The Development and Meiotic Behavior of Asymmetrical Isochromosomes in Wheat

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ABSTRACT

To determine which segments of a chromosome arm are responsible for the initiation of chiasmate pairing in meiosis, a series of novel isochromosomes was developed in hexaploid wheat (*Triticum aestivum* L.). These isochromosomes are deficient for different terminal segments in the two arms. It is proposed to call them "asymmetrical." Meiotic metaphase I pairing of these asymmetrical isochromosomes was observed in plants with various doses of normal and deficient arms. The two arms of an asymmetrical isochromosome were bound by a chiasma in only two of the 1134 pollen mother cells analyzed. Pairing was between arms of identical length whenever such were available; otherwise, there was no pairing. However, two arms deficient for the same segment paired with a frequency similar to that of normal arms, indicating that the deficient arms retained normal capacity for pairing. Pairing of arms of different length was prevented not by the deficiency itself, but rather, by the heterozygosity for the deficiency. Whether two arms were connected via a centromere in an isochromosome or were present in two different chromosomes had no effect on pairing. This demonstrates that in the absence of homology in the distal regions of chromosome arms, even if relatively short, very long homologous segments may remain unrecognized in meiosis and will not be involved in chiasmate pairing.

THE term "iso-chromosome" was proposed by DARLINGTON (1939) to describe a metacentric chromosome consisting of two identical arms connected by a centromere. Such chromosomes are produced by misdivision of the centromeres. Because the two arms of an isochromosome are formed by sister chromatids of the original chromosome arm, they are identical in every respect. Isochromosomes have been used for a variety of purposes, including the allocation of genes to chromosome arms in secondary trisomics (see SYBENGA 1992), but they are perhaps most useful in studies on mechanisms of chromosome pairing and synapsis.

Premeiotic applications of colchicine in bread wheat (*Triticum aestivum* L.) lowered the frequency of pairing of normal homologous arms but did not affect pairing of the two arms of isochromosomes (DRISCOLL and DARVEY 1970). This suggested that the process of pairing consisted of two stages, the alignment of homologues and synapsis, with colchicine disrupting the former. Because pairing of the arms connected into an isochromosome was not affected by colchicine, it became clear that pairing success depended on the arrangement of chromosomes in the nucleus. However, the mechanisms controlling the arrangement of chromosomes leading to chiasmate pairing remain a mystery (for review, see MAGUIRE 1988; LOIDL 1990).

CALDECOTT and SMITH (1952) described a type of chromosome aberration in barley that resulted from an X-ray-induced reciprocal exchange of the distal segments of the opposing arms of a pair of homologues. Following the exchange, the distal (telomeric) regions of the two arms of each of these chromosomes were identical, the regions proximal to the centromere were different. Such chromosomes were called pseudo-isochromosomes. The formation of pseudo-isochromosomes after irradiation was observed in several other organisms (MORRIS 1955; KOO 1958; WATANABE 1973), but MORRIS (1955) pointed out that they can also be produced by crossing over in pericentric inversions. In all instances studied, pseudo-isochromosomes regularly formed rings in metaphase I of meiosis, demonstrating that the homology of the distal segments of chromosome arms was sufficient for successful pairing, notwithstanding lack of homology in the proximal regions of the arms.

Chromosomes with a structure opposite to that of pseudo-isochromosomes have never been described, and their meiotic behavior can only be a matter of speculation. Such chromosomes would be bibrachial with identical proximal regions flanking the centromere, but the distal (telomeric) regions of the two arms would be different. They could be produced in several different ways. This paper reports the development of isochromosomes composed of arms of unequal length by a combination of centric misdivision of a univalent and a chromatid-type breakage-fusion-bridge cycle (BFB). The resulting isochromosomes had arms deficient for different segments. It is proposed to call these novel chromosomes "asymmetrical isochromosomes."

