Recognition of Homeology by the Wheat *Ph1* Locus

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ABSTRACT

Chromosome $1A^m$ of *Triticum monococcum* is closely homeologous to *T. aestivum* chromosome *1A* but recombines with it little in the presence of the wheat suppressor of homeologous chromosome pairing, *Ph1*. In the absence of *Ph1*, the two chromosomes recombine as if they were completely homologous. Chromosomes having either terminal or interstitial segments of chromosome $1A^m$ in *1A* were constructed and their recombination with *1A* was investigated in the presence of *Ph1*. No recombination was detected in the homeologous ($1A^m/1A$) segments, irrespective of whether terminally or interstitially positioned in a chromosome, whereas the levels of recombination in the juxtaposed homologous (*1A/1A*) segments was normal or close to normal relative to completely homologous *1A* chromosomes. These observations show that *Ph1* does not regulate chromosome pairing by premeiotic chromosome alignment and a mitotic spindle-centromere interaction, as has been suggested, but processes homology along the entire length of chromosomes.

ALLOHEXAPLOID bread wheat, *Triticum aestivum* L. ($2n = 6x = 42$; genomes AABBDD) originated from hybridization of three diploid species. In spite of the close relationship among the genomes of these species, wheat chromosomes pair only homogenetically at metaphase I (MI). Metaphase I pairing between homeologous chromosomes is also virtually absent in wheat haploids (RILEY 1960; MCGUIRE and DVOŘÁK 1982). The suppression of pairing between wheat homeologous chromosomes is primarily due to the activity of the *Ph1* locus on chromosome *5B*, which is evidenced by high levels of homeologous chromosomes pairing in wheat *5B*-nullihaploids, *5B*-nullisomics, interspecific hybrids lacking chromosome *5B*, and recessive mutants at the *Ph1* locus (OKAMOTO 1957; RILEY and CHAPMAN 1958; SEARS and OKAMOTO 1958; RILEY 1960; SEARS 1977; GIORGI and CUOZZO 1980).

A number of hypotheses have been suggested to explain the mechanism by which *Ph1* controls heterogenetic chromosome pairing. It was speculated that *Ph1* may control the long range recognition of homologues, although a specific mechanism was not suggested (RILEY 1960), or that *Ph1* may shorten the duration of meiotic prophase, allowing homologues to pair but not providing sufficient time for homeologues to pair (RILEY 1968). Since meiosis was found to be of a similar length in the *Ph1* and *ph1* plants the latter hypothesis was abandoned (BENNETT *et al.* 1974).

FELDMAN *et al.* (1966) observed univalents, multivalents and interlocking of bivalents in plants with increased dose of the *5BL* arm and concluded that *Ph1* controls heterogenetic chromosome pairing by suppressing premeiotic association of chromosomes. Premeiotic association was concluded to be partially suppressed at two doses of *Ph1*, eliminating pairing between homeologues (FELDMAN *et al.* 1966; FELDMAN 1993). At higher doses of *Ph1*, premeiotic association was hypothesized to be suppressed also between homologues, resulting in overall reduction of pairing, its randomness, and interlocking of bivalents. Measurements of distances between telocentrics in root tip metaphase plates seemingly supported this hypothesis since homologous telocentrics were found to be closer to each other than homeologous telocentrics (FELDMAN *et al.* 1966). Since telocentrics for opposite arms of a chromosome were found to be associated in *Ph1* plants, and since *Ph1* plants were found to be more colchicine sensitive than *ph1* plants, it was concluded that interactions of mitotic spindle microtubules with the centromeres are responsible for premeiotic association of chromosomes and, consequently, for the suppression of heterogenetic chromosome pairing at meiosis (FELDMAN *et al.* 1966, 1973; FELDMAN 1968, 1993).

These assumptions have been reinforced by the observation that application of colchicine on premeiotic cells phenocopies the effects of increased doses of *Ph1* (DRISCOLL *et al.* 1967; DOVER and RILEY 1973). In wheat and other plants, multivalents are observed after treatments of meiocytes with low concentrations of colchicine and are assumed to reflect homeologous chromosome pairing induced by disruption of homologous chromosome associations (FELDMAN 1968; DOVER and RILEY 1973; JACKSON and MURRAY 1983). DOVER and RILEY (1973) and AVIVI and FELDMAN (1973) found that the colchicine-sensitive period is either during the premeiotic mitosis or the transition between mitosis and

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G_1. On the basis of these observations, DOVER and RILEY (1977) modified the somatic association hypothesis and suggested that alignment of chromosomes occurs when chromosomes are contracted in the last premeiotic anaphase or telophase.

