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Meiotic mutants of rye *Secale cereale* L. II. The nonhomologous synapsis in desynaptic mutants *sy7* and *sy10*

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Abstract We studied the expression and inheritance of two spontaneous mutations found in different populations of rye *Secale cereale* L. that cause high univalent frequency in meiosis and low fertility. Both mutations were inherited as monogenic recessives. For each of the mutations the corresponding gene symbols (*sy7 and sy10*) were suggested although their allelism has not been studied. These mutants differ in chiasma frequency and in the number of univalents per meiocyte. Electron microscopy of the whole-mount surface-spread synaptonemal complexes (SCs) from microsporocytes of both mutants revealed that during meiotic prophase I random synapsis began and progressed that involved not only homologous but also non-homologous chromosomes. SCs were formed with frequent changes of pairing partners (switches) and intrachromosomal foldbacks of unpaired axial elements. As a result, incompletely synapsed, non-homologous and multivalent SCs were formed in mutants by the stage analogous to pachytene in normal plants. In *sy7* a maximum in the number of switches and foldbacks were observed at zygotene, whereas in *sy10* this occurred at pachytene. We suggest that it is the process of recognition of homology that is impaired in both mutants. This leads to indiscriminate synapsis and prevents chiasma formation. Both mutants may be classified as desynaptic.

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Introduction

The term "desynapsis" is used in cases when, by the stage of diakinesis and metaphase I (MI), bivalents transform into univalents because of the lack of chiasmata. In some cases, this is a result of the incomplete synapsis of homologous chromosomes at pachytene (Khristolyubova and Safonova 1982; Fedotova et al. 1992). In a desynaptic mutant of maize (Timofeeva and Golubovskaya 1991) and achiasmatic mutant of *Allium fistulosum* (Jenkins and Okumus (1992), the indiscriminate synapsis of axial elements of chromosomes in meiocytes has been reported. In both cases multivalent synaptonemal complexes (SCs) as well as foldbacks of lateral elements were found. In both species the multivalent SCs and the foldbacks were formed through non-homologous synapsis of extensive chromosome regions. The non-homologous SCs were assumed to be ineffective in supporting chiasmata formation, resulting in univalent formation.

In a desynaptic maize mutant, Maguire et al. (1991) found that at pachytene the central region of the SC was significantly wider (statistically) that its counterpart in normal plants and the the SCs twisted less. The pattern of separation of the distal heterozygous heterochromatic knob between four chromatids in MI bivalents indicated the normal occurrence of crossingover in the mutants, but the chiasma frequency was considerably reduced and univalents were observed. The authors came to the conclusion that there was a failure in chiasma maintenance in spite of normal crossingover in the mutant.

In this paper, data are presented on the inheritance and expression of two partially sterile mutations of diploid rye *Secale cereale* in which both of the above-mentioned types of non-homologous synapsis, multivalents and foldbacks, were found. This indiscriminate synapsis is assumed to be the reason for reduced chiasma frequency and significant number of univalents observed in the mutants.

Materials and methods

Two partially sterile, spontaneous mutant forms of diploid winter rye *Secale cereale* L., *ms7* and *ms10*, were used. The mutant forms have been isolated from populations of 'Vyatka' variety and weedy rye (Sosnikhina and Smirnov 1990). The inheritance of each of the mutations was studied in segregating inbred progenies by calculating fertile, and sterile and partially sterile plants. The additional calculation of the plants with normal meiosis and with desynapsis was performed in some progenies that segregated ms10 mutants.

For light microscopy, spikes were fixed with Newcomer fluid and stained with acetocarmine, following which squash preparations of the anthers were prepared. Whole-mount surface-spreads of the prophase I microsporocytes stained with silver nitrate were prepared for electron microscopy by methods described elsewhere (Fedotova et al. 1989; Sosnikhina et al. 1992). The spreads were examined and photographed in the JEM-100B electron microscope

Results

Inheritance

17 inbred progenies segregating for mutant *ms7* plants the ratio of fertile and sterile plus partially sterile plants was 130:53 and fitted well to the expected monogenic ratio (χ^2=1.50; P>0.20). Partially sterile and sterile plants appeared to be homozygotes for a recessive allele. The cytological study showed that fertile plants had normal meiosis whereas sterile and partially sterile ones had univalents in MI; these univalents were assumed to be characterized by abnormalities in chromosome synapsis. We designated the recessive allele responsible for the abnormal synapsis *sy7*. The partially sterile mutant plants set up to 6% seeds after selfing and 19–56% seeds after open pollination. The fertile sibs from the same progenies set from 17% to 83% seeds after selfing. Thus, the mutant allele *sy7* was expressed to a higher degree in microsporogenesis than in megasporogenesis.

