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Review

Cytogenetics of Synaptic Mutants in Higher Plants

P.R.K. Koduru and M.K. Rao
Department of Botany, Andhra University, Waltair (India)

Contents

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1 Introduction

In all sexually reproducing organisms the homologous chromosomes synapse longitudinally during zygotene and pachytene, forming bivalents. During later stages of prophase I, when this synapsis lapses, bivalents are maintained by the chiasmata until anaphase I, at which time the homologous chromosomes segregate to opposite poles. The zygotene pachytene synapsis seems to be a prerequisite for the crossing-over and chiasma formation. Like several other events of meiosis, homologous synapsis and chiasma formation are known to be under genetic control, and over the last 60 years mutations affecting these events have been reported in 126 species belonging to 93 genera of higher plants (Table 1). The phenomenon of failure of pairing during prophase I is referred to as asynapsis or desynapsis (Darlington 1937). From a review of literature it can be concluded that lack of pairing is found in (1) interspecific or intergeneric hybrids, (2) apomictic lines, (3) individuals carrying a mutation for this character (genotypic abnormality), (4) aneuploids and (5) in plants under physiological stress (Darlington 1937). Unlike in hybrids and apomicts, in the other cases the lack of pairing is not due to lack of chromosome homology but to other factors, and the subsequent restoration of normal factors restores normal pairing.

Among the various kinds of non-pairing, genotypic abnormality has received wide attention from cytologists and geneticists and the present article deals with this aspect. Asynapsis was once broadly used to denote the lack of chromosome pairing during late prophase I stages; its usage now has been restricted to those cases in which there is no pachytene pairing, as was originally proposed by Randolph (1928), and desynapsis is used to denote the falling apart of the synapsed homologues due to their inability to generate or retain chiasmata (Li et al. 1945; Rieger et al. 1976). However, since a distinction is not always possible as some species are not amenable to analysis at pachytene, Riley and Law (1965) suggested that 'synaptic mutants' is a better alternative term to describe the lack of prophase I chromosome pairing.

Prakken (1943) first proposed a classification of the synaptic mutants into weak, medium strong and strong types on the basis of the frequencies of bivalents and univalents in meiocytes. In the

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Table 1. Species of higher plants in which synaptic mutants have been reported (excluding those cited by Katayama 1964)

Species	Origin	Genetics	Author(s)
Aegilops triarisatata	Sp	1R	Lacadena et al. 1971
A. ventricosa	Sp	–	Katayama 1931
Allium cepa	Sp, I	–	Kaul 1975*; Konvička et al. 1974; Koul 1962*; Gohil et al. 1971*
A. consanguineum	Sp	–	Gohil et al. 1971*
A. odorum	Sp	–	Vig et al. 1965
A. sativum	Sp	–	Gohil et al. 1971
A. tuberosum	Sp	–	Gohil et al. 1971*; Mathur et al. 1965
Amorphophallus campanulatus	Sp	–	Magoon et al. 1967*+
A. konjac	Sp	–	Watanabe 1963
Anemone	Sp	–	Moffett 1932*
Apples	Sp	–	Heilborn 1930*
Artemisia douglasiana	Sp	–	Estes 1971*
Avena sativa	Sp	1R	Thomas 1973
Avena strigosa	Sp	1R	Dyck 1964; Dyck et al. 1965; Thomas et al. 1966
Bothriochloa	Sp	1R	Chheda et al. 1961
Brassica campestris	Sp	1R	Stringam 1970*
B. oleracea	I	1R	Gottschalk et al. 1971*; 1972; Konvička et al. 1971
Capsicum annuum	I	–	Katiyar 1977; Ramalingam 1977; Lakshmi et al. 1977*
Chlorophytum laxum	Sp	–	Sheriff et al. 1974+
Citrus aurantifolia	Sp	–	Iwamasa et al. 1963
Clementine mandarin	I	–	Eliseev 1974
Coix lacryma-jobi	Sp	–	Rao 1975
Coleus	Sp	–	Vasudevan et al. 1967*
Collinsia tinctoria	Sp	1R	Mehra et al. 1972+; Rai 1967
Colocasia anti-quorum	Sp	–	Krishnan et al. 1970*
Cyamopsis tetra-gonoloba	Sp	–	Sohoo et al. 1975
Dactylis	I	–	McCollum 1958
Eleusine coracana	I	–	Seetharam et al. 1975
Glycine max	Sp	1R	Hadley et al. 1973; Hadley et al. 1964+; Palmer 1974*
Gossypium arboreum	Sp	1R	Ramaiah et al. 1941
G. hirsutum	Sp	1R, 2R	Bahavandoss et al. 1968; Brown 1948, 1958; Smithson 1974; Weaver 1971
G. gossypioides	Sp	2R	Menzel et al. 1955+
Hieracium	Sp	–	Rosenberg 1917+
Hordeum jubatum	Sp	1R	Wagenaar 1964
Hordeum vulgare	Sp, I	1R	Ramage et al. 1972; Ahokas 1977; Enns et al. 1962; Fedak 1973; Kartel et al. 1977, 1978; Kasha et al. 1960; Manzyuk et al. 1975, 1977; Riley et al. 1966; Scheuring et al. 1975; Sethi et al. 1970; Sharma et al. 1974; Srivastava 1974; Swietlinska et al. 1970; Tyagi et al. 1975; Wagenaar 1960
Hypochoeris radicata	Sp	1R	Parker 1975*
Ipomoea batatas	SP	–	Rajendran et al. 1975
I. gracilis	Sp	–	Jones 1970
Lilium hybrids	Sp	–	Ribbands 1937
Lolium perenne	Sp	1R	Ahloowalia 1969a*, 1969b, 1972; Myers 1945; Omara et al. 1978*
Lycopersicon esculantum	Sp, I	1R	Clayberg 1958+; Het Ram Kalia 1962+; Lamm 1944; Moens 1969+*
Matthiola incana	Sp	1R	Armstrong et al. 1934*; Philip et al. 1931
Nicotiana glutinosa	Sp	–	Shambulingappa 1966
N. tabacum	Sp	1R	Clausen et al. 1944; Swaminathan et al. 1959
Oryza perennis	Sp	–	Shastry et al. 1963
O. sativa	Sp, I	1R	Chao et al. 1960, 1961; Ratho et al. 1973; Yamaguchi 1974; Wang et al. 1965
Pelargonium crispum	Sp	1R	Tokumasu 1974
Paeonia	Sp	–	Hicks et al. 1934
Paspalum commer-sonii	Sp	–	Christopher 1971+; Pi et al. 1974*+
P. conjugalum	Sp	–	Christopher 1971+
P. longifolium	Sp	–	Pi et al. 1974*+
P. secans	Sp	–	Snyder 1961+
Pennisetum orientale	Sp	–	Jauhar et al. 1969
P. ramosum	Sp	–	Jauhar et al. 1971*
P. typhoides (P. americanum)	Sp, I	1R, 2R	Dhesi 1973; Dhesi et al. 1973*; Dhesi et al. 1975; Jauhar

