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Review

Mutant genes affecting higher plant meiosis*

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1 Summary. That meiosis is conditioned by a large number of genes majority of which are present in a dominant state, is evidenced by the detection of numerous monogenic recessive mutant genes which affect the premeiotic, meiotic and post-meiotic course of events. These genes are site- and stage-specific, and a few are sex specific. Of these, the most prevalent are the mutant genes affecting male meiosis and causing male sterility (*ms* genes) and those inhibiting synapsis and chiasma formation (synaptic genes) and leading to gametic sterility. Majority of the mutant genes affect the entire chromosomal complement but a few influence only specific chromosomes of a complement so that the chromosomes behave differentially within a genome of the same species. Some mutant genes alter chromosome form and function, others modify integrity, degree of spiralization, movement and migration of chromosomes. Their cytogenetic behaviour, genetic significance and breeding utility are described and discussed.

Key words: Meiotic mutants – Recombinational variations – Synaptic mutations

* Dedicated to Professor Dr. Werner Gottschalk, Director, Institute of Genetics, University of Bonn, Federal Republic of Germany, on his 65th birthday for his excellent scientific contribution and humane nature

2 Introduction

Meiosis, genetically significant activity of a biological organism, comprises highly coordinated physiological, biochemical, cytogenetical and phenotypical events that lead to gene recombination, chromosome reduction and gamete formation. Unlike mitosis, meiosis is characterized by a slow rate of DNA synthesis, the absence of a S-period between the two divisions of meiosis, pairing of homologous chromosomes, crossing-over and reduction of chromosome number. Whereas DNA replication is complete before mitosis, it is slightly incomplete before meiotic division. The S-phase of meiosis is slow and of long duration; it is short and quick in mitosis. Though meiosis is a continuous process with specific and precise events, it has been partitioned into various stages that characterize certain cytological features and genetic events. These events are controlled by a large number of genes presently denoted as meiotic genes. The majority of these are

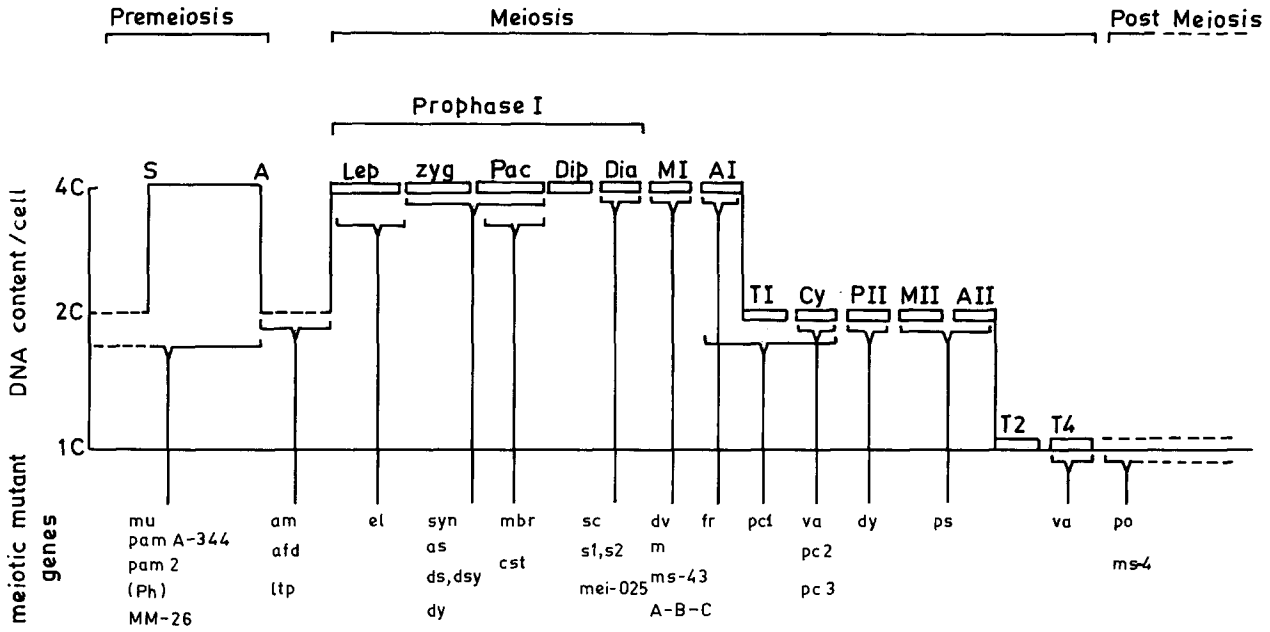

Fig. 1. Time of action of some meiotic mutants in higher plants

present in a dominant state. The mutation of any of these genes causes meiotic anomalies that affect gametic fertility. The presence of a large number of such genes is evidenced by the isolation of various mutants that affect meiotic division and/or its associated phases. Since the isolation of the first well-described mutant *c(3)G* in *Drosophila* by Gowen and Gowen (1922), many meiotic mutants have been isolated and characterized in plants and animals. The mutant genes affect viable gamete production by impairing different stages beginning from premeiotic mitoses to the reinitiation of gametophytic mitosis. Depending on the meiotic stage over which the genes act, they are classified as (a) premeiotic genes – the genes controlling meiotic initiation, (b) meiotic genes – genes controlling the meiotic course, and (c) post meiotic genes – the genes controlling post-meiotic events and gametogenesis. In the following, the action of mutant genes over these meiotic stages is discussed.

3 Premeiotic mutant genes

3.1 Premeiotic mitosis

Premeiotic division, a mitotic-type cell division, is unique as the switching on or off of meiotic processes occurs only after its successful and normal culmination. Any disturbance during this division causes deviations in meiosis and affects gametic fertility. Thus, this stage is the foundation stone on which the whole meiotic architecture is based. Its uniqueness can be deduced from the occurrence of some mutants of barley, *Brassica campestris*, pearl millet, pea, maize and rice (Smith 1942; Stringam 1970; Pantulu and Manga 1971; Gottschalk and Kaul 1974; Golubovskaya and Mashnenkov 1977; Kitada et al. 1983), in which this stage is disturbed (Table 1). In all these mutants the anomaly is conditioned by single recessive genes. In a barley mutant, Smith (1942) observed up to 112 bivalents per meiocyte due to defective cytokinesis during the last premeiotic mitoses. In the '*mu*' mutant of pearl millet (Pantulu and Manga 1971), cytokinesis fails in the majority of the sporogenous cells and the chromosomes undergo repeated duplications at least 8 times so that meiocytes having up to 256 nuclei are formed. The cells exhibit an erratic meiotic course and produce nonviable male and female gametes. Whereas the meiotic process occurs in multiploid sporocytes in pearl millet, in a pea mutant meiosis does not occur at all and the sporogenous cells degenerate along with the tapetum (Gottschalk and Kaul 1974). However, in a maize mutant (Golubovskaya and Mashnenkov 1977), 20% of the meiocytes undergo an abnormal meiosis after which the meiocytes degenerate. In another pearl millet mutant (Murthy, unpublished), the fusion of the majority of the sporogenous cells after the last premeiotic mitotic division results in meiocytes with varying levels of ploidy. Due to the lack of proper space, some of the bivalents fail to orientate at M I on the M I spindle and lag behind. Degeneration occurs after the irregular separation of chromosomes at AI and normal second meiotic divisions. In the rice mutant MM-26, several PMCs are interconnected due to an incomplete last premeiotic cytokinesis (Kitada et al. 1983). In addition, anomalies such as the presence of tetraploid cells and less condensed chromosomes lead to about 80% pollen and seed sterility.

