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Chromosomal regions associated with segregation distortion in maize

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Abstract Segregation distortion skews the genotypic frequencies from their Mendelian expectations. Our objectives in this study were to assess the frequency of occurrence of segregation distortion in maize, identify chromosomal regions consistently associated with segregation distortion, and examine the effects of gametophytic factors on linkage mapping. We constructed a simple sequence repeat (SSR) linkage map for a LH200/LH216 F_2Syn3 (i.e., random-mated three times) population, and compared the segregation distortion in this map with the segregation distortion in three published linkage maps. Among 1,820 codominant markers across the four mapping populations, 301 (17%) showed segregation distortion ($P < 0.05$). The frequency of markers showing segregation distortion ranged from 19% in the Tx303/CO159 mapping population to 36% in the B73/Mo17 mapping population. A positive relationship was found between the number of meioses and the frequency of segregation distortion detected in a population. On a given chromosome, nearly all of the markers showing segregation distortion favored the allele from the same parent. A total of 18 chromosomal regions on the ten maize chromosomes were associated with segregation distortion. The consistent location of these chromosomal regions in four populations suggested the presence of segregation distortion regions (SDRs). Three known gametophytic factors are possible genetic causes of these SDRs. As shown in previous research, segregation distortion does not affect the estimate of map distance when only one gametophytic factor is present in an SDR.

Keywords Segregation distortion · Maize · Linkage map · Gametophytic factor

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Introduction

The law of segregation, which is the most fundamental law in Mendelian genetics, relies on: (1) a predictable transmission of alleles from a parent to its offspring, and (2) a predictable formation of genotypes from the transmitted alleles. Segregation distortion, which is defined as a deviation of the observed genotypic frequencies from their expected values, violates the law of segregation and renders conventional genetic theory and analysis to be invalid.

Segregation distortion was first reported in maize by Mangelsdorf and Jones (1926). On the basis of linkage between the gametophyte factor *Ga*1 (formerly *Ga* or *Ga*9) and the *Su* allele for starchy endosperm, Mangelsdorf and Jones found that pollination with *Ga*1 pollen only, or with *ga*1 pollen only, led to normal genotypic ratios. But because pollen-tube growth is faster in pollen with *Ga*1 than with *ga*1, a mixture of *Ga*1 and *ga*1 pollen led to an excess of the genotypes with the linked *Su* allele. Segregation distortion in maize was subsequently reported by Burnham (1936), Rhoades (1942), Longley (1945), Helentjaris et al. (1986), Wendel et al. (1987) and Gardiner et al. (1993). Segregation distortion has also been reported in other crop species including rice (*Oryza sativa* L.) (Nakagahra 1972; McCouch et al. 1988; Xu et al. 1997), barley (*Hordeum vulgare* L.) (Graner et al. 1991; Heun et al. 1991; Devaux et al. 1995), sorghum (*Sorghum bicolor* L.) (Pereira et al. 1994), tomato (*Lycopersicon* sp.) (Paterson et al. 1988),

Table 1 Number and frequency of molecular markers with segregation distortion in four maize populations

Population	Progenies		Markers	
	Type	Number	Total	Distorted (%)
LH200/LH216	F_2Syn3	351	160	55 (34%)
B73/Mo17[a]	Recombinant inbreds	208	201	73 (36%)
Mo17/H99[b]	$F_{6:7}$	186	141	41 (29%)
Tx303/CO159[c]	F_2	54	1,318	132 (10%)
Total		799	1,820	301 (17%)

[a] MaizeDB, panel of stocks ID #105417
[b] MaizeDB, panel of stocks ID #134046
[c] MaizeDB, panel of stocks ID #57244

alfalfa (*Medicago sativa* L.) (Echt et al. 1994) and coffee (*Coffea* sp.) (Ky et al. 2000).