Table 1. (continued)

Species	Origin	Genetics	Author(s)
			1969; Koduru 1980+; Koduru et al. 1978*; Krishna Rao et al. 1978a+, 1978b; Lakshmi et al. 1979*. Lakshmi et al. 1978*; Minocha et al. 1968*, 1975; Pantulu et al. 1976; Patil et al. 1962; Singh et al. 1977; Subbarao 1976*, 1978, 1980
Pepper	I	–	Morgan 1963+
Picea abies	Sp	–	Jonsson 1973*
Pinus sylvestris	Sp	–	Runqvist 1968*
Pisum sativum	Sp, I	1R	Ezhova et al. 1977; Gostimsky 1976; Gottschalk et al. 1971*, 1964, 1976*, 1975+, 1965; Klein 1969a*, 1969b*, 1970+, 1971; Klein et al. 1972*+, 1971, 1972, 1976; Narsinghani et al. 1976
Red clover	Sp	–	Whittington 1958
Ribes nigrum	Sp	–	Vaarama 1949
Rumohra aristata	Sp	–	Bhavanandan 1971
Secale africanum	Sp	–	Singh 1977
S. cereale	Sp, I	1R	Giraldez et al. 1976, 1978; Kolobaeva 1974, Morrison 1956, Rees 1955
S. montanum	Sp	–	Singh 1977
S. vavilovii	Sp	–	Singh 1977
Solanum wendlandii	Sp	–	Chennaveeraiah et al. 1968
Sorghum	I, Sp	1R	Franzke et al. 1952; Pritchard 1965; Ramulu 1970; Ross et al. 1954; Sadasivaiah et al. 1965*
S. purpureosericeum	Sp	1R	Magoon et al. 1961*
S. vulgare	Sp	1R	Stephens et al. 1965+
Suaeda fruticosa	Sp	–	Malick et al. 1960
Tradescantia	Sp	–	Vosa 1961
T. bracteata	I	–	Dowrick 1957
Trifolium pratense	Sp	–	Strzyxewska 1976*+
Triticum	Sp, I	1R	Wagenaar 1960+; Zschege 1963
Triticum aestivum	Sp		Bayliss et al. 1972a; 1972b
T. durum	I	1R	Bozzini et al. 1971*; Martini et al. 1966+
T. vulgare	Sp	1R	Okamoto 1963; Pao et al. 1948; Sears 1952; Zhirov
Tulipa hageri	Sp	–	Couzin et al. 1973*
Uvularia perfoliata	I	–	Dowrick 1957
Vicia faba	I	1R	Sjodin 1970*+
Watermelon	I	1R	Kihara et al. 1972
Zea mays	Sp, I	1R	Baker et al. 1969+; Banu et al. 1972; Burnham 1963; Dempsey 1958, 1959; Golubovskaya et al. 1976, 1977; Maguire 1977a, 1978*; Miller 1963*+; Nel 1973; Sinha, 1967; Sinha et al. 1969
Zebrina pendula	Sp	–	Venkateswarlu et al. 1968

Sp = spontaneous; I = induced; 1R = 1 recessive gene; 2R = 2 recessive genes; * = observed pachytene pairing; + = no pachytene pairing

weak types, only a few univalents are formed in the majority of the meiocytes. Further, these are characterised by high chiasma frequency, low frequency of irregularities at MI and AI and relatively high fertility; they can be maintained as pure lines. In medium strong types, many univalents are formed in most of the cells. Meiotic irregularities will be frequent, leading to relatively high sterility. In complete or strong mutants all chromosomes in most of the meiocytes form univalents very early in meiosis with high frequency of irregularities and almost total sterility. This system of classification may be regarded as arbitrary because the cytological behaviour of the mutants varies greatly in different seasons of an year or under different environmental conditions.

2 Morphology and Cytology

2.1 Diploids

Except in seed setting, marked morphological differences between the mutants and normals have not been observed in a majority of cases. Krishnaswamy et al. (1949) noticed non-heritable vegetative proliferation of the basal parts of the spike of the desynaptic pearl millet, while Patil and Vohra (1962) noticed more tillering and dwarfism in the desynaptic plants than in normals of the same species. Roy and Jha (1958) also observed luxuriant vegetative growth, profuse branching and decreased fruit size in the desynaptic plants of *Abelmoschus esculantus*. In barley, the desynaptic plants were dwarf and showed defective

heading of the spikes from the boot leaves (Srivastava 1974). In the desynaptic mutants of *Sorghum*, Krishnaswamy et al. (1957) observed slow seedling growth and prolonged anthesis. Sadasivaiah and Magoon (1965) in *Sorghum* and Vasudevan et al. (1967) in *Coleus* also reported stunted growth of the desynaptic mutants. In *Ipomoea gracilis* (Jones 1970), pea (Gostimsky 1976) and *Capsicum* (Ramalingam 1977) the mutants were late flowering. Soost (1951) noticed a decreased size of the anthers of the mutants than the normals in tomato while Catcheside (1939) in *Oenothera* and Sharma and Reinbergs (1974) in barley observed under-developed indehiscent anthers in the desynaptic mutants. However, a distinct morphological type which could serve for easy identification of synaptic mutants in a segregating population has not been detected so far.