In segregating progenies with the mutant form *ms10*, there were more sterile plants than fertile ones. Segregation differed from both the monogenic and digenic types, with the total ratio of 78 fertile:90 sterile plants fitting well to the expected trigenic ratio 27:37 with complementary interaction of three dominant genes (χ^2=0.93; P>0.30). The cytological investigation revealed that the mutant *ms10* sterile plants were not uniform. Some of them had many univalents in MI, whereas others had two different types of meiotic abnormalities, such as irregular chromosome condensation in prophase I and compact chromosomes in MI (to be published elsewhere), which were expressed independently of each other. Only plants with univalents at diakinesis – MI were analyzed in this paper. The calculation of plants as to the normal and impaired chromosome pairing gave the ratio of 112:35, which fitted the expected one for monogenic inheritance (χ^2=0.19; P>0.50). This meant that, in the case of the mutant form ms10, the impairment of chromosome pairing was determined by a recessive allele of one gene, which we designated *sy10*. The homozygotes for the mutant allele *sy10* also had significantly lower fertility both in cross-pollination (0–30%) and inbreeding (0–15%).

Light microscopy

Partially sterile *sy7* and *sy10* plants had an increased number of open bivalents with one chiasma and a high number of univalents compared to the normal plants with no univalents (Table 1, Figs. 1 and 2).

The mutants had different numbers of univalents in their meiocytes. *sy10* mutant plants were arbitrarily placed into two groups: those with a relatively low (3.2) mean number of univalents per meiocyte (group 1) and those with a high (7.8) mean univalent number (group 2). The effect of mutations *sy7* and *sy10* is not only unequally expressed in different plants but also in the anthers of different florets and spikelets. The variability among meiocytes for number of univalents shows that the behavior of different chromosomes may be individual and independent of one another (Table 2). The impairment of homologous pairing of different chromosomes appeared to occur at random. The distribution of meiocytes with different number of univalents in mutant *sy10* plants of both groups fits the Poisson distribution (for group 1, χ^2=11.63; P>0.05; for group 2, χ^2=8.86, P>0.10). In *sy7* mutant plants the distribution of meiocytes with different number of univalents does not fit either Poisson or binomial distributions due to the high frequency of cells with 14 univalents (Table 2).

Interchromosomal chromatin connections of non-chiasmatic origin were observed in meiocytes of *sy10* mutants at diakinesis and MI. These connections were often seen between non-homologous chromosomes. The proportion of meiocytes with such chromatin connections varied from 15% to 75% in different mutant plants (Fig. 3) The main type of abnormality at the anaphase I (AI) stage was chromosome lagging (Table 3). The number of laggards correlated with the mean number of univalents at the MI stage (for *sy10* mutants r_s=0.82; P>0.01). At the AI stage many chromosome bridges (double, triple and multiple) were observed in the *sy7* mutants which may be evidence of the abnormal formation of chiasmata in prophase (U-exchanges). Chromosomal fragments were found in diakinesis and MI meiocytes of the *sy7* mutants.