Table 1. Monogenic recessive premeiotic mutant genes in higher plants

Species	Authors	Source
Brassica campestris (2n = 20)	Stringam 1970	Ethyleneimine
Hordeum vulgare (2n = 14)	Smith 1942	sp[a]
Oryza sativa (2n = 24)	Kitada et al. 1983	sp
Pennisetum americanum (2n = 14)	Koduru 1980; Pantulu and Manga 1971	sp, EMS
Pisum sativum (2n = 14)	Gottschalk and Kaul 1974	X- and γ-rays
Zea mays (2n = 20)	Golubovskaya 1979; Golubovskaya and Mashnenkov 1975, 1977; Rhoades 1956	sp

[a] Spontaneous

3.2 Meiotic entry

Initiation of the meiotic process or the 'meiotic switch-on' represents another critical step for a successful occurrence of meiosis. That this process likewise is gene controlled is evidenced by the 'ameiotic' maize mutant (*am*) found by Rhoades (1956) in which meiotic switch-on does not occur at all though premeiotic divisions occur normally and the sporogenous cells attain meiotic cell configurations by having larger cell and nuclear volume, relaxed chromosome morphology and the presence of biaccuminate metaphase spindles: Instead, the sporogenous cells undergo two to three ameiotic mitoses and degenerate. This anomaly is conditioned by a recessive gene '*am*', located on the short arm of chromosome 5 and leads to complete male sterility and partial female sterility. Whereas meiosis is completely eliminated in the ameiotic maize mutant, in a pearl millet mutant (Koduru 1980) a mitosis-like process occurs in the meiocytes which degenerate after the formation of restitution nuclei. Likewise, in a maize mutant '*afd*' (absence of the first division) (Golubovskaya and Mashnenkov 1975; Golubovskaya 1979), first meiotic division is like mitosis and the second meiotic division is also abnormal, resulting in complete male-female sterility. These premeiotic mutants provide an evidence that the information for a cell to undergo meiosis or mitosis is under genetic control and is available much prior to the initiation of the actual process.

4 Meiotic mutant genes

Meiotic prophase is a vital stage of cell division as homologous chromosomal synapsis, crossing-over and chiasma building occur during this stage. Genetically it is the most significant stage of meiosis as gene recombination occurs here. That all these events are under the genetic control of specific genes is evidenced by the isolation of mutant genes influencing the process of homologous chromosome pairing. Whereas some mutant genes completely suppress synapsis, others inhibit it to varying degrees. Some other mutant genes permit synapsis but cause the precocious pulling apart of the synapsed chromosomes so that no chiasmata are formed between them. During zygotene-pachytene, when synapsis occurs, chromosomes are thin, long and tangled. Thus, this stage is not easily amenable to cytological analysis in most plants. Therefore, differentiation between the complete absence of synapsis (asynapsis) and the initial synapsis followed by quick withdrawl of synapsis (desynapsis) is not always possible. Hence we adopt the term *'synaptic mutants'*, initially used by Riley and Law (1965) for those mutants in which chromosomal synapsis and/or chiasma formation and maintenance are inhibited or impaired. In all these mutants, univalents in varying frequency occur during the diakinesis-metaphase I stages.

4.1 Synaptic mutant genes

Crossing-over, a post leptotene event, is a basic requisite for gene recombination. Effective homologous chromosome pairing is a necessary prerequisite for crossing-over to occur. The pairing is brought about by formation of synaptonemal complexes (SCs). This formation is gene regulated and mutation of the regulatory genes leads to defective SC formation and consequently to univalence. This occurs in an asynaptic wheat mutant (LaCour and Wells 1970) in which lateral elements of the SC are normal but where the central element which binds the two homologues is absent. Consequently no homologous pairing occurs, meiosis continues abnormally and the gametes formed are sterile. Thus, an initial defect in SC formation disturbs the whole course of microsporogenesis and gametic fertility. A synaptic mutant gene in maize, *dsy*-A344 (Golubovskaya and Mashnenkov 1976) also causes defective SC formation and the SC consists of interrupted strands lacking the central element in some regions along the homologues. In another mutant of tomato, SCs are normal but the homologues fall

apart precociously (Moens 1969) due to defective SC formation caused by a mutant gene.

4.2 Recombinational variations

Like other mutant genes, synaptic mutant genes alter the degree of synapsis and crossing-over. It is easy to identify the genes that decrease synapsis or chiasma formation, add irregularities to the meiotic course and impair gametic fertility. But it is hard to identify those that increase degree of synapsis and/or chiasma formation and crossing-over and do not interfere with the meiotic course or gametic fertility. Some mutant genes increase chiasma frequency and recombination between certain chromosomal segments, whereas others reduce it (Table 3). This is indicated below:

4.2.1 Reduced recombination. Many meiotic mutants usually develop apparently normal SCs and the initial synapsis in them is nearly normal. But the mutant genes suppress or inhibit chiasma formation, thereby hastening the separation of homologues and reducing gene recombination. This occurs in a barley and a maize mutant. In barley, the gene reduces chiasma frequency leading to a two-fold reduction in gene recombination between certain marked genes located on chromosome 2 (Enns and Larter 1962). In a maize mutant, such a gene drastically decreases chiasma frequency and recombination of the genes located on chromosomes 5 and 3 (Nel 1973, 1979). The dominance of the wild allele is incomplete as the reduction is appreciable in the heterozygous $+/as$ genotype.

4.2.2 Normal recombination. Some genes do not alter crossing-over between the loci of homologues. Thus in a synaptic maize mutant, a normal level of crossing-over occurs between *sh-wx* genes of the *C-wx* linkage group (Beadle 1933) of chromosome 9. In as_1 and as_4 tomato mutants (Moens 1969), the cross-over frequency in the distal region of chromosome 1 (nucleolar organizer) is not altered.