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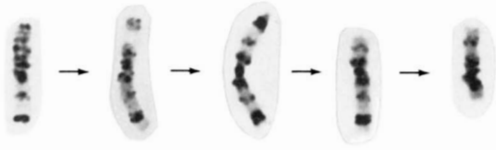

FIGURE 1.—The development of asymmetrical isochromosome 1–1 in bread wheat. From left to right: normal chromosome *1B*; chromosome *1B* with an rtd on L; isochromosome $1BL_{RTD}$; isochromosome 1; asymmetrical isochromosome 1-1.

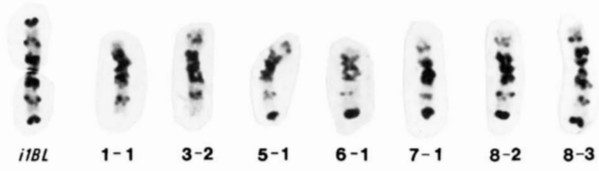

FIGURE 2.—Asymmetrical isochromosomes of bread wheat analyzed in this study. The first chromosome on the left was constructed for demonstration purposes from two normal long arms of chromosome *1B*.

MATERIALS AND METHODS

All experiments were performed using various cytogenetic stocks of hexaploid bread wheat, *T. aestivum* L. cv "Chinese Spring" (CS). These stocks, involving monosomics, ditelosomics, a disomic substitution of a rye (*Secale cereale* L.) chromosome *1R* for chromosome *1B*, and a chromosome *1B* of CS with a reverse tandem duplication (RTD) on the long arm were either taken from the collection of CS aneuploids developed by the late Dr. E. R. SEARS and maintained by the author or developed by the author. Meiotic behavior of the $1B_{RTD}$ was described previously (LUKASZEWSKI 1995).

Analyses of mitotic chromosomes were by C banding as described by LUKASZEWSKI and XU (1994). For the analyses of meiotic chromosome pairing, anthers with a majority of pollen mother cells (PMCs) in metaphase I were fixed in a mixture of three parts of absolute alcohol to one part of glacial acetic acid at room temperature for several hours, refrigerated for 3–6 mo, and C-banded according to GIRALDEZ *et al.*, (1978). Between 50 and 174 PMCs from each combination were analyzed.

All terms in this article are used in the classical cytogenetic sense. The term "deficiency" is used in the sense proposed by BRIDGES (1917), as a loss of an acentric terminal segment of a chromosome arm. Deletion is a loss of an interstitial segment. The nomenclature of wheat chromosomes with deficiencies is the same as used previously (CURTIS *et al.* 1991). Bibrachial chromosome *1B* with the RTD is referred to as $1B_{RTD}$; any chromosome constructs involving only the long arm of this chromosome are referred to as $1BL_{RTD}$.

RESULTS

Development of the asymmetrical isochromosomes: In a study unrelated to this one, a $1B_{RTD}$ line of CS was crossed to the *1R*(*1B*) substitution line of CS and double monosomics 20″ + $1B_{RTD}$′ + *1R*′ were selected, grown and allowed to self-pollinate. Among 155 of their progeny screened by C banding, five plants with one copy each of an isochromosome $1BL_{RTD}$ were isolated (Figure 1). Additionally, there were 28 other misdivision products of $1B_{RTD}$ (telocentrics, isochromosomes *1BS*, and various *1B-1R* fusion products), for a 21.3% misdivision frequency.

Plants with isochromosomes $1BL_{RTD}$ were grown and their progeny screened again. The RTDs in the arms of the isochromosomes initiated the chromatid-type BFB cycle (LUKASZEWSKI 1995). If the BFB cycles were initiated in both arms simultaneously, ring chromosomes were produced (which could have also resulted from crossing over involving a duplicated inverted segment of an RTD on one arm and a corresponding segment in the normal orientation on the other arm). Several of these chromosomes were observed. The BFB cycle initiated in only one arm of an isochromosome $1BL_{RTD}$ resulted in breakage of that arm, producing a deficiency, whereas the other arm retained the RTD (Figure 1). Eight such isochromosomes, numbered 1 through 8, were recovered. The deficient arms of these eight isochromosomes, having lost the RTD, became stable; the other arms retained the RTD and were still capable of breakage.