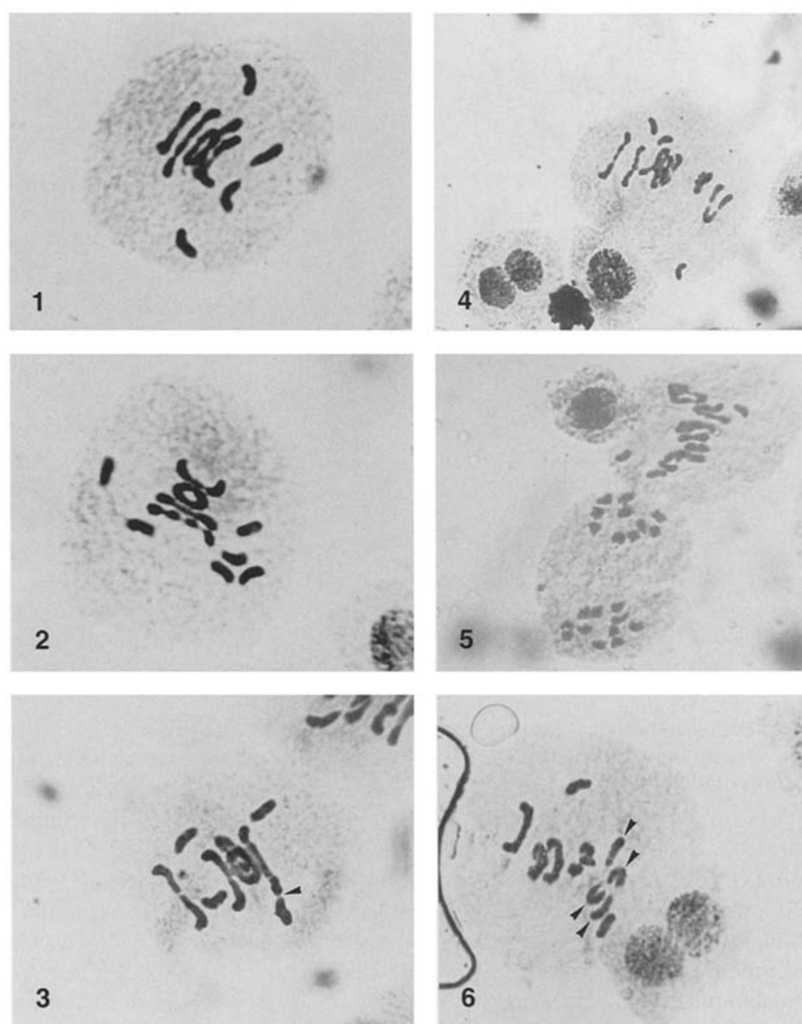
In both types of mutants abnormalities at the later stages of meiosis were determined by impairments in the first division. High frequencies of tetrads with micronuclei (44–96%) and polyads (2–9%) were found.

Of the plants studied cytologically, 2 triploids (Fig. 4 and 5) and 2 plants heterozygous for translocations (Fig. 6) were found in the inbred progenies segregating for the *sy10* mutation.

Thus, the meiocytes of both synaptic rye mutants contained univalents at the MI stage, and this occurrence caused the decrease in the fertility of the mutants. A negative correlation exists between the mean number of univalents per cell and seed set (r_s=-0.805; P>0.01) for *sy10* mutants.

Table 1 Chromosome pairing at diakinesis and MI in desynaptic rye mutants *sy7* and *sy10* and in normal plants

Mutant and group	Diakinesis		Metaphase I				
	Number of cells studied	Number of chiasmata per bivalent: mean (±SE)	Number of cells studied	Chromosome pairs		Meiocytes with univalents (%)	Number of univalents per meiocyte: mean& (range)
				Without chiasmata (%)	With one chiasma (%)		
Normal	200	2.25 (±0.1)	200	0	2.0	13.5	0.38 (0–2)
sy7	26	0.64 (±0.06)	79	44.1	48.0	96.0	8.0 (4–12)
sy10 group 1	120	1.59 (±0.11)	274	6.5	32.4	80.9	3.2 (2–4)
sy10 group 2	50	1.29 (±0.15)	290	22.1	35.1	98.6	7.8 (4–12)



Figs. 1–6 Meiosis in desynaptic rye mutants or plants from the inbred progeny segregating the mutants: **Fig. 1** Metaphase I from *sy10*. 2 univalents, 5 open (rod) bivalents and 1 closed (ring) bivalent

Fig. 2 Metaphase I from *sy7*: 6 univalents, 3 rods and 1 ring bivalent

Fig. 3 Metaphase I from *sy10*. 4 univalents are present and 1 of these is connected with a bivalent by chromatin thread (*arrowhead*)

Figs. 4–5 Meiosis in a triploid plant from the inbred progeny segregating *sy10* mutants

Fig. 4 Metaphase I: trivalent, univalents and bivalents can be seen

Fig. 5 Anaphase I: 21 chromosomes can be counted in the upper cell

Fig. 6 Metaphase I in a diploid plant from the inbred progeny segregating *sy10* mutants. This plant is heterozygous for a translocation. 1 tetravalent (*arrowheads*), 2 univalents and 4 bivalents are visible

Electron microscopy

Forty-five meiocyte nuclei from 14 normal plants, 37 from 2 *sy7* mutant plants and 68 from 7 *sy10* mutant plants were studied with an electron microscope.

