Proc. Natl. Acad. Sci. USA
Vol. 92, pp. 6645–6649, July 1995
Plant Biology

Differentiation between homoeologous chromosomes 1A of wheat and $1A^m$ of *Triticum monococcum* and its recognition by the wheat *Ph1* locus

JORGE DUBCOVSKY, MING-CHENG LUO, AND JAN DVOŘÁK

Department of Agronomy and Range Science, University of California, Davis, CA 95616

Communicated by S. J. Peloquin, University of Wisconsin, Madison, WI, April 10, 1995

ABSTRACT **In most allopolyploid plants, only homogenetic chromosome pairing occurs in meiosis, as a result of the recognition of genome differentiation by the genetic system regulating meiotic chromosome pairing. The nature of differentiation between chromosomes of closely related genomes is examined here by investigating recombination between wheat chromosome 1A and the closely related homoeologous chromosome $1A^m$ of *Triticum monococcum*. The recognition of the differentiation between these chromosomes by the *Ph1* locus, which prevents heterogenetic chromosome pairing in wheat, is also investigated. Chromosomes 1A and $1A^m$ are shown to be colinear, and it is concluded that they are differentiated "substructurally." This substructural differentiation is argued to be recognized by the *Ph1* locus. In the absence of *Ph1*, the distribution and frequencies of crossing over between the 1A and $1A^m$ homoeologues were similar to the distribution and frequencies of crossing over between 1A homologues. The cytogenetic and evolutionary significance of these findings is discussed.**

Although polyploid plant species contain two or more related pairs of genomes, their meiosis is usually diploid-like, with almost exclusively homogenetic chromosome pairing. Bread wheat (*Triticum aestivum* L., $2n = 6x = 42$) is a good example of this paradox. Bread wheat originated from hybridization of three diploid species, *T. urartu* Thum. (A genome) (1–3), an extinct or undiscovered population closely related to *T. speltoides* (Tausch) Gren. (B genome) (4, 5), and *T. tauschii* (Coss.) Schmalh. (D genome) (6, 7). Although the chromosomes of these species have been subjected to significant differentiation, they are still capable of limited chiasmatic pairing at metaphase I (MI) in their diploid hybrids (8). However, there is very little MI pairing among the chromosomes of the A, B, and D genomes in wheat haploids (9). Nullisomy for the long arm of chromosome 5B—or a recessive mutation, *ph1b*, at the *Ph1* locus on this arm—restores MI pairing among the homoeologous chromosomes of the A, B, and D genomes (10–13). This indicates that *Ph1* plays a fundamental role in the discrimination between homologous and homoeologous chromosomes in wheat. In wheat breeding, *Ph1* represents a barrier to introgression of alien germ plasm, and recessive mutants at this locus (13, 14) are important genetic tools for obtaining introgression of alien chromosome segments with economically important genes into wheat. To understand the mechanism by which *Ph1* distinguishes homoeologous chromosomes from homologous chromosomes, it is necessary to understand (*i*) the nature of the differentiation between homoeologous chromosomes and (*ii*) the physiological mode of the action of the *Ph1* gene. The present investigation addressed both components of this problem.

A traditional hypothesis has been that chromosomes lose homology by structural rearrangements that alter the sequence of loci by chromosome inversions, translocations, duplications, etc. This hypothesis has led to the concept of segmental chromosome differentiation of homoeologous chromosomes (15). According to the segmental-differentiation hypothesis, homoeologous chromosomes are mosaics of homosequential segments and structurally altered segments. Structural chromosome heterozygosity may not manifest itself cytogenetically (may be cryptic), and the absence of inversion bridges or multivalents in hybrids does not constitute conclusive evidence against this hypothesis (15).

Alternatively, chromosomes of related genomes may be differentiated by the accumulation of "substructural" changes. Substructural differentiation [formerly referred to as "nonstructural" differentiation (16)] is assumed to leave chromosomes homosequential but alters their homology at the level of nucleotide sequences. This can range from nucleotide substitutions to various types of nucleotide sequence rearrangements, including insertions and deletions. Differentiation was shown to involve all chromosomes in a genome and to occur throughout the entire lengths of chromosome arms (16–18).

