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ORIGINAL PAPER

Identification and characterization of segregation distortion loci along chromosome 5B in tetraploid wheat

Sunil Kumar · Bikram S. Gill · Justin D. Faris

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Abstract Segregation distortion genes are widespread in plants and animals and function by their effect on competition among gametes for preferential fertilization. In this study, we evaluated the segregation distortion of molecular markers in multiple reciprocal backcross populations derived from unique cytogenetic stocks involving the durum cultivar Langdon (LDN) and wild emmer accessions that allowed us to study the effects of chromosome 5B in isolation. No segregation distortion of female gametes was observed, but three populations developed to analyze segregation of male gametes had genomic regions containing markers with skewed segregation ratios. One region of distortion was due to preferential transmission of LDN alleles over wild emmer alleles through male gametes. Another region required the presence of LDN 5B chromosomes in the female for preferential fertilization by male gametes harboring LDN alleles indicating that the corresponding genes in the female gametes can govern genes affecting segregation distortion of male gametes. A third region of distortion was the result of preferential transmission of wild emmer alleles over LDN alleles through male gametes. These results indicate the existence of different distorter/meiotic drive elements among different genotypes and show that distortion factors along wheat chromosome 5B differ in chromosomal location as well as underlying mechanisms.

Keywords Wheat · Segregation distortion · Gamete · Meiotic drive · Recombination

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S. Kumar
Department of Plant Sciences, Loftsgard Hall,
North Dakota State University,
Fargo, ND 58105, USA

B. S. Gill
Department of Plant Pathology, 4024 Throckmorton
Plant Sciences Center, Kansas State University,
Manhattan, KS 66506, USA

J. D. Faris (✉)
USDA-ARS Cereal Crops Research Unit,
Northern Crop Science Laboratory,
1307 18th Street North, Fargo, ND 58105, USA
e-mail: justin.faris@ars.usda.gov

Introduction

Segregation distortion is the deviation of observed genetic ratios from the expected Mendelian ratios of a given genotypic class within a segregating population. Distorted segregation ratios may result from gametophytic competition resulting in preferential fertilization, or abortion of the male or female gametes or zygotes (Lyttle 1991). Genetic elements that cause segregation distortion may be potent evolutionary forces (Sandler and Novitski 1957). Theory suggests that these 'meiotic drive' elements are highly important for the evolution of recombination, heteromorphic sex chromosomes, sex ratios, and reproductive isolation (for reviews, see Hurst et al. 1996; Werren and Beukeboom 1998; Hurst and Werren 2001; Jaenike 2001). There is a variety of mechanisms that can cause segregation distortion and in most systems act in the male gametes (Taylor and Ingvarsson 2003). Another generalization of segregation distortion studies is that the underlying mechanisms do not distort meiosis per se but rather alter the products of meiosis by causing chromosome breakage and/or aborting gametes that do not carry the driving allele.

Segregation distortion has been observed in populations of a wide variety of organisms including fungi, plants,

insects, and mammals (see Lyttle 1991 for review). The most well studied and characterized example is *Segregation Distorter* (*SD*) in *Drosophila melanogaster*. The SD chromosome from heterozygous *SD/SD+* males is preferentially transmitted due to induced dysfunction of *SD+* spermatids (Sandler et al. 1959). The *SD* gene encodes a mutant RanGAP protein, which is required for nuclear transport and other nuclear functions (Merrill et al. 1999). The defect in nuclear transport may be the main cause of sperm dysfunction (see Kusano et al. 2003 for review).

Mechanisms of segregation distortion have been less studied in plants, but genomic regions harboring markers with segregation ratios that do not conform to the expected Mendelian ratios are encountered often and have been reported in many crop species including barley (*Hordeum vulgare*) (Graner et al. 1991; Heun et al. 1991; Zivy et al. 1992; Kleinhofs et al. 1993; Devaux et al. 1995), pearl millet (*Pennisetum glaucum*) (Busso et al. 1995; Liu et al. 1996), rice (*Oryza sativa*) (Causse et al. 1994; Harushima et al. 1996; Yamagishi et al. 1996; Xu et al. 1997), maize (*Zea mays*) (Wendel et al. 1987; Dufour et al. 2001; Lu et al. 2002), and wheat (*Triticum aestivum*) (Zhang and Dvorak 1990; Faris et al. 1998; Messmer et al. 1999; Peng et al. 2000). In maize, gametes possessing the gametophyte factor *Ga1* linked to the *Su* allele for starchy endosperm had faster pollen tube growth compared to gametes with the *ga1* allele when fertilizing *Ga1/ga1* or *Ga1/Ga1* pistils (Mangelsdorf and Jones 1926). Thus, a mixture of *Ga1* and *ga1* pollen resulted in an excess of genotypes with the linked *Su* allele. Recently, the maize *aberrant pollen transmission 1* (*apt1*) gene was isolated and found to be required for pollen tube growth and likely involved in membrane trafficking (Xu and Dooner 2006). In the wheat pollen killer systems, male gametes are produced that carry the non-driving allele, but they are rendered inviable later in development (Loegering and Sears 1963).