Segregation distortion has frequently been found during the construction of genetic linkage maps. In maize, Wendel et al. (1987) observed that 11 of 17 (65%) segregating allozyme loci showed significant segregation distortion in an F_2 population. Gardiner et al. (1993) detected chromosomal regions associated with segregation distortion on chromosomes 1, 2, 3 and 5. In tomato, Paterson et al. (1988) reported that 48 of 70 (68%) markers at 21 distinct regions had distorted ratios in an interspecific backcross. In rice, Xu et al. (1997) found chromosomal regions associated with marker-segregation distortion in six segregating populations.

If a gene that causes segregation distortion is segregating in a population, then markers close to it would tend to exhibit distorted ratios (Zamir and Tadmor 1986). And if several populations are segregating for the same gametophyte factors or other unknown genes that cause segregation distortion, then these populations will exhibit segregation distortion at the same chromosomal regions. Molecular-marker analysis in several populations is therefore useful for finding common regions with segregation distortion (i.e., *segregation distortion regions* or SDRs) and for future identification of yet-unknown genes that cause segregation distortion in these regions. There has been no comparative study of segregation distortion in different maize populations. Our objectives in this study were to assess the frequency of occurrence of segregation distortion in maize, identify chromosomal regions consistently associated with segregation distortion, and examine the effects of gametophytic factors on linkage mapping.

Materials and methods

Primary linkage map

We constructed a genetic linkage map for LH200/LH216 F_2Syn3, which was developed by random mating the (LH200 × LH216)F_2 population for three generations. LH200 is related to B37 and B73, which were both derived from the Iowa Stiff Stalk Synthetic population. LH216 is related to Mo17 and LH123, which were both derived from the Lancaster Sure Crop population. LH200 and LH216 were developed by Holden's Foundation Seeds. The population was random-mated to expand the linkage map and permit a better resolution of marker distances. We analyzed 351 plants in this population with 160 simple sequence repeat (SSR) markers. The SSR primers were synthesized by Research Genetics Incorporated (Huntsville, Ala., USA). The primers were developed by the following companies or research institutions (primer codes in parentheses): Brookhaven National Laboratory (bnlg), Pioneer Hi-Bred International (phi), DuPont (dupssr), Asgrow (A), University of Missouri-Columbia (umc) and Monsanto (a, mer).

The PCR and electrophoresis of SSR amplification products were conducted at the Monsanto laboratory in Ankeny, Iowa. The SSR methods were described in MaizeDB (ftp://ftp.agron.missouri.edu/pub/methods/ssrmethods.html). The reaction constituents were: Tris-HCl 10 mM, pH 8.3; $MgCl_2$ 2.5 mM; dATP, dCTP, dGTP, dTTP 0.2 mM each; forward- and reverse-primers 0.33 µM each; Ampli*Taq* Gold (Perkin Elmer) 0.5 units; Cresol Red 0.002%; genomic DNA 20 ng; and 2.43% glycerol. ddH_2O brought the total reaction volume to 15 µl. The PCR reaction was carried out in a touchdown fashion. The cycling profile included: (1) activation of Ampli*Taq* Gold for 10 min at 95 °C; (2) 30 cycles of denaturation at 94 °C for 45 s, annealing at 55 °C for 45 s, and extension at 68 °C for 45 s; and (3) a final extension at 72 °C for 5 min. After the thermocycler amplification, the SSR plates were stored in a 4 °C refrigerator until eletrophoresis. Gels were photographed by Stratagene Eagle Eye and were scored visually. The Kosambi mapping function (Kosambi 1944) was used in MAPMAKER/EXP 3.0b software (Lincoln et al. 1992) to construct a linkage map.

Published populations

To examine the consistency of the locations of chromosomal regions that exhibited segregation distortion, we compared the LH200/LH216 F2Syn3 linkage map with published maps for three other populations (Table 1). The B73/Mo17 RI 1997 population (MaizeDB, http://www.agron.missouri.edu/ssr.html, panel of stocks ID #105417) comprised 208 recombinant inbreds that were analyzed with 201 SSR or RFLP markers (Senior et al. 1996). The Mo17/H99 $F_{6:7}$ RI population (MaizeDB, panel of stocks ID #134046) comprised 186 recombinant inbreds that were analyzed with 141 codominant markers (Austin and Lee 1996). The Tx303/CO159 F_2 1995–1998 population (MaizeDB, panel of stocks ID #57244) comprised 54 F_2 plants that were analyzed with 1,318 codominant markers (Gardiner et al. 1993).

