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The effect of a deficiency and a deletion on recombination in chromosome 1BL in wheat

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Abstract To test two models of chiasma allocation and the distribution of crossing-over in chromosomes, genetic mapping was performed in normal, deletion and deficiency chromosome arms 1BL of wheat, *Triticum aestivum* L. Shortening of the chromosome arm, either by a deletion of the proximal half of the arm or by a deficiency of the terminal quarter of the arm's length, significantly reduced the frequency of multiple crossovers but did not affect the distribution of the distal, presumably the first, crossover in the arm. In the deficiency chromosome, the recombination rate in the terminal segment was much higher than that in the same segment of the complete arm. This suggests that recombination frequency is not an inherent characteristic of a segment but depends on the segment's position on the centromere-telomere axis. These observations support the classical model of chiasma distribution along the chromosome based on the point of pairing initiation, chromosome length and the positive chiasma interference. The study also demonstrates that the distribution and frequency of recombination in a chromosome segment can be manipulated. Therefore, even the segments with very low recombination frequencies could be saturated with large numbers of crossover events to produce high-density genetic maps.

Keywords Recombination · Crossing over · Distribution · Deletion · Deficiency · Wheat

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Introduction

Chiasmata are the cytological expressions of crossing-over. Apart from their genetic function, in all organisms with chiasmate meiosis they are also responsible for the proper disjunction of chromosomes in anaphase-I. Because of their importance, the distribution and frequencies of chiasmata are under strict genetic control (John 1990). The control mechanisms responsible for the pattern of chiasma distribution and, therefore the pattern of crossing-over, have been of great interest for decades. Mather (1937) proposed that the pattern of chiasma distribution was a function of the length of the chromosome arm and positive chiasma interference. Positive chiasma interference is a mechanism that prevents, or reduces the probability of, additional chiasmata in the vicinity of an already established chiasma. This limits the total number of chiasmata per chromosome arm and increases the probability that each chromosome pair forms at least one chiasma. In Mather's model, each chromosome arm will have at least one chiasma regardless of its length, but longer chromosomes have a greater probability of forming multiple chiasmata.

In wheat, *Triticum* sp., chiasmata are localized and distal. In breadwheat (*Triticum aestivum* L.), genetic mapping of the physical attributes of chromosomes (Dvorak and Chen 1984; Lukaszewski and Curtis 1993) and deletion mapping of genetic markers (Gill et al. 1996a, b; Werner et al. 1992; Mickelson-Young et al. 1995) has shown that crossing-over was indeed concentrated in the terminal segments of the chromosome arms and was practically absent from the proximal halves of the arms. Such an uneven distribution of recombination creates large differences between the physical and genetic distances. Based on two extreme cases, it was estimated that this difference might be as large as 153-fold (Lukaszewski and Curtis 1993).

It is now becoming evident that recombination in plants is concentrated in, if not outright limited to, unique DNA sequences (Schnalbe et al. 1998). Physical mapping of a considerable number of polymorphic

cDNA markers showed their clustering in the vicinity of the telomeres and in isolated intercalary islands. Such gene-rich islands may cover only small fractions of the genome (Künzel et al. 2000). Based on these observations, Gill et al. (1996a, b) proposed that the pattern of crossing-over in wheat was determined by the distribution of such gene-rich islands. This implies that the pattern of crossing-over is an inherent characteristic of a chromosome arm and that it cannot be modified experimentally unless the gene content is changed.

We tested these two models of the distribution of crossing-over by examining the frequency and distribution of crossovers in two structural variants of the long arm of chromosome 1B of wheat. We assumed that if Mather's model was correct, the removal of a portion of the arm should not affect the general pattern of crossing-over, with recombination still concentrated in the terminal regions of the modified arms. However, if the hypothesis of Gill et al. (1996a, b) was correct, the removal of the recombining, distal, part of an arm should reduce the ability of that arm to form chiasmata, especially in the distal region; removal of the proximal part of the arm should have no effect on the parameters of recombination.