In prophase I of meiocytes of normal fertile plants normal SCs of bivalents were formed that did not differ from the SCs of normal rye plants.

At early zygotene (the "bouquet" stage) of meiocytes of the mutant plants the axial elements of chromosomes began to synapse and make SCs, but not only between the homologs. In both mutants one of two SC lateral elements sometimes changed pairing partners to form SCs with the axial element of a non-homologous chromosome (Fig. 7). Such sites of switches in pairing partners were easily observed in the spreads of mid-zygotene nuclei. The regions

Table 2 The distribution of meiocytes with different numbers of univalents at MI in desynaptic rye mutants *sy7* and *sy10*

Mutant and group	Number of meiocytes studied	Mean number of univalents per meiocyte	The proportion (%) of meiocytes with the following number of univalents							
			0	2	4	6	8	10	12	14
sy7	79	8.0	3.8	11.4	8.9	13.9	13.9	6.3	6.3	35.5
sy10,group 1	247	3.2	19.1	32.0	24.7	12.5	5.3	3.2	2.4	0.8
sy10, group 2	290	7.8	1.3	5.9	11.7	20.3	21.7	18.6	12.1	8.4

Table 3 Abnormalities in AI in *sy7* and *sy10* plants

Mutant and group	Number of cells studied	Types of abnormal cells (%)				
		Total	Lagged chromomes	Bridges	Fragments	Unequal-division
Normal	500	6.7	3.8	3.6	0	0
sy7	98	95.0	45.0	72.7	8.9	8.2
sy10, group 1	1146	33.7	27.4	3.9	2.1	0
sy10, group 2	903	69.1	52.3	2.4	2.9	10.9

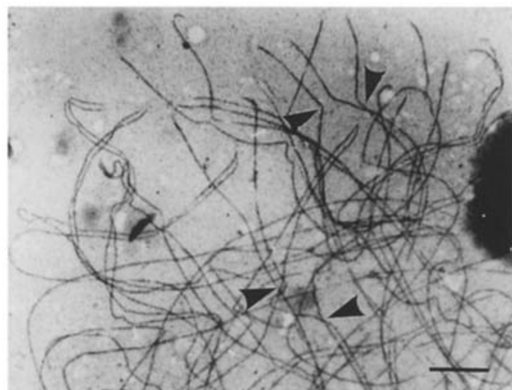


Fig. 7 Electron micrograph of part of an early zygotene nucleus from the *sy10* mutant. The ends of both the axial elements and of the SCs demonstrate the "bouquet" configuration.The *arrowheads* indicate the sites of exchange (switches) of the axial elements aligned at a distance (*upper pair*) or of the axial element and the lateral element of SC (*lower pair*). *Bar*: 2 μm

of unpaired axial elements were found between the segments of SC, and they linked the SC segments after several switches into some kind of a chain (Fig. 8). Internal unpaired segments of lateral elements which made foldbacks were also observed at this stage (Fig. 8).

In addition, breaks in the axial (lateral) elements were found in 97.5% of the zygotene nuclei of the mutants.

As a result of indiscriminate synapsis, some regions of axial elements inevitably remain asynapsed, and one should have criteria, independent from the completeness of SC formation, for identifying the pachytene stage. Unfortunately, such indirect criteria are lacking for meiotic prophase I in rye. On the basis of our experience, it is impossible to identify the pachytene stage in rye on the grounds of nucleolus morphology as has been done in maize (Anderson et al. 1988), and our technique did not enable us to make prophase I staging using the criteria of kinetochore staining, as was done by Sherman et al.(1989). Following Gillies (1985), if the synapsis was not complete because of irregularities, we considered those cells in which the ends of the SCs' were distributed randomly over the nuclear periphery to be at pachytene; this in contrast to the grouped SC ends observed at zygotene stage.

In spite of indiscriminate synapsis during zygotene, almost completely formed SCs with only small asynaptic regions at the sites of switches and bases of foldbacks were observed in some pachytene cells, we considered these cells to be late pachytene. However, in some pachytene cells extensive non-synapsed axial element regions were observed (Fig. 9). Because of non-homologous synapsis, it was possible to observe chains composed of SC segments connected by non-synapsed segments of axial elements similar to those found in zygotene cells.