Segregation analysis

At each locus, the allele from the first parent (i.e., LH200, B73, Mo17 or Tx303) was denoted as *A* whereas the allele from the second parent (i.e., LH216, Mo17, H99 or CO159) was denoted as *B*. The expected allelic ratio for both F_2 and RI populations was 1:1 (*A:B*). The expected genotypic ratio was 1:2:1 (*AA:AB:BB*) for F_2 populations and 1:1 (*AA:BB*) for recombinant-inbred populations. The observed ratios were tested for deviation from their expected values with a chi-square goodness-of-fit test ($P < 0.05$) for each marker. Individuals with a missing genotype at a locus were excluded from the analysis for that locus. We declared the presence

of a segregation distortion region (SDR) when three or more closely linked markers exhibited significant segregation distortion in one or more of the four populations. The most-skewed marker in an SDR was considered the most-likely location of a distorting factor.

Results and discussion

Segregation distortion in LH200/LH216 F_2Syn3

A total of 55 (34%) out of the 160 SSR loci in LH200/LH216 F_2Syn3 showed significant ($P < 0.05$) segregation distortion (Table 2). Among these 55 loci, 30 (55%) were skewed towards the LH200 genotype, 20 (36%) were skewed towards the LH216 genotype, and five (9%) were skewed towards the heterozygous genotype (Table 2). The 55 distorted markers were unevenly distributed among the ten chromosomes of maize. No distorted markers were detected on chromosome 7, whereas 14 distorted markers were detected on chromosome 3.

Except for A1138 on chromosome 2, A1890 on chromosome 4 and mer161 on chromosome 10, all distorted markers on a given chromosome were skewed towards the same parent. On chromosome 9, for example, all ten markers with segregation distortion were skewed towards LH200. Maximum distortion was centered on phi033 (Table 2, Fig. 1). This result strongly indicated that genetic factors for segregation distortion existed on most chromosomes and they determined the direction of skewness of the markers on the same chromosome.

Frequency of molecular markers with segregation distortion in four populations

Averaged across all markers, all four populations displayed the expected Mendelian gene frequency for the two parental alleles. The frequency of the *A* allele varied from 49.6% to 50.8% among the four populations. The normal segregation at the genome level in the four populations indicates that there were no systematic physiological or genetic factors skewing segregation across the genome. Out of the 1,820 markers used across all four populations, 301 (17%) exhibited segregation distortion (Table 1). The lowest frequency (10%) of distorted markers was found in Tx303/CO159 F_2 1995–1998, an F_2 population that underwent two generations of meiosis.

Veldboom and Lee (1994) analyzed F_3 families (Maize DB, panel of stocks ID #98795) as well as recombinant inbreds of the Mo17/H99 cross. Only nine (9%) of 102 markers had distorted ratios among F_3 families, whereas 41 (29%) of 141 markers had distorted ratios among recombinant inbreds (Table 1). LH200/LH216 F_2Syn3 and B73/Mo17 RI had about 35% of distorted markers. LH200/LH216 F_2Syn3, B73/Mo17 RI and Mo17/H99 $F_{6:7}$ RI each underwent at least five generations of meiosis (either selfing or intermating). Xu et al. (1997) found that RI populations had significantly higher frequencies of distorted markers than doubled-haploid, backcross and F_2 populations. All these results indicate that segregation distortion accumulates with additional generations of meiosis, i.e., there is a positive relationship between the number of generations of meiosis (mating) and the frequency of segregation distortion.