Materials and methods

Chromosome constructs and mapping populations

Genetic mapping was performed in a normal 1BL arm of hexaploid wheat cv Chinese Spring (abbreviated $1B_{cs}$) and its two midgets: deletion chromosome Del1B45L, where the proximal 45% of the arm's length was removed, and deficiency chromosome Df1B23L missing the terminal 23% of the normal arm (Fig. 1). The midget chromosomes were produced from chromosome 1B of cv Chinese Spring by a combination of breakage-fusion-bridge cycles and centric misdivision (Lukaszewski 1997a). The source of polymorphism for mapping were substitutions of chromosomes 1B of cv Timstein ($1B_{tim}$) and *Triticum dicoccoides* ($1B_{dic}$) into Chinese Spring, obtained from the late Dr. E.R. Sears.

For mapping in the complete long arm of 1B, the ditelocentric 1BL line of CS (Dt $1BL_{cs}$) was crossed with the two single-chromosome substitution lines, CS $1B_{tim}$ and CS $1B_{dic}$, and the resulting F_1's were backcrossed as females to the CS Dt1BS line. The same protocol was followed for mapping in the deletion telocentric chromosome Del1B45L. To generate mapping populations for the Df1B23L chromosome, the deficiency breakpoint had to be transferred, by crossing-over, to chromosomes $1B_{tim}$ and $1B_{dic}$. The plants with $Df1B23L_{dic}$ were crossed to plants with the telocentric deficient chromosome $Df1BL23_{cs}$ and all steps from this point on were identical to those for the complete arms and the deletion chromosomes.

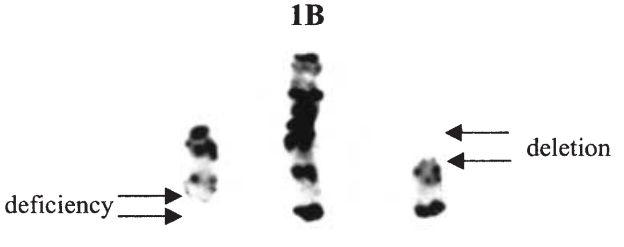

Fig. 1 Normal C-banded chromosome 1B of Chinese Spring (middle) and its midgets, Df1BL23 (left) and Del1BL45 (right), used in genetic mapping

The backcross kernels were individually screened by SDS polyacrylamide-gel electrophoresis (SDS-PAGE), and germinated and screened by C-banding to identify the 1B chromosomes present. Resulting plants were grown and samples of their leaf tissue collected, DNA extracted by the CTAB method and mapped using microsatellite markers. In each mapping population about 150 kernels were screened by SDS-PAGE and germinated. Because of the inevitable losses due to the poorer germination of cut kernels, and other causes, final mapping backcross populations consisted of 117 to 130 individuals each.

For the analysis of meiotic chromosome pairing, anthers with a majority of the pollen mother cells (PMCs) in metaphase-I (MI) were fixed in a mixture of 3 parts ethanol and 1 part glacial acetic acid and refrigerated for several months. Squash preparations were made and C-banded according to Giraldez et al. (1979). All cells available for analysis were scored.

Genetic mapping

The genetic linkage maps of the chromosome constructs were developed using eight markers: the centromere, the *Glu-B1* locus, and six microsatellite markers: *Xgwm124*, *Xgwm131*, *Xgwm140*, *Xgwm259*, *Xgwm268* and *Xpr3100*. All *Xgwm* markers were kindly provided by Dr. M.S. Röder of the Institute fur Pflanzengenetik und Kulturpflanzenforschung, Gatersleben, Germany (Röder et al. (1998); the *Xpr3100* marker was purchased from the John Innes Centre, Norwich, UK. The PCR-amplified DNA fragments generated using the provided primers were separated on denaturing acrylamide gels (7% acrylamide, 6 M Urea and 1 × TBE) run in 1 × TBE at 1,250 V. The PCR conditions were similar to those provided by the sources of the primers.