A widely accepted explanation of the physiological mechanism by which *Ph1* prevents pairing and recombination between differentiated (homoeologous) chromosomes is the somatic-association hypothesis proposed by Feldman *et al.* (19). This hypothesis postulates that the pattern of meiotic pairing in wheat is predetermined by the distance (association) between homologous and homoeologous chromosomes in the nucleus that persists throughout the entire life cycle (for review, see ref. 20). It is believed that *Ph1* affects chromosome pairing by modifying spindle proteins and primarily acts on the centromere (19). Although indirect evidence in support of this hypothesis has been reported (19, 21–23), direct evidence is lacking and the validity of the hypothesis has been questioned on several grounds (24–27).

Although it is not known at which time point of meiosis *Ph1* acts, a large body of evidence shows that its ultimate effect is manifested at MI. Therefore, recombination or chiasmatic pairing at MI can be equally employed in investigation of the effects of *Ph1* on meiotic chromosome pairing in wheat. Both approaches are employed here to examine the nature of differentiation, and its recognition by *Ph1*, between a single pair of closely related homoeologous chromosomes, 1A of wheat and $1A^m$ of *T. monococcum* L. ($2n = 2x = 14$). *T. monococcum* is closely related to *T. urartu*, the source of the wheat A genome, as indicated by the observation that their chromosomes form seven MI bivalents in their hybrids (28).

MATERIALS AND METHODS

Plants. An amphiploid nullisomic for 1A was developed from the cross *T. aestivum* cv. Chinese Spring (CS) monote-

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Abbreviations: cM, centimorgan(s); CS, Chinese Spring; MI, metaphase I; PMC, pollen mother cell; RFLP, restriction fragment length polymorphism; RSL, recombinant substitution line.

losomic 1AL × *T. monococcum* ssp. *aegilopoides* G1777 and was five times backcrossed to CS monotelosomic (Mt) 1AL (29). A disomic recombinant substitution line (RSL) was ultimately produced, RSL1A^{rec}. The substituted chromosome was largely 1A^{m} which had a distal region of the long arm recombined with CS1A within a 7-centimorgan (cM) interval between *Xmwg984* and *Xmwg710*. All backcross plants since BC_2 had the same recombined chromosome. Ditelosomic substitution lines for both arms of 1A^{rec} were developed.

Six mapping populations were developed (Table 1). Except for the *T. monococcum* ssp. *aegilopoides* population used to construct a genetic map of *T. monococcum* 1A^{m}, the mapping populations were monosomic RSLs in the isogenic background of CS (Table 1). A modified procedure of targeted homoeologous recombination (30) was used to develop a population of RSLs from recombination between RSL1A^{rec} and CS1A in the *ph1* background (Fig. 1). Chromosome 1A was replaced by *1E* of *Lophopyrum elongatum* (Host) Löve during the production of triple-monosomic *ph1b* in which *ph1b* induced homoeologous recombination between CS1A and 1A^{rec}. In the *Ph1* plants, 1E does not pair with 1A. Four triple-monosomic plants, designated 6, 7, 16, and 17, each hemizygous for *ph1b*, were used in crossing with monotelosomic 1AL to produce monosomic RSLs (Table 1). Since *ph1b* was available only in the CS genetic background, and since 1A in the triple-monosomic *ph1b* plants was contributed by the *ph1b* stock, the chromosomes targeted for homoeologous recombination, 1A^{rec} and 1A, were heterozygous for the 1A^{m} segment but homozygous (CS) for the 1A segment. The same was true for the *Ph1* control populations [1A^{rec} × CS and 1A^{rec} × CS double-ditelosomic 1A (henceforth DDt1A)] (Table 1). MI chromosome pairing was investigated by determining the percentages of pollen mother cells (PMCs) in which CS1A and 1A^{rec} paired with CS telosomes 1AS and 1AL in progeny from crosses of CS and RSL1A^{rec} with DDt1A.