Plants with isochromosomes 1 through 8 were self-pollinated and crossed to normal CS. In the generation following selfing, isochromosomes were sought in which the RTD arm had undergone the BFB cycle and lost a segment. This resulted in asymmetrical isochromosomes consisting of two deficient arms of *1BL* (with each arm being deficient for a different distal segment, see Figure 2). In the generation resulting from crosses to normal CS, plants with one of the isochromosomes and a normal 1B were selected and grown. The long arm of the normal *1B* was expected to pair and recombine with the RTD arms of the isochromosomes. Such events would result in asymmetrical isochromosomes consisting of one deficient and one normal arm of *1BL*.

The asymmetrical isochromosomes, selected either after the second round of the RTD-initiated breakage or after recombination with normal *1BL*, were isolated and numbered 1-1, 1-2, 2-1, 2-2, 2-3, etc. All isochromosomes within each family share the same breakpoint in one arm; the breakpoints in the other arm are different. Because the intent of this study was to observe pairing of the asymmetrical isochromosomes at meiosis and the telomeric C-band on *1BL* greatly facilitates identification of this chromosome arm, preference was given to isochromosomes consisting of one normal *1BL* arm and one deficient *1BL* arm (Figure 2).

Meiotic pairing of the asymmetrical isochromosomes: Pairing pattern of seven asymmetrical isochromosomes *1BL* was observed (Figure 2), either as monoisosomic, diisosomics or in various combinations with other chromosomes like normal or deficient chromosome *1B*, for a total of 12 chromosome combinations. The numbers of the PMCs analyzed ranged from 50 to 174 per combination, for the total of 1134.

Meiotic pairing of the asymmetrical isochromosome 1-1 was observed in a plant with chromosome constitution 20″ + 1-1 + $1B_{RTD}$. Among 126 PMCs analyzed, the two deficient arms of this isochromosomes were paired with each other in two cells; in four cells, the longer

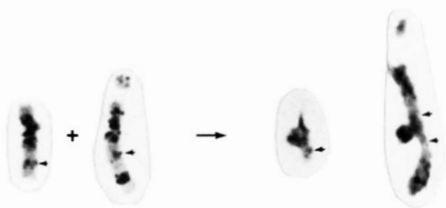

FIGURE 3.—Meiotic metaphase I pairing of asymmetrical isochromosome 1-1 with $1B_{RTD}$ in bread wheat. From left to right: mitotic chromosome 1-1; mitotic $1B_{RTD}$; metaphase I ring formed by 1-1; rod bivalent resulting from pairing of the longer arm of 1-1 with the long arm of $1B_{RTD}$. Arrowheads point to the identifying C-bands, normally in the middle of *1BL*.

arm of the isochromosome 1-1 was paired with the duplicated inverted segment of $1B_{RTD}$. (Figure 3). In the remaining 120 PMCs, the arms of isochromosome 1-1 remained not paired and it was present as a rod univalent.

Isochromosomes 3-2 and 6-1 were monosomic in plants with chromosome constitution 20″ + 3-2′ and 20″ + 6-1′. Among 50 and 85 PMCs analyzed, respectively, the two isochromosomes were always present as rod univalents, with arms never bound by a chiasma.

Isochromosomes 5-1 and 7-1 were combined with their precursors, chromosomes 5 and 7, respectively. Both isochromosomes 5-1 and 7-1 are deficient in one arm while the other arms are complete arms *1BL* (Figure 2). Consequently, in each combination studied there were four copies of at least a portion of *1BL*: one pair of arms deficient for identical segments, one normal *1BL* and one $1BL_{RTD}$. Among 100 and 75 PMCs analyzed in these two combinations, respectively, no intra-isochromosome pairing (two arms of an isochromosome bound by a chiasma to form a ring) was observed. However, the deficient arms of the isochromosomes 5-1 and 7-1 paired with the corresponding deficient arms of their precursor chromosomes 5 and 7, with 73 and 83% frequencies, respectively. Normal long arms of isochromosomes 5-1 and 7-1 paired with the RTD arms of chromosomes 5 and 7 with 45 and 55% frequencies, respectively.