Faris et al. (1998) analyzed the degree and direction of segregation distortion in an *Aegilops tauschii* F_2 population using molecular markers and found regions with significantly skewed ratios on chromosomes 1D, 3D, 4D, 5D, and 7D. The most severely distorted regions were on chromosome 5D, which were subsequently analyzed in reciprocal backcross populations. Severe distortion of markers along chromosome 5D was observed in the male population whereas no distortion was observed in the female.

In addition to the work by Faris et al. (1998) others have shown that the homoeologous group 5 chromosomes of wheat and its relatives possess factors associated with segregation distortion (Jiang and Gill 1998; Faris et al. 2000; Peng et al. 2000). Wild emmer wheat [*T. turgidum* ssp. *dicoccoides* (Körn. ex Asch. & Graebner) Aarons ($2n = 4x = 28$, AABB genomes)] is the wild progenitor of cultivated durum (macaroni) wheat (*T. turgidum* ssp. *durum* L., $2n = 4x = 28$, AABB genomes) (Zohary 1970). Wild emmer readily hybridizes with durum, and its chromosomes pair and recombine with those of durum in a normal fashion. *T. turgidum* ssp. *dicoccoides* has provided useful genes for durum wheat improvement for traits such as disease resistance (Gerechter-Amitai and Stubbs 1970; Nevo et al. 1985, 1986, 1991; Fahima et al. 1998; Stack et al. 2002; Kumar et al. 2007) and quality (Joppa and Cantrell 1990). Dr. L. R. Joppa (USDA-ARS retired) developed a large number of cytogenetic stocks in tetraploid wheat, many of which involved disomic chromosome substitution lines in which pairs of chromosomes from *T. turgidum* ssp. *dicoccoides* were substituted for pairs of homologous chromosomes in the *T. turgidum* ssp. *durum* cultivar 'Langdon' (LDN) background (Joppa 1993). These cytogenetic stocks have proven to be valuable tools for genetic analyses because they essentially allow the effects of single chromosomes to be studied in isolation. Here, we exploited these cytogenetic stocks for the development of populations that contain recombinant 5B chromosomes involving different *T. turgidum* ssp. *dicoccoides* accessions in a homozygous LDN background, and which allowed us to study and compare the transmission of gametes through the male and female without the possibility of nucleo-cytoplasmic effects.

Materials and methods

Plant materials

The *T. turgidum* ssp. *durum* cultivar LDN and the LDN-*T. turgidum* ssp. *dicoccoides* accession Israel A chromosome 5B disomic substitution line [LDN-DIC 5B(IsA)] were used as parents for four of the six populations described in this research. The two other populations employed LDN and a LDN-*T. turgidum* ssp. *dicoccoides* chromosome 5B disomic substitution line derived from PI478742 [LDN-DIC 5B(742)]. All the LDN-DIC substitution lines were developed by Dr. Leonard Joppa (USDA-ARS retired). The LDN-DIC 5B(IsA) lines were described in Joppa and Cantrell (1990).

To analyze the transmission of gametes through the female, two populations were produced by backcrossing the F_1 derived from LDN x LDN-DIC 5B(IsA) as the female with LDN-DIC 5B(IsA) and LDN each as the male (Fig. 1). The pedigrees of these populations are LDN/LDN-DIC 5B(IsA)//LDN-DIC 5B(IsA) and LDN/LDN-DIC 5B(IsA)//LDN, and they are designated IsAf and LDNf, respectively. A third female population, designated 742f, was developed by backcrossing the F_1 derived from LDN x LDN-DIC 5B(742) as the female with LDN-DIC 5B(742) as the male [pedigree: LDN/LDN-DIC 5B(742)//LDN-DIC 5B(742)].