The development of synaptonemal complexes in *T. aestivum* euhaploids is limited compared to the *T. aestivum 5B*-nullihaploid but this is greatly dependent on the genotype. For example, in euhaploids (*Ph1*) derived from cv. Kedong, up to 90% of the chromosome complement was observed to be synapsed compared to a maximum of 41% in euhaploids derived from cv. Chinese Spring (WANG 1988). Yet, both types of haploids are essentially achiasmatic at MI (WANG 1988). Exchanges of pairing partners associated with extensive multivalent pairing were observed during early phases of synapsis in *Ph1* hexaploid wheat (HOLM 1986, 1988a,b; HOLM and WANG 1988), wheat *Ph1* haploids (WANG and HOLM 1988), and interspecific hybrids having *Ph1* (GILLIES 1987; WANG and HOLM 1988). The presence of multivalents in early prophase in both *Ph1* and *ph1* genotypes but their virtual absence in late pachytene and MI in the *Ph1* genotypes lead HOBOLTH (1981) and GILLIES (1987) to question the existence of premeiotic alignment of wheat chromosomes and to suggest that *Ph1* may regulate the rate of pairing or the timing of crossing over. HOLM and WANG (1988) suggested that *Ph1* affects both synapsis and crossing over by regulating heteroduplex formation.

To investigate the role of the centromere and telomere in the recognition of homeology, MI chromosome pairing and recombination were examined in *Ph1* plants with chromosome pairs in which a terminal homologous segment was present in otherwise homeologous chromosomes with a homeologous centromere or a terminal homeologous segment was present in otherwise homologous chromosomes with a homologous centromere (DUBCOVSKY *et al.* 1995; DVOŘÁK *et al.* 1995). In these chromosomes, recombination was essentially absent in the homeologous segments in the *Ph1* state.

The homeologous segments of these chromosome pairs were composed of genetic material of *T. aestivum* chromosome *1A* and *T. monococcum* chromosome $1A^m$. Although the genome of *T. monococcum* is closely related to the *A* genome of *T. aestivum* (CHAPMAN *et al.* 1976), *T. monococcum* chromosomes recombine very little with the *A* genome chromosomes if they are individually introgressed into *T. aestivum,* indicating that they are differentiated from them (PAULL *et al.* 1994; DUBCOVSKY *et al.* 1995). In the absence of *Ph1,* however, the level of recombination between $1A^m$ and *1A* does not differ from that between *T. aestivum 1A* homologues (DUBCOVSKY *et al.* 1995).

In the present study, the levels of recombination in interstitial homeologous segments composed of genetic material of *T. monococcum* $1A^m$ and *T. aestivum* in otherwise homologous chromosomes (homologous centromere and telomere) were quantitatively compared with the levels of recombination in (1) a chromosome pair having a terminal homeologous segment (homeologous telomere and homologous centromere), (2) a completely homologous chromosome pair in the presence of *Ph1,* and (3) a homeologous chromosome pair in the absence of *Ph1.* It is hypothesized that if the *Ph1* locus controls chromosome pairing by regulating premeiotic alignment of chromosomes, the recognition of homoeology from homology is impaired in homeologous interstitial segments inserted into otherwise homologous chromosomes in the *Ph1* plants.

MATERIALS AND METHODS

Chromosomes and mapping populations: Maps employed in this work (Table 1) were constructed from segregation in populations of recombinant substitution lines (RSLs). Wheat RSLs are lines in which a recombined chromosome is substituted either as a monosome or a disome for a homologue (or homeologue) of a recipient line. Hence, while individual lines in a population of RSLs differ in the recombined chromosome, they are isogenic for the remaining 20 chromosome pairs. In the populations employed here, chromosome *1A* was recombined whereas the remaining 20 chromosome pairs were of cv. Chinese Spring (CS) (Table 1). Each population was developed by crossing a specific F_1 with CS monotelosomic *1AL* (female) and selecting monosomics in the progeny (Table 1); only male meioses were consequently sampled in each RSL population.

Four RSL populations were developed here (Table 1). The initial material for the development of the four populations was RSL$1A^{rec}$, in which CS*1A* was replaced by *T. monococcum* chromosome $1A^m$ recombined with CS*1A* in the distal region of the long arm (Figure 1) (DUBCOVSKY *et al.* 1995; DVOŘÁK and DUBCOVSKY 1995). The RSL was crossed with the recessive CS *ph1b* mutant, and double monosomic *1A-*$1A^{rec}$ *ph1b* plants were selected. A population of 96 RSLs was developed by crossing the double monosomic *1A-*$1A^{rec}$ *ph1b* plants with CS monotelosomic *1AL* and used for a map construction (DUBCOVSKY *et al.* 1995). Two RSLs, designated numbers 82 and 104 (Figure 1), were selected for work described here. RSL82 harbored a *1A/*$1A^m$ chromosome with a terminal *T. monococcum* segment in the short arm and RSL104 harbored a *1A/*$1A^m$ chromosome with two interstitial *T. monococcum* segments (Figure 1) (for genotypes of these two chromosomes see DVOŘÁK and DUBCOVSKY 1995). The two RSLs were crossed with (1) CS and (2) a disomic substitution line in which chromosome *1A* of *T. aestivum* cv. Cheyenne (henceforth Cnn) was substituted for CS*1A* (henceforth DSCnn*1A*) (MORRIS *et al.* 1966). The F_1s were crossed as males with CS monotelo-*1AL,* producing thereby four RSL populations (Table 1). Genetic maps based on two additonal RSL populations in the CS background (Table 1) developed previously (DUBCOVSKY *et al.* 1995) were used. One was a population based on recombination between Cnn*1A* and CS*1A* in the presence of *Ph1* and was used to construct a map of wheat chromosome *1A,* and the other was a population based on recombination between $1A^{rec}$ and CS*1A* in the absence of *Ph1.* The latter was used to construct a map based on homeologous recombination of CS*1A* with the $1A^m$ portion of $1A^{rec}$ (DUBCOVSKY *et al.* 1995). The numbers of RSLs scored for construction of the maps are specified in Table 1.