2.1.1 Early Prophase I

Very few workers have analysed the early prophase stages of the synaptic mutants, due, obviously, to the inherent difficulties of chromosome analysis at these stages. Soost (1951) observed the occurrence of well-filled nucleus with single but lightly staining threads at the late leptotene stage in tomato mutants. Krishna Rao and Koduru (1978a) observed at these stages tight coiling and breakage of chromonema. Koduru (1980) reported the absence of any stages comparable to leptotene and zygotene in a complete asynaptic mutant of pearl millet. Further, this plant showed premeiotic condensation of chromatin material in the PMCs.

Pachytene is considered to be the critical stage to assign a mutant to either of the classes of the synaptic mutants, but in cases of partial failure of pairing even pachytene analysis may not help to decide to which class the mutant belongs (Riley and Law 1965).

2.1.2 Diplotene

This is the first stage at which the univalents can be seen in these mutants. The frequency of univalents increases by this stage, due to falling apart of the synapsed homologues. Such univalents usually lie close to each other (Östergren and Vigfusson 1953; Jauhar 1969; Jauhar and Singh 1969). Patil and Vohra (1962) in pearl millet and Sadasivaiah and Magoon (1965) in *Sorghum* mutants reported mostly terminal chiasmata in desynaptics by this stage.

2.1.3 Diakinesis

The pairing relations of the homologues are mostly studied at diakinesis because studies at MI and AI are not easy due primarily to the presence of several univalents even before the onset of AI, which usually remain scattered in the spindle.

The univalents at diakinesis are usually longer and exhibit a fuzzier appearance than those involved in bivalent formation in normal plants, which is supposed to be a result of undercoiling. Chiasma frequency declines from diplotene to diakinesis and will have a direct bearing on the number of bivalents. In *Allium consanguineum* Gohil and Koul (1971) reported that meiosis in the desynaptic plants began with the formation of eight bivalents, having two to five chiasmata per bivalent, but before the end of prophase chromosomes of most of the bivalents fell apart.

2.1.4 Metaphase I

As a consequence of the formation of univalents during prophase I, MI is characterised by abnormal spindle development and defective equatorial congression of the univalents. Such irregularities are the indirect effects of the mutant condition as indicated by similar irregularities in the meiosis in non-mutant forms carrying univalents resulting from hybridity or aberration and equivalent meiotic abnormalities in the mutants of the same type in different species (Gottschalk and Konvička 1972; Baker et al. 1976).

Position of Univalents: Univalents tend to remain scattered (John and Lewis 1965) more towards the poles or the periphery of the spindle and their distribution seems to depend on the time of their formation. Thus, if they were formed just before MI, the homologous univalents would lie close to each other with their kinetochores directed towards the spindle axis and the arms towards the outside. Further, the polar distribution of the univalents decreases with a simultaneous increase in the equatorial distribution as the number of bivalents per cell increases (Östergren 1951; Östergren and Vigfusson 1953).

Several other factors seem to influence the position of the univalents in the spindle: (a) the structure of the meiotic chromosomes, (b) stage of development of metaphase I – thus the distribution will be more equatorial towards the late metaphase I stage and (c) the interchromosomal effects. The position of the univalents is independent of the type of synaptic variation and the direction of the arms. The homologous univalents may lie on the same spindle arc or on separate spindle arcs which are close to each other.

Random vs Non-random Bivalent Formation: In all the reported cases of synaptic mutants, variation in the number of bivalents per cell was reported. The inter PMC varia-

tion in the frequency of bivalents in the mutants suggests that the response of different chromosomes of a genome to the influence of the mutated gene may be variable and independent. The Poisson and binomial analyses of the PMCs with varying number of bivalents in the partial and complete asynaptic mutants of *Nicotiana* showed that the bivalent formation in partial mutants did not conform to either type of distribution and hence was non-random (Swaminathan and Murty 1959). In view of this finding Swaminathan and Murty (1959) subjected the published data on maize, *Pisum*, rye, wheat and others to similar analyses and concluded that in complete mutants a bivalent may be formed at random. In partial mutants, however, as the mean number of bivalents per cell approached the equivalent of half of the potential number of bivalents, a gradual departure from the Poisson distribution was evident, ending in a very wide deviation in cases where n/2 or more bivalents were formed or where more than the number of bivalents constituting a genome in an alloploid species were formed. From these analyses it appears that when conditions conducive to normal chromosome pairing are inadequate, only certain homologues undergo synapsis and generate a few chiasmata, while others fail to pair. In fact, Rees (1958) showed in *Scilla* that different chromosome pairs responded differentially to abnormal cellular conditions while Jain (1957) reported that in *Lolium* specific chromosomes showed specific responses to changes in environmental factors. Thus, within the chromosome complement of a species there may be differences among the different chromosomes concerning their requirements for the initiation of pairing. Under such conditions, in the case of allopolyploids, the different genomes may have different requirements in order to undergo synapsis.

However, Sreenath and Sinha (1968) pointed out that deviation from the binomial distribution of the bivalent frequency in the desynaptics can arise not only from a differential behaviour of the different chromosomes within a meiocyte, as postulated by Swaminathan and Murty (1959), but also because of differences between cells within a population. The reason for the deviation can be inferred from the pattern of deviation from binomial distribution which can be tested by the Models 1 and 2 proposed by Sreenath and Sinha (1968). The analysis of the data of Miller (1963) on asynaptic corn by them revealed that the pattern of deviation from binomial distribution conformed to their Model 2 and therefore differences between cells within a population could be inferred as the reason for the deviation in corn. This situation may arise out of (a) variation in chromosome behaviour in different regions of the same anther, (b) variation between the anthers of the same floret and (c) variation in different spikelets. Further, it is important to note that, as pointed out by Sreenath and Sinha (1968), conformation to Model 2 would not eliminate the existence of intracellular differences in chromosomal behaviour but would emphasise the relatively greater contribution of intercellular differences for the pattern of variation. Subba Rao (1976) applied the models of Sreenath and Sinha (1968) to desynaptic pearl millet and inferred that differential behaviour of homologous pairs within a meiocyte was the basis for the deviation from binomial distribution in this species. Thus, there appear to be different factors contributing to the non-randomness of bivalent formation in the synaptic mutants of different species.