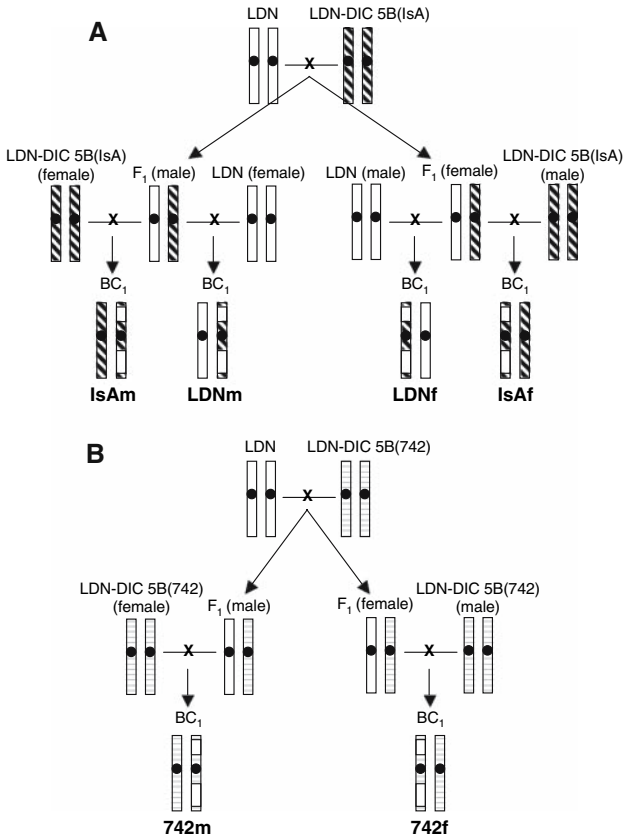

Fig. 1 A schematic description of the development of backcross populations to assess the transmission of gametes through the male and female. **a** The F_1 plant derived from LDN x LDN-DIC 5B(IsA) was used as the male parent in crosses to LDN-DIC 5B(IsA) and LDN to produce the IsAm and LDNm populations, respectively. The same F_1 was used as the female parent in crosses to LDN-DIC 5B(IsA) and LDN to produce the IsAf and LDNf populations, respectively. **b** The F_1 plant derived from LDN x LDN-DIC 5B(742) was used as the male and female parent in crosses to LDN-DIC 5B(742) to produce the 742m and 742f populations, respectively

Similarly, transmission of gametes through the male was evaluated in two populations generated by crossing the LDN x LDN-DIC 5B(IsA) F_1 as the male parent to LDN-DIC 5B(IsA) and LDN each as the female. The pedigrees of these populations are LDN-DIC 5B(IsA)//LDN/LDN-DIC 5B(IsA) and LDN//LDN/LDN-DIC 5B(IsA), and they are designated IsAm and LDNm, respectively (Fig. 1). A third male population was developed by crossing the LDN x LDN-DIC 5B(742) F_1 as the male to LDN-DIC 5B(742) as the female and designated 742m [pedigree: LDN-DIC 5B(742)//LDN/LDN-DIC 5B(742)]. Each population consisted of 88 individuals used for mapping. All populations consisted of a pair of recombinant 5B chromosomes in a homozygous LDN background. Therefore, we were able to evaluate the effects of chromosome 5B in isolation. Furthermore, all populations existed in LDN cytoplasm and therefore nuclear-cytoplasmic interactions were not present.

Molecular mapping

Eleven molecular markers along chromosome 5B known to be polymorphic between LDN and LDN-DIC 5B(IsA) were chosen for the development of genetic linkage maps in each population. Seven of the ten markers (*Xbcd873, Xbcd1871, XksuA3, XksuH1, Xfcg3, Xfcg4*, and *Xpsr580*) were generated by RFLP analysis. The RFLP probe sources and contributors are as follows: BCD (barley cDNA), by M. Sorrells; PSR (wheat genomic DNA) by M. Gale; KSU (wheat genomic and cDNA) by B. Gill; and FCG (wheat genomic DNA) by J. Faris. Methods of DNA isolation, restriction enzyme digestion, gel electrophoresis, Southern blotting, probe labeling and hybridization, and membrane washing were performed as described in Faris et al. (2000). After the probes were hybridized to membranes, the membranes were exposed to phosphorimaging screens for 3-12 h and scanned with a Typhoon 9410 variable mode imager (GE Healthcare Inc., USA).

The remaining three markers including *Xbarc109, Xbarc74, Xbarc59* (Song et al. 2005) and *Xfcp393* (J. Faris, unpublished) were microsatellite, or simple sequence repeat (SSR) markers. Conditions for PCR amplification and gel electrophoresis were described in Liu et al. (2005).

Data analysis

Linkage maps were constructed using the computer program Mapmaker v2.0 (Lander et al. 1987) for Macintosh with an LOD of 3.0 and the Kosambi mapping function (Kosambi 1944). The correctness of marker orders was validated using the 'Ripple' command with a LOD threshold of 3.0. Markers were subjected to a Chi-squared test for fit to a 1:1 ratio in the male and female BC populations using the computer program QGENE (Nelson 1997). Significant differences in total map lengths and in map distances between adjacent loci were determined comparing the number of crossovers using a contingency Chi-squared test.