The centromere marker (presence of a bi-armed vs a telocentric chromosome) was determined cytologically by C-banding. The identity of the *Glu-B1* locus was determined by SDS-PAGE using standard protocols. Proportions of various classes of chromosomes in different populations were compared using the χ^2 test.

Results

Structural polymorphism in the terminal regions of chromosome arms of wheat adversely affects their ability to pair and form chiasmata (Curtis et al. 1991; Lukaszewski 1997b). Chromosome arms 1BL and Del1B45L were structurally identical in the terminal regions and their MI-pairing was normal. Large structural difference in the proximal regions had no effect on MI-pairing. To generate deficiency stocks with identical structure in the terminal regions and with adequate polymorphism for genetic mapping, the Df23 breakpoint from chromosome $Df1B23L_{cs}$ had to be transferred to chromosomes $1B_{tim}$ and $1B_{dic}$ by crossing-over. Owing to the complete reliance of wheat chromosomes on the terminal initiation of pairing (Lukaszewski 1997b), a chromosome deficient for the terminal 23% of its length could not be expected to pair regularly with a complete arm and an attempt of direct transfer failed. Therefore, the transfer was accomplished in two steps. In the first step, the Df1B12L breakpoint was transferred from chromosome 1B of CS deficient for the terminal 12% of the long arm to $1B_{tim}$ and $1B_{dic}$. In the second stage, among 78 progeny screened involving $Df1B12L_{dic}$ and $Df1B23L_{cs}$, two re-

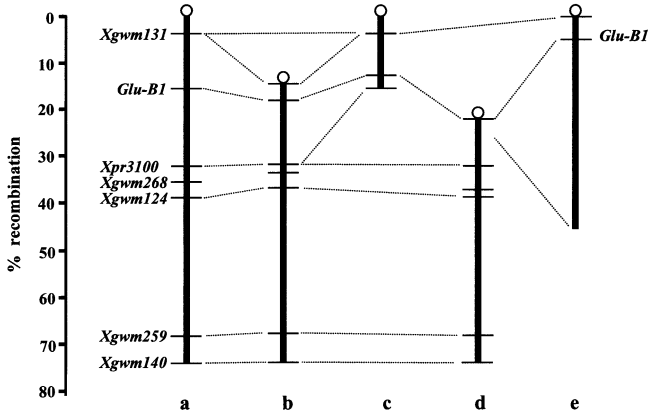

Fig. 2a–e Uncorrected genetic maps of chromosome $1B_{dic}$ and its midgets. **a** Genetic map of the entire long arm, **b** genetic map of the long arm generated by the distal, presumably first, crossover, **c** genetic map of 1BL generated by the additional crossovers, **d** genetic map of and $Del1BL45_{dic}$, **e** genetic map of $Df1BL23_{dic}$. Apart from a small reduction in the *Glu-B1–Xpr3100* interval, the map of $Del1BL45_{dic}$ is identical to the map of the normal 1BL generated by the first crossover. The length of the Df1BL23 map was estimated based on the MI pairing frequency. *Dotted lines* connect the same markers on different maps