4.2.3 Increased recombination. Certain synaptic genes partially inhibit synapsis but considerably enhance crossing-over in the synapsed regions of the homologues. This occurs in some synaptic mutants of maize in which recombination per cent between marked genes in the distal region of the short arm and in the centromere region increases appreciably (Rhoades 1947; Rhoades and Dempsey 1949; Dempsey 1958, 1959; Miller 1963). A similar generalized increase in recombination occurs in synaptic mutants of *Lolium* and maize (Omara and Hayward 1978; Sinha and Mohapatra 1969). In tomato, different synaptic mutant genes differentially affect the recombination between marked genes located on different chromosomes. Thus while a synaptic mutant gene as_1 has no effect on recombination between the genes on chromosome 2, a 2.4 fold increase in recombination occurs between three genes in the mutant having the synaptic mutant gene, as_4. Another synaptic mutant gene as_b increases recombination differently between some major genes. These synaptic mutant genes also differ in increasing recombination in the distal regions of chromosome 2. This

Table 3. Influence of synaptic mutant genes[a] on recombination

Plant species	Authors	Genotype	Effect on recombination
Hordeum vulgare (2n = 14)	Enns and Larter 1962	*ds/ds*	Two-fold reduction between marked loci on chromosome 2
Lycopersicon esculentum (2n = 24)	Soost 1951	as_1/as_1 as_4/as_4	Normal level in *ds-wo* region of chromosome 1
		as_1/as_1	No effect on marked loci on chromosome 2
	Moens 1969	as_4/as_4	Two-fold increase between *d-aw-wv* genes
		as_b/as_b	1.5 times increase between *ds-wv* genes and 2.3 times between *d-wv* genes
Zea mays (2n = 20)	Beadle 1933; Dempsey 1958, 1959; Miller 1963; Rhoades and Dempsey 1949	*as/as*	Normal level or increase in *Ws3-Lg-Gl*$_2$ of chromosome 2S, *C-Sh* and *Sh-Wx* segments of 9S and the *A*$_2$*-Bt-Pr* region of chromosome 5
	Nel 1973, 1979	*as/as*	Reduction in the *Gl*$_6$*-Lg*$_2$*-A* region of chromosome 3. Reduction is more in male than in female flowers
	Nel, 1973, 1979	*As/as*	Reduction in *Gl*$_6$*-Lg*$_2$*-A* interval of chromosome 3 and *A*$_2$*-Bt-Pr* region of chromosome 5

[a] Monogenic recessive

increase is different in the different synaptic mutants, but each synaptic mutant gene has a certain capacity for increasing the recombination frequency between a specified set of genes (Moens 1969).

4.2.4 Chiasma frequency and distribution. Chiasma formation and terminalization is under genetic control. Genes causing defects in recombination are basically of two types (Baker et al. 1976): (a) those reducing chiasma frequency per cell but not affecting chiasma distribution, e.g. *Crepis* (Richardson 1935), *Vicia faba* (Sjödin 1970), *Lycopersicon esculentum* (Soost 1951), pearl millet (Murthy 1982) etc. In them, chiasma distribution is directly proportional to the length of the chromosome; (b) those altering chiasma distribution irrespective of the reduction in chiasma frequency, e.g. rye (Prakken 1943), *Vicia faba* (Sjödin 1970), pea (Koller 1938), *Oenothera* (Catcheside 1939), wheat (Li et al. 1945), maize (Beadle 1933; Miller 1963), tomato (Soost 1951; Moens 1969) and pine (Runqvist 1968). In a synaptic mutant of rye (x=7) which has three acrocentric chromosomes, all 14 chromosome arms have the same probability of forming chiasmata (Prakken 1943). Thus, the short arms possess an equal capacity to generate chiasmata as the long arms. When only a few chiasmata occur, they are in fact localized in the short instead of the long arms. This is in contrast to the situation in normal rye plants where the chiasma formation is more frequent in longer than in short arms. Similarly in a synaptic *V. faba* mutant, chiasma frequency is equal in all six bivalents (Sjödin 1970) though the plant has a single very long, and five short, chromosome pairs. Thus, reduction has occurred in the long rather than in the short chromosomes. A nearly similar situation occurs in the other species mentioned above. Alteration in chiasma localization and distribution brought about by the mutant genes indicates that both chiasma frequency and its distribution and localization are gene controlled. In a meiotic mutant of rye, although chiasma frequency is normal, its distribution is completely random among cells, within chromosomes and along bivalents (Jones 1967). A high positive correlation occurs between those (Jones 1974) suggesting a common genetic control. In contrast, in *Lolium perenne,* the three components of chiasma distribution (i.e. between cell variance, within cell variance and distribution along bivalents) are not correlated (Karp and Jones 1982; Jones and Karp 1983). This has led to the proposition of two levels of control on chiasma formation and distribution. The chiasma frequency, between and within cell variance and distribution of chiasmata within bivalents are interrelated by a system which operates at a common level (level I) of control. Mutation in level I results in changes in all three components, as happens in the distributional mutant of rye. When level I is normal, mutation in level II affects only one of the components. Inbreeding exposes the effect of the recessive alleles affecting both levels of control. Increase in recessive homozygosity at level I causes a reduction in chiasma frequency and enhances cell and bivalent variance. Level II operates as a regulatory system and causes different patterns of variations in the chiasma components (Karp and Jones 1982). Thus a major and a polygenic system appears to control formation and distribution of chiasmata in higher plants.

4.3 Chromosome specific control

The examples of non-random participation of chromosomes in bivalent formation and the non-random distribution of chiasmata over the different chromosomes provide clear indication of some degree of bivalent autonomy in chiasma formation. In other words, there is an indication of different genetic controls over different chromosomes of the complement. An unequivocal demonstration of this phenomenon is provided by the gene controlled chromosome specific chiasma variation seen in *Hypochoeris radicata* (Parker 1975) and *Crepis capillaris* (Tease and Jones 1976). For instance, in *H. radicata,* a synaptic mutant gene inhibits chiasma formation in chromosome pair IV in all the PMCs uniformly. In the other three chromosome pairs, the chiasma frequency is increased probably due to a compensatory effect. Thus the total chiasmata per cell equal the norm. In *C. capillaris,* three non-allelic mutant genes affect three different chromosomes in different plants. Unlike in *H. radicata,* no interchromosomal chiasma compensation occurs in these mutants. Thus total chiasma frequency is reduced in them.

These above mentioned examples indicate the existence of two different levels of genetic control over chiasma formation, viz. a) genes controlling chiasma building of the chromosome complement and b) genes controlling chiasma formation of single specific chromosome pairs. Since both systems have not been detected conjointly in most of the investigated species, it appears that numerous major genes condition synapsis and chiasma formation of all the chromosomes, and only a few genes are chromosome specific. This needs to be investigated further.