Meiotic pairing of the isochromosome 8-2 was observed in four combinations: single copy plus normal chromosome *1B* (20″ + 8-2 + *1B*), single copy plus a pair of normal chromosomes *1B* (20″ + 8-2 + *1B*″), in a diisosomic (20″ + 8-2″) and in a diisosomic plus normal *1B* (20″ + 8-2″ + *1B*). Among 129, 87, 80 and 54 PMCs analyzed, respectively, no intra-isochromosome pairing was observed, nor did the deficient arms of the isochromosomes pair with the long arms of chromosome *1B*. In the two diisosomics 8-2, the deficient arms were paired in 85 and 90% of cells, respectively. In the two combinations where two normal arms of *1BL* were present (20″ + 8-2 + *1B*; 20″ + 8-2″) they paired with 86 and 95% frequency, respectively. In the two combinations where three normal arms of *1BL* were present (20″ + 8-2 + *1B*″; 20″ + 8-2″ + *1B*), the average number of *1BL* arms paired per PMC was 2.26.

Pairing of the asymmetrical isochromosome 8-3 was studied in three combinations: in a single dose with one normal chromosome *1B* (20″ + 8-3 + *1B*), in a single dose with a single chromosome *1B* deficient for the same segment as the deficient arm of isochromosome 8-3 (20″ + 8-3 + Df*1B*, produced by recombination of the deficient arm of chromosome 8 with *1BL*) and in a diisosomic (20″ + 8-3″). Samples of 174, 99 and 75 PMCs were analyzed in the three combinations, respectively. In no instance was intra-isochromosome pairing observed, and there was no pairing between the deficient and normal arms of the isochromosomes with the deficient or normal long arm of chromosome *1B*. When two arms deficient for the same segment were present, as in the diisosomic or in the combination with deficient *1B*, the deficient arms paired with each other with 91 and 93% frequency, respectively. When pairs of normal chromosome arms were present, as in the 20″ + 8-3 + *1B* and the diisosomic, they paired with each other with 90 and 99% frequency, respectively.

Overall, among the 1134 PMCs analyzed, intra-isochromosome pairing was observed in only two cells (both were in the combination of isochromosome 1-1 with $1B_{RTD}$, Figure 3). In four cells, also in the combination involving isochromosome 1-1 with $1B_{RTD}$, the longer arm of 1-1 paired with the inverted duplicated segment of $1B_{RTD}$ (Figure 3). In the remaining 1128 cells, pairing was exclusively between arms of the same length. If a chromosome arm of the same length was not present, both arms of an isochromosome remained unpaired (Figure 4).

In the plants diisosomic for isochromosomes 8-2 and 8-3, the two isochromosomes frequently formed one large ring (89.7% of the 155 PMCs observed), but pairing was always between arms of the same length: the deficient arm of one isochromosome paired with the deficient arm of the other isochromosome, and the normal arm paired with the normal arm (Figure 4). Overall, among 483 cells having pairs of deficient arms either as diisosomics or in combinations of an asymmetrical isochromosome with a deficient *1B* or the precursor chromosomes, the average pairing frequency of the deficient arms was 85.5%, whereas among 458 cells having pairs of normal long arms of chromosome *1B*, the average pairing frequency of the normal *1BL* arms was 91%. In no instance did a deficient arm, either of an asymmetrical isochromosome or of a deficient *1B*, pair with a normal *1BL* arm.