Results

Construction of genetic linkage maps

The markers used to develop linkage maps of chromosome 5B in the six populations were selected based on their distribution in previously published genetic maps of wheat chromosome 5B (Nelson et al. 1995; Gill et al. 1996; Faris et al. 2000; Haen et al. 2004; Sourdille et al. 2004) to give complete chromosome coverage. The marker *XksuH1* was not polymorphic between LDN and LDN-DIC 5B(742). Therefore, the 742f and 742m maps consist of ten markers, whereas the LDNf, LDNm, IsAf, and IsAm maps have

eleven markers. Comparison of the genetic maps with the 5B physical map indicates that markers and recombination frequencies are not evenly distributed along the chromosome, which is a previously reported phenomenon (Gill et al. 1996; Faris et al. 2000) (Fig. 2). All markers mapped at an LOD > 3.0, and the order of markers among the six maps agreed with the order of markers on the physical map.

Recombination

Among the six populations, map lengths ranged from 112 cM for the LDNm population to 168 cM for the LDNf population (Fig. 2). Comparisons of the total number of crossovers observed among all populations indicated that only the LDNf and LDNm populations had significantly ($P < 0.01$) different recombination frequencies. No other significant differences in recombination frequencies along chromosome 5B were detected among all other populations (data not shown). However, average map lengths of male and female populations were 122 and 151 cM, respectively, which suggests that recombination frequencies tend to be higher in female meiosis compared to male meiosis.

A contingency Chi-squared test was used to test for significant differences in recombination frequencies of each marker interval along the maps of each female population with the corresponding intervals along the maps of the male populations generated from the corresponding reciprocal cross, i.e. marker intervals in LDNm vs LDNf, IsAf vs IsAm, and 742f vs. 742m. Only two intervals differed significantly, and both were identified when comparing the LDNf and LDNm populations (Fig. 2). The interval defined by markers *Xbcd873* and *Xbcd1871* accounted for 32.2 cM on the LDNf map but only 15.2 cM on the LDNm map ($P < 0.05$). The second interval delineated by markers *Xfcg3* and *Xfcp393* accounted for 43.8 and 19.1 cM ($P < 0.05$) on the LDNf and LDNm maps, respectively. Comparisons of recombination frequencies of all other marker intervals between corresponding male and female populations revealed no other significant differences.