4.4 Cytoplasmic anomalies

Cytoplasm constitutes the medium for cell divisional activities. Topographical alterations in it lead to abnormal cytokinetical activities. This is known to occur in a meiotic rice mutant (MM-22) in which vacuolation of the cytoplasm occurs during diakinesis and persists until microspore formation (Kitada et al.

1983). This vacuole pushes the chromosomes as well as the spindle apparatus to one side of the PMC. Whereas chromosomal behaviour is normal, cytokinesis is incomplete in the vacuolar area of the cell, and about half of the pollen grains possess the vacuole and degenerate.

4.5 Chromosome coiling

Chromosome coiling, an event necessary for proper synapsis, chiasma terminalization and disjunction of homologues of A I, is also under genetic control. This is evidenced by mutant genes that either decrease or increase the degree of chromosome coiling. In certain mutants of *Matthiola incana* (Frost 1919; Lesley and Frost 1927; Philip and Huskins 1931), chromosome coiling is decreased. In these the chromosomes are relatively uncoiled during meiosis and thus are long and elongated at M I. Male meiosis proceeds normally in these mutants. Whereas there is no alteration in chiasma frequency in *Matthiola,* in maize (Rhoades and Dempsey 1966) and barley (Burnham 1946) it is decreased. In a mutant *'el'* (elongated) microsporogenesis probably proceeds normally but female meiosis II is totally absent in the majority of the cells. In about 20% of the cells even the first meiotic division is absent. This anomaly leads to the production of 2n eggs. This mutant shows increased recombination near the centromeric regions (Nel 1975) in both the sexes.

Unlike some genes which decrease chromosome contraction, others enhance chromosome coiling, disturb the meiotic course and impair gametophytic fertility. In an atomic bomb irradiation induced barley mutant (Moh and Nilan 1954) the chromosomes are abnormally short and univalence is high at MI. Further meiotic divisions are abnormal. In a mutant of *Lathyrus odoratus* (Upcot 1937) bivalents are short and chiasmata are terminalized completely, leading to total pollen sterility. A similar mutant in *Pisum sativum* was obtained in pea after 5 kR γ-ray treatment (Kaul, unpublished). The anomaly in all these mutants is conditioned by single recessive genes. It appears that the genes increasing chromosome coiling do not necessarily inhibit chiasma frequency. This is evidenced in some *Alopecurus* populations (Johnsson 1944). In these populations chiasma frequency is nearly similar although some progenies have chromosomes with reduced chromosome coiling and others have increased chromosome coiling. Different fertile segregants of *Lolium perenne* are known to differ both in the chromosome size and chiasma frequency per cell (Thomas 1936).

4.6 Chromosome stickiness

Some mutant genes cause chromosomal stickiness in *Zea mays* (Beadle 1932, 1937; Golubovskaya 1977), rye (Sosnikhina 1973), *Collinsia tinctoria* (Mehra and Rai 1970) and *Alopecurus myosuroides* (Johnsson 1944). Except in *Alopecurus,* in which the anomaly is conditioned by two recessive genes s_1 and s_2, in others the control is by single recessive genes. These genes may act either during prophase I or M I. Except in the mutant *mei-025* of maize (Golubovskaya 1977), chromosomal stickness is usually followed by extensive chromosome fragmentation, spindle anomalies and abnormal meiosis. In *Collinsia* (Mehra and Rai 1972), the sticky mutant c^{st} is allelic to a desynaptic gene c^{ds}. In safflower, *Carthamus tinctorius,* three major genes *A, B, C* are responsible for non-specific stickiness of chromosomes at M I. Plants with genotype *A-, B-, C-* are normal and those with *aa, bb, C-* or *aa B-* or *aa bb cc* are sterile indicating epistatic gene interaction between genes *B* and *C*. These genes act by delaying the meiotic timing sequence in the sterile plants (Carapetian and Knowles 1976; Carapetian and Rupert 1977).

4.7 Chromosome breakage and defective repair

Chromosomal integrity is a basic requisite for the manoeuvrability of chromosomes during cell division. Breakdown of integrity results in disruption and alteration of linkage groups and loss of important genetic material, thereby causing lethality in many instances. Genetic control of chromosomal integrity is evidenced from the isolation of mutants specifically affecting the chromosomal integrity, feebly or drastically. Minor alterations are not detectable at the submicroscopic level, but the major ones are visualised through alterations of chiasma configurations. Instead of the usual X-type, U-type chiasma formation occurs in such mutants. In some of them A I bridges, acentric fragments and laggards not associated with inversion heterozygosity occur (Lewis and John 1966). Many such genes cause breakages at chromosomal, chromatid or subchromatid levels. In some, reunions follow breakages and in others reunions are either delayed or absent. This is evidenced from the account to follow.

In a rye (Jones 1968, 1969) and some pea mutants (Klein 1969b; Klein and Baquar 1972), mutant gene induced chromosome breakages and irregular reunions occur during meiosis. Such anomalies are coupled with the formation of U-type chiasmata indicating that the chiasmata anomaly is the cause of bridges and fragments. In these mutants, the chromosomal breakages followed by irregular reunions lead to the anomalous chiasma building and to the subsequent chromosomal bridges, fragments and laggards. In *Triticum,* a mutant gene *fr* (Smith 1936) causes extremely high non-localized chromosome fragmentation so that up to 70 fragments occur at A I. Further meiosis is stopped in it. On the other hand, in a similar pearl millet mutant *mbr*

(meiotic breakage) (Murthy 1982; Koduru et al. 1982) meiosis continues until to the end although the mutant gene causes a variable degree of chromosome fragmentation during pachytene. The fragmentation is non-random among bivalents. The meiotic stages are diffuse and asynchronous throughout the entire spike even after its emergence in this mutant whereas in normal plants meiosis is confined to a particular segment of the spike while it is completely enclosed in the boot leaf. Thus the sequence of the meiotic course is delayed. Another interesting feature of this mutant is that in the plants with partial chromosome fragmentation, different chromosomes differ in their fragmentation capacity (Table 4). On the other hand, in PMCs with extreme chromosome fragmentation, the chromatin is shattered. In some cells, univalence of variable degree occurs. Such cells undergo meiosis and complete it much prior to the ones having fragments where meiotic completion is delayed considerably. Callose wall formation is triggered around all cells after microspore formation by the normal PMCs. Since the meiotic course is considerably delayed in the meiocytes having fragments, callose wall deposition occurs around meiocytes that have different meiotic stages. Thus giant cells are