DISCUSSION

The meiotic behavior of asymmetrical isochromosomes studied here demonstrates that a deficiency of ≤50% of a chromosome arm (as in isochromosome 5-1) does not significantly reduce the ability of that chromosome arm to pair. Whenever two chromosome

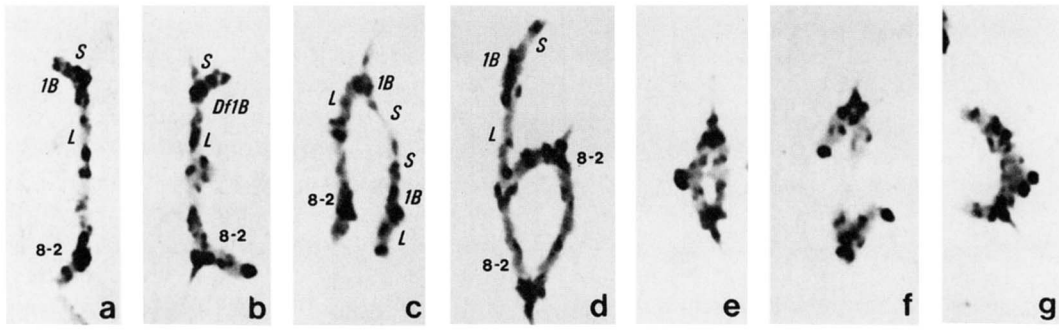

FIGURE 4.—Meiotic configurations involving asymmetrical isochromosome 8-2 in bread wheat. (a) Rod bivalent with a normal *1B*, paired in the long arm (L). (b) Rod bivalent with a deficient *1B*, paired in the deficient arm. (c) Trivalent with two normal *1B* chromosomes; the deficient chromosome is paired in L. (d) "Frying pan" trivalent formed by two 8-2 chromosomes and one normal *1B*. Both arms of 8-2 are paired. (e) Ring bivalent of two 8-2 chromosomes. (f and g) Rod bivalents formed by two 8-2 chromosomes paired in the deficient and normal arms, respectively.

arms deficient for the same segment were present, they paired with each other with frequencies quite similar to that of normal arms. While the deficiency did not significantly affect pairing of an arm, heterozygosity for a deficiency effectively prevented pairing, even when proximal ≤80% of the arm lengths were identical and regardless of whether the two arms were connected by a centromere. When an arm of a normal or deficient isochromosome had a choice of pairing partner, either deficient for the same segment or normal, pairing was exclusively between arms of the same length. Not one instance of pairing of arms of different length was observed whenever a better-matched pairing partner was available.

BARLOW and DRISCOLL (1981) postulated that a low frequency of metaphase I pairing of deficient chromosome arms in wheat was a result of desynapsis, because the amount of recombination in the progeny exceeded pairing frequency. However, CURTIS *et al.* (1991) showed that the increased amount of recombination in the progenies of deficiency heterozygotes relative to the metaphase I pairing frequency was completely explained by gametic selection, and no contribution of desynapsis was apparent. In the study reported here, desynapsis would have produced unpaired arms of isochromosomes in MI with chromatids of different length. Since five of the seven isochromosomes had one arm terminating in a prominent C band, the presence of such unequal chromatids would have been easily detected, but none was observed. This suggests that not desynapsis but either lack of synapsis (asynapsis) or inability to form chiasmata in synapsed arms was responsible for the absence of chiasmate pairing of the arms of asymmetrical isochromosomes in metaphase I.

The observations in this study are practically identical to those of the pairing behavior of deficient chromosome arms not connected in isochromosomes (CURTIS *et al.* 1991). Deficiency *per se,* even covering the entire distal half of an arm, does not impair the ability of that arm to pair; it is the heterozygosity for a deficiency, of even a small segment, which severely impairs pairing. On the other hand, deletions of very long proximal segments, regardless of whether they are in homozygous or heterozygous condition, have little effect on pairing (CURTIS *et al.* 1991; A. J. LUKASZEWSKI, unpublished data). True to this, two arms of a pseudo-isochromosome, with homologous terminal regions and heterologous proximal regions, regularly pair to form a ring (CALDECOTT and SMITH 1952; MORRIS 1955), whereas two arms of an asymmetrical isochromosome, with perfect homology (they originate from sister chromatids) for the entire length of the shorter of the two arms but with heterologous terminal segments, do not pair with each other. The pairing success of two arms is determined in the terminal (telomeric) region: the presence of homology in the proximal regions, the physical proximity of these regions in an isochromosome, or the identity of the centromere itself (CURTIS *et al.* 1991) do not affect pairing success.