Chiasma Frequency and Distribution: In general the effect of mutant synaptic genes is to reduce chiasma frequency and/or alter chiasma distribution. Therefore, the frequency of chiasmata per cell will be less in the mutants compared to normals (Gottschalk and Pietrini 1965; Konvička and Gottschalk 1971). In many cases a correlation between the univalent frequency and chiasma frequency was demonstrated. Giraldez and Lacadena (1978) among others observed an increase in the frequency of terminal chiasmata with a reduction in the mean chiasmata and bivalents per cell. This terminal position of chiasmata might result either from a rapid terminalization of chiasmata in the mutants or from the initiation of pairing at the ends rather than at all parts simultaneously (Soost 1951). John and Henderson (1962) have demonstrated the actual terminal localization of chiasmata in asynaptic cells of locusts. A more specific case of localization was reported in rye, (Prakken 1943), in which the asynaptic genotypes showed the formation of chiasmata in the short arms of the chromosomes, which is never the case in normal genotypes where the chiasmata are confined to long arms. This was explained on the assumption that short arms usually pair rapidly and generate more chiasmata under disturbed cellular conditions than long arms.

In *Hypochoeris radicata* the mutant gene affected a single pair of specific chromosomes while the others were not affected (Parker 1975). Thus, in this genotype, the mutant gene controlled the chiasma formation on a chromosomal basis. Miller (1963), Runquist (1968), Moens (1969), Klein (1969b) and others have also noticed that the mutant gene altered the chiasma formation differentially in different bivalents of the same genome. Richardson (1935) in *Crepis* and John and Naylor (1961) in *Schistocera gregaria* observed that small chromosomes formed univalents more frequently than long chromosomes, while Koller (1938) in *Pisum*, Rees (1957) in *Locusta*, John and Henderson (1962) in *Schistocera paranensis* and Sjodin (1970) in *Vicia faba* noticed a greater reduction of chiasma frequency in the long chromosomes than in the short chromosomes; also the long chromosomes formed univalents more frequently than the short chromosomes. This might be because the short chromosomes pair and generate chiasmata earlier than the long chromosomes, which do not pair at all because of disturbed time relations (Darlington and Haque 1955). Thus, the effect of synaptic genes may be different on different chromosomes within the same genome and even on different parts of the chromosome.

However, not all synaptic mutant genes alter chiasma distribu-

tion, as can be inferred from the reports of the non-localization of chiasmata in the asynaptic genotypes of *Crepis* (Richardson 1935), *Lycopersicon esculantum* (Soost 1951), *Vicia faba* (Sjodin 1970) and in others. In corn (Beadle 1933), *Pisum* (Koller 1938), *Avena* (Thomas and Rajhathy 1966), rye grass (Ahloowalia 1969a) pearl millet (Dhesi et al. 1973), *Lolium* (Omara and Hayward 1978), and in many other species, a strong positive relationship between the number of chiasmata per cell and the number of bivalents was also noticed, which can be expected with random distribution of chiasmata over all the chromosomes. Bozzini and Martini (1971) in *Triticum durum* noticed a strong positive relationship between the number of chiasmata per bivalent and the mean chiasmata per cell. In rye (Prakken 1943), corn (Sinha and Mohapatra 1969), *Vicia faba* (Sjodin 1970) and *Lolium* (Omara and Hayward 1978) a negative relation between the mean chiasmata per bivalent and the frequency of bivalents per cell was noticed.

Cases of variability in the expression of the mutant genes have also been reported. In rye, Rees and Naylor (1960) and Rees (1962) observed significant differences in the chiasma frequencies between the PMCs derived from different regions of same anther, which they attributed to the difference in the nutrient level in the different regions of the anther and also to the different times at which the PMCs of different regions of an anther entered meiosis. In barley, significant differences in the expression of the desynaptic genes between the anthers of the same floret, between florets and between tillers of a plant were also noticed; these were attributed to the influence of modifying factors and also to the physiological differences between the organs under consideration (Wagenaar 1964)[1].

2.1.5 Anaphase I

Metaphase I and anaphase I cannot be distinguished from one another in total desynaptic or asynaptic mutants as there is no clear equatorial orientation of univalents: they usually remain scattered in the spindle more towards the poles. Person (1955) coined the term 'meta-anaphase' to describe this stage of meiosis. Apart from these orientation irregularities, anaphase I is characterised by (a) lagging chromosomes, (b) formation of bridges and fragments (c) irregular distribution of univalents (d) misdivision of the centromeres of the univalents (e) formation of polyads (f) development of micronuclei (g) formation of restitution nucleus and (h) blockage of meiosis. These irregularities usually increase as the mutant grows older (Klein and Milutinović 1972).

Behaviour of Univalents: Bivalents usually undergo reduction division but the behaviour of the univalents varies in different synaptic mutants. Johnson (1944), Celarier (1955), Sjodin (1970) and Palmer (1974) believe that in strong types with all univalents, the univalents are not situated on the equatorial plane, but move undivided to poles. However, in the literature several strong and medium types were reported in which the univalents underwent equational division at first anaphase. Equational division of univalents at AI was also observed in total asynaptic apomictic species (Rosenberg 1917). Koller (1938) and Östergren (1951) believe that the univalents which are nearer to the equatorial plate divide, while Andersson (1947), Smith (1936), Catcheside (1939) and Richardson (1935) reported the division of the univalents irrespective of their position in relation to the equatorial plate. In *Pisum* the metaphase I orientation of the univalents is influenced by the number of bivalents and also by mutual interference between them (Klein and Quednau 1976). Johnsson (1944) argued that the formation of a well organised metaphase I plate by the few bivalents that do form in partial mutants gives enough time to univalents to divide at AI in medium strong types of the synaptic mutants. But the AI division of the univalents in strong types with all univalents suggests that the presence of bivalents at MI plate is not a prerequisite for the AI division of univalents. Also, not all medium strong types showed the AI division of univalents. Therefore, the orientation of bivalents at MI and the division of the univalents at AI are to be regarded as independent events conditioned by independent factors.