Table 4. Mutant genes[a] causing chromosome breakage

Species	Authors	Origin
Allium cepa (2n = 16)	Koul 1962	sp
Brassica campestris (2n = 20)	Stringam 1970	sp, ethyleneimine
Collinsia tinctoria (2n = 14)	Mehra and Rai 1970	X-ray
Pennisetum americanum (2n = 14)	Koduru et al. 1982; Lakshimi et al. 1979; Murthy 1982; Rao and Koduru 1978	sp
Pisum sativum (2n = 14)	Klein 1969b, 1970; Klein und Baquar 1972	X-ray
Scilla sibirica (2n = 12)	Rees 1952	sp
Secale cereale (2n = 14)	Jones 1967, 1968; Rees 1962	sp, X-ray
Sorghum purpureo-sericeum (2n = 10)	Magoon et al. 1961	sp
Triticum sps. (2n = 28)	Smith 1936	sp
Zea mays (2n = 20)	Beadle 1937	sp

[a] Monogenic recessive

Table 5. Mutant genes[a] affecting M I and M II spindles

Plant species	Gene symbol	Authors	Stage of action
Agropyron cristatum (2n = 14)	*dy*	Tai 1970	M I
Alopecurus myosuroides (2n = 14)	–	Johnsson 1944	M II
Clarkia exilis (2n = 14)	*m*	Vasek 1962	M I, M II
Cymopsis tetragonoloba (2n = 14)	–	Sohoo and Gill 1975	M I
Pennisetum orientale (2n = 14)	–	Jauhar and Singh 1969	M I
Pisum sativum (2n = 14)	–	Klein 1969a	M I
Ribes nigrum (2n = 16)	–	Vaarama 1949	M I
Solanum commersonii (2n = 24)	sy^2	Johnston et al. 1981	M I
	–	Hanneman and Rheude (see Peloquin 1982)	M I, M II
Solanum phureja (2n = 24)	*ps*	Mok and Peloquin 1975a, b	M II
Suaeda fruticosa (2n = 36)	–	Malick and Tandon 1960	M I
Tradescantia sp. (2n = 12)	–	Celarier 1955	M I
Zea mays[b] (2n = 20)	*dy*	Clark 1940	M I
	ms-43	Golubovskaya 1977	M I

[a] Monogenic recessive
[b] Sex specific

also formed along with pollen grains. This gene does not disturb the physiological development of the anthers inferring thereby a different genetic control of traits. The normal occurrence of premeiotic mitosis in this mutant indicates that the gene action is restricted only to meiosis. Certain synaptic mutants also show extreme nonlocalized meiotic chromosome breakage (Table 4).

4.8 Spindle form and function

Both spindle organization and its function are under genetic control (Table 5), the genes are of divisional-type-specific and/or sex-specific. Some mutant genes completely suppress spindle formation and others hinder its normal development. Complete spindle absence occurs in some mutants of *Tradescantia* (Celarier 1955), *Suaeda fruticosa* (Malick and Tandon 1960), *Artemisia maritima* (Koul 1965), *Pennisetum orientale* (Jauhar and Singh 1969), *Zea mays* (Golubovskaya 1977), etc. In these mutants, segregational anomalies or the complete blockage of meiosis occurs so that no fertile gametes are produced. Some mutant genes induce defects in spindle development which leads to the formation of uni- and multi-polar spindles in addition to some normal bipolar spindles, e.g. *Zea mays* (Beadle 1933), *Artemisia vulgaris* (Koul 1964), *Collinsia tinctoria* (Mehra 1975), pearl millet (Murthy 1982) etc. These anomalies lead to chromosome segregations of more than two poles, and as a result micronuclei and laggards are formed. In *Zea mays* (Beadle 1933; Baker and Morgan 1969) and *Pennisetum* (Krishnaswamy et al. 1949; Koduru 1980) such genes lead to the formation of crescent shaped spindles.

That the spindle mechanism in micro- and mega-sporogenesis may be under separate genetic control is inferred from the existence of sex specific mutants in maize and potato. In maize, a mutant gene *dv* (Clark 1940) causes spindle divergence, aberrant chromosome contraction and movements in some PMCs. Spindle divergence leads to more than two chromosome groups. At A II each group functions independently and a multispored pollen grain results. This mutant gene is male sex specific and it acts only on the microsporogenesis and only a little stainable pollen is produced by this mutant. Megasporogenesis and female fertility are normal in this mutant. Another similar mutant gene *ms-43* induces complete male sterility without impairing female fertility in maize (Golubovskaya 1977). On the other hand, a mutant gene in *Agropyron cristatum* affects M I spindle only and causes multipolar spindles in micro-as well as in mega-sporogenesis (Tai 1970).

4.8.1 Divisional specificity. The formation and proper functioning of M I and M II spindles is a prerequisite for normal meiotic completion. Whereas the M I spindle is meiotic and thus biaccuminate, the M II spindle is mitotic and barrel-shaped. That these two divisions are under independent genetic control is evidenced by the isolation of meiotic mutants wherein the M I spindle is abnormal but the M II spindle is normal. This is known in *Clarkia exilis* (Vasek 1962) and *Pisum sativum* (Klein 1969 a) in which two spindles instead of one occur at M I. The M II spindle formation and subsequent divisional processes are normal. In a mutant of *Artemisia vulgaris,* the M II spindle is either absent or poorly developed (Koul 1964). M I chromosomal congregation is poor and no anaphase separation of chromosomes occurs. The cells form restitution nuclei and no viable pollen is ever produced by the mutant. In a *Solanum commersonii* mutant, both M I and MII spindles are absent and diploid spores with the parental genotype are formed (Hanneman and Rheude, see Peloquin 1982). In potato, two non-allelic mutant genes pc_1 and pc_2 cause second division restitution by premature cytokinesis after first meiotic division (Mok and Peloquin 1975 a) and the second meiotic division is absent. Whereas gene pc_1 causes irregular A I, with the subsequent disintegration of the chromatids at telophase I and cytokinesis after telophase I, the mutant pc_2 does not possess the first two characteristics, but cytokinesis occurs in this mutant at prophase II (Mok and Peloquin 1975 b). The authors have isolated another apparently allied mutant gene designated as pc_3. Another mutant gene *sy* causes a complete first division restitution after an abnormal first division followed by a normal second division, resulting in two 2n eggs (Iwanaga and Peloquin 1979, 1980). A mutant gene *va* of maize, located on chromosome 7, causes the absence of cytokinesis after A I or A II, thereby leading to several chromosomal anomalies and partial to complete gametic sterility (Beadle 1932). In a meiotic mutant (MM-23) of rice, incomplete cytokinesis after normal first and second meiotic divisions leads to interconnected pollen grains and to nearly total pollen and seed sterility (Kitada et al. 1983).