Segregation distortion regions

Among the 301 markers showing aberrant segregation in the four populations, 259 (86%) markers were located in putative SDRs. Eighteen SDRs were identified, and 16 of them were found in at least two populations (Fig. 1). SDR4.1 was detected in all the four populations. The SDRs were identified on all ten chromosomes of maize, but they were unevenly distributed over the ten chromosomes. Chromosomes 5, 6, 7 and 9 had one SDR. Chromosomes 1 and 2, which were the two largest chromosomes, had three SDRs.

All 23 RFLP markers showing segregation distortion detected by Gardiner et al. (1993) were in SDR1.3, SDR2.3, SDR3.1 or SDR5.1. All 11 allozyme markers showing segregation distortion detected by Wendel et al. (1987) were in SDR1.3, SDR2.3, SDR3.1, SDR6.1 or SDR8.1. The results from our study and from these previous studies provide strong evidence for the presence of heritable genetic factors for segregation distortion.

The size of SDRs varied from one bin unit (Gardiner et al. 1993) in SDR2.1, SDR8.1 and SDR10.2 to four bins in SDR8.2. Most of the SDRs had a consistent map location over the four populations. SDR1.3, SDR3.1, SDR3.2, SDR4.2 and SDR7.1 had almost the same map location for their most-severely distorted marker among the four populations. Some SDRs had the same distorted markers detected in two or more populations. For example, *nc*003 in SDR2.2 was distorted in both LH200/LH216 F_2Syn3 and B73/Mo17 RI 1997. Markers *umc*68 and *phi*085 in SDR5.1 were distorted in LH200/LH216 F_2Syn3, Mo17/H99 $F_{6:7}$ RI and Tx303/CO159 F_2 1995–1998. Marker *umc*7 in SDR8.2 was distorted in B73/Mo17 RI 1997 and Tx303/CO159 F_2 1995–1998 (Table 3).

On the other hand, some SDR map locations, as indicated by the most-severely distorted markers in the region, comprised a wide chromosomal region with a range of two to four bins, such as SDR1.2, SDR4.1 and SDR8.2 (Fig. 1). Further studies are needed to determine whether or not these large SDRs comprise two or more smaller SDRs.

Causes of segregation distortion

A variety of physiological and genetic factors could cause segregation distortion (Grant 1975, pp 228–250). Mechanisms for preferential segregation include pollen-tube competition (Mangelsdorf and Jones 1926; Levin and Berube 1972; Liedl and Anderson 1993), pollen lethals (Rick 1966), preferential fertilization (Schwemmle

Table 2 SSR markers showing segregation distortion ($P < 0.05$) in the LH200/LH216 F_2Syn3 maize population, the numbers of homozygotes and heterozygotes at each marker locus, and their direction of skewness