From the observations of Soost (1951), Clayberg (1958), Person (1958), Stringam (1970), Sheriff and Rao (1974) and Koduru (1980) it appears that there is some relationship between the complete lack of pachytene pairing (true asynaptic types) and the AI division of the univalents. One of the factors controlling this behaviour of the univalents is the possible precocious centromere division which has been suggested to occur in *Alopecurus* (Johnsson 1944), *Picea* (Andersson 1947), tomato (Lamm 1944, Clayberg 1959) and pearl millet (Koduru 1980). In the asynaptic genotypes of pearl millet, PMCs just before the onset of meiosis showed 14 compacted masses of chromatin each with a pair of separated chromatin fibres (probably separated chromatids). These structures developed into C-mitotic like chromosomes by MI and underwent equational division at AI (Koduru 1980). Thus premeiotic individualization of chromatids inhibits chromosome pairing (Huskins and Smith 1934; Koduru 1980), while the precocious division of the centromere leads to the orientation of the univalents at MI and equational division at AI (Koduru 1980).

Anaphase I behaviour of univalents also depends on the time at which they are formed and on the type of orientation of its two centromeres at metaphase I. When amphitelic orientation is produced (bipolar orientation of univalents), equational division at anaphase I follows (Bauer et al. 1961; Luykx 1970). Giraldez and Lacadena (1976), working with asynaptic rye, observed two types of univalents. In type I the univalents behave as true univalents, i.e. both members of a pair can divide equationally or reductionally independently of one another while in type II both the members of a homologous pair always divide reductionally. All univalents of a PMC will have the same probability of belonging to type I. The type of orientation (syntelic or amphitelic) of the centromeres of univalents depends on the time at which they are formed in relation to the organisation of MI. Thus, if they were formed too early, they had greater chances of orienting amphitelically and hence would divide equationally at AI and if they were formed too late they would always orient syntelically and hence undergo reduction division (Giraldez and Lacadena 1976). Whatever the factors controlling the AI division of univalents, the result

[1] See note added in proof.

is that the chromosomes will have single gene strings and a centromere (Darlington 1939) by the second division, which sometimes may lead to the blockage of meiosis.

Chromosome Breakage: There are several reports of chromosome breakage in various synaptic mutants. These breaks owe their origin to the errors occurring in crossover processes during the synaptic stage (Lewis and John 1966; Jones 1968, 1969; Klein 1969b, 1970). Klein (1969b) pointed out that pairing and chromosome breakage are essential for crossing-over to occur in the synaptic mutants, during which U-type reunions (reverse chiasmata) occur between the sister chromatids, leading to bridges and fragments, instead of X-type reunions, leading to crossing-over. However, synapsis is not a prerequisite for the occurrence chromosome breakage. In desynaptic mutants fragments and bridges occur when chromosome pairing is normal with normal breakage frequency and broken ends preferentially recombine U-like rather than X-like and/or remain open. In asynaptic mutants fragments and bridges occur when synapsis is impaired and breakage occurs at normal level, with the broken ends remaining open to give rise to fragments and/or recombine with those of homologous or non-homologous chromosomes to result in bridge formation (Klein 1969b).

An analysis of chromosome breakage through meiosis in pearl millet revealed that fragmentation did not proceed with meiosis but occurred at a specific developmental stage during meiosis or even during premeiotic interphase (Krishna Rao and Koduru 1978a). Magoon et al. (1961) in *Sorghum*, Rees (1952, 1955) in *Scilla* and rye also traced the chromosome breakage to the pachytene stage. However, it has been shown in *Pisum* (Klein 1969b, Klein and Baquar 1972) that chromosome breakage occurs only when crossing-over takes place (at the four strand stage of pachytene) and there was a direct relation between the level of crossing-over and breakage. This supports the hypothesis that chromosome breakage in meiosis arises out of errors in crossover process. Non-random distribution of breaks was reported in rye (Rees and Thompson 1955) and pearl millet (Krishna Rao and Koduru 1978a). Extreme fragmentation of chromatin resulting in blasted appearence of nuclei was reported in desynaptic mutants of *Pisum* (Klein 1970) and pearl millet (Lakshmi et al. 1979). But it is not known whether this has any relation to errors in synapsis and crossing-over discussed above. Jones (1968) pointed out that in general these highly irregular meiotic errors could have been due to reduced control over the general meiotic process due to genetic imbalance caused by inbreeding or hybridisation or gene mutations.

Spindle Behaviour: In some of the reported cases of synaptic mutants, the spindle is normal and bipolar (Krishnaswamy et al. 1949; Prakken 1943; Seetharam et al. 1975; Krishna Rao and Koduru 1978a; etc) but the more usual condition seems to be an abnormal spindle formation, which was reported in a large number of species. In *Tradescantia* (Celarier 1955), *Pennisetum* (Jauhar and Singh 1969), *Suaeda* (Malick and Tandon 1960) and clustor bean (Sohoo and Gill 1975) there was no spindle formed while in desynaptic *Pisum sativum*, Klein (1969a) reported the occurrence of two spindles in the first division of meiosis. In species with defective spindles the spindle fibres may differ in their pulling ability which usually results in the development of irregularly grouped chromosomes (Östergren and Vigfusson 1953).