4.9 Second division

Second meiotic division, a mitotic type of cell division, differs from first division in having a lower chromosome number, recombined gene sequences and barrel-shaped spindles at metaphse. This division is likewise influenced by certain mutant genes. For instance, in *Datura stramonium,* the gene *dy* causes the absence of the second meiotic division (Satina and Blakeslee 1935). After a normal first division, the PMCs undergo a prolonged interphase during which post-meiotic chromosome replication occurs. This is followed by division simulating pollen mitosis and diploid pollen are form-

ed. In a potato mutant *ps*, first division restitution is caused by parallel spindles at meiosis II (Mok and Peloquin 1975 a, b). As a result 2n microspores are produced following cytokinesis. Whereas the gene *ps* causes first division restitution, another mutant gene causes second division restitution through suppression of cytokinesis leading to 2n spore formation (Mok and Peloquin 1975 b). In an alfalfa mutant (Vorsa and Bingham 1979), a single recessive gene controls formation of parallel spindles at second meiosis and produces 2n pollen grains. In some other alfalfa mutants (Pfeiffer 1982), 2n egg production is caused by the normal occurrence of both meiotic divisions accompanied by the absence of cytokinesis after A II.

5 Post-meiotic mutants

Unlike *ms* mutant genes, the majority of which impair post-meiosis, relatively few meiotic mutant genes influence post-meiotic events. Such mutant genes do not influence the meiotic course but the postmeiotic spore developmental stages. For instance, in the 'polymitotic' maize mutant, supernumerary divisions in microspores continue until one chromosome is left in a cell. The cells finally degenerate and no viable male gametes are formed by this mutant. The anomaly is conditioned by a single recessive gene '*po*' (Beadle 1929) located on the satellite of chromosome 6. Another maize mutant *ms-4* allelic to *po* also causes premature post-meiotic mitoses (Golubovskaya and Urbach 1981).

6 Genetic characterization of meiotic mutant genes

6.1 Impaired gametic fertility

Harmonious and the normal course of meiosis ensures gametic fertility. Control and coordination of he meiotic process depends in turn upon the regulated action of a large number of genes, most of which are dominant and non-allelic. This becomes apparent from such cytogenetically well-analysed diploids as maize, barley, tomato, pea, etc. In these, each meiotic gene acts precisely over a specific meiotic event and mutation of any of these genes impairs steps of a highly coordinated meiotic process and reduces the gametic fertility. The majority of the meiotic mutations are recessive (Table 2) and the mutant alleles exhibit variable degrees of penetrance and expressivity (Kalia 1962; Gottschalk and Kaul 1974). The gene action is influenced by genotype (G), environment (E) and G×E interaction. Dominant meiotic mutant genes are few and known in *Crepis* (Hollingshead 1930) and *Phleum* (May and Kasha 1980). A digenic recessive control is known in wheat (Smith 1936) and cotton (Beasley and Brown 1942; Menzel and Brown 1955; Weaver 1971). Duplicate recessive gene interaction occurs in *Triticum* (Hayter and Riley 1967) and *Fragaria* (Reighter and Jelenkovic 1979). In a dioecious *Rumex acetosa* (Löve 1943), the Y-linked inheritance of asynapsis and male sterility is known.

Multiple site regulation of chromosome behaviour, form and function in bread wheat is evidenced by the existence of pairing suppressors as well as promotor genes on different homoeologous and homologous chromosomes (Table 6). For normal homologous pairing a specific balance of these genes is necessary. The presence of such a high number of closely interacting genes may be due to the polyploid nature of this crop. Similar chromosome pairing control systems operate in such allopolyploids as *Avena* (Gauthier and McGinnis 1968; Rajathy and Thomas 1972), *Festuca* (Jauhar 1975), tobacco (Kimber 1961), *Bothriochloa* (Chheda and Harlan 1962) and *Chrysanthemum* (Watanabe 1983).

6.2 High mutation rate

The presence of a large number of spontaneously arisen meiotic mutant genes indicates their high mutational rate and control of meiosis by a large number of such genes. The majority of these meiotic mutant genes impair homologous synapsis and/or chiasma formation. Thus, nearly 130 species belonging to 100 genera of higher plants exhibit synaptic mutations (Table 2, also see (Gottschalk and Kaul 1980 a, b; Koduru and Rao 1981). Compared to others, the detection of these mutants is easier as they cause distinct cytological anomalies, disturb meiosis and reduce gametic fertility. On the other hand, the influence of other types of meiotic mutant genes over meiosis and fertility is feeble so that they pass undetected and are either shed off or retained depending upon their usefulness to population-fitness and survival-value. Another genetic feature of these meiotic mutant genes is their high rate of non-allelic mutations. Whereas allelic mutations are hard to differentiate, non-allelic mutations have been identified. Thus, over 15 loci have been assigned to the synaptic mutants in barley (Ramage and Eckhoff 1981), five each in tomato (Soost 1951; Moens 1968, 1969) and soybean (Hadley and Starnes 1964; Palmer 1974; Palmer and Kaul 1983), three each in *Brassica campestris* (Stringam 1970) and pearl millet (Murthy 1982).

6.3 Polygenic nature

The meiotic mutant genes predominantly are major genes with distinct cytogenetic effect and a precise action on sporogenesis. The majority of these are recessive although a few are dominant. However, some