The sites of switches were Y shaped structures (Fig. 10), whereas such sites in the translocation heterozygote looked like four-prong, cross-shaped structures. In 63% of the pachytene nuclei of the mutant plants we found segments of axial elements in the form of foldbacks (Fig. 9), some of which belonged to univalents. The SCs of bivalents could be found in some nuclei but in most cases it was difficult to distinguish them from parts of the multivalents. Such parts probably appeared as a result of breaks in the lateral elements of SCs at the switch points or at the bases of foldbacks (Figs. 9 and 10). That was the reason why we did not calculate the number of bivalent SCs per nucleus. Only 1.3% of the pachytene nuclei of the mutants did not have breaks in SCs. These breaks might be considered as artifacts although they were never found in the meiocytes of normal plants.

The number of breaks in the lateral elements of SCs increased at diplotene. However, the switches and foldbacks

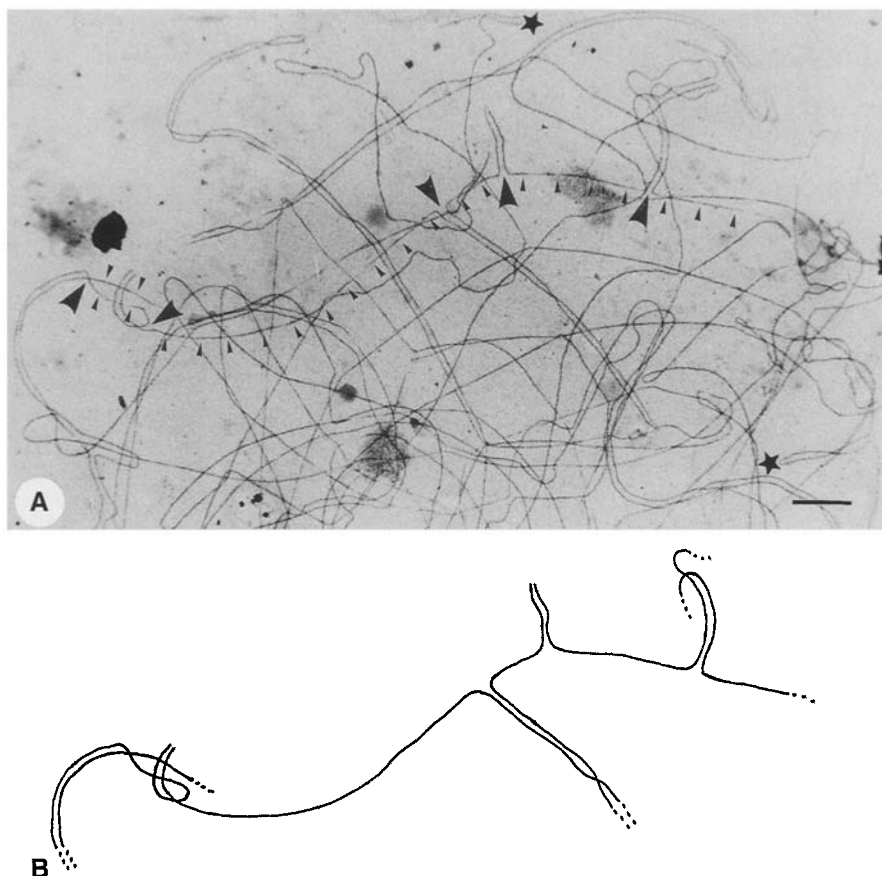


Fig. 8A, B Electron micrograph (**A**) and drawing (**B**) of part of the mid-zygotene nucleus from the *sy10* mutant. *Large arrowheads* indicate switch-points, *small arrowheads* indicate unpaired axial elements linking SC segments into a chain. The chain is shown in (**B**). *Asterisks* mark foldbacks at their tops. *Bar*: 2 μm

still could be found. While the number of foldbacks and switches changed from zygotene to diplotene, this change occurred differently in the two mutants (Fig. 11). There were, however, common trends in the changes of switches and foldbacks in both mutants in early (zygotene) and late (diplotene) prophase I. The *sy10* mutants had a maximum total number of SC abnormalities at late pachytene, *sy7* mutant plants at zygotene (Fig. 11).