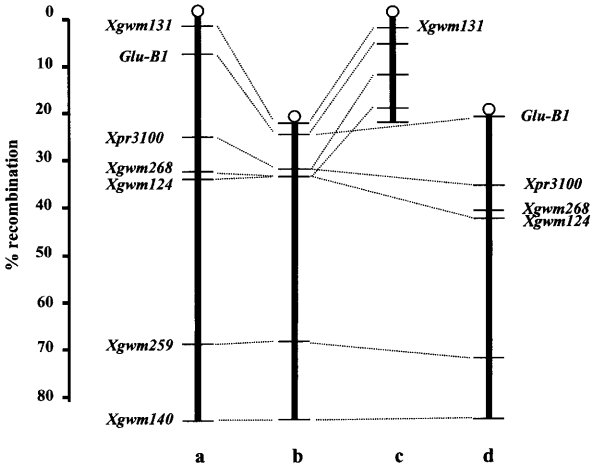

Fig. 3a–d Uncorrected genetic maps of chromosome $1B_{tim}$ and the deletion chromosome $Del1BL45_{dic}$. **a** Genetic map of the entire long arm, **b** genetic map of the long arm generated by the distal, presumably first, crossover, **c** genetic map of 1BL generated by the additional crossovers, **d** genetic map of and $Del1BL45_{dic}$. The map of $Del1BL45_{dic}$ is practically identical to the map of the complete 1BL generated by the first crossover in the arm. *Dotted lines* connect the same markers on different maps

combinant chromosomes $Df1B23L_{dic}$ were isolated. No recombinants were found among a total of 167 progeny from the cross involving chromosome $Df1B12L_{tim}$ screened in two rounds of selection, and this part of the experiment was abandoned.

Among 190 pollen mother cells in the MI of meiosis in the $1BL_{cs}$ + $1B_{dic}$ combination the two arms were paired in 173 cells (91.1%). Among 41 (1B + 1BL) bivalents suitable for detailed analysis, 33 had clearly terminal chiasmata and eight had intercalary chiasmata, presumably accompanied by additional, terminal chiasmata. In this combination, 124 progeny chromosomes were successfully analyzed, of which 49 were parental, 61 showed single exchanges and 14 showed double exchanges. A total of 89 exchanges were detected and the sum of the recombination frequencies detected in the individual inter-marker segments was 71.8%.

In the $1BL_{cs}$ + $1B_{tim}$ combination, 224 MI PMCs were analyzed and the two arms were paired in 213 cells (95.1%). No observations on the distribution of chiasmata were made. Among 130 progeny chromosomes analyzed, 46 were parental (non-recombined), 55 showed single exchanges, 27 double exchanges and two triple exchanges for the total number of 115 detected crossover events. The sum of all detected recombination events was 87.7%. While chromosome $1BL_{tim}$ paired with a higher frequency, and was genetically longer than $1BL_{dic}$, these differences were not statistically significant.

In the combination $Del1BL45_{cs}$ + $1B_{dic}$, MI pairing of the two arms in question was 89.3% in 103 PMCs scored and 90.7% in 224 scored cells of the $Del1BL45_{cs}$ + $1B_{tim}$ combination. All chiasmata appeared to be terminal. Deletion of the proximal 45% of the arm's length removed marker *Xgwm131*. The sums of all detected recombination frequencies for the remaining six inter-marker intervals were 53.7% and 68.5% for $Del1B45L_{dic}$ and $Del1B45L_{tim}$, respectively (Figs. 2 and 3). The reduction of the genetic lengths of the deletion chromosomes appears to have been caused primarily by significant reductions in the frequencies of multiple crossovers relative to normal, complete, chromosomes. There were 23 chromosomes with multiple crossovers in the combined Del1B45L populations and 43 in the combined complete 1B populations ($\chi^2 = 8.08$ $P < 0.01$). Apart from the reduction of the frequency of multiple crossovers, the recombination frequencies of the deletion chromosomes were similar to the recombination frequencies of the corresponding regions of the complete chromosomes (Figs. 2 and 3).

In the $Df1BL23_{cs}$ + $Df1B23L_{dic}$, MI pairing was 88.4% among 173 cells scored and all chiasmata observed were terminal. Deficiency for the terminal 23% of the arm removed five distal markers making direct monitoring of recombination in most of the arm impossible. Among 116 progeny scored, only eight single-crossover events were detected, all in the *Xgwm131–Glu-B1* interval. The remaining 108 progeny were parental. As there were no markers in the vicinity of the telomere, the total recombination frequency of the arm can only be estimated on basis of its MI pairing frequency.