Construction of maps and their comparisons: Nuclear

TABLE 1

Mapping populations of monosomic or disomic recombinant substitution lines for the investigation of recombination

Designation of population	Status of *Ph1*	Number of RSLs	Parentage	Reference
RSL82 × CS	*Ph1*	55	CS mt*1AL* × (CS RSL82 × CS)	Present data
RSL82 × DSCnn*1A*	*Ph1*	92	CS mt*1AL* × (CS RSL82 × DSCnn*1A*)	Present data
RSL104 × CS	*Ph1*	66	CS mt*1AL* × (CS RSL104 × CS)	Present data
RSL104 × DSCnn*1A*	*Ph1*	104	CS mt*1AL* × (CS RSL104 × DSCnn*1A*)	Present data
DSCnn*1A* × CS	*Ph1*	101	CS mt*1AL* × (DSCnn*1A* × CS)	DUBCOVSKY *et al.* (1995)
RSL$1A^{rec}$ × CS	*ph1*	96	CS mt*1AL* × (RSL$1A^{rec}$ × CS)	DUBCOVSKY *et al.* (1995)

RSL, recombinant substitution line.

DNAs were isolated according to DVOŘÁK *et al.* (1988). The sources of clones used as probes for the detection of restriction fragment length polymorphisms (RFLPs) were described by DUBCOVSKY *et al.* (1995). Southern blotting and DNA hybridization were performed as described earlier (DUBCOVSKY *et al.* 1994). RFLP maps were constructed with the computer program Mapmaker/EXP 3.0 (LANDER *et al.* 1987; LINCOLN *et al.* 1992) using KOSAMBI function (KOSAMBI 1943). To test the statistical significance of differences in the lengths of individual intervals between maps, interval lengths in cM were converted back into percentage of recombination using the KOSAMBI function, variances of the estimates of percentages of recombination were calculated according to ALLARD (1956), and the significance of differences in the lengths of specific intervals between maps were tested by the z-test.

Metaphase I (MI) chromosome pairing: CS double ditelosomic *1A* (henceforth DDt*1A*) was crossed with RSLs 82 and 104. Immature spikes were collected from the F_1 plants. Pollen mother cells (PMCs) were stained with acetocarmine and squashed. Slides were systematically scanned and pairing at MI of the *1AL* and *1AS* telosomes was recorded. Since it is very difficult to score chiasmata in wheat, the MI pairing frequency reflects the number of PMCs in which specific chromosome arms were paired.

RESULTS

Recombination and MI pairing of the *1A/1A*^{m} chromosome of RSL82 with CS*1A* and Cnn*1A*: The *1A/1A*^{m} chromosome in RSL82 has a terminal *1A*^{m} segment in the short arm (Figure 1). The exchange point is within interval *Xpsr688-Xabg500* (see DVOŘÁK and DUBCOVSKY 1995). The *1A*^{m} segment is a minimum of 32.4 cM long (the distance of *Xpsr688* from the terminal locus *Nor9*) and a maximum of 37.5 cM long (the distance of *Xabg500* from *Nor9*) in terms of recombination between *1A*^{rec} and CS*1A* in the absence of *Ph1* (Figure 2).

On the *ph1* RSL*1A*^{rec} × CS map, interval *Nor9-Xpsr688* in the *1A*^{m} segment was 32.4 cM long (Figure 2). In the presence of *Ph1*, recombination was not observed in this interval in either the RSL82 × CS population or the RSL82 × DSCnn*1A* population (Figure 2).

In the short proximal homologous segment in the short arm, interval *Xpsr688-XTri* was 5.3 cM on the RSL82 × DSCnn*1A* map, which is not significantly different ($P = 0.24$) from 8.9 cM, the length of this interval on the RSl*1A*^{rec} × CS *ph1* map (Figure 2). In the long arm, no interval on the RSL82 × DSCnn*1A* map significantly differed from a corresponding interval on the DSCnn*1A* × CS map (Figure 1).

MI pairing of the *1A/1A*^{m} chromosome of RSL82 with telosomes *1AS* and *1AL*: Telosomes *1AS* and *1AL* could be unequivocally distinguished from any other chromosome at MI and, hence, their pairing frequencies with the *1A/1A*^{m} chromosome could be determined by scoring the frequencies of PMCs in which a telosome paired with a complete chromosome. CS telosome *1AS* was paired with the *1A/1A*^{m} chromosome of RSL82 in 9.8% of the MI PMCs (Table 2). The *1AL* telosome paired with the *1A/1A*^{m} chromosome in 98% of the MI PMCs.