Thus, our study of the meiotic prophase in both *sy7* and *sy10* mutants with an electron microscope revealed that multivalent SCs had formed by the end of pachytene in both cases. Since both mutants have many univalents at diakinesis and MI, they should inevitably be classified as desynaptics.

Discussion

Our study of inbred progenies which segregate *ms7* and *ms10* partially sterile mutants has revealed that meiosis with a high number of univalents is determined by a recessive allele of one gene, designated as *sy7* and *sy10*, respectively. It is microsporogenesis which is mainly impaired in homozygotes for the recessive allele in both cases, and this is true for all known desynaptic mutants (Gottschalk and Kaul 1980; Gottschalk 1987). It should be mentioned that, although both spontaneous rye mutations were obtained independently from different populations, we cannot consider them to be mutations of different genes until a test on allelism is performed.

According to the literature, the number of synaptic genes in a genome is probably around several dozens (Gottschalk and Kaul 1980; Koduru and Rao 1981; Kaul and Murthy 1985). One can assume that the reason for the occurrence of univalents in MI of some desynaptic mutants is the lower probability of the formation of chiasmata as a result of incomplete formation of SCs and /or their precocious degradation in meiotic prophase. However, the formation of chiasmata in *sy7* and *sy10* rye mutants was reduced by non-homologous synapsis which resulted in non-homologous SCs: multivalents and foldbacks.

Taking this into consideration, we would like to discuss the effect of *sy7* and *sy10* mutations in the framework of the general problem of the mechanisms of homologous chromosome synapsis.

At present pairing and synapsis are believed to involve several independent stages: (1) the recognition of homologues, (2) the attraction of homologues by means of SC formation, (3) the precise synapsis of homologous DNA sequences in the frames of SC (see Loidl 1990 for the review). Recently, Hawley and Arbel (1993) again considered pairing and synapsis to be two different meiotic events. Pairing was assumed to be the primary alignment of homologous chromosomes during leptotene, while synapsis was defined as the intimate association of homo-