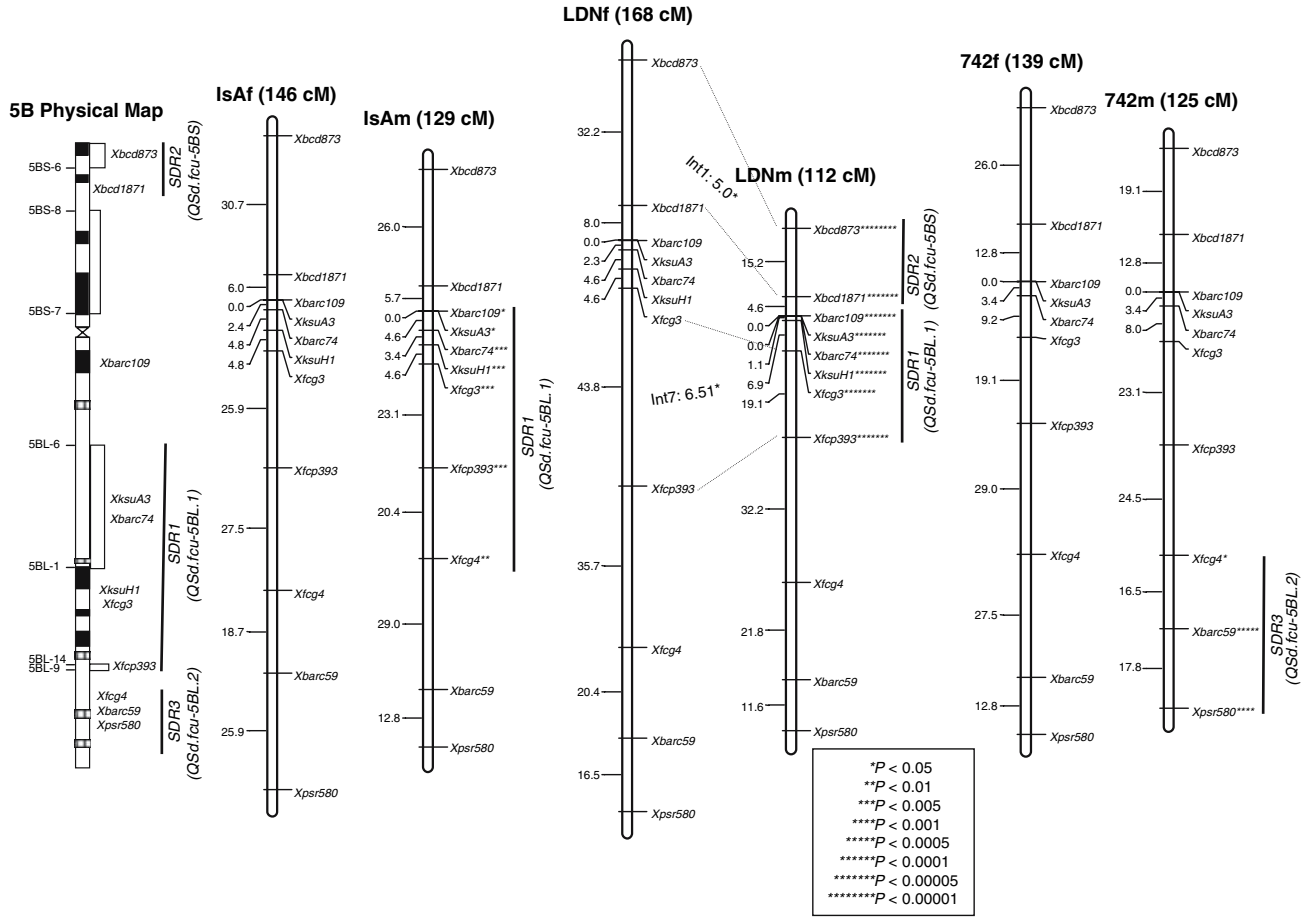

Fig. 2 The physical map of chromosome 5B based on deletion breakpoints (summarized from Gill et al. 1996; Faris et al. 2000; Sourdille et al. 2004) and the chromosome 5B genetic linkage maps developed in the IsAf, IsAm, LDNf, LDNm, 742f, and 742m populations. Markers with significantly distorted segregation ratios are indicated with *asterisks*, and the degrees of significance (*P*-values) are indicated in the *box* below the genetic maps. Numbers within the intervals defined by *dotted lines* between the LDNf and LDNm maps indicate the significant Chi-squared value calculated from a contingency Chi-squared test to determine significant differences in recombination frequencies

Segregation distortion

No segregation distortion was detected in any of the female populations because all markers along chromosome 5B in the LDNf, IsAf, and 742f populations conformed to the expected Mendelian segregation ratio of 1:1. Seven markers in the IsAm population had segregation ratios that deviated significantly ($P < 0.05$) from the expected ratio of 1:1 (Fig. 2). These markers spanned a genetic distance of 56.1 cM on the long arm and were distorted due to a greater than expected number of heterozygotes and a less than expected number of genotypes homozygous for IsA alleles (Fig. 3). The center of distortion was depicted by markers *XksuH1*and *Xfcg3*, which are physically located near the middle of the long arm within the deletion bin delineated by breakpoints 5BL-1 and 5BL-14 (Fig. 2). Both markers segregated in a ratio of 29 homozygotes: 59 heterozygotes ($\chi^2 = 10.2$; $P < 0.005$) (Table 1). This region of segregation distortion will hereafter be referred to as segregation distortion region 1 (SDR1).

Eight markers on the LDNm map had distorted segregation ratios (Fig. 2). The eight markers spanned a genetic distance of 46.9 cM extending from the distal end of the short arm to near the middle of the long arm, but the markers on the short arm had the most severely distorted segregation ratios. The marker *Xbcd873*, which is located at the distal end of 5BS (Fig. 2), defined the epicenter of distortion (Table 1, Fig. 3). This marker segregated in a ratio of 84 homozygotes: 4 heterozygotes ($\chi^2 = 72.7$; $P < 0.00001$) (Table 1). All markers with distorted segregation ratios in this region, which we will refer to as SDR2, were skewed due to a greater than expected number of genotypes homozygous for LDN alleles and less then expected number of heterozygous genotypes (Table 2, Fig. 3).

Three markers spanning 34.3 cM within the distal 25% of the long arm of chromosome 5B had distorted segregation ratios in the 742 m population (Fig. 2). The markers in this region, hereafter referred to as SDR3, were distorted due to a greater than expected number of genotypes homozygous for LDN-DIC 5B(742) alleles and less than expected number of heterozygotes (Table 2, Fig. 3). Of the three markers, *Xbarc59* was the most severely distorted and segregated in a ratio of 61 homozygotes: 27 heterozygotes ($\chi^2 = 13.1$; $P = 0.0003$) (Table 1).