In some of the synaptic mutants longer than the normal spindles, which take a crescent shape in order to accommodate themselves in the limited cell space have been reported (Beadle 1933; Krishnaswamy et al. 1949; Baker and Morgan 1969; Koduru 1980). The unusual elongation of the spindle is facilitated by the absence of any well-organised metaphase plate and thus Beadle (1933) was able to observe a correlation between the number of bivalents and the length of the spindle axis. The presence of bivalents at the metaphase I plate was supposed to check the elongation of the spindle by necessitating the exertion of separation forces by the fibres on the bivalents. In mutants with long spindles the chromosomes at the centre will have a greater chance to organise into a third group, resulting in micronuclei at the end of the first division.

Inspite of defective chromosome congression and defective spindles, a majority of the PMCs of the synaptic mutants were reported to show bipolar distribution. Synapsis and spindle organisation thus appear to be governed by independent mechanisms. This could also be postulated from the occurrence of mutations affecting synapsis or spindle formation (Clark 1940; Vaarama 1949; Martini 1966). Thus the lack of normal equatorial congression leading to irregular metaphase and anaphase distribution does not seem to be sufficient to destroy the poleward movement of chromosomes (Martini and Bozzini 1966).

2.1.6 Telophase I

Irrespective of the distribution pattern of chromosomes at AI, those that reach the poles organise dyad nuclei and the lagging chromosomes develop into micronuclei at telophase I. Blockage of further progress of meiosis was observed in *Oenothera* (Catcheside 1939), *Sorghum* (Sadasivaiah and Magoon 1965), *Alopecurus* (Johnsson 1944) rye (Prakken 1943), *Allium cepa* (Koul 1962) *Amorphophallus* (Magoon and Sadasivaiah 1967), *Pennisetum orientale* (Jauhar and Singh 1969) and pearl millet (Koduru 1980).

2.1.7 Second Division

Second division, wherever studied, was largely normal in the majority of cases except for such irregularities such as laggards, bridges, fragments and inactive polar movement. These irregularities owe their origin to disturbances at the

AI. Consequently, at the end of meiosis tetrad as well as micronuclei formation, up to 18 in cotton (Beasley and Brown 1942), has been reported. However, in a few cases such as *Hordeum* (Ekstrand 1932), *Triticum* (Smith 1936), *Alopecurus* (Johnsson 1944), *Sorghum* (Krishnaswamy et al. 1957) and pearl millet (Krishnaswamy et al. 1949), normal tetrad formation was reported.

2.1.8 Pollen Development and Fertility

In general the pollen fertility of the synaptic mutants will be reduced and variable depending on the intensity of mutation. In *Trifolium*, Whittington (1958) observed total sterility of the mutants. The two exceptions were *Allium* (Leven 1939, 1940) and *Paspalum* (Christopher 1971). Normal fertility was reached in *Allium*, because of restitution at AI and normal division at AII, while in *Paspalum* normal division of univalents at AI was followed by restitution at AII. The developing microspores usually degenerate soon after the formation of the pollen grain wall. However, Srivastava (1974) observed degeneration at the tetrad stage in barley while Celarier (1955) noticed degeneration of the microspores in the male gametophytic stage in *Tradescantia*. Sjodin (1970) pointed out that pollen fertility was not related to univalent frequency, but Thomas and Rajhathy (1966) noticed an inverse relation between univalent frequency and pollen fertility. The pleiotropic effects of the mutant gene(s)-like breakage, stickiness and spindle abnormalities, will also contribute to the pollen sterility.

2.1.9 Female Meiosis and Embryosac Development

Most of the information available on chromosome behaviour during megasporogenesis in the synaptic mutants was inferred from the seed-setting ability of the mutants after pollination with fertile pollen from the normal plants. Prakken (1943) reported the same type of abnormalities that occurred in the PMC meiosis. Davies and Jones (1974) in rye and Johnsson (1973) in *Picea* showed that the genetic mechanism controlling chiasma frequency in both the male and female was alike and expressed identically in the two sexes. From a comparison of pollen fertility and seed setting on controlled or open pollination in several species, the intensity of irregularities on the female side was deduced to be less than on the PMC side. But in rice (Katayama 1964) and *Brassica* (Gottschalk and Konvička 1971) the desynaptic genes were observed to have more effect on megasporogenesis than on the male side. Bergner et al. (1934) in *Datura*, Roy and Jha (1958) in *Abelmoschus*, Whittington (1958) in *Trifolium*, Martini and Bozzini (1966) in wheat, Gottschalk and Konvička (1975) in pea, Srivastava (1974) in rye, Golubovskaya and Mashnenkov (1976, 1977) in corn and Koduru (1980) in pearl millet reported complete female sterility, while Nelson and Clary (1952) reported normal female fertility in corn. In the asynaptic cotton embryosac development was found to be absent (Weaver 1971).

3.2 Polyploids

Compared to the diploids there are fewer reports on the occurrence of synaptic mutants in polyploids (Newton and Pellew 1929; Levan 1939, 1940; Beasley and Brown 1942; Darlington and Janaki-Ammal 1945, Myers 1945; Morrison 1956; McCollum 1958; Mathur and Tandon 1965; Vig and Meharotra 1965; Thomas and Rajhathy 1966; Ahloowalia 1969a; Estes 1971; Gohil and Koul 1971; Kolobaeva 1974; Pi and Chao 1974; Koduru and Krishna Rao 1978; Subba Rao 1978). In autopolyploids desynapsis was mostly expressed as reduced chiasma frequency, reduced multivalent formation, an increased univalent frequency and reduced fertility. In tetraploid *Allium tuberosum* (Vig and Meharotra 1965), as in *A. ascalonicum* (Darlington and Haque 1955), the PMCs from the central part of anther showed asynapsis which they attributed to disturbed time relations in the anther development. In the asynaptic tetraploids of *Avena*, as in asynaptic corn (Beadle 1933), Thomas and Rajhathy (1966) observed a positive correlation between mean chiasma frequency and the mean number of bivalents per cell, indicating, in addition, a randomness in the failure of chiasma formation in the desynaptic tetraploids. The mutants showed monogenic recessive inheritance. At the end of first division in the achiasmatic *Artemisia*, restitution nuclei were developed in all PMCs. followed by normal second division which lead to the formation of mostly euploid 4x gametes. In *Paspalum longifolium*, the octoploids obtained from 4x asynaptic plants showed normal pairing. At the 12x level the spontaneously occurring plants showed normal pairing while those induced from 6x asynaptic genotypes showed asynapsis. In mutants of both ploidy levels, restitution nuclei developed at the end of the first division. The second division was comparatively normal (Pi and Chao 1974).