In the RSL82 × DSCnn*1A* population, the length of the proximal homologous segment, measured from *Xpsr688* to *XTri*, was 5.3 cM. Recombination in the interval *XTri*-centromere could not be determined in the RSL82 × DSCnn*1A* population. Interval *XTri*-centromere is 3.8 cM on the RSL*1A*^{rec} × CS map and 3.4 cM on the map of chromosome *1A*^{m} in *T. monococcum* (DUBCOVSKY *et al.* 1995). Adding an average of the two estimates, 3.6 cM, to the 5.3 cM, the length of the proximal pairing region is 8.9 cM in terms of recombination. This value is not significantly different from 4.9 cM calculated from the MI pairing frequency of the *1A/1A*^{m} chromosome with the *1AS* telosome (9.8 × 0.5), assuming that the telosome was paired with the complete chromosome by a single chiasma. Agreement between the two estimates suggests that chiasmata, like crossovers, were only in the homologous segment in the short arm.

Recombination of the *1A/1A*^{m} chromosome of RSL104 with CS*1A* and Cnn*1A*: The *1A/1A*^{m} chromosome in RSL82 has two interstitial *1A*^{m} segments, one in each arm (Figure 1). In the short arm, the exchange points are within the 5.1-cM *XChs3-XksuE18* interval on the distal side of the *1A*^{m} segment and within the 2.5-cM *Xbcd1124-Xcdo1188* interval on the proximal side (Figure 1; DVOŘÁK and DUBCOVSKY 1995). In the long arm, the exchange points are within the 4.2-cM *XksuE8-Xabc152.2* interval on the proximal side of the *1A*^{m} segment and within the 7.2-cM *Xmwg984-Xmwg710* interval on the distal side (Figure 1; see also DVOŘÁK and DUBCOVSKY 1995; DUBCOVSKY *et al.* 1995).

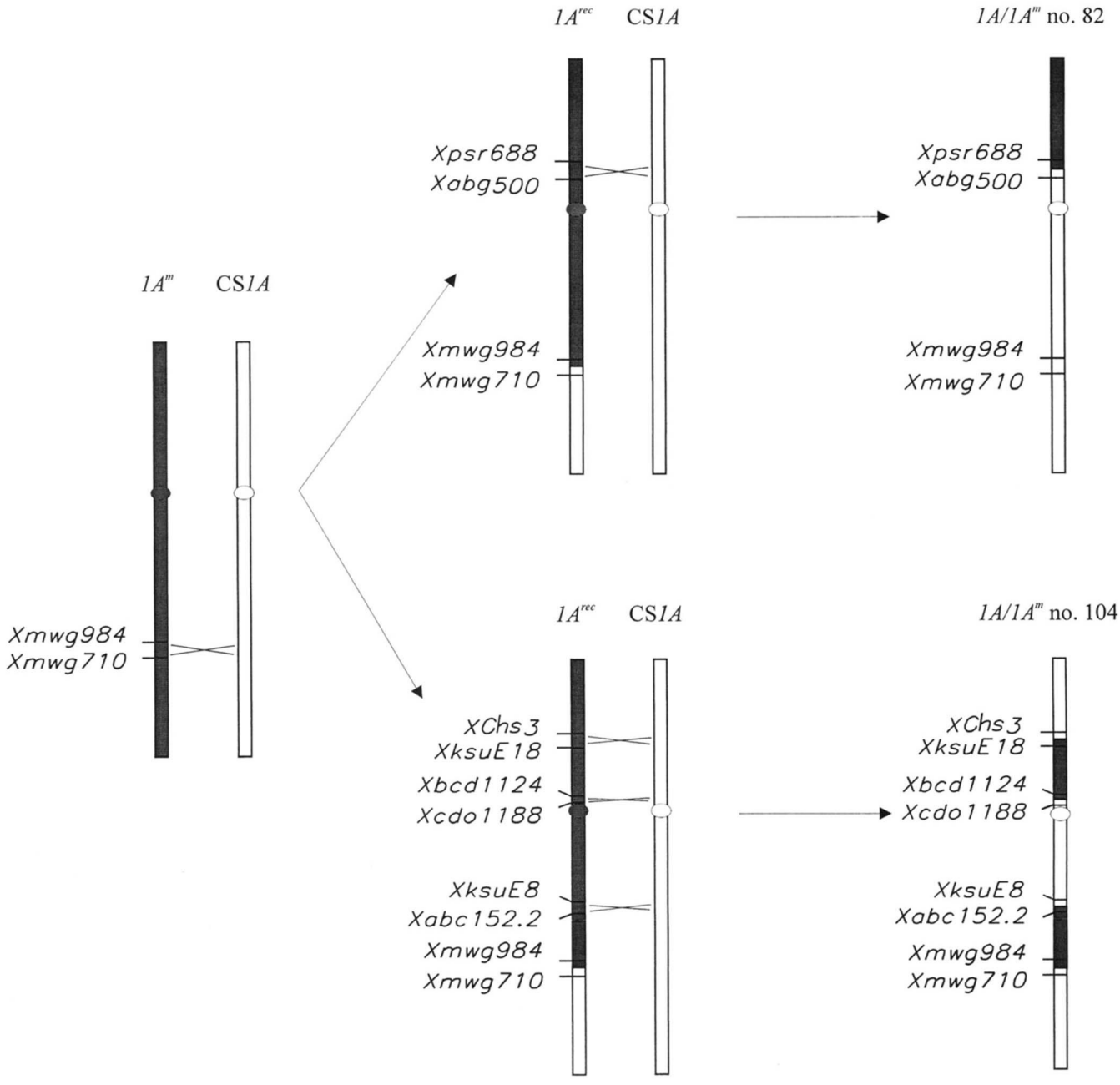

FIGURE 1.—The origin of the recombinant *1A/1A*^{m} chromosomes in RSL*1A*^{rec} and RSLs numbers 82 and 104 by recombination between genetic material of *T. monococcum* chromosome *1A*^{m} (■) and that of Chinese Spring chromosome *1A* (□). Markers flanking the crossover points are shown and the centromeres are indicated by ○.