SSR locus	Chromosome	Number of homozygotes		Number of heterozygotes	χ^2 value	Direction of skewness
		LH200 allele	LH216 allele			
phi011	1	78	41	225	40.6	Heterozygote
dupssr25	2	76	62	197	11.6	Heterozygote
asg088	2	77	61	201	13.2	Heterozygote
a1233	2	78	61	200	12.7	Heterozygote
mer161	10	77	56	197	15.1	Heterozygote
a1138	2	105	65	178	9.4	LH200
a1325	3	120	46	161	33.6	LH200
phi029	3	113	74	151	12.8	LH200
a1638	3	113	65	161	14.4	LH200
a1452	3	107	54	151	18.3	LH200
phi053	3	117	67	154	17.5	LH200
a1601	3	113	71	160	11.9	LH200
a1117	3	120	72	158	16.5	LH200
a2241	3	116	73	152	14.9	LH200
a1449	3	123	62	144	27.7	LH200
a1951	3	123	72	146	22.3	LH200
a1160	3	129	76	144	26.8	LH200
bnlg197	3	122	59	135	31.8	LH200
dupssr17	3	115	64	143	20.2	LH200
a1605	3	131	60	143	37.1	LH200
a1890	4	116	63	168	16.5	LH200
phi048	5	110	73	161	9.4	LH200
phi085	5	119	75	150	16.9	LH200
a1346	5	130	66	140	33.7	LH200
a1885	5	132	53	162	37.5	LH200
asg066	9	106	56	184	15.9	LH200
a1209	9	113	67	167	12.7	LH200
asg124	9	111	66	165	12.3	LH200
asg093	9	118	62	160	19.6	LH200
phi065	9	115	64	162	16.1	LH200
dupssr06	9	112	71	143	15.2	LH200
phi028	9	122	69	145	23.0	LH200
phi033	9	131	71	143	31.0	LH200
a1583	9	115	63	157	17.5	LH200
a1724	9	104	55	174	15.1	LH200
a2248	2	55	119	163	24.7	LH216
bnlg490	4	58	105	183	13.9	LH216
umc005	4	48	148	144	66.8	LH216
phi079	4	57	117	174	20.7	LH216
a1755	4	60	101	168	10.4	LH216
phi026	4	42	121	185	37.3	LH216
asg115	6	64	117	167	16.7	LH216
a1732	6	64	116	160	17.1	LH216
bnlg345	6	54	129	153	36.2	LH216
phi070	6	84	114	143	14.2	LH216
a1065	8	54	130	147	39.0	LH216
bnlg240	8	56	126	160	30.1	LH216
a2181	8	55	143	138	56.8	LH216
asg105	8	51	128	158	36.5	LH216
a2082	8	52	118	157	27.2	LH216
a1067	8	50	118	142	32.0	LH216
asg025	8	51	118	169	26.6	LH216
a1839	10	56	119	174	22.7	LH216
a1518	10	70	112	152	13.3	LH216
phi059	10	49	109	185	23.1	LH216

1968; Gadish and Zamir 1986) and selective elimination of zygotes (Rick 1963). In maize, the most-commonly reported genetic factors associated with the distorted segregation ratio are gametophytic factors (*ga*) (Mangelsdorf and Jones 1926; Burnham 1936; Jain 1967; Pfahler 1975; Neuffer et al. 1997).

*Ga*1 was mapped on chromosome 4 (Emerson et al. 1935) in the region of bin 4.02 (Pioneer Composite 1999 in Maize DB, panel of stocks ID #258944; Bins Map). SDR4.1 was mapped nearby to *Ga*1 (Fig. 1), and it was detected in all four populations. The two parental inbreds of each of the four populations may have had different