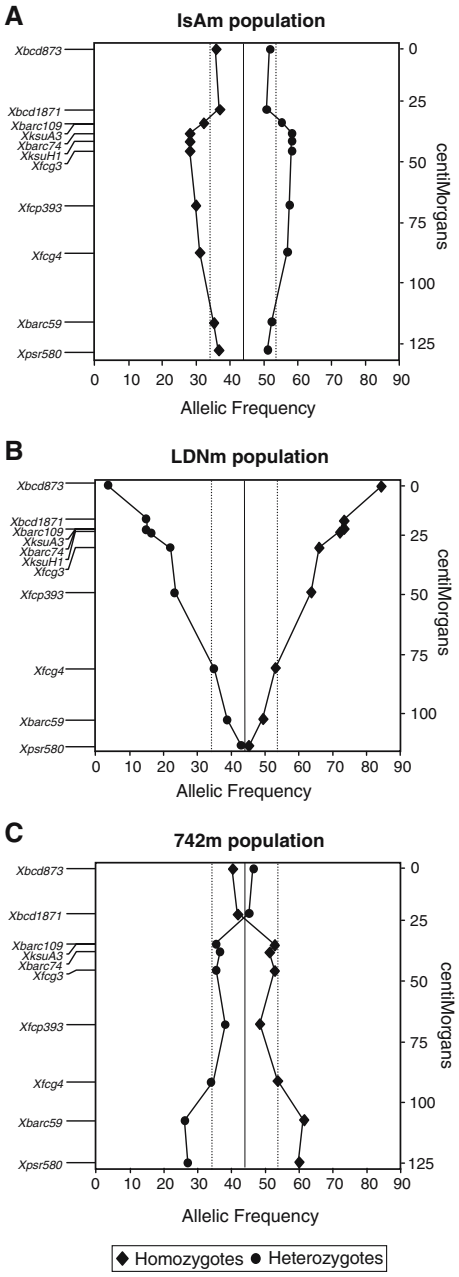

Fig. 3 Genotype frequencies as a function of the genetic linkage map. Frequencies of genotypes along chromosome 5B in the IsAm (**a**), LDNm (**b**), and 742m (**c**) backcross populations are plotted along the genetic linkage map. The *x*-axis indicates the genotypic values observed for each marker, and the *y*-axis corresponds to the genetic linkage map. The *legend* indicates the genotype that each line represents. *Fine dotted lines* indicate the significance threshold of $P < 0.05$

Discussion

Analysis of marker segregation ratios in the reciprocal backcross populations revealed severe segregation distortion as a result of mechanisms acting on male gametes. Three different regions of segregation distortion involving transmission of male gametes were identified. SDR1 clearly showed skewed segregation ratios in the IsAm population where the F_1 derived from LDN x LDN-DIC 5B(IsA) was backcrossed as the male to LDN-DIC 5B(IsA); and when the same F_1 plant was backcrossed as the male to LDN, SDR2, the region of most severe distortion, was observed. SDR3 was only observed in the 742m population indicating there is allelic variation among the different distorter loci.

Table 1 Analysis of markers showing the most severe segregation distortion in the IsAm, LDNm, and 742m populations

Population	SD region	Marker	Map position	Arm	χ^2	*P*	No. homozygotes	No. heterozygotes
IsAm [LDN-DIC 5B(IsA)//LDN/ LDN-DIC 5B(IsA)]	SDR1	*XksuH1, Xfcg3*	39.7, 44.3	5BL	10.2	0.0014	29	59
LDNm [LDN//LDN/LDN-DIC 5B(IsA)]	SDR2	*Xbcd873*	0	5BS	72.7	<0.00001	84	4
742m [LDN-DIC 5B(742)//LDN/ LDN-DIC 5B(742)]	SDR3	*Xbarc59*	107.4	5BL	13.1	0.0003	61	27

Table 2 Summary of observations of chromosome 5B segregation distortion regions SDR1, SDR2, and SDR3 in populations designed to analyze the transmission of gametes through the male

Population	SD Region	Segregation classes		Favored allele	Comments/conclusions
		Homozygotes	Heterozygotes		
IsAm	SDR1	Deficient	Excess	LDN	Distortion due to competition among male gametes
	SDR2	Expected	Expected	–	No distortion because LDN 5B alleles not present in female
	SDR3	Expected	Expected	–	Distortion not observed because the driving allele observed in *T. turgidum* ssp. *dicoccoides* PI478742 is not present in IsA or LDN
LDNm	SDR1	Excess	Deficient	LDN	Distortion likely due to competition among male gametes, but the degree of distortion may be obscured due to linkage with SDR2
	SDR2	Excess	Deficient	LDN	Distortion due to preferential fertilization by male gametes carrying LDN alleles due to the presence of corresponding LDN alleles in female gametes
	SDR3	Expected	Expected	–	Distortion not observed because the driving allele observed in *T. turgidum* ssp. *dicoccoides* PI478742 is not present in IsA or LDN
742m	SDR1	Expected	Expected	–	Distortion not observed because no polymorphism between *T. turgidum* ssp. *dicoccoides* PI478742 and LDN for the driving allele
	SDR2	Expected	Expected	–	Distortion not observed because no polymorphism at distortion locus, or because LDN 5B alleles not present in female gametes. Evaluation of a male population derived from the LDN x LDN-DIC 5B(742) F_1 backcrossed to LDN is needed
	SDR3	Excess	Deficient	PI478742	Distortion due to competition among male gametes

In accordance with McIntosh et al. (1998), we propose to designate the segregation distortion regions SDR1, SDR2, and SDR3 as *QSd.fcu-5BL.1, QSd.fcu-5BS*, and *QSd.fcu-5BL.2*, respectively.