3 Genetics

3.1 Inheritance

In most of the synaptic mutants investigated, except in *Crepis* (Hollingshead 1930) where monofactorial dominant inheritance was reported, monogenic recessive inheritance was observed. In wheat (Smith 1936), cotton

[2] See note added in proof

(Beasley and Brown 1942, Menzel and Brown 1955, Weaver 1971, Smithson 1974) and pearl millet (Lakshmi et al. 1979) digenic recessive inheritance of the mutant condition was reported. Hayter and Riley (1967) reported duplicate gene activity affecting meiotic chromosome pairing at low temperature in *Triticum*. In *Rumex* the asynaptic gene followed Y – linked inheritance (Löve 1943).

In a few species more than two non-allelic recessive synaptic mutant genes have been identified- three each in Soyabean (Hadley and Starnes 1964; Palmer 1974) and *Brassica* (Stringam 1970), five each in barley (Ramage and Soriano 1972) and tomato (Soost 1951) and more than 20 in *Pisum* (Gottschalk 1973, 1975; Gottschalk and Klein 1976). The gene symbols, *as*, for the asynaptic genotypes and, *ds*, for the desynaptic genotype, were proposed. Ramage and Soriano (1972) proposed the use of three letters – *aes* and *des* to designate the genes for asynaptic and desynaptic conditions respectively, followed by numbers to represent the number of loci, then again letters to represent the mutational event at each locus.

In species with habitual cross pollination such as rye, *Hordeum* and pearl millet, prolonged inbreeding results in genetically controlled reduction in chiasma frequency and hence in some degree of desynapsis in some families. This phenomenon was reported to be under polygenic control (for a detailed account see Rees 1955, 1961; Gale and Rees 1970; Pantulu and Manga 1972).

3.2 Crossing-over and Recombination

Crossing-over, a prerequisite for gene recombination, is a post-leptotene event (Henderson 1970). All sexually reproducing higher organisms develop synaptinemal complexes for an effective crossing-over (Moses 1968). Moens (1969) demonstrated the development of a synaptinemal complex (S.C.) in the desynaptic mutants of tomato. These structures subsequently were destroyed early as the paired homologues fell apart subsequent to pachytene. La Cour and Wells (1970) observed the development of the components of S.C. in asynaptic wheat, which later disorganised owing to the lack of effective synapsis of the chromosomes. Thus, in asynaptic mutants where pairing is not even attempted, the formation of normal S.C. is doubtful (Catcheside 1977), though the individual elements of S.C. may be formed.

The absence or reduction in chiasmata in the synaptic mutants should result in reduced recombination. Enns and Larter (1962) observed more than a two-fold reduction in per cent recombination between marked loci on chromosome 2 in homozygous desynaptic plants of barley. In corn, heterozygosity for the *as* gene reduced recombination between the marked genes on chromosome 5 while the per cent recombination between marked loci on chromosome 3 was not affected (Nel 1973). These differences were explained on the basis of the differential sensitivities of the factors of these two regions affecting recombination to the influence of *As/as* genotype[3]. Beadle (1933) found normal levels of crossing-over between the *sh-wx* region of the *C-wx* linkage group in asynaptic plants of corn. Miller (1963) observed only reductional division for such cytological markers as the abnormal 10 at metaphase I in partial asynaptic corn. In such cases the asynaptic and desynaptic genes would have an effect on the linkage maps. Maguire (1978) observed that crossing-over and chiasma maintenance in desynaptic mutants of corn are controlled by different mechanisms and that the mutant gene had no effect on crossing-over but influences the maintenance of chiasmata. In the as_1 and as_4 mutants of tomato Soost (1951) observed a normal level of crossing-over between marked genes (*ds*, *Wo*) located distally on the nucleolar chromosome (chromosome 1).

On the other hand, Rhoades (1947), Rhoades and Dempsey (1949), Dempsey (1958, 1959), Miller (1963) and Moens (1969) noticed higher recombination for marked loci in the progenies of the synaptic mutants. In tomato, different asynaptic genes were found to affect the per cent recombination between marked loci to varying degrees. Thus, the as_1 gene had no effect on the map distance of marked loci on chromosome 2, whereas the as_4 gene showed a 2.4 fold increase in the map distance of the marked loci (*d-aw-wv*) and the as_b locus showed a 1.5 times increase between *d-aw* and a 2.3 fold increase between the *d-wv* loci. Further, for the two distal regions examined, in as_4 the increase in per cent recombination was the same, while in as_b both regions were affected to different degrees whereas both of them have not decreased the per cent recombination in the proximal regions. All three mutants increased the coefficient of coincidence to different levels (Moens 1969). Moens (1969) explained these results on the assumption that these genes cause interference with the process of genetic exchange at meiosis, either directly or indirectly through changes prior to exchange. These reports indicate that in spite of the absence of cytological synapsis and chiasmata at diakinesis and MI, recombination could take place in the synaptic genotypes. This could be explained on the assumption that in desynaptics, if the chiasmata are strongly localised at terminal regions and if there is a lack of strong terminal affinity, by metaphase I univalents with crossover chromatids will appear (Moffett 1932). Alternatively, the increased recombination found in some of the mutant genotypes for the marked loci may be due to compensation of the loss of recombination in some other parts of the genome in these mutants, as observed in cotton (Giles 1961, Stephens 1961) and *Drosophila* (Schultz and Redfield 1951; Suzuki 1962). Supporting this compensation in genetic recombination, compensation for chiasma formation in the desynaptic mutants was reported in corn (Sinha and Mohapatra 1969) and *Lolium* (Omara and

[3] See note added in proof

Hayward 1978). Miller (1963) also noticed that the frequency of crossing-over in short synaptic regions like distal segments of short arms and the centromeric region of asynaptic corn was more than in the corresponding regions of normal plants, a situation similar to compensatory chiasma formation.