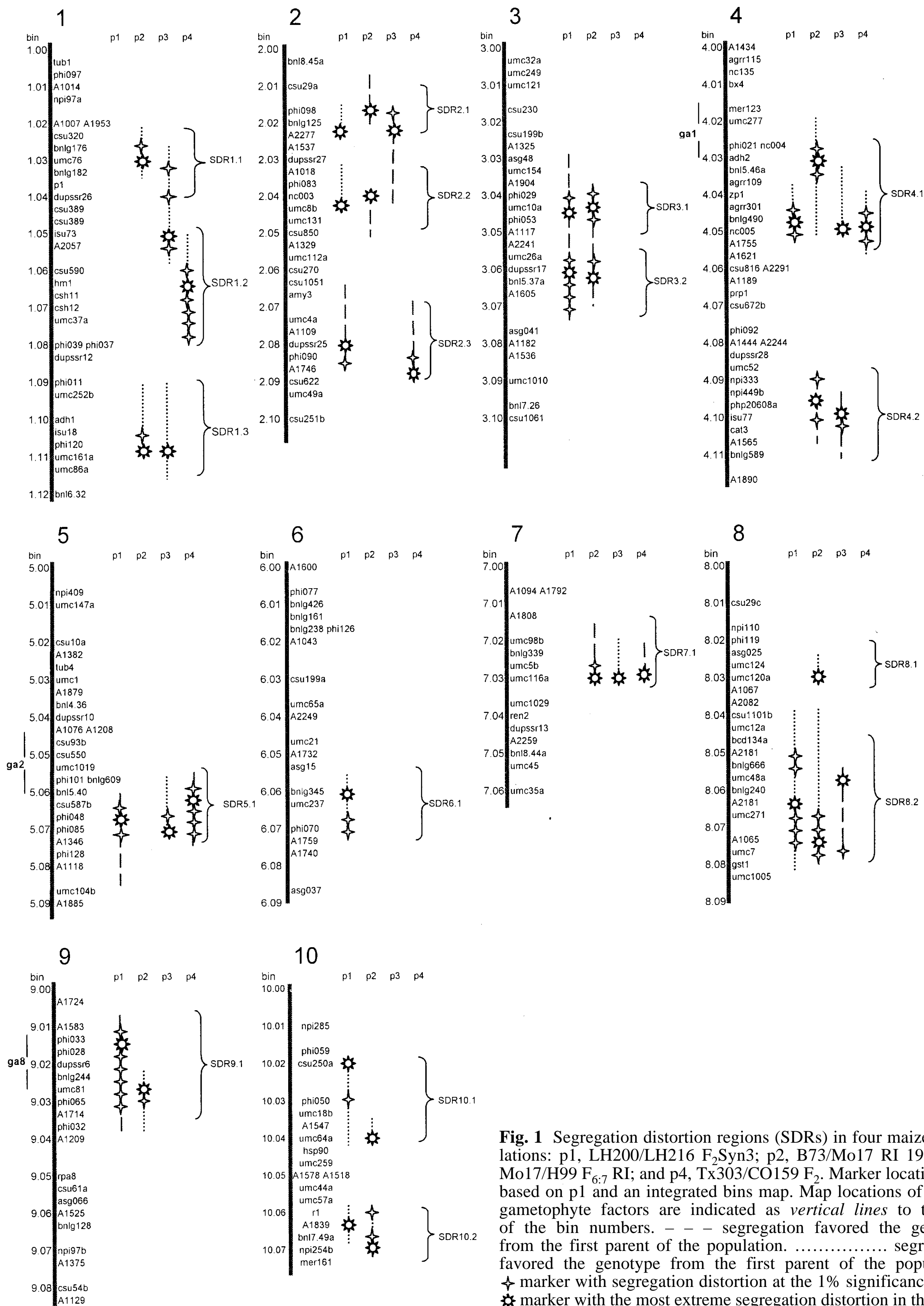

Fig. 1 Segregation distortion regions (SDRs) in four maize populations: p1, LH200/LH216 F_2Syn3; p2, B73/Mo17 RI 1997; p3, Mo17/H99 $F_{6:7}$ RI; and p4, Tx303/CO159 F_2. Marker locations are based on p1 and an integrated bins map. Map locations of known gametophyte factors are indicated as *vertical lines* to the left of the bin numbers. – – – segregation favored the genotype from the first parent of the population. …………… segregation favored the genotype from the first parent of the population. ✧ marker with segregation distortion at the 1% significance level. ✡ marker with the most extreme segregation distortion in the SDR

alleles at the *Ga*1 locus. The *ga*2 allele is located near SDR5.1, and *ga*8 is located in SDR9.1. Only three out of 18 SDRs were detected close to the locations of five known gametophytic factors (Fig. 1). This result suggests that gametophytic factors may not be the only genetic reason for segregation distortion. On the other hand, there may be other gametophytic factors that have not been identified and mapped.

The excess of one of the homozygous genotypes, in 50 out of the 55 loci (91%) exhibiting segregation distortion in LH200/LH216 F_2Syn3, is consistent with the effects of gametophytic factors. Consider a locus (*A*) that is linked with a recombination frequency of *r* with a gametophytic factor (*G*). An inbred with genotype *AG* is crossed with an inbred with genotype *ag*. We assume that segregation distortion occurs in one sex only (the male, for convenience of discussion). The proportion of male gametes that are successful in fertilization is denoted by *p* for gametes with the *g* allele, and $(1-p)$ for gametes with the *G* allele. The value of *p* is therefore equivalent to the fitness associated with the *g* allele. Normal segregation occurs when *p* is equal to 0.50. Otherwise, the proportion of male gametes produced upon meiosis are $(1-p)(1-r)$ for *AG*; *pr* for *Ag*; $(1-p)r$ for *aG*; and $p(1-r)$ for *ag*. The frequency of the *A* allele is therefore $1-[p(1-r)+r(1-p)]$. The frequencies of the female gametes are unaffected. With a frequency of 0.50 for both the *A* and *a* alleles among female gametes, the frequency of *AA* genotypes therefore increases from 0.25 to $0.50\{1-[p(1-r)+r(1-p)]\}$, when both *r* and *p* are less than 0.50. Therefore, a gametophytic factor causes an excess of one homozygote and a deficiency of the other homozygote, but the frequency of the heterozygote remains constant at 0.50.