On the *ph1* RSL*1A*^{rec} × CS map, the short arm interval *XksuE18-Xbcd1124* is 21.9 cM long and the long arm interval *XksuE8-Xmwg984* is 28.1 cM long. In the presence of *Ph1,* no recombination was observed in these intervals in either the RSL104 × CS population or the RSL104 × DSCnn*1A* population (Figure 3).

The interstitial segment in the short arm is flanked by two *1A* segments. The *1A*^{m} segment terminates proximally to *Xbcd1124.* The proximal *1A* segment was <2.5 cM, the distance of *X-bcd1124* to the centromeric marker *Xcdo1188* on the *ph1* RSL*1A*^{rec} × CS map (Figure 3). Recombination could not be investigated in this interval due to lack of polymorphism between Cnn*1A* and CS*1A.* In the distal *1A* segment, interval *Xmwg60-XGli1* was 21.4 cM on the DSCnn*1A* × CS map (DUBCOVSKY *et al.* 1995). On the RSL104 × DSCnn*1A* map, the interval was 38.0 cM long, which is significantly greater ($P < 0.05$) than 21.4 cM (Figure 3). The internal interval *XGli1-XGlu3* was also significantly longer (Figure 3). The most proximal interval of the segment, *Xmwg60-XksuE18,* which is 11.6 cM on the *1A*^{rec} × CS map, was shorter on the RSL104 × DSCnn*1A* map because it is composed of homologous and homeologous segments (Figures 1 and 3).

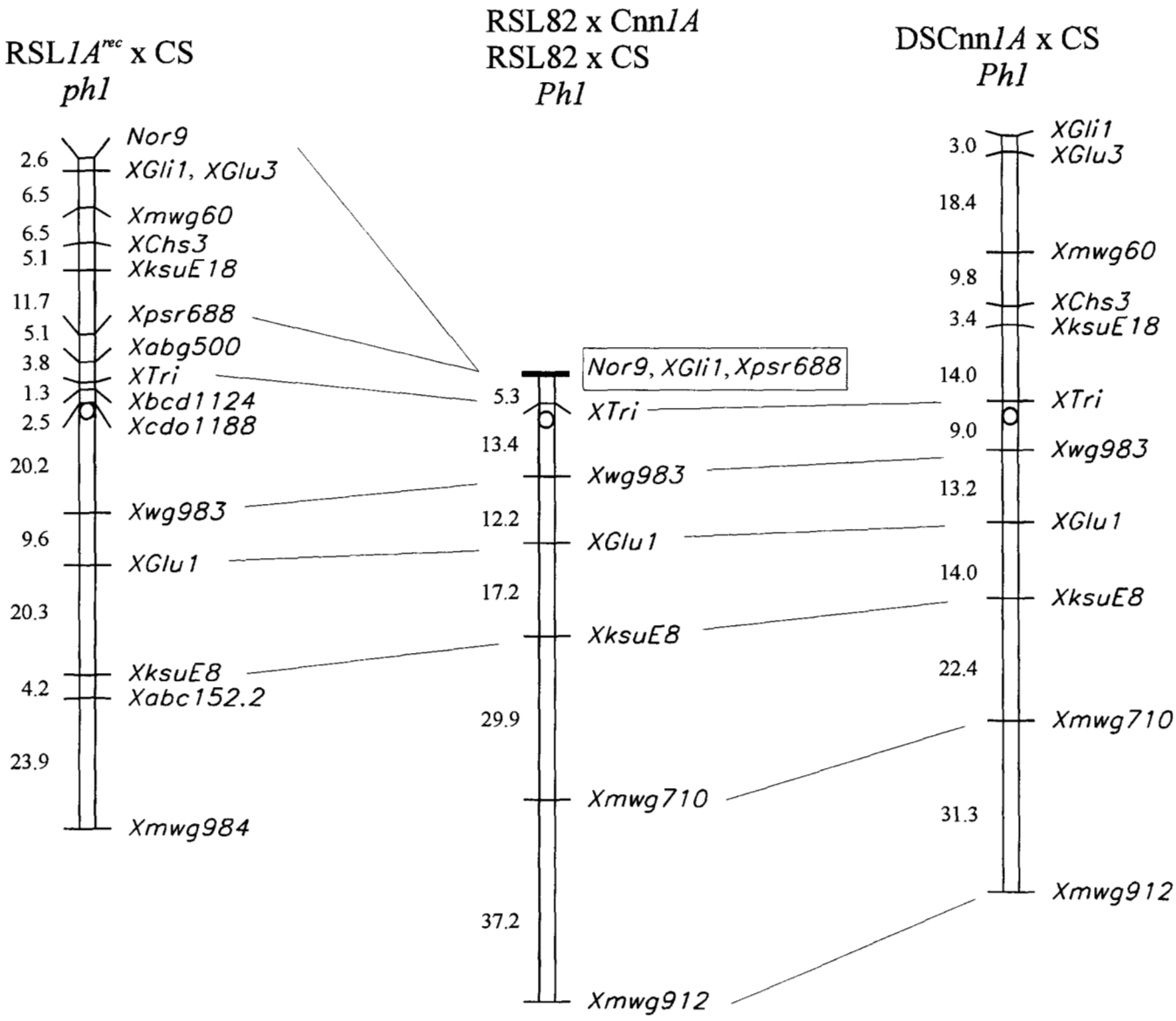

FIGURE 2.—Recombination between RFLP markers in a chromosome pair composed of *1A/1A*^{m} recombinant chromosome of RSL82 and complete Cnn*1A* and CS*1A* chromosomes in the presence of the *Ph1* locus (RSL82 × Cnn*1A* and RSL82 × CS). In the RSL82 × CS population, recombination could be investigated only in the homeologous segment, since only the homeologous segment was polymorphic. Markers within the homeologous segment are boxed. Maps based on recombination between genetic material of chromosome *1A*^{m} and that of CS*1A* in RSL*1A*^{rec} × CS in the absence of *Ph1* (left) and between CS*1A* and Cnn*1A* chromosomes in the presence of *Ph1* (right), reported by DUBCOVSKY *et al.* (1995), are shown for comparison. The positions of the centromeres are indicated by ○.

The interstitial homeologous segment in the long arm is flanked by proximal and distal homologous segments in RSL104 × DSCnn*1A*. The lengths of intervals in the proximal segment from *Xabc152.2* to *Xbcd1124* did not significantly differ from those on the *ph*1 RSL*1A*^{rec} × CS map (Figure 3). An exception was interval *XksuE8-Xabc152.2* that showed zero length on the RSL104 × DSCnn*1A* map but was 4.2 cM long on the RSL*1A*^{rec} × CS map. Presumably, genetic material in this interval was mostly homeologous. In the distal homologous segment, interval *Xmwg710-Xmwg912* was 33.0 cM long on the RSL104 × DSCnn*1A* map and 31.3 cM on the DSCnn*1A* × CS map (Figure 3).

MI pairing of the *1A/1A*^{m} chromosome of RSL104 with telosomes *1AS* and *1AL*: CS telosome *1AS* was paired with the *1A/1A*^{m} chromosome in 80.0% of the MI PMCs (Table 2). Telosome *1AL* was paired with the long arm of the *1A/1A*^{m} chromosome in 98% of the MI PMCs (Table 2).

In the RSL104 × CS population, there were two homologous segments in the short arm. The proximal homologous segment was <2.5 cM long, as pointed out above. The length of the terminal segment, measured from *XGli1* to *XksuE18*, was 43.8 cM in terms of recombination. Recombination in the terminal interval *XGli1-Nor9* could not be determined. This interval is 2.6 cM

TABLE 2

Frequencies of MI pollen mother cells in which Chinese Spring telosomes *1AS* and *1AL* were seen to be chiasmatically paired with *1A/1A*^{m} chromosomes of RSLs 82 and 104

Cross	PMCs scored	Percentage of PMCs with a telosome paired with a *1A/1A*^{m} chromosome	
		1AS	*1AL*
DDt*1A* × RSL82	184	9.8	98.0
DDt*1A* × RSL104	130	80.0	98.0

PMC, pollen mother cell.

on the RSL*1A*^{rec} × CS map (DUBCOVSKY *et al.* 1995). The sum of the lengths of the pairing regions is, therefore, ~49 cM, which is similar to the 55 cM calculated from 80% of MI PMCs in which telosome *1AS* paired with *1A/1A*^{m} (0.8 × 0.5 and converted to cM with the KOSAMBI 1943 mapping function). The two estimates closely agree, which suggests that chiasmata, like the crossovers, were only in the homologous segments in the short arm. The essentially complete MI pairing of the *1AL* telosome makes similar calculations meaningless for the long arm.

DISCUSSION

Recombination in the homeologous segments: No recombination was detected between wheat chromosome *1A* and each of the *T. monococcum* segments of the *1A/1A*^{m} chromosomes in RSLs 82 and 104 in the presence of *Ph1*. This was true irrespective of whether a homeologous segment was terminal or interstitial. Since extensive recombination was observed in the same intervals in the absence of *Ph1* (DUBCOVSKY *et al.* 1995), the absence of recombination in the presence of *Ph1* must be caused by the activity of *Ph1*. A possibility that

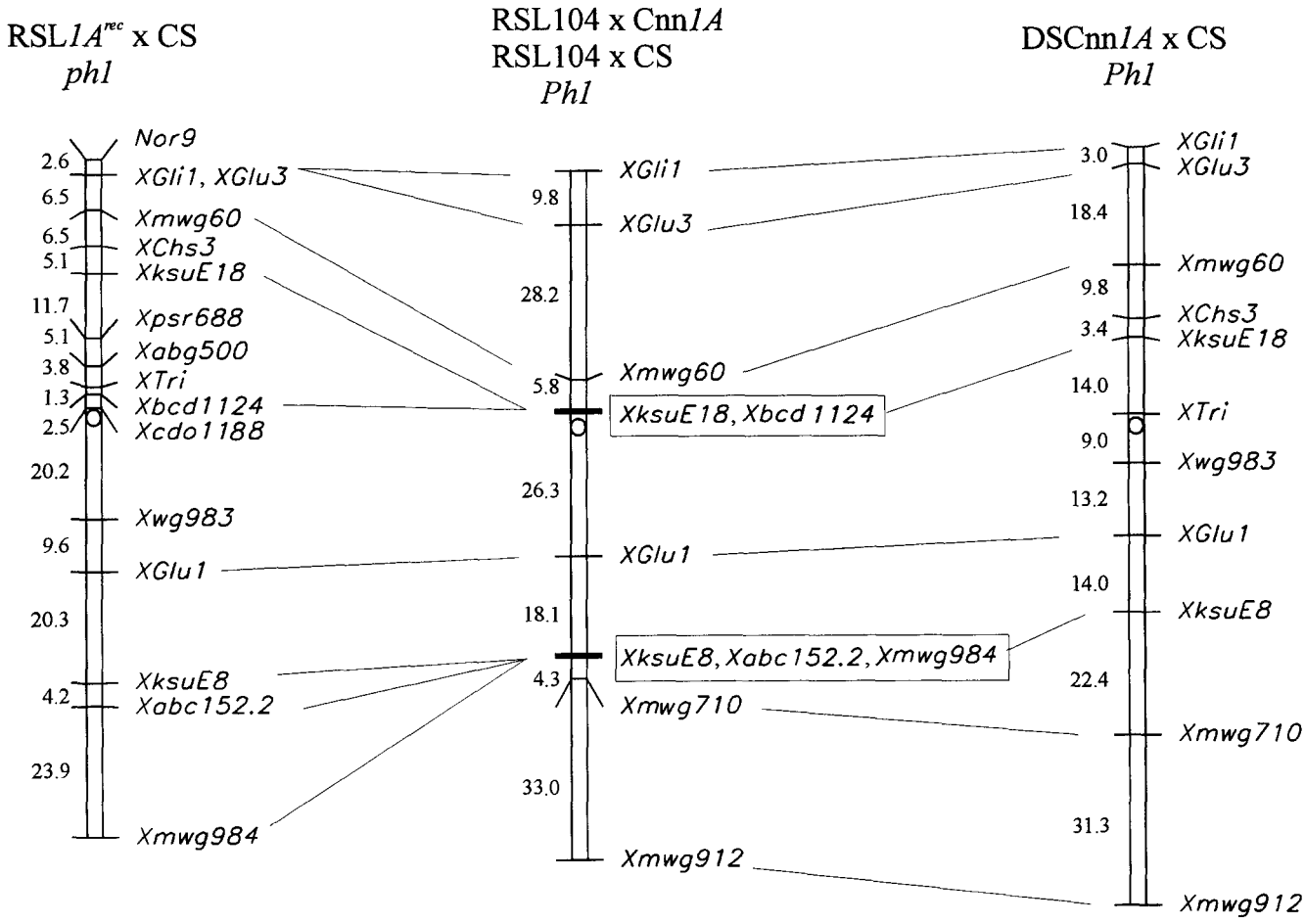

FIGURE 3.—Recombination between RFLP markers in a chromosome pair composed of *1A/1A*^{m} recombinant chromosome of RSL104 and complete Cnn*1A* and CS*1A* chromosomes in the presence of the *Ph1* locus (RSL104 × Cnn*1A* and RSL104 × CS). In the RSL104 × CS population, recombination could be investigated only in the homeologous segment, since only homeologous segment was polymorphic. Markers within the homeologous segments are boxed. Maps based on recombination between genetic material of chromosome *1A*^{m} and that of CS*1A* in RSL*1A*^{rec} × CS in the absence of *Ph1* (left) and between CS*1A* and Cnn*1A* chromosomes in the presence of *Ph1* (right), reported by DUBCOVSKY *et al.* (1995), are shown for comparison. The positions of the centromeres are indicated by ○.

some other factor was the causal agent is extremely unlikely in view of the fact that (1) the *Ph1* and *ph1* populations in which recombination was compared were isogenic except for *1A*, and (2) the same result was obtained with three different homeologous segments and two different wheat *1A* chromosomes.

Recombination of two other *1A/1A*^{m} chromosomes, harbored in RSL*1A*^{rec} and RSL21, with CS*1A* and Cnn*1A* in the *Ph1* genetic background was investigated previously (DUBCOVSKY *et al.* 1995). The *1A/1A*^{m} chromosome of RSL*1A*^{rec} was a *T. monococcum* chromosome with a terminal segment of *1AL*. Crossovers regularly occurred in the terminal homologous segment but were virtually absent from the proximal, homeologous portion of the long arm and the entire short arm. The *1A/1A*^{m} chromosome in RSL21 was largely CS*1A* that had a terminal segment of *1A*^{m}*S* <2.6 cM long in the absence of *Ph1*. In the presence of *Ph1*, no recombination was detected between this *1A*^{m}*S* segment and *1A* (DUBCOVSKY *et al.* 1995).