Segregation distortion region 1

The markers within SDR1 on the IsAm map were skewed in favor of LDN alleles whereas the same markers on the female map segregated in a Mendelian fashion. This indicates that SDR1 is the result of gametophytic competition among male gametes resulting in preferential fertilization of females by male gametes carrying LDN alleles at this locus. In the female population, the pollen was homogeneous and therefore there was no opportunity for male gametophytic competition, which resulted in the markers in SDR1 segregating in a Mendelian fashion. Therefore, an important property of SDR1 is that it operates solely in male gametes effectively governing gametophytic competition.

Faris et al. (1998) reported three different segregation distorter loci on the homoeologous chromosome 5D of *Ae. tauschii*, the D-genome progenitor of hexaploid bread wheat. The markers identified in SDR1 in our IsAm population correspond to those identified in the *QSd.ksu-5D.2* region identified by Faris et al. (1998) in *Ae. tauschii*. It is possible that SDR1 on the long arm of chromosome 5B is caused by a homoeoallele of the segregation distorter at the *QSd.ksu-5D.2* locus on chromosome 5D in *Ae. tauschii*.

Skewed segregation ratios of markers have been observed in other wheat populations that correspond to SDR1 along 5B (Blanco et al. 1998; Messmer et al. 1999; Faris et al. 2000) and the homoeologous regions of chromosomes 5A (Luo et al. 2000) and 5D (Faris et al. 1998). Devaux et al. (1995) reported a region of segregation distortion on barley chromosome 7(5H) that is in the vicinity of SDR1 and may represent a homoeologous distorter region in barley. Furthermore, segregation distortion regions of rice chromosomes 3 (Matsushita et al. 2003) and 9 (Harushima et al. 1996; Xu et al. 1997) are known to have colinearity with Triticeae group 5 chromosomes (Ahn et al. 1993; Van Deynze et al. 1995), including SDR1 identified in this study. It is possible that gametophytic genes have been conserved through evolution and may be present in many grass species.

Segregation distortion region 2

SDR2 was the most severely distorted of the three SDRs and was identified only in the LDNm population, which is the only male population developed by backcrossing to LDN as the recurrent parent. The same F_1 plant used to generate the IsAm population was used to generate the LDNm population, and therefore the only difference between the LDNm and IsAm populations is the source of chromosome 5B in the female parent. However, it was clearly evident that SDR2 was the result of extensive segregation distortion of male gametes in the LDNm population, but not in the IsAm population. This indicates LDN female gametes preferentially "attracted" male gametes harboring LDN alleles of a gene or factor underlying SDR2. The same attraction did not occur by IsA female gametes because SDR2 was not distorted in the IsAm population.

The mechanism underlying segregation distortion in SDR2 may be similar to the *Ga1* system in maize. In that system, *Ga1* and *ga1* pollen grains are equally competitive when fertilizing *ga1/ga1* pistils, but *Ga1* pollen grains are preferentially transmitted over *ga1* pollen grains when fertilizing pistils of *Ga1/Ga1* or *Ga1/ga1* genotype (Mangelsdorf and Jones 1926; Emerson 1934). Similarly, our work indicates that LDN and IsA alleles at SDR2 are equally competitive on pistils with IsA alleles, but preferential transmission of LDN alleles occurred through the male when fertilizing pistils with LDN alleles. It is possible that the female and male factors responsible for the preferential fertilization resulting in SDR2 are the same gene, but our experimental material is not sufficient to make this determination. However, we can be sure that both the male and female factors involved in causing the segregation distortion in SDR2 reside on chromosome 5B.

It is most likely that the gametophytic competition resulting in SDR1 observed in the IsAm population also operated in the LDNm population. However, the severity of distortion that occurred at SDR2 in LDNm may have obscured the effects of SDR1 making it difficult to determine.