DNA Hybridization. Southern hybridization of DNAs with probes listed in Figs. 2 and 4 was done as described (31). Probes were prepared either by excising inserted DNA fragments with a restriction endonuclease or by PCR amplification followed by purification with a Magic PCR purification kit (Promega).

Map Construction and Statistical Comparisons. The goodness of fit of segregation of each pair of alleles was tested with χ^2. Linkage maps were constructed with MAPMAKER/EXP 3.0 (32, 33) and JOINMAP 1.4 (34) using the Kosambi function (35). Distances presented in Figs. 2–4 were obtained with MAPMAKER/EXP 3.0. The significance of the differences of the recombination fractions in the same intervals in different maps was determined by the *Z* test.

RESULTS

Polymorphism. Restriction fragment length polymorphism (RFLP) between RSL1A^{rec} and CS and between disomic substitution line Cheyenne 1A in CS (68) (DSCnn1A) and CS was investigated with 558 (75 probes) and 615 (42 probes) probe–enzyme combinations, respectively. A mean of 62.1% probe–enzyme combinations showed a polymorphism in the

Table 1. Mapping populations

Cross	*Ph1*	Type	No.
T.m.a. G2528 × *T.m.a.* G1777	–	F_3 families	76
RSL1A^{rec} × CS	–	RSLs	96
RSL1A^{rec} × CS	+	RSLs	36
RSL1A^{rec} × CS DDt1A	+	RSLs	59
DSCnn1A × CS	+	RSLs	101
RSL21 × DSCnn1A	+	RSLs	147

T.m.a., *T. monococcum* ssp. *aegilopoides*; DDt, double ditelosomic; DS, disomic substitution line; Cnn, wheat cv. Cheyenne.

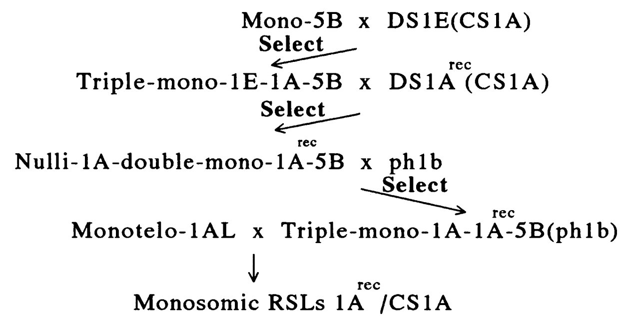

FIG. 1. Scheme for development of monosomic RSLs in the *ph1b* background. Monosomic, monotelosomic, and nullisomic are abbreviated as mono, monotelo, and nulli, respectively.

former comparison and 12.5% in the latter comparison ($P < 0.01$).

Recombination and MI Pairing Frequencies Between 1A^{rec} and 1A. In the presence of *Ph1*, only 2 of 59 chromosomes derived from the cross involving DDt1A and only 1 of 36 chromosomes from the cross involving CS were recombined in the homoeologous region (Fig. 2). Since both chromosomes with recombined short arm were derived from the cross involving DDt1A as the male parent, the inferred length of 2.2 cM for the short arm (Fig. 2) is an overestimate because of the selection for pollen grains from the PMCs in which the 1AS telosome paired at MI (59). The recombination was adjusted for this bias to 1.1 cM (Fig. 3) by use of a formula for a backcross employing a monotelodisomic as a male (59), which is applicable to the backcross progeny of both a monotelodisomic and a double monotelotrisomic.

CS telosomes 1AS and 1AL paired at MI with CS1A in 86.2% and 100.0% of PMCs, respectively, and with 1A^{rec} in 0.76% and 91.8% of PMCs, respectively (Fig. 3). In the short arm, the crossover frequency of 0.38% calculated from MI chromosome pairing (0.5 × MI pairing frequency) did not significantly differ from the genetic length based on recombination (1.1 cM). However, there was a great discrepancy between these two estimates in the long arm, indicating a high incidence of crossovers in the homologous segment (Fig. 3).

