* F_2 map were not distorted in this population.

All 10 markers making up the 1704F map segregated in a Mendelian fashion. The order of markers along the 1704M map agreed with the order on the 1704F map.

DISCUSSION

Analysis of the F_2 population revealed regions of segregation distortion on chromosomes *1D*, *3D*, *4D*, *5D*, and *7D*. Major regions of distortion on chromosomes *1D*, *3D*, *4D*, and distorted *region 1* of *5D* were the result of excess TA1691 homozygotes and a deficiency of TA1704 homozygotes, while the number of heterozygotes was normal. Deviation of distorted *region 2* of chromosome *5D* was caused by excess heterozygotes and a lack of TA1704 homozygous genotypes, while there was no deviation in the number of TA1691 homozygotes. Distorted *region 3* of *5D* was caused by a lack of heterozygotes and excess TA1691 homozygotes, with the expected number of TA1704 homozygotes. Chromosome *7D* was the only chromosome with distorted regions in which marker segregation ratios deviated because of excess TA1704 homozygotes and a deficiency of TA1691 homozygotes, while the frequency of heterozygotes was as expected.

A region of 56.2 cM consisting of eight markers on *1DL* was skewed in favor of TA1691 homozygotes and had a deficiency of TA1704 homozygotes, while the frequency of heterozygotes was as expected. Telocentric *1DS* has poor competitive ability in the monotelodisomic condition and suffers from postmeiotic loss after frequent pairing failure in microsporocytes (Sears and Sears 1978). Female gametes function in the nullisomic *1D* condition, but functional pollen almost always has a complete *1D* chromosome. It is therefore likely that a genetic selection factor for gametophyte competition existed on *1DL*. This factor may be related to the *1DL* locus of wheat described by Maan (1992), which causes segregation distortion by nucleo-cytoplasmic interactions. Following McIntosh *et al.* (1994), we propose the symbol *QSd.ksu-1D* to designate this locus.

The major region of segregation distortion on chromosome *3D* was located distally on the short arm. To our knowledge, there are no known hybrid sterility or gametophyte genes located on this arm. We propose the symbol *QSd.ksu-3D* to designate this locus.

Four markers clustered on the short arm of chromosome *4D* had segregation ratios that deviated from the expected ratio. A hybrid male sterility (*ms*) gene is known to exist on *4BS* (Sears 1966), and it is possible that a homeoallele exists on *4DS*. We propose the symbol *QSd.ksu-4D* to designate the locus responsible for the segregation distortion.

A more in-depth investigation of markers on the distal tip of *7DS* will be needed to determine if the distorted segregation ratio exhibited by *Xpsr160* was caused by random chance or if it is actually linked to a distorter locus. The major region of distortion on this chromosome was clustered proximally on the short arm. Despite repeated attempts, Sears and Sears (1978) were unable to recover a telo-*7DL* plant in "Chinese Spring." It is therefore possible that the distorted segregation observed on *7DS* was caused by a gametophyte selection factor. This distortion factor is likely homologous to the *Sd-1* locus identified on chromosome *7Ag* of *L. ponticum* (Zhang and Dvorák 1990). We propose the symbol *QSd.ksu-7D* to designate the distortion factor on *7D* of *Ae. tauschii*.

The results of this study indicate that chromosome *5D* possesses at least three distorter loci. Though distorted *regions 1* and *2* were adjacent, they differed in their directions of distortion. *Region 1* was skewed in favor of TA1691 homozygotes and in disfavor of TA1704 homozygotes, while there was no deviation in the number of heterozygotes. *Region 2* was skewed in favor of heterozygous genotypes and was deficient in TA1704 homozygotes, while the number of TA1691 homozygotes was normal. Therefore, it seemed that these two regions of distortion were caused by the expression of two different distorter loci with dissimilar modes of action. The locus responsible for the distortion of markers in *region 1* must lie close to the centromere of *5D*, but it is unclear whether the locus lies on the short or long arm. The marker with the highest degree of distortion in this region (*Xcdo677*) is known to lie on *5DS* near the centromere (Gill *et al.* 1996), but markers in this region with similar degrees of distortion are known to lie on *5DL*.

Region 3 consisted of only two markers with distorted segregation ratios, but they exhibited a direction of distortion that differed from that of *region 1* and *region 2*. The ratios of these markers lacked heterozygotes and had excess TA1691 homozygotes, while there was no deviation in the number of TA1704 homozygotes. We propose the symbols *QSd.ksu-5D.1*, *QSd.ksu-5D.2*, and *QSd.ksu-5D.3* to designate the segregation distorter loci in *regions 1*, *2*, and *3*, respectively, of chromosome *5D*.