Recombination was suppressed in the LDNm population relative to the LDNf population. The entire linkage map of the LDNf chromosome 5B was 168 cM compared to only 112 cM for the LDNm map. Recombination frequencies tended to be higher in most marker intervals along the LDNf map compared to the corresponding intervals of the LDNm map (Fig. 2). However, only intervals 1 and 7 defined by marker pairs *Xbcd873-Xbcd1871* and *Xfcg3-Xfcp393*, respectively, had significantly higher recombination frequencies in the LDNf population compared to the LDNm population. It is interesting to note that both of these marker intervals correspond to the approximate locations of SDR1 and SDR2. We cannot rule out the possibility that the segregation distortion loci influenced the estimation of recombination in the LDNm population. The estimation of recombination distance is not likely to be affected if segregation distortion is due to a single locus (Bailey 1949; Lorieux et al. 1995a, b). However, Lorieux et al. (1995a, b) showed that this may not be true when segregation distortion is caused by two linked gametophytic factors. In this case, estimations of recombination frequency and map distances become biased. Indeed, our results suggest that segregation distortion along chromosome 5B in the LDNm population is caused by two separate but linked factors, which may have affected the estimation of recombination to some degree. However, because the overall recombination frequencies in the LDNm and LDNf populations were only significantly different from each other, and neither had significantly different recombination frequencies compared to the other four populations, we cannot ascertain whether recombination was increased relative to normal in the LDNf population or if it was decreased relative to normal in the LDNm population, or if a combination of both scenarios occurred.

Segregation distortion region 3

Backcross populations 742f and 742m were developed using the LDN-DIC 5B(742) substitution line to determine if segregation distortion caused by SDR1 and SDR2 could be detected in the male when a different *T. turgidum* ssp. *dicoccoides* accession was used as the chromosome 5B donor in the LDN background. Whereas markers within SDR1 and SDR2 did not have significantly skewed segregation ratios, three markers within SDR3 at the distal end of the long arm of 5B did exhibit segregation distortion. This region was not skewed in the populations involving the *T. turgidum* ssp. *dicoccoides* IsA 5B substitution indicating that different *T. turgidum* ssp. *dicoccoides* accessions

harbor allelic variation for meiotic drive elements. Segregation ratios of markers within SDR3 were skewed in favor of LDN-DIC 5B(742) alleles indicating that *T. turgidum* ssp. *dicoccoides* accession PI478742 carries a meiotic drive element within the region. This is contrary to the SDR1 and SDR2 regions in the IsAm and LDNm population, which were skewed in favor of LDN alleles. The mechanism underlying segregation distortion in SDR3 is likely similar to that of SDR1 in that competition among male gametes occurs leading to the preferential transmission of gametes carrying LDN-DIC 5B(742) alleles at this locus.

Conclusions

Together, these results demonstrate that LDN harbors alleles at SDR1 that allow male gametes to be preferentially transmitted over gametes carrying IsA alleles, but not PI478742 alleles (Table 2). *T. turgidum* ssp. *dicoccoides* accession PI478742 harbors factor(s) at SDR3 that allow its gametes to be preferentially transmitted over gametes having LDN alleles, whereas there is no competition between gametes carrying LDN and IsA alleles at this locus. LDN harbors alleles at SDR2 that allow gametes to be preferentially transmitted over gametes carrying IsA alleles, but only when the female harbors the necessary alleles on 5B. Evaluation of a BC population developed by crossing the F_1 derived from LDN x LDN-DIC 5B(742) with LDN is needed to determine if the LDN alleles that cause segregation distortion at SDR2 would have a competitive advantage over *T. turgidum* ssp. *dicoccoides* PI478742 alleles.

As mentioned above, there is evidence that the meiotic drive element underlying SDR1 has been observed in other experimental material, but to our knowledge, regions of segregation distortion corresponding to SDR2 and SDR3 have not been observed in other wheat populations. Taylor and Ingvarsson (2003) cite two explanations for why segregation distortion factors may be important in evolution, but go unobserved. First, meiotic drive elements without deleterious effects might rapidly become fixed. Second, genetic conflict could arise due to meiotic drive elements having deleterious effects, which would then cause inadvertent selection for alleles at other loci that suppress their effects. It is possible that the meiotic drive element underlying SDR2 has become fixed in cultivated durum and bread wheat populations thereby allowing it to go undetected in intraspecific crosses. However, the SDR2 driving allele observed in LDN is polymorphic with the *T. turgidum* ssp. *dicoccoides* IsA accession, and likely other wild progenitor accessions, which allowed the effects of the driving allele to be observed. Because the driving allele underlying SDR3 is associated with *T. turgidum* ssp. *dicoccoides* accession PI478742, it is possible that this element is associated with a deleterious effect and therefore has become fixed among cultivated durum and bread wheat.

Many important agronomic genes are known to exist on chromosome 5B of common wheat and durum, and *T. turgidum* ssp. *dicoccoides* is a common source of novel allelic variation used by breeders and geneticists for germplasm improvement. When transferring genes known to exist on chromosome 5B from *T. turgidum* ssp. *dicoccoides*, or when developing segregating mapping populations for marker identification or map-based cloning, breeders and geneticists should strongly consider using the F_1 plant as the female for backcross population development and take into account the mechanisms underlying the manifestation of distortion in regions such as SDR2. In this way, breeders may be able to circumvent segregation distortion and potentially suppressed recombination, which could otherwise complicate breeding efforts and genetic analyses.

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