In the absence of *Ph1*, 70 monosomes were recombined due to one or more crossovers in a total of 96 monosomic RSLs. Sixteen RSLs derived from triple-monosomic *ph1b* plant 6 showed restriction fragments only for 1A^{m} alleles at the loci distal to *X5SDna1* in the short arm; the restriction fragments for 1A alleles were absent. These 1A nulls were always accompanied by increased signal levels of 1D restriction fragments. Obviously, triple-monosomic *ph1b* plant 6 acquired a recombined 1D/1A chromosome instead of 1A from the *ph1b* stock. The remaining three triple-monosomic *ph1b* plants, nos. 7 (62 RSLs), 16 (6 RSLs), and 17 (12 RSLs), acquired an intact 1A from the *ph1b* stock. In the *ph1* state, the presence of a distal 1D region in 1A of plant 6 reduced recombination in the proximal region by 62% in comparison with plants 7, 16, and 17. The short-arm markers of the sixteen plants were, therefore, excluded from the data. The recombination in the long arm in family 6 did not significantly differ from that in the remaining three families.

The segregation at all loci fit the expected 1:1 ratio. Recombination was found along the entire homoeologous region (Fig. 2). Eighty-two markers were mapped in the 123.3-cM interval between *XNor* in the short arm and *Xmwg984* in the long arm. Among the 70 recombined monosomes, 1A was recombined with a chromosome other than 1A^{rec} in only 3 (nos. 12, 16, and 19). Both alleles distal to *Xmwg706* (RSL12) and *Xbcd808* (RSL16), and the entire region from *XNor* to *Xmwg733* (RSL19), were absent. Chromosome numbers and allele dosages were determined from 7–10 F_2 progeny of these