Discussion

The overall pairing and recombination frequencies of the complete 1BL chromosome arm observed in this study

were well within the expected values for wheat (Sallee and Kimber 1978). Pairing was in the 90 to 95% range and the total genetic lengths observed suggested an average of about 1.6 crossovers per arm. Reduction of the physical length of chromosome arm 1BL by deletion of the proximal 45% of the arm had no significant effect on its ability to pair with the normal 1BL but resulted in a significant reduction in the average number of crossovers per arm to 1.2 ($\chi^2 = 4.77$ $P < 0.05$). In may be noted that the original chromosome Del1B45L of Chinese Spring was produced by a combination of the breakage-fusion-bridge cycle with centric misdivision (Lukaszewski 1997a) and its centromere was derived entirely from the centromere of chromosome 5B. The identity and the structure of this centromere had no effect on the fidelity of pairing of the deletion chromosomes.

Recombination frequencies of the deletion chromosomes were considerably lower than those of the complete arms and the reduction was primarily by lower frequencies of multiple crossovers per arm. The distribution of the distal markers in the maps of the complete and deletion chromosomes was practically identical (Figs. 2 and 3). If the genetic maps of the complete chromosomes are separated into portions generated by the distal, presumably the first, chiasma/crossover in the arm and the additional, proximal crossovers (Figs. 2 and 3), it becomes evident that the reduction in the overall recombination frequencies of the deletion chromosomes was primarily by the lower frequency of multiple crossovers. The distribution of the distal, presumably first, crossovers in the arm was not affected by the removal of the proximal one-half of the arm's length. This agrees with the chiasma allocation model of Mather (1937) and by the general relationship between chromosome length and chiasma frequency (Rees and Narayan 1988)

Removal of the distal 23% of 1BL eliminated five of the eight markers from the arm and made monitoring of recombination difficult. The total recombination frequency of this chromosome can only be inferred from its MI pairing. Based on the observed 88.4% MI pairing frequency, the minimum genetic length of this arm was 44.2 cM assuming a single chiasma/crossover per arm. If an arbitrary assumption is made that each of the eight detected crossover events in the *Xgwm131–Glu-B1* interval were accompanied by an exchange in the *Glu-B1*–telomere interval, the total recombination frequency of Df1B23L would be 51.1%. Taking into account the physical distance of the positive chiasma interference in the B-genome chromosomes of wheat (Curtis and Lukaszewski 1992), multiple crossovers in such proximity do not appear likely.

Regardless of the assumptions, it is evident that most recombination in the Df1B23L chromosome was in the *Glu-B1*–telomere interval. This interval shows a considerable increase in the coefficient of exchange (Lindsley and Sandler 1977), here understood as an average crossover frequency per chromosome relative length unit, as compared to its coefficient of exchange in a structurally normal chromosome. This coefficient of exchange can be calculated by estimating the most-likely relative physical positions of various markers and breakpoints in normal and Df1B23L chromosomes. The *Xpr3100* marker was completely linked with the telomere of the Df1BL17L chromosome among 23 recombined $Df1B17L_{dic}$ chromosomes and 17 recombined $Df1B17_{tim}$ chromosomes. These chromosomes were missing the distal 17% of the long arm. Therefore, the maximum physical distance between the Df1B23L breakpoint and *Xpr3100* is about 6% of the arm. The *Glu-B1* locus is present on the 1BL-8 and 1BL-9 chromosomes produced by Endo (1988), which are deficient for about 26% of the arm but is absent from the Df1B31L chromosome (Lukaszewski, unpublished), and from Endo's 1BL-2, both of which are deficient for the terminal 31% of the arm. Two translocations of 1DL to 1BL that remove *Glu-B1* cover the area up to 27% of the relative arm's length from the telomere (Lukaszewski, unpublished). The corresponding loci *Sec-2* in 1RL and *Glu-D1* in 1DL when translocated to 1RL were located physically at about 23–27% of the relative length from the telomere (Lukaszewski et al. 2000). Consequently, the *Glu-B1* locus must be located at about 26–27% of the arms' length from the telomere, that is within the terminal 3–4% of the Df1B23L chromosome, and the physical distance separating *Xpr3100* and *Glu-B1* must be about 9–10% of the arm. In the complete 1BL, this *Xpr3100–Glu-B1* segment was responsible for 20.2% recombination (Figs. 2, 3), or about 2% recombination per unit of relative length. One-third of this segment that is present in Df1B23L, or the terminal 3% of Df1B23L distal to *Glu-B1*, was responsible for a minimum 37.2% recombination or 10–12% recombination per relative length unit. This is about a five-fold higher recombination rate relative to that in a normal chromosome. This recombination rate may also be higher than that in similarly located terminal segments of a normal 1BL. The *Xpr3100*–telomere segment covers 17% of the arm's length and was responsible for 39.5% recombination frequency. Taking into account that the terminal 10% of 1BL is occupied by a C-band (Gill et al. 1991) that is believed not to participate in recombination, the 7% of the arm's length between *Xpr3100* and the telomeric C-band produced 39.5 cM of uncorrected map length. This gives a rate of 5–6% recombination per relative unit length; about one-half of that for the terminal 3% segment of Df1B23L. However, if there is a clear centromere–telomere gradient of crossovers across an entire arm, it may also be present in segments such as the terminal recombining 7% of 1BL, and the recombination rate in the distal regions of these segments may in fact be greater than the whole-segment average.