Meiotic behavior of deficient chromosomes in wheat may help to explain several aspects of chromosome pairing. First, pairing failure of long homologous segments of the asymmetrical isochromosomes and of deficient and normal chromosomes (CURTIS *et al.* 1991) indicates that homology per se is not a sufficient condition for chiasmate pairing. If not all homologous regions pair, the absence of pairing does not necessarily indicate the absence of homology. Homology appears to be only a prerequisite for pairing. Second, the proper alignment of homologous regions in the vicinity of the telomeres is essential for pairing success. This is in full agreement with numerous earlier observations on meiotic behavior of chromosomes in a range of organisms from barley, where pairing configurations in double translocation heterozygotes were dependent on the arrangement of the chromosome ends (KASHA and BURNHAM 1965), maize, where the proximal 60% of the chromosome arms were found incapable of the initiation of pairing (BURNHAM *et al.* 1972) to grasshopper, where the translocation-induced misalignment of the telomeres at the point of attachments to the nuclear membrane prevented pairing of a chromosome arm (MOENS *et al.* 1989).

The assumption that all homologous chromosome

segments present in a nucleus faithfully pair at meiosis severely complicated models of chromosome pairing (MAGUIRE 1984, 1988; LOIDL 1990). This assumption seems to have been based on the regular formation of characteristic pachytene or metaphase I configurations in translocation, duplication or inversion heterozygotes. Unfortunately, these aberrations are identified primarily by the specific meiotic configuration they produce. Consequently, if the ability of an aberration to form a specific meiotic configuration is used to identify this aberration itself, the fact that the configuration is produced is not necessarily indicative of the pairing process lest the argument becomes circular. In other words, if the ability of a translocated, duplicated or an inverted segment to pair with its counterpart in a normal chromosome is used to identify this segment, then it is no surprise that it pairs or it would have never been identified. It would seem that most of the translocations, inversions, or duplications now available are only a subset of all such aberrations present, and this subset was selected for the ability to form typical meiotic configurations. Consequently, from the point of view of pairing initiation, these aberrations are not necessarily a representative sample and their behavior should not be generalized. When chromosome aberrations are identified by means different from their meiotic pairing behavior, such as C banding in this study, it becomes clear that not all regions of homology, even if extensive, engage in chiasmate associations. There are numerous examples where long stretches of homology never engage in chiasmate pairing, including the deficient chromosome arms discussed here and the barley, maize and grasshopper examples mentioned above, an inversion of ~90% of a rye chromosome arm, which has never been observed to form chiasmate bonds with its normal counterpart (A. J. LUKASZEWSKI, unpublished data), or wheat-rye translocations, the pairing behavior of which was dependent mainly on the pairing partner available (LUKASZEWSKI 1994).