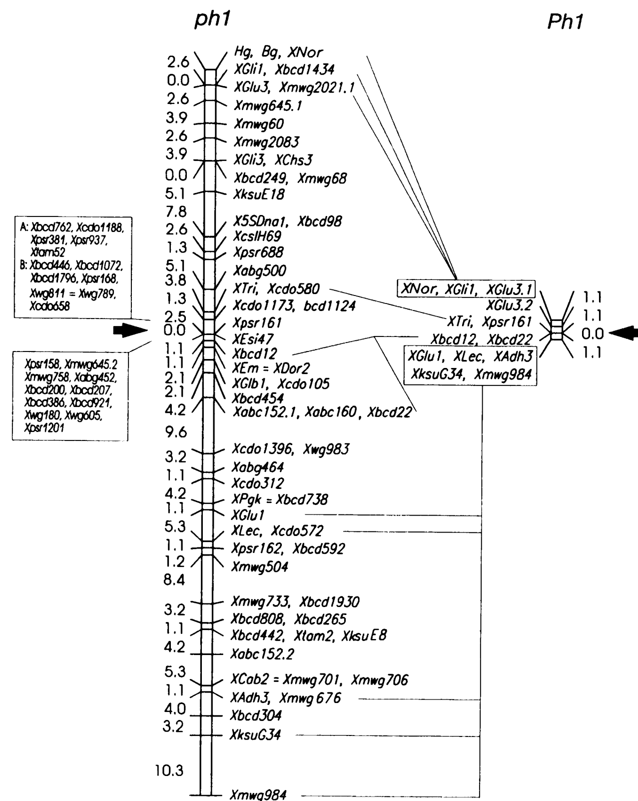

FIG. 2. Linkage maps constructed from recombination between $1A^m$ region of $1A^{rec}$ and CS1A in the genetic background of CS in the presence (*Ph1*) or absence (*ph1*) of the *Ph1* locus. The positions of the centromere, inferred by telocentric mapping, are indicated by arrows. Centromeric markers that showed no recombination in the short arm are listed in the upper box and those in the long arm are listed in the lower box to the left of the *ph1* map. The A group of the short-arm markers is distal to the B group, which was determined by the mapping of a deletion–duplication in RSL37 (see *Results*). The morphological loci were *Hg* (hairy glume) and *Bg* (black glume). The remaining loci were mapped by hybridization of DNA probes: *XNor* [pTa750.15 (36)], *XGlu3* [pTdUCD1 (37)], *XGli3* and *XGli1* [pcP387 (38)], *XChs3* (39), *X5SDna1* [pTa794 (40)], *XTri* [Tri25-11 (41)], *XEsi47* [pESI47 (42)], *XEm* [p1015 (43)], *XDor2* [pMA1959 (44)], *XGlb1* (45), *XPgk* (46), *XGlu1* [pDY10A/KS⁻ (47)], *XLec* [pNVR20 (48)], *XCab2* [pKG1490 (49)], *XAdh3* [p3′NTR (50)]. Other loci were mapped with probes developed as described: *Xabg* and *Xabc* (51); *Xmwg* (52); *Xbcd*, *Xcdo*, and *Xwg* (53); *Xglk* (54), *Xtam* (55), *Xksu* (56), *XcsiH69* (57), and *Xpsr* (58). Note that 3.3 cM in the *Ph1* map equals 2.2 cM after the adjustment for selection favoring euploid pollen grains.

RSLs. Complete agreement was found between the number of chromosomes present (nulli-, mono-, or disomy) and the increased dosage of the 1D alleles, indicating that CS1A was recombined with CS1D in all three RSLs. Hence, 95.7% of the recombined chromosomes originated from recombination between 1A and $1A^{rec}$, and 4.3% from recombination between 1A and 1D; $1A^{rec}$ did not recombine with any other chromosome except for 1A.

In one RSL (no. 37), markers *Xtam52, Xcdo1188, Xpsr937, Xpsr381*, and *Xbcd249* were heterozygous and all markers distal to *XGli3* were nulls. This allelic arrangement of the short arm cosegregated in eight progeny plants. A likely possibility is that the monosome of RSL37 originated from an unequal crossover in the inverted orientation distal to *XGli3* between 1A and $1A^{rec}$, followed by formation of a dicentric bridge and breakage proximal to the $1A^{rec}$ centromere, resulting in a duplication–deletion.

Colinearity of 1A and $1A^m$ Chromosomes. Linkage maps of $1A^m$ in *T. monococcum* and 1A in *T. aestivum* based on the CS