Table 2. Synaptic mutant genes in higher plants

Plant species	Authors	Origin
Aegilops triaristata (2n = 28)	Lacadena and Piqueras 1971	sp
Allium ascalonicum (2n = 16)	Darlington and Haque 1955	sp
A. cepa (2n = 16)	Konvička and Gottschalk 1974	sp, γ-rays
Alopecurus myosuroides (2n = 14)	Johnsson 1944	sp
Arachis hypogea (2n = 40)	Patil and Mouli 1977	X-ray
Avena sativa (2n = 42)	Thomas 1973	sp
Avena strigosa (2n = 14)	Dyck 1964; Dyck and Rajathy 1966	sp
A. abyssinica × *A. barbata* (2n = 28)	Thomas and Rajathy 1966	sp
Bothriochloa hybrids (2n = 40)	Chheda and DeWet 1961	sp
Brassica campestris (2n = 20)	Stringam 1970	sp, Ethyleneimine
B. oleracea (2n = 18)	Gottschalk and Konvička 1971, 1972; Kinvička and Gottschalk 1971	γ-ray
Capsicum annuum (2n = 24)	Rajarao and Aniel Kumar 1983; Sadanandam and Subhash 1983	sp, Colchicine
Cassia tora (2n = 26)	Katayama 1953	Atom bomb
Citrullus vulgaris (2n = 22)	Kihara and Saito 1972	γ-ray
Collinsia tinctoria (2n = 14)	Rai 1967; Mehra and Rai 1972	X-ray
Corchorus olitorius (2n = 14)	Mitra and Singh 1971; Paria and Basak 1980; Paria et al. 1978	EMS, X-ray sp
Crepis capillaris (2n = 6)	Hollingshead 1930; Tease and Jones 1976	sp
C. capillaris × *C. tectorum*[a] (2n = 6)	Hollingshead 1930; Richardson 1935	sp
Cyamopsis tetragonoloba (2n = 14)	Sohoo and Gill 1975	sp
Datura stramonium (2n = 24)	Bergner et al. 1934; Blakeslee 1928; Blakeslee and Avery 1934	sp
Eleusine coracana (2n = 36)	Seetharam et al. 1975	EMS
Fragaria annanasa[b] (2n = 56)	Reighter and Jelenkovic 1979	sp
Glycine max (2n = 40)	Hadley and Starnes 1964; Palmer 1974; Palmer and Kaul 1983; Winger et al. 1977	sp
Gossypium arboreum (2n = 26)	Ramiah and Gadkari 1941	sp
G. gossypoides[b] (2n = 52)	Menzel and Brown 1955	sp
G. hirsutum[b] (2n = 52)	Bahvandas and Veluswamy 1968; Brown 1948; Hutchinson and Gadkari 1935; Weaver 1971	sp
G. hirsutum × *G. barbadense*[b] (2n = 52)	Beasley and Brown 1942	sp
Hordeum sativum (2n = 14)	Moh and Nilan 1954; Sethi et al. 1970	Atom bomb
H. jubatum (2n = 14)	Wagenaar 1960 b	sp
H. vulgare (2n = 14)	Burnham 1946; Enns and Larter 1960; Kasha and Walker 1960; Ramage and Eckhoff 1981; Sharma and Reinbergs 1974; Srivastava 1974; Tyagi and Das, 1975; Wagenaar 1964	sp, X-ray, γ-ray, EMS
Hypochoeris radicata (2n = 8)	Parker 1975	sp
Lens culinaris (2n = 14)	Sinha 1980	sp
Liatris ligulistylis (2n = 20)	Gaiser 1950	sp
Lolium perenne (2n = 14)	Ahloowalia 1969, 1972; Omara and Hayward 1978	sp
Lycopersicon esculentum (2n = 24)	Kalia 1962; Moens 1968, 1969; Soost 1951	sp
Matthiola incana (2n = 14)	Armstrong and Huskins 1934; Lesley and Frost 1927	sp
Nicotiana rustica × *N. tabacum* (2n = 48)	Swaminathan and Murty 1959	sp

Table 2 (continued)

Plant species	Authors	Origin
N. sylvestris (2n = 24)	Goodspeed and Avery 1939	X-ray
N. tabacum (2n = 48)	Clausen and Cameron 1944; Swaminathan and Murty 1959	sp
Oenothera erythrina (2n = 14)	Catcheside 1939	sp
Oryza sativa[b] (2n = 24)	Chao et al. 1960; Katayama 1961; Kitada et al. 1983; Misra and Shastry 1969; Ramanujam and Parthasarthy 1935; Ratho and Misra 1973; Wang et al. 1965	sp, EMS, X-ray, Thermal Neutrons
Pelargonium crispum (2n = 18)	Tokumasu 1974	sp
Pennisetum americanum (2n = 14)	Dhesi et al. 1973; Minocha et al. 1975; Murthy 1982; Pantulu and Subbarao 1976; Subbarao 1976, 1978, 1980	sp, colchicine
Phaseolus mungo (Vigna mungo) (2n = 22)	Goswami 1980; Jana 1962; Kumar and Gupta 1978	γ-ray, X-ray
Phleum nodosum[a] (2n = 14)	May and Kasha 1971, 1980	sp
Pisum sativum (2n = 14)	Ezhova et al. 1977; Gostimsky 1976; Gottschalk 1968; Gottschalk and Pietrini 1965; Gottschalk and Baquar 1971; Gottschalk and Konvička 1975; Gottschalk and Klein 1976; Gottschalk and Kaul 1980a, b; Klein 1969a, 1970; Klein and Milutinovic 1971; Koller 1938	sp Neutrons X-ray
Raphanus sativus (2n = 18)	Dayal 1977	sp
Rumex acetosa (Y-linked, 2n = 12 + XY_1Y_2)	Löve 1943	sp
Secale cereale (2n = 14)	Prakken 1943; Kolobaeva 1974	sp
Solanum commersonii (2n = 24)	Johnston et al. 1981	sp
S. phureja (2n = 24)	Okuwagu and Peloquin 1981; Peloquin 1982	sp
S. tuberosum (2n = 48)	Iwanaga and Peloquin 1979, 1980	sp
Sorghum purpureo-sericeum (2n = 10)	Magoon et al. 1961	sp
S. almum × *S. vulgare* (2n = 40)	Pritchard 1965; Ramulu 1970	sp, X-ray
Sorghum hybrids (2n = 20)	Franzke and Ross 1952; Ross et al. 1960	sp, X-ray
S. subglabrens (2n = 20)	Krishnaswamy and Meenakshi 1957	X-ray
S. durra × *S. subglabrens* (2n = 20)	Krishnaswamy and Meenakshi 1957; Krishnaswamy et al. 1958	sp
S. vulgare (2n = 20)	Stephens and Schertz 1965	–
Tradescantia sp. (2n = 12)	Celarier 1955	sp
Triticum aestivum (2n = 42)	Okamoto 1963; Pao and Li 1948; Sears 1952; Wagenaar 1960a; Zhirov et al. 1973	sp
T. aestivum (duplicate genes)	Hayter and Riley 1967	sp
T. durum (2n = 28)	Bozzini and Martini 1971; Martini and Bozzini 1966	sp, X-ray
Triticum sp. (2n = 28)	Li et al. 1945; Zchege 1963	sp
T. monococcum (2n = 28)	Smith 1936	sp
Vicia faba (2n = 12)	Sjödin 1970	EMS, neutrons, γ-rays
Zea mays (2n = 20)	Beadle 1933; Baker and Morgan 1969; Burnham 1963; Golubovskaya 1977; Golubovskaya and Mashnenkov 1976, 1977; Golubovskaya and Urbach 1981; Miller 1963; Nelson and Clary 1952; Powers and Dahl 1937; Sinha 1967	sp, NMU, EMS, X-ray

[a] One dominant and [b] two recessive; in the remaining (including rice and cotton) monogenic recessive gene control; sp = spontaneous