Aside from gametophytic factors, many naturally occurring gene mutants may cause segregation distortion of linked loci: *dek* (defective kernel genes), *ms* (male-sterile genes) and *emb* (the development of embryo-specific mutations) are three possible mutants (Neuffer et al. 1997, pp 310–319). But these mutant genes are unlikely be present in crosses among LH200, LH216, Mo17, B73 and H99, which have been or are being used as parents of commercial hybrid cultivars. A breeder would have easily selected against any segregation distortion due to defective kernels, male sterility or defective embryos. This result indicates that segregation distortion in elite germplasm is most-likely due to gametophytic factors, whose effects are not easily seen. The molecular-marker approach we used in this study is therefore especially appropriate for studying segregation distortion in elite germplasm.

Implications for linkage mapping

As Bailey (1949) and Lorieux et al. (1995a, b) indicated, the estimation of recombination distance remains unaffected if segregation distortion is due to only one locus in an SDR. Consider a gametophytic factor (*G*) that is flanked by two loci, *A* and *B*. The recombination frequencies are r_1 between *A* and *G*, and r_2 between *G* and *B*. The genotype of the F_1 between the *AAGGBB* and *aaggbb* cross is depicted as:

$$\frac{A \quad r_1 \quad G \quad r_2 \quad B}{a \qquad\quad g \qquad\quad b}$$

The frequencies of the male gametes are as follows: $(1-p)(1-r_1)(1-r_2)$ for *AGB*; $(1-p)(1-r_1)r_2$ for *Agb*; $pr_1(1-r_2)$ for *Agb*; pr_1r_2 for *AgB*; $p(1-r_1)(1-r_2)$ for *agb*; $p(1-r_1)r_2$ for *agB*; $(1-p)r_1(1-r_2)$ for *aGB*; and $(1-p)r_1r_2$ for *aGb*. Segregation distortion affects a backcross population (to either *ABC/ABC* or to *abc/abc*) when the F_1 is used as the male parent, but not when the F_1 is used as the female parent. In a backcross population with the F_1 as the male parent, the estimates of recombination frequency are:

$$\hat{r}_1 = pr_1(1-r_2)+pr_1r_2+(1-p)r_1(1-r_2)+(1-p)r_1r_2 = r_1$$

and

$$\hat{r}_2 = (1-p)(1-r_1)r_2+pr_1r_2+p(1-r_1)r_2+(1-p)r_1r_2 = r_2$$

These results indicate that, as was previously found by Bailey (1949) and Lorieux et al. (1995a, b), the estimates of recombination frequency are unaffected by segregation distortion due to a single gametophytic factor. The estimates of r_1 and r_2 remain unchanged if the order of loci is *A-B-G*. Lorieux et al. (1995a, b) found, however, that these results do not hold true when segregation distortion in an SDR is caused by more than one gametophytic factor. With two linked gametophytic factors, the estimates of recombination frequency and, consequently, map distance become biased. The implications of SDR on the estimation of map distances therefore depend on the number of genetic factors that cause segregation distortion in a given SDR. We speculate that in most instances only one gametophytic factor is present in an SDR. Interval mapping methods for mapping loci that cause segregation distortion (Vogl and Xu 2000) would be helpful in resolving the number of segregation distortion loci in each SDR.

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