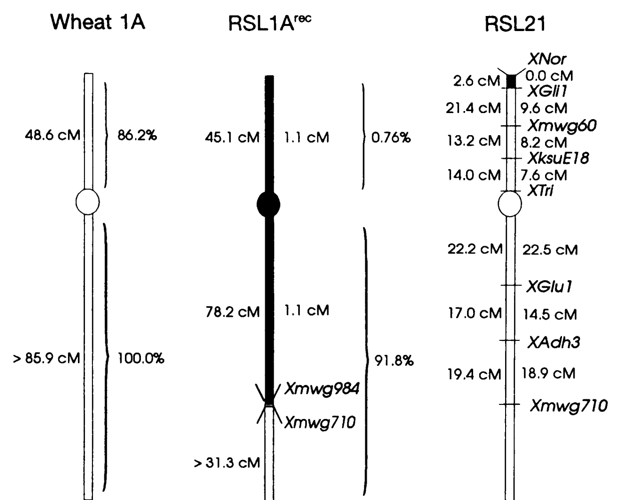

FIG. 3. The structure of wheat chromosome 1A and recombined $1A/1A^m$ chromosomes in $RSL1A^{rec}$ and RSL21. Chromosome $1A^m$ segments are black and those of 1A are white. Genetic lengths (cM) of the 1A intervals are from the CS1A × Cnn1A *Ph1* map (see Fig. 4) and are shown to the left of the chromosomes. Genetic lengths (cM) of the $1A^m$ intervals are from the $RSL1A^{rec}$ × CS1A *ph1* map (Fig. 2) and are also shown to the left of the chromosomes. Genetic lengths (cM), if determined, of these intervals based on recombination between chromosome $1A^{rec}$ and CS1A and between $1A/1A^m$ of RSL21 and Cnn1A in the presence of *Ph1* are shown to the right of the chromosomes. Pairing frequencies, in terms of the percentages of cells in which a chromosome paired with CS telosomes 1AS and 1AL, are shown to the right of the chromosomes. The MI pairing frequencies with CS telosomes of wheat 1A were determined in 51 cells, and those of $1A^{rec}$ in 1118 cells for the short arm and 61 cells for the long arm.

× DSCnn1A mapping population were compared to determine the relative order of markers in the two chromosomes. The two maps were colinear for 19 common loci (Fig. 4). Except for *XAga7* in *T. monococcum* ($P = 0.03$), all loci segregated in the expected 1:2:1 ratio. In only two intervals, *XEm–XGlu1* ($P < 0.01$) and *XGli1–Xmwg60* ($P < 0.05$) were the maps significantly different; the *T. monococcum* map was shorter in both intervals. These findings indicated that $1A^m$ and 1A have the same linear arrangement of loci and, except for two regions, show a similar distribution of crossovers.

Recombination of RSL21 Monosome with Cnn1A. In monosomic RSL21, derived from triple-monosomic *ph1b* plant 17, the recombined monosome was CS1A except for a short region distal to *XGli1* in the short arm, which was $1A^m$ (Fig. 3). Among 147 monosomes from the cross Mt1AL × (RSL21 × DSCnn1A), no recombination was found in the terminal homoeologous region, but recombination was observed in all intervals in the juxtaposed homologous region (Fig. 3). However, compared with recombination between Cnn1A and CS1A (Fig. 4), recombination in the homologous region was reduced (Fig. 3). The greatest reduction ($P < 0.05$) was in the interval closest to the homoeologous region (Fig. 3). The reductions in the other two intervals were not statistically significant (Fig. 3). No reduction in the homologous recombination occurred in the long arm (Fig. 3).

Pattern of Recombination Between Homoeologues. Twenty-two intervals in the map based on homoeologous (*ph1*) recombination between CS1A and $1A^{rec}$ were compared with the map based on recombination between *T. aestivum* homologues CS1A and Cnn1A. Only the intervals *Xcdo105–Xwg983, Xpsr162–Xbcd808*, and *XGlu3–Xmwg60* were significantly different ($P < 0.01$, 0.05, and 0.05, respectively). In the first and second intervals, the CS1A × $1A^{rec}$ map was longer, whereas in the third it was shorter. The existence of a closely linked locus that is paralogous to *Xbcd808* in 1A (61) makes the