Table 6. Genes regulating chromosome pairing in bread wheat and related species

Plant species	Pairing suppressor genes	Authors
T. aestivum (2n = 40)	3AS	Driscoll 1972; Mello-Sampayo and Canas 1973
	3DS	Driscoll 1972; Mello-Sampayo 1968, 1971 a; Mello-Sampayo and Canas 1973; Riley et al. 1960; Upadhya and Swaminathan 1967
	4D	Driscoll 1973
	5BL	Driscoll et al. 1970; Feldman 1966 a, 1968; Feldman and Avivi 1973; Feldman et al. 1966, 1972; Okamoto 1957; Riley 1960, 1968; Riley and Chapman 1958, 1964; Sears 1975, 1976; Upadhya and Swaminathan 1967; Wall et al. 1971
T. timopheevi (2n = 28)	–	Feldman 1966 b
Secale cereale (2n = 14)	5RL	Riley et al. 1973
	Pairing promoter genes	
T. aestivum (2n = 42)	2AS	Riley et al. 1960; Sears 1954
	2B, 3B	Zhirov et al. 1973
	3AL	Mello-Sampayo and Canas 1973
	3BL	Kempanna and Riley 1962; Sears 1954
	3DL	Driscoll 1972; Mello-Sampayo and Canal 1973; Mello-Sampayo 1971 a
	5AL	Feldman 1966 a, 1968; Riley et al. 1966
	5BS	Riley and Chapman 1967
	5D	Bayliss and Riley 1972; Feldman 1966 a, 1968, 1971; Hayter and Riley 1967; Riley 1966; Riley et al. 1966; Viegas et al. 1980
T. speltoides (2n = 14)	–	Riley et al. 1961
T. tripsacoides (2n =)	–	Riley and Law 1965
Aegilops longissima (2n = 14)	–	Mello-Sampayo 1971 a

genes exhibit a polygenic inheritance and action. These mainly influence the degree of pairing and chiasma formation. Such genes occur in rye, *Lolium*, *Sorghum* and pearl millet. These being outbreeders, conserve such polygenic meiotic mutant genes in a heterozygous state in their genome but remain fertile because of the low number of such polygenes. Upon inbreeding, their number increases and dosage effect appears. They express their increased effect with each generation of selfing. In wheat, an inbreeder, the reverse occurs. Here the meiotic mutant genes are expressed after the species is outbred. Thus probably a positive interchromosomal nonallelic genetic interaction of the type homozygous × homozygous in the inbreeders and heterozygous × heterozygous in the outbreeders maintains chromosome pairing and chiasma frequency in these plant populations. A breakdown in this balance may also occur following meiotic mutations or by reversals imposed in their normal breeding system. These cause anomalous meiosis and gametic fertility reductions. Accordingly, alterations in the breeding system may counteract the evil effect of meiotic mutations and vice versa. This needs investigation.

6.4 Time and site specificity

One of the major genetic properties of meiotic mutant genes is their time and site specificity. Both these are highly interdependent since meiosis is a continuous process with an extreme precision of timing and sequencing. Thus, meiotic mutant genes act specifically at certain meiotic stages and act either on the chromosome form or its function. For instance, whereas asynaptic genes inhibit chromosome synapsis during early prophase, desynaptic genes suppress chiasma building during pachytene-diplotene. Some meiotic mutant genes do not influence chromosomes but spindle development, whereas others prevent cytokinesis and cell division. Some other genes in maize, barley and pea effect MI spindle while others affect MII spindle. Both gene types impair gametic fertility almost equally.

6.5 Sex-specificity

Some meiotic mutant genes exhibit extreme sex-specificity, the most common ones being those causing either male or female sterility (Gottschalk and Kaul 1974). Whereas the female sterile (*fs*) mutants are difficult to

perpetuate, male sterile (*ms*) mutants are maintained through natural cross pollinations and segregations. This has led to the detection of a large number of *ms* mutants compared to *fs* mutants (Gottschalk and Kaul 1974; Gottschalk and Klein 1976; Frankel and Galun 1977).

6.6 Chromosome specificity

The majority of meiotic mutant genes influence entire chromosomal complements nearly equally. But in *Hypochoeris radicata* (Parker 1975) and *Crepis capillaris* (Tease and Jones 1976), specific genes exist that control pairing behaviour of certain chromosomes. Such genes are rare and exist in addition to the usual meiotic mutant genes in a genome.

7 Site of genetic recombination

Recombination nodules (RN) are the dense round or elliptic or irregular bodies associated with the central element of SC. They exhibit a high correlation with frequency, and/or the distribution of chiasmata has been observed in several organisms (Carpenter 1979). In rye, except for NOR regions, all the bivalent arms have at least one large RN at pachytene (Abirached-Darmency et al. 1983). By diakinesis both chiasmata along the bivalents and RN are mostly distally localized indicating a high correlation between distribution of chiasmata and RN along the bivalents. In plants meiotic mutants have not been used to test this relationship. But three recombination defective mutants of *Drosophila* have been used in such experiments (Carpenter 1979). Two of the three mutants *mei-218* and *mei-41* have low and randomly distributed RN. The third mutant *mei-9* has the similar number and distribution of RN as in the wild type. Carpenter (1979) has suggested that either RN are not functional in the mei-9 or were not the regions where the recombination takes place. Direct relationship between the constitutive heterochromatic in chiasma formation have been suggested by Loidl (1982). Using *Allium flavum, A. carinatum* and *A. sipyleum,* the author has observed that the chiasmata form close to C-bands in the euchromatic regions. It is assumed that chiasma occurs at repetitive sequences located in heterochromatin. If it is so, it will be interesting to use such comparative chromosome banding and Electron Micrographic studies of normal plants and meiotic mutants to find a correlation between the C-bands or repeated sequences and the RN.

8 Conclusions

I. Meiotic mutant genes provide evidence that meiosis is under the control of a large number of dominant genes whose action is precisely coordinated and highly regulated.

II. Compared to other meiotic mutations, the mutation rate of the genes controlling chiasma development and building is extremely high.

III. The mutations boost up genetic variability in the otherwise inflexible and conservative meiotic system. This results in aneuploid gamete production and highly variable progenies, some of which have enhanced variability (Palmer and Heer 1976; Gottschalk and Milutinovic 1973; Subbarao 1976).

IV. Many allelic and nonallelic meiotic mutant genes affect the same meiotic phase. This occurs in maize (Golubovskaya 1979; Golubovskaya et al. 1980) in which two nonallelic genes act on the same meiotic phase nearly simultaneously or successively. Some isoallelic meiotic mutant genes also exist in the plant genomes of pea, tomato, barley and soybean. Whether allelic meiotic mutations, all affecting the same meiotic stage arise due to mutations in repeated or unique DNA is not known. Compared to *Drosophila* the allelic mutation rate of *Pisum sativum* is very high. This high rate may be due to the presence of 70% repeated DNA in pea genome, whereas *Drosophila* has only 25% repeated sequences.

V. The meiotic mutant genes that prevent crossing-over in combination with genes such as *ps* which causes first division restitution can provide genetic fixity to an organism and desirable recombinations can be maintained uniformly (e.g. in potato; Peloquin 1982). Thus intact germplasm has been transferred from economically useful diploid potato to tetraploid cultivars using meiotic mutant genes.

VI. In wheat, genes for resistance from alien chromosomes to wheat chromosomes were transferred by inducing homoeologous pairing after suppressing the activity of *Ph* or deleting it from the 5B chromosome (Riley et al. 1968; Sears 1973).

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