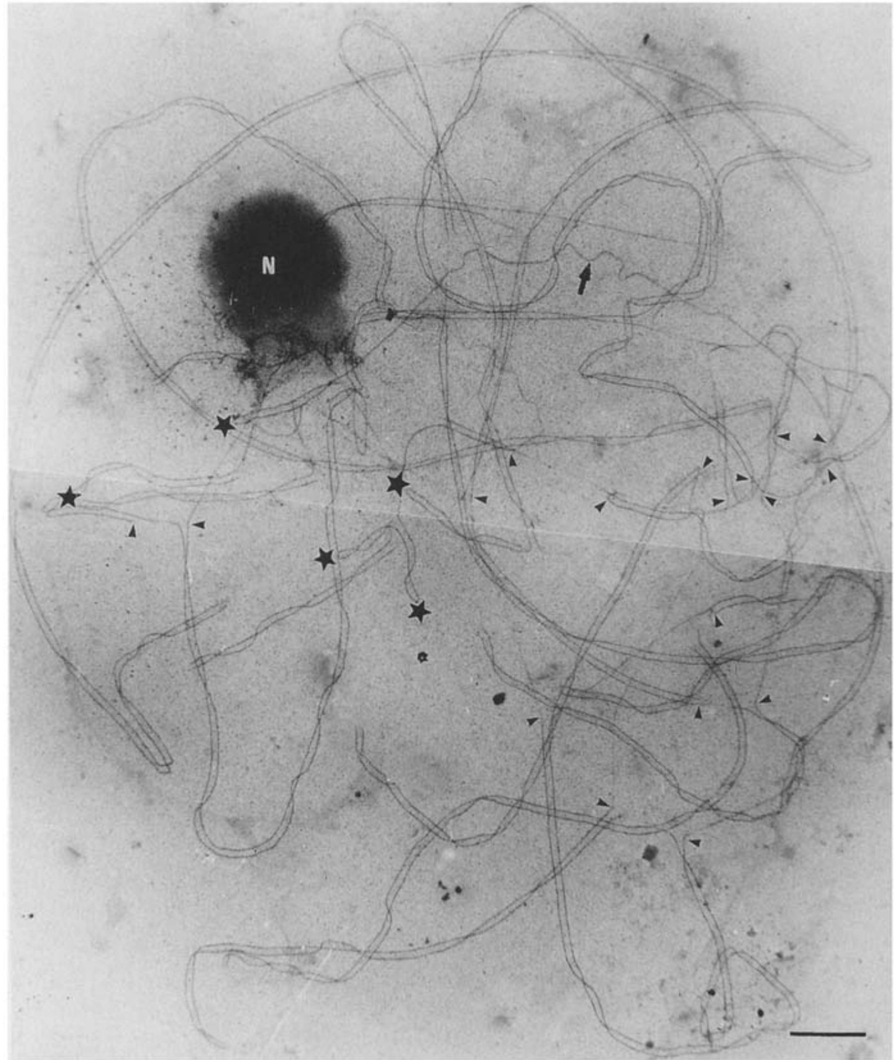


Fig. 9 Electron micrograph of pachytene in desynaptic mutant *sy10*. *Small arrowheads* indicate the most evident switch-points, the large *arrow* indicates an unpaired axial element, and the foldbacks are indicated by *asterisks* (*N* nucleus). *Bar*: 2 μm

logues in the SC. The SC may be in some respect indifferent to homology and this is especially obvious in the cases of SC formation in monohaploids and formation of multivalent SCs in allopolyploids (see Holm and Wang 1988; Gillies 1989; Loidl 1990; Chatterjee and Jenkins 1993, and the literature cited therein). These facts provide evidence for the relative independence of the stage of homologous recognition of chromosomes from the stage of SC formation.

The evidence is insufficient in our work to suggest the primary impairments in *sy7* and *sy10* rye mutants. Meanwhile two points are obvious: neither the ability to form axial elements nor the capacity of these elements to join into SC are impaired in either mutant. One can assume that it is the process of primary recognition of homologous sites during initial pairing that is affected by the mutations. If SCs are formed between non-homologous chromosomes, the differences in DNA nucleotide sequence may prevent chiasma formation which will lead to precocious desynapsis.

It is worthwhile to compare the cytogenetic effect of the *sy7* and *sy10* loci in rye with that of the *Ph* locus of common wheat. It is well known that the *Ph* locus prevents the synapsis of homoeologous chromosomes (Sears 1976; Holm and Wang 1988 and the literature cited therein) and determines the occurrence of only bivalents in MI. However, a study of SC in *Triticum aestivum* has revealed the formation of multivalent SCs at zygotene and their subsequent correction into bivalents by pachytene (Jenkins 1983). Switches as well as foldbacks were observed in *T. aestivum* at zygotene. The *Ph* locus obviously controls some mechanism of correction that eliminates the abnormalities of synapsis caused earlier by possibly inexact homologous recognition due to the presence of three homoeologous genomes (Jenkins 1983; Holm and Wang 1988). In rye, impaired homology recognition is probably characteristic of the mutants *sy7* and *sy10*, whereas *Sy7* and *Sy10* normal alleles determine the correct recognition.

Thus, the study of *sy7* and *sy10* rye mutants has revealed that diploid species carry genes whose normal alleles prevent non-homologous synapsis. Such non-homologous synapsis could be caused by repeated DNA sequences of short homologous regions in non-homologous chromosomes that could arise as a result of chromosome rearrange-

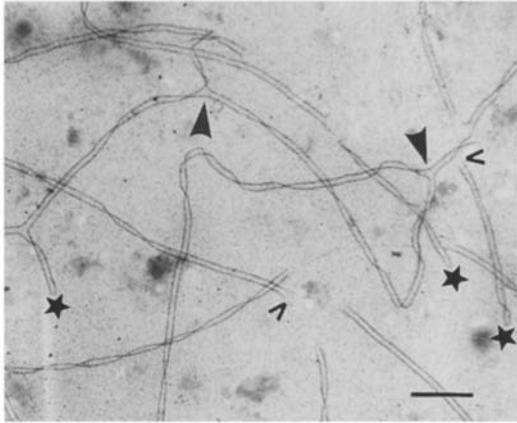


Fig. 10 Electron micrograph of part of the late pachytene nucleus in the desynaptics *sy10*. *Solid arrowheads* indicate triradial configurations at the switch-points, foldbacks are indicated by *asterisks*, and *open arrowheads* indicate breaks (or gaps) at the bases of the foldbacks. *Bar*: 2 μm