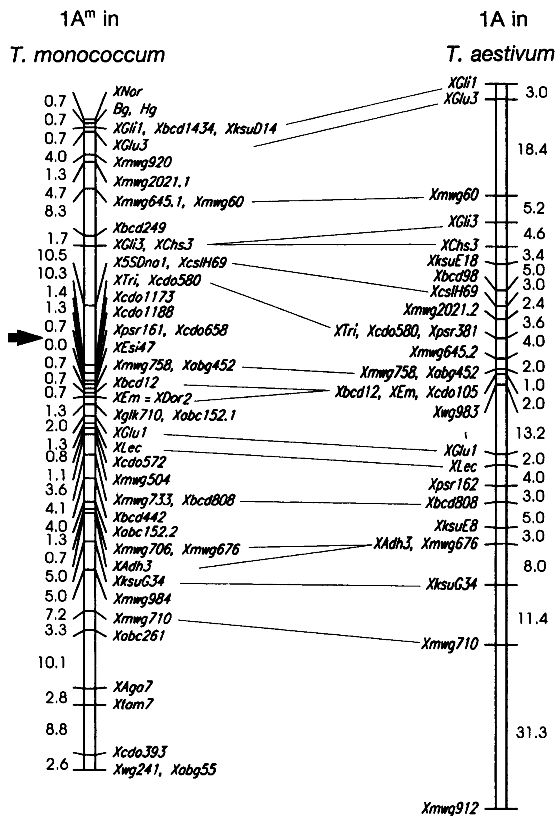

FIG. 4. Linkage map of 1Aᵐ constructed with *T. monococcum* mapping population G1777 × G2528 and that of 1A constructed with mapping population of DSCnn1A × CS1A RSLs. Probes and their sources are described in Fig. 2 except for *XAga7*, which was mapped with probe WE:AGA7 (60). Distances are given (cM).

second difference questionable. The intervals *Xcdo105–Xwg983* and *XGlu3–Xwmg60* are within intervals—*XEm–XGlu1* and *XGli1–Xmwg60*, respectively—in which the *T. aestivum* 1A map differed from the *T. monococcum* 1Aᵐ map (Fig. 4). The interval lengths in the homoeologous region near its border with the homologous region in the *ph1* CS1A × 1A^{rec} map were not shorter than the same intervals in the map based on recombination between Cnn1A and CS1A in the presence of *Ph1*.

DISCUSSION

Although recombination occurred in the homoeologous region between 1A^{rec} and 1A in both the presence and the absence of *Ph1*, it was 56 times higher, 123.3 cM vs. 2.2 cM, in the absence of *Ph1* than in its presence. While recombination in the presence of *Ph1* was dramatically reduced in the homoeologous region of the 1A^{rec} long arm, crossing-over had to be present in the homologous region. This is indicated by high MI pairing between the long arm of 1A^{rec} and CS telosome 1AL. The agreement between the genetic lengths of the short arm of 1A^{rec} estimated from homoeologous recombination and homoeologous MI chromosome pairing shows that there was a reasonably good agreement between MI chromosome pairing and crossovers. That crossovers frequently occur in homologous regions bordering homoeologous regions in the presence of *Ph1* was directly shown by the investigation of recombination between the 1A/1Aᵐ monosome of RSL21 and Cnn1A.

Poor homoeologous recombination in the presence of *Ph1* could not be due to a genetic background that was foreign to the *T. monococcum* chromosome; when *Ph1* was absent, the *T. monococcum* segment recombined in this background as in its own background. The poor recombination was also not caused by structural differences between 1Aᵐ and 1A; they were found to be colinear. Also, no evidence was found for cryptic structural differences, since the two chromosomes recombined as if they were undifferentiated once *Ph1* was removed.

Feldman and collaborators (19, 21–23) hypothesized that *Ph1* suppressed recombination between homoeologues by altering a fixed spatial relationship between homologues and homoeologues in the nucleus via interaction of chromosomes with spindle proteins; this fixed spatial relationship was argued to persist throughout the entire life cycle. The present results contradict this hypothesis. In the presence of *Ph1*, crossing-over between 1A^{rec} and CS1A efficiently occurred in the distal homologous region, as indicated by almost complete MI pairing between telosome 1AL and 1A^{rec}, even though the two chromosomes had homoeologous centromeres. Recombination precipitously ceased in the juxtaposed homoeologous region even when the chromosomes shared homologous telomeres and a homologous distal region. Observations on the synapsis of chromosomes in the presence and absence of *Ph1* also argue against the possibility that *Ph1* prevents synapsis and recombination between homoeologues by altering some fixed arrangements of chromosomes in the nucleus (27). Moreover, no differences could be found in the distribution of homologous chromosomes in the root tips of plants in which *Ph1* was active and inactive (24, 25). Analysis of PMCs somatically reduced by premeitotic multipolar divisions has revealed that wheat homologues are not associated in premeiotic cells (26).