3.3 Interaction with B-Chromosomes

The action of a desynaptic gene on the behaviour of B-chromosomes was reported in pearl millet. The desynaptic gene affected both pairing and chiasma formation in standard and deficient B-chromosomes present in the desynaptic genotypes, as it does on A-chromosomes. On the other hand, the presence of three standard Bs increased the A-chromosome bivalent frequency in desynaptic plants while the presence of standard and deficient Bs increased the univalent frequency of A-chromosomes in mutant plants (Pantulu and Subba Rao 1976).

3.4 Factors Influencing Pairing

Both cellular and environmental factors are known to influence the synaptic behaviour of the chromosomes of these mutants. The mutants are less buffered against the fluctuations in the environmental factors than normal genotypes and hence a slight change in the environment may have pronounced effect on these unstable genotypes (Darlington 1958).

3.4.1 Environmental Factors

As early as the 1930s environmental factors were recognised as influencing chromosome synapsis. In the desynaptic mutants of *Nicotiana sylvestris* high temperature and low humidity induced asynapsis while high temperature and high humidity favoured chromosome pairing (Goodspeed and Avery 1939). Thus humidity is the factor having a relatively higher effect on the functioning of the synaptic genes. Prakken (1943) also noticed that relative humidity of the air and the soil had an influence on the extent of variation in the synaptic mutants of rye.

Barber (1942) in *Fritillaria*, Soost (1951) in tomato and Ahloowalia (1969b) in rye grass noticed a negative relationship between temperature and the bivalent frequency. Thus, in rye grass, at 28 ± 2°C the degree of desynapsis was more while at 11 ± 2°C there was normal chromosome pairing. Beyond 30°C meiosis was blocked at diakinesis and the mutant produced polyploid gametes. Further, Soost (1951) noticed that the temperature at the time of early prophase had the greatest effect on the bivalent formation. In *Tradescantia* and *Uvularia perfoliata* an increase in temperature up to 30°C increased the chiasma frequency; further increases had negative effects and resulted in desynapsis (Dowrick 1957).

On the other hand, a positive relationship between the temperature and bivalent frequency was reported in some species. In *Citrus*, asynapsis was expressed at low temperatures (10°C) but chromosome pairing was normal at higher temperatures (Iwamasa and Iwasaki 1963). Li et al. (1945) and Pao and Li (1948) in common wheat, Chao and Hu (1961) and Wang et al. (1965) in rice also noticed a positive relation between temperature and bivalent frequency in the mutants. Thus, at 10°C the manifestation of the mutant gene in wheat is total, resulting in total desynapsis. The frequency of bivalents increased with a rise in temperature (Li et al. 1945). In the hexaploid wheat Bayliss and Riley (1972a) also showed that in the absence of chromosome 5D, a recessive allele, *ltp*, on chromosome 5A was unable to stabilize chiasma frequency at low temperatures, resulting in a partial or complete failure of the mechanism of zygotene chromosome pairing (Asynapsis). Subsequently, they (Bayliss and Riley 1972b) found that the temperature sensitive stage was between the last premeiotic mitosis and the start of DNA synthesis (G_1 period).

These reports clearly indicate the sensitivity of the synaptic genes and their products to changes in the environmental factors. Under such conditions different genes may show different responses, which may be due to the differences in their genotypic adaptability.

3.4.2 Experimental Studies

Riley and Miller (1966) observed that x-ray treatment increased the number of chromosomal rearrangements in desynaptics of rye, while Swietlinska and Evans (1970) observed no difference in type or frequency of induced aberrations between the mutant and normal genotypes of rye. Thomas (1973) observed a somatic association of chromosomes in desynaptic oats.

Ahloowalia (1969b) reported an increased chiasma frequency and bivalent frequency in the desynaptic mutants of rye grass treated with 0.01% aqueous phenobarbitol at 20°C. Since barbiturates were known to develop hydrogen bonds with DNA bases it was suggested that the synapsed homologues were held together by hydrogen bonds. Therefore, the synaptic mutants might be lacking or defective in a chemical that participates in maintaining hydrogen bonds in the coiled structure of the paired chromosomes.

In normal genotypes of *Lolium* (Law 1963) and rye (Bennett and Rees 1970) an increase in mean chiasma frequency was observed with an increase in ion content of potassium, calcium and phosphate available to the plant. In desynaptic barley, Fedak (1973) reported an increase in the number of ring bivalents with addition of mineral phosphate. Using a desynaptic line of pearl millet, Dhesi et al. (1975) also observed increased chiasma frequency and decreased desynapsis with increase in the application of phosphate and potassium to the soil. Thus, there appears to be some relationship, both in normal and desyn-

aptic genotypes, between the level of mineral supply (phosphate, potassium and calcium) to the plant and the chiasma frequency in the PMCs. An attempt by Lakshmi et al. (1979) to find out whether the desynaptic mutants of pearl millet had a deficiency of phosphate (P) and potassium (K) in their plant body, particularly at the time of meiosis in the PMCs, and whether the positive response in chiasma frequency was due to a partial or complete compensation of deficiency due to increased availability of substances in the soil, failed to reveal any significant differences between the normals and the desynaptics. However, before coming to any conclusion on the role of minerals on synapsis and chiasma frequency, a detailed analysis of the mineral composition of the PMCs of desynaptic and normal plants is desirable.

3.4.3 Time Relations

The time at which the meiocytes enter meiosis may influence the synaptic behaviour of the homologues. Thus, a first meiotic prophase which begins too late, gives the chromosomes time to divide, hence they fail to pair (Darlington and Haque 1955); if it begins too early, there is interference with the despiralisation of the chromosomes, which prevents pairing (Sax and Sax 1935). In the asynaptic mutants of rye, Prakken