If the requirement for perfect recognition of all homologous segments in meiotic chromosome pairing is removed, the model of pairing initiation can be considerably simplified. It has been known for some time, and recently studied in detail in maize (DAWE *et al.* 1994), that in the early meiotic prophase the telomeres congregate in a tight knot on the nuclear membrane, forming the "leptotene bouquet." DAWE *et al.* (1994) clearly demonstrated that only the telomeric regions, and not interstitial or proximal regions of chromosome arms, come into close physical proximity in the leptotene bouquet. The role of this structure in the initiation of pairing has been downplayed (MAGUIRE 1988; LOIDL 1990) because it would not account for the recognition and pairing of interstitial segments in translocations or inversions. Instead, quite elaborate structures and mechanisms have been postulated to explain the recognition and pairing of such segments (for review, see LOIDL 1990). However, the leptotene bouquet, or a similar configuration, may be the effective means of pairing initiation by bringing the telomeres of the homologous chromosome arms into sufficient physical proximity to initiate synapsis (FUSSELL 1987). As a general rule, the synaptonemal complex is initiated at, or close to, the attachment of the telomeres to the nuclear membrane, and secondary initiation sites appear later. GILLIES (1985) observed ≤76 synaptonemal-complex initiation points per nucleus in diploid rye ($n = 7$), and these new initiation points appeared until late in the zygotene. Zygotene in rye lasts ~11–12 hr (BENNETT and KALTSIKES 1973; ROUPAKIAS and KALTSIKES 1977). Also, there seems to be a sufficient time-lag between the initiation of synapsis at the ends of chromosome arms and at the interstitial sites (C. B. GILLIES, personal communication) to speculate that chromosome condensations alone may drive the progression of synapsis along the arm by bringing various interstitial regions of the arms into contact. Once synapsis is initiated at or near the telomeres, the position of a pair of homologous arms in the nucleus may become fixed. At the distal end, the telomeres are attached to the nuclear membrane, while the synaptonemal complex initiated as a result of homologue contact during leptotene bouquet stage binds the two arms to each other. On the opposing end of the arms, the centromeres are confined to a limited space of the nucleus in the general Rabl's orientation (FUSSELL 1987). With the two ends of the arms fixed in space, the progression of chromosome condensation, from very thin threads many times longer than the diameter of the nucleus at the onset of zygotene to relatively short and well-defined bivalents at the beginning of pachytene, would be expected to bring the two arms into register and into physical proximity appropriate for synapsis. If the structure of the two arms is similar, homologous segments are automatically juxtaposed. However, if homology is absent in the telomeric regions during the initial contact of the two arms in the leptotene bouquet, as in deficiency heterozygotes, in asymmetrical isochromosomes, or in heterozygotes of many other chromosome aberrations, the arms do not synapse, chiasmata will not be formed and the arms will not be paired at metaphase I. The failure of two arms of an asymmetrical isochromosome to form chiasmate bonds suggests that interstitial initiation of synapsis may be dependent on the successful initiation of synapsis in the telomeric region. If synapsis in the distal (telomeric) regions fails, no back up mechanism appears to be present to assure contact between nontelomeric regions of homologous chromosome arms, and these arms will fail to form chiasmate associations. The alternative explanation of the observed phenomena is that because the synaptonemal complex is indifferent to homology (LOIDL 1990), synapsis of the arms of unequal length could still be initiated at the telomeres, but the homologous regions would not be juxtaposed for the chiasmata to form. However, if this was the case, the offset in juxtapositioning of homologous segments

should decrease toward the centromere, and proximal chiasmata should be formed with noticeable frequencies, especially in the chromosome aberrations involving relatively short distal segments, like isochromosome 8-3 in this study (Figure 2). However, the two chiasmata involving two arms of isochromosome 1-1 observed here, and all chiasmata binding deficient with normal arms observed by CURTIS *et al.* (1991), as well as pairs of arms with different deficiencies (A. J. LUKASZEWSKI and C. A. CURTIS, unpublished data), always appeared terminal for the shorter of the two arms bound. The study on Chloealtis by MOENS *et al.* (1989) has shown that translocation of a small segment of a B-chromosome to the distal end of chromosome 7 misaligned the normal and translocated arms at their point of attachment to the nuclear membrane and synapsis was not between the translocated and nontranslocated arms but between the nontranslocated arm of chromosome 7 and a heterologue. These observations suggest that heterozygosity for deficiency prevents synapsis between arms of different length and chiasmata never have a chance to form.

Together with a considerable body of evidence on the pairing failure of long homologous segments of chromosomes, the observations on the meiotic behavior of the asymmetrical chromosomes clearly point to the importance of the proper alignment of the telomeres for pairing success. Interestingly, 30 years ago, when it was discovered that the telomeres of chromosomes were attached to the nuclear membrane, SVED (1966) suggested that a pairing initiation site at the attachment point of the telomeres of homologues to the nuclear membrane explained many aspects of the pairing behavior of chromosomes, including low multivalent frequencies in autopolyploids, low frequency of bivalent interlocking and the behavior of inversions. A role of such pairing initiation site could be fulfilled by any segment of a chromosome located in the vicinity of the telomere which comes in contact with its homologous partner in the leptotene bouquet stage.

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