It could be argued that the observation that recombination did not reach the normal levels in the homologous region bordering homoeologous region in the short arm of 1A/1Aᵐ of RSL21 in the presence of *Ph1* supports the somatic-association hypothesis. However, the association would have to be mediated by the telomeres, since the centromeres were homologous in this case. That there was virtually no recombination in the long-arm homoeologous region between 1A^{rec} and CS1A in the *Ph1* state, although the arms had homologous telomeres, shows that the telomere cannot be the site where homoeology is distinguished from homology by *Ph1*. While it is not clear why 1A homologous recombination was reduced in the vicinity of the homoeologous segment in the presence of *Ph1*, our data consistently show that *Ph1* distinguishes homoeology from homology along the entire chromosome.

Reduction of proximal homologous recombination also occurred in triple-monosomic plant 6, in which 1A had a distal region replaced by 1D. This plant was, however, *ph1*, and the reduction in recombination of 1D/1A with 1A was most likely caused by pairing of the 1D segment of 1D/1A with 1D.

The MI chromosome pairing in hybrids between different wheat cultivars is reduced in the A and B genomes in comparison to that in the inbred parental lines; for some chromosome arms, such as the short arms of 1B and 6B, MI pairing can be reduced by as much as 40% (16). These MI pairing reductions were shown to be caused by factors along the entire lengths of chromosomes rather than a single factor in a specific region, such as the telomere or the centromere (17, 18, 62). RFLP between CS1A and 1A^{rec} is 5-fold higher than that between CS1A and Cnn1A. It should, therefore, not be surprising to find poor recombination between 1A and 1A^{rec} in the presence of *Ph1* if *Ph1* would recognize substructural polymorphism between chromosomes.

The extensive polymorphism between 1A and 1A^{rec} did not markedly reduce recombination once *Ph1* was removed; only 1 of the 22 investigated intervals was significantly shorter (2 were significantly longer) in the map based on recombination between homoeologous chromosomes in the absence of *Ph1* than in the map based on recombination between homologous chromosomes Cnn1A and CS1A. It is likely, therefore, that the reductions in MI pairing between homologous chromosomes

from different wheat cultivars, as well as 1A and $1A^{rec}$, are caused by *Ph1*. Since genes with strong activity such as *Ph1* have not been detected in diploids, except for B chromosomes (63), it is not surprising that crossover frequencies are not usually affected by intraspecific substructural polymorphism at the diploid level (18).

Except for several major translocations involving a few of the 21 wheat chromosomes (64), comparative mapping has not revealed structural differences between homoeologous chromosomes of the wheat A, B, and D genomes (61, 65, 66). It could be argued that these chromosomes do differ structurally but the differences are cryptic (15)—i.e., too small to be detected in low-density comparative maps. However, in the absence of *Ph1*, recombination between 1A and $1A^{rec}$ was significantly lower in only one interval than that between wheat homologues, arguing against the possibility that the poor recombination along the entire $1A^{m}$ segment in the presence of *Ph1* was caused by structural differences. Since the substructural differentiation was sufficient to almost eliminate crossing-over in the homoeologous region of $1A^{rec}$ in the presence of *Ph1*, the same form of differentiation may be able to preclude crossing-over between the homoeologous chromosomes of the wheat A, B, and D genomes in the presence of *Ph1*.

The A, A^{m}, B, and D genomes exemplify genomes constituting the chromosome complements of segmental allopolyploids (15). As shown here for chromosomes 1A and $1A^{m}$, the chromosomes are not mosaics of homologous and homoeologous segments, as assumed by the segmental-allopolyploidy concept, but show a uniform differentiation along their entire lengths. Hence, the concept of segmental differentiation and segmental allopolyploidy appears to have no factual basis and should be abandoned, as has already been argued on the basis of other evidence (67).

We thank Ronda Bransteitter and Amrita Desai for excellent technical assistance and Dr. R. S. Kota for providing us with seeds of the $1A^{rec}$ substitution line. This project was supported by Operating Grant CA-D-ARS-5717-CG from the U.S. Department of Agriculture–National Research Initiative Competitive Grants Program. J. Dubcovsky also thanks the Argentinian Research Council (CONICET) for a fellowship.

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