Mapping of the corresponding distorted regions in the reciprocal BC populations revealed extreme segregation distortion of markers in the 1691M and 1704M populations. All the markers in the 1691F and 1704F populations segregated in a Mendelian fashion. Distortion of ratios in the 1691M population favored TA1691 homozygous genotypes and segregation ratios in the

1704M population were skewed in favor of heterozygotes. This indicates that there is a factor(s) present on chromosome *5D* of the TA1691 accession that caused preferential selection for TA1691 alleles. Because the extreme segregation distortion was only observed in the 1691M and 1704M populations, the selective interaction was likely the result of gametophyte competition among pollen. In the 1691F and 1704F populations, the pollen was homogenous and therefore pollen competition did not exist. Meiosis of these hybrids was normal and the pollen was viable (data not shown). Furthermore, no chromosome aberrations or differences in seed set were observed in reciprocal crosses. This provides further evidence that the mechanism of selection acted on the gametes and not the zygote.

Marker loci that gave distorted segregation ratios in distorted *region 2* of the *5D* F_2 linkage map also gave distorted ratios in the 1691M population, but it is unclear whether or not this region was distorted in the 1704M population because the direction of distortion observed in the BC will not differ from *region 1* as in the F_2 population. It is likely that either *region 2* of the 1704M map was not distorted, and the observed segregation distortion of *XksuH1* was caused by linkage to *region 1*, or *region 2* was in fact distorted and the gamete competition was less intense, resulting in *Xwg530* giving a normal segregation ratio. It is possible that a nucleo-cytoplasmic interaction exists such that the TA1704 cytoplasm modifies the selection for TA1691 gametes, resulting in less segregation distortion.

Distorted *region 3* of the *5D* F_2 map was also observed on the 1691M map but not on the 1704M map. This suggested that the gametophytic gene(s) responsible for the segregation distortion observed in *region 3* is manifested by an ovule effect that is specific for the TA1691 genotype. Because 1704M was in TA1704 cytoplasm, the stigma, style, and ovary were of TA1704 genotype, and these interactions were less intense or did not exist. As a result, marker loci of 1704M corresponding to distorted *region 3* of the *5D* F_2 map were not distorted. To test this hypothesis, segregation of markers will need to be tested from the following BCs: TA1691 × (TA1704 × TA1691), (TA1704 × TA1691) × TA1691, TA1704 × (TA1691 × TA1704), and (TA1691 × TA1704) × TA1704.

In plant systems, euploid male gametes usually have a competitive advantage over male gametes with extra chromosomes. The expected transmission frequencies of most alien chromosomes in a wheat genetic background are ~0.05 and 0.25 for male and female gametes, respectively (Hyde 1953; Riley 1960). Jiang and Gill (1997) studied the transmission frequencies of a telocentric chromosome *5HL* from *Elymus trachycaulus* (Link) Gould ex Shinners in a genetic background of common wheat. They found that this telocentric chromosome was transmitted in 20% of the female gametes and in 97% of the male gametes.

Other evidence for preferential male transmission factors present on *5DL* is the fact that ditelosomic *5AS*, *5BS*, and *5DS* are among the few unavailable ditelosomic lines of Chinese Spring wheat. Researchers have been unable to recover ditelosomic *5AS*, *5BS*, and *5DS* plants from the progenies of selfed plants with the chromosome constitution of 20″ + *5AS*″ + *5AL*′, 20″ + *5BS*″ + *5BL*′, and 20″ + *5DS*″+*5DL*′, respectively. Furthermore, when heterozygous *5A*, *5B*, or *5D* deletion lines were selfed, homozygous deletion plants were never recovered if the deleted chromosome segment was >50% of the long arm (Endo and Gill 1996). It is likely that the same genes responsible for preferential male transmission and gametic competition caused the segregation distortion of marker ratios on chromosome *5D* in this study.

The distorted segregation of markers in these regions of *5D* have also been observed in the homologous regions of barley chromosome *5H* (Graner *et al.* 1991; Kleinhofs *et al.* 1993; Devaux *et al.* 1995). Furthermore, comparative mapping experiments have demonstrated that a large portion of wheat homoeologous group *5* chromosomes, including the regions of segregation distortion identified in this study, have homology to rice chromosome *9* (Van Deynze *et al.* 1995; Ahn *et al.* 1993). Regions of segregation distortion have been identified on rice chromosome *9* in doubled haploid, recombinant inbred (Xu *et al.* 1997), and F_2 (Harushima *et al.* 1996) populations. It is possible that these gametophyte genes have been conserved through evolution and may be present in many grass species.

The genetic mechanism of distortion on chromosome *5D* appears to be different from those reported for the gametocidal chromosomes of other Aegilops species (Endo and Tsunewaki 1975; Maan 1975; Endo 1990). In this study, preferential selection of TA1691 alleles was only observed through the male. On the contrary, gametocidal chromosomes cause abortion of gametes lacking the gametocidal chromosome, which results in partial sterility and exclusive transmission of these chromosomes through both male and female gametes (Endo 1990).

Ae. tauschii is considered an important source of novel resistance genes that can be easily introgressed into elite germplasm of hexaploid wheat. Knowledge of the chromosomal locations and activation of segregation distortion loci will aid breeders in designing appropriate crossing schemes and help them predict the frequency at which a given allele will be transmitted to the progeny. Once the performance of a genotype can be predicted, a breeder may be able to maximize transmission of desired alleles or to preferentially exclude deleterious alleles.

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