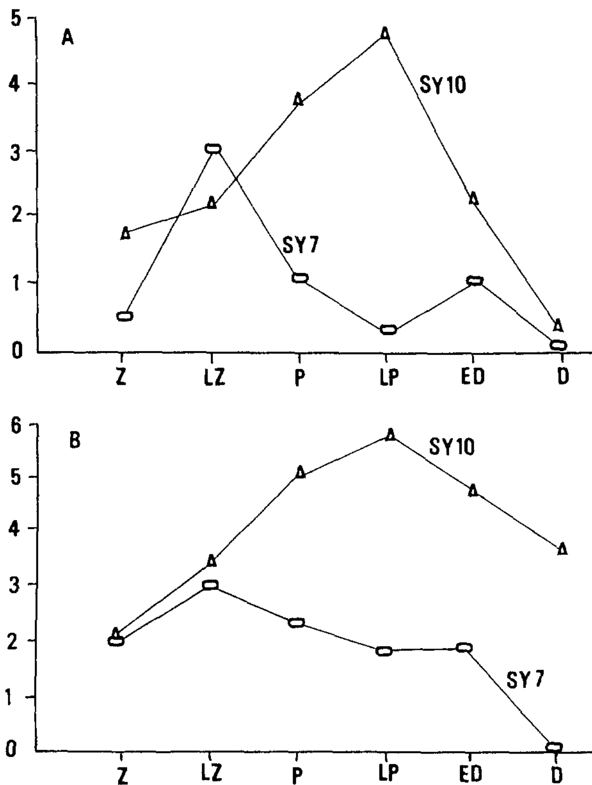


Fig. 11A, B Dynamics of changes in the number of switches (**A**) and foldbacks (**B**) from early zygotene to diplotene in mutants *sy7* and *sy10*. *Abscissa*: meiotic stages [*Z* zygotene (except LZ), *LZ* late zygotene, *P* pachytene (except LP), *LP* late pacytene, *ED* early diplotene, *D* diplotene (except ED)]. *Ordinate*: mean number of switches or foldbacks per nucleus

ments. Sears (1976) called attention to the problem of the existence of such genes in diploid species when he discussed the possible origin of the *Ph* locus in wheat.

It may be suggested that in the *sy7* rye mutant some transformation of multivalents and elimination of non-homologous synapsis do occur at late zygotene (Fig. 11) similar to such process in common wheat. However, it occurs probably too late for chiasmata formation due to the slow degradation of SC in foldbacks and the persistence of some of them up to early diplotene. In the *sy10* mutant the elimination of non-homologous contacts begins only at late pachytene, actually at the time when of SC degradation begins (Fig. 11). As a result, non-homologous partners of multivalents transform into univalents, while homologous ones may form open bivalents. The relatively high number of foldbacks at diplotene is proof that this particular type of non-homologous synapsis is more difficult to eliminate. It can be assumed that the breaks of the lateral elements occurred at pachytene in the mutant plants as a correction step of heterologous synapsis and resolution from interlocking. On the other hand, it can also be assumed that the unequal synapsis of heterologous chromosomes in mutant nuclei results in additional tension. During the spreading procedure this tension leads to an increased number of breaks in both the lateral elements and SCs at the bases of foldbacks and switch-points relative to those found in normal plants;, consequently, these are artifacts. In summary, the disturbance of the process of homologous recognition leads to random participation of different chromosomes in SC formation and to subsequent random formation of a number of univalents after multivalent degradation at the end of prophase I.

The data obtained in our study show that in the rye mutants studied multivalents do not transform into bivalents as they do in polyploid species. It is possible that in these mutants non-homologous chromosome synapsis occurs by the pathway observed in haploids where some multivalents may persist during prophase I (Gillies 1974; De Jong et al. 1991; Loidl et al. 1991), and even in MI when the associations of two or more chromosomes may be observed (Sosnikhina and Smirnov 1981).

If, in haploids or mutants like *sy7* and *sy10*, non-homologous synapsis occurs due to the existence of short homologous regions in non-homologous chromosomes (the consequences of chromosome rearrangements during genome evolution) then crossingover in such regions may lead to new chromosomal rearrangements. This may be the possible explanation for the occurrence of two translocation heterozygotes in the inbred progenies segregating *sy10* mutants found in our material.

Thus, we suggest that the diploid rye genome contains genes that ensure the exact homologous recognition as well as later transformation of multivalent associations.

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