Thus, all data so far obtained show complete absence of recombination in the homeologous segments of chromosome pairs composed of homologous and homeologous segments in the presence of *Ph1*. Recombination is absent from the homeologous region(s) irrespective of whether the telomere is homologous and the centromere homeologous (RSL*1A*^{rec} × CS population), or the centromere homologous and the telomere homeologous (RSL21 × DSCnn*1A*, RSL82 × DSCnn*1A*, RSL82 × CS), or both the telomere and centromere homologous (RSL104 × DSCnn*1A*, RSL104 × CS). The absence of recombination from a homeologous segment is also independent of its length; the lengths of homeologous segments ranged from 2.6 to 111.7 cM.

Recombination in homologous segments: Compared to recombination between completely homologous chromosomes, recombination frequency in the homologous segment proximal to the homeologous segment was slightly reduced in the short arm in the RSL82 × DSCnn*1A* population. However, considering the fact that the proximal end of the homeologous segment, which shows no recombination, was included in the compared interval, there is no real difference between the two recombination values. Nor was recombination reduced in the homologous segment proximal to the interstitial homeologous segment in the long arm in the RSL104 × Cnn*1A* population. However, in the previous investigation of recombination in a chromosome pair composed of a proximal homologous segment and a distal homeologous segment in the short arm (population RSL21 × DSCnn*1A*, DUBCOVSKY *et al.* 1995), recombination was significantly reduced in the homologous segment compared to that between complete CS*1A* and Cnn*1A* chromosomes (DUBCOVSKY *et al.* 1995).

Recombination frequencies in homologous segments distal to homeologous segments showed no reductions in recombination; in the short arm, such a segment showed a significant increase in recombination compared to completely homologous chromosomes, suggesting some intra-arm compensation for the absence of crossovers in the proximal homeologous segment. However, no increase in recombination was observed in the terminal homologous segment in the long arm. A factor that may be responsible for the inconsistency is the length of the homeologous segment relative to the length of the homologous segment in the context of the total genetic length of an arm. The proximal homeologous segment from which crossovers were excluded accounted for about a half of the length of the linkage group of the short arm but it accounted for only a small fraction of the length of the linkage group of the long arm. High correlation between the lengths of homologous segments and the MI chiasmatic pairing frequencies of chromosomes composed of homologous and homeologous segments with a complete homologue in the presence of *Ph1* also shows that the absence of crossovers in the proximal homeologous segments is not compensated or only partially comensated by recombination increase in the distal homologous segments (DVOŘÁK *et al.* 1995). If such compensation were complete, chromosome pairs composed of homologous and homeologous segments would pair in 100% of PMCs, irrespective of the length of the homologous segment.

In conclusion, there appears to be no universal effect of a homeologous segment on recombination in a juxtaposed homologous segment in the presence of *Ph1*. The effect may depend on factors such as (1) the lengths of the homeologous and homologous segments relative to each other, (2) the total genetic length of the arm, and (3) the positions of the segments relative to each other in the arm. If a homologous segment is proximal, the presence of a distal homeologous segment may have no effect, or may reduce recombination in the homologous segment. If a homologous segment is distal, there may again be either no effect on recombination in the homologous segment or the homologous segment may show elevated recombination. This variation is likely related to the distal position of the first chiasma, and the interference of the first chisama with the position of the the second, proximal chiasma, as pointed out by LUKASZEWSKI (1995).

The action of *Ph1*: The findings reported here and previously (DUBCOVSKY *et al.* 1995) show that the activity of *Ph1* essentially eliminates recombination from the homeologous segments in chromosome pairs composed of homologous and homeologous segments in the same way as it does from completely homeologous chromosome pairs. If premeiotic alignment of homologues and exclusion of complete homoeologues from such alignment were the strategy by which *Ph1* prevented heterogenetic chromosome pairing, interstitial homeologous segments would be expected to behave as homologous, and recombine with a similar frequency

as they do in the absence of *Ph1*. Alternatively, homeologous segments could cause the rest of a chromosome pair to behave as homeologous and recombine poorly or not at all, so that such chromosomes would act entirely as homeologues. Neither alternative was encountered. Recombination was absent from the homeologous segments but normal or close to normal levels of recombination occurred in the juxtaposed homologous segments (see above). These facts strongly argue against a possibility that *Ph1* regulates homeologous meiotic pairing by premeiotic alignment of chromosomes as suggested by FELDMAN *et al.* (1966, 1973), FELDMAN (1968, 1993), and DOVER and RILEY (1977). No recombination occurred in the interstitial homeologous segments when the centromeres and both telomeres were homologous. It is, therefore, very unlikely that an interaction of mitotic spindle with the centromere is the target of the *Ph1* action, as assumed by the premeiotic association hypotheses (see Introduction). Investigation of synapsis has failed to reveal an alignment of chromosomes at the onset of meiotic prophase, which also argues against a possibility that a meiotic pairing pattern is predetermined by somatic or premeiotic alignment of chromosomes in wheat (HOBOLTH 1981; HOLM 1986; HOLM and WANG 1988; GILLIES 1987). The fact that crossing over abruptly ceased at boarders between homology and homoeology shows that homology is scrutinized along the entire length of a chromosome. This is consistent with the assumption that *Ph1* regulates homology recognition at the level of individual DNA heteroduplexes (HOLM and WANG 1988), although the exact mechanism is unknown.

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