The observations made in this experiment suggest that the recombination frequency of a chromosome segment depends primarily on its location on the telomere–centromere axis. If a segment is moved toward the telomere, as it was in the case here when terminal segments were removed, its recombination frequency increases dramatically. At the same time, because of the reduction of the total chromosome length, the recombination frequency of the proximal regions may be reduced, primarily because of a reduced probability of multiple crossovers but perhaps also because of a more-distal concentration of chiasmata

in the shorter arms. Similar phenomena were observed by Devos et al. (1995) who compared genetic map distances of homoeologous segments in group-4 chromosomes of wheat which, because of a structural rearrangement, were present in arms of different length and in different positions relative to the telomere. Coupled with the fact that midget chromosomes of wheat are fully capable of chiasmate pairing, even when they are missing the regions which in the normal arms are completely responsible for chiasma formation and recombination, these observations do not support the notion that the pattern of recombination in the chromosomes of wheat is an inherent characteristic of a given segment, perhaps dictated by the distribution of the DNA sequences capable of crossing-over. The ability to pair and recombine of chromosome constructs composed of chromatin originating from various regions of the original arms suggests that such sequences must be distributed along the entire chromosomes with sufficient density to allow for normal chiasmate pairing and recombination of any cytologically identifiable segment of euchromatin. What determines the presence/absence of crossovers is the position of a segment on the telomere–centromere axis. These observations support the model of chiasma distribution of Mather (1937) with correction for the telomeric/distal pairing initiation. Any chromosome arm or its fragment, if accompanied by a homologous partner with sufficient structural similarity in the terminal region, is capable of forming a distal chiasma. Depending on the length of such a chromosome, the positive chiasma interference distance, and perhaps the total amount of time available for chiasma formation, additional chiasmata may be formed in the proximal regions of the same arm.

This study demonstrates that the frequency of crossing-over in a given chromosome segment can be manipulated experimentally. In wheat, such manipulation would involve by the use of existing, and perhaps newly generated, stocks of terminal deletions (Endo 1988) that shorten the arms and can effectively move any desired chromosome segment to the vicinity of the telomere. Such physical location imposes regular and frequent recombination on the segment when a structurally similar pairing partner is available, even if the segment originates from the regions of a chromosome arm that normally do not recombine. While this approach may require a substantial initial time-investment in the creation of proper genetic stocks, especially if a step-wise strategy of transferring the breakpoint must be employed, the resulting high crossover frequency in the targeted region would permit the generation of high-density genetic maps in reasonably sized populations, thereby eliminating the current limitations on chromosome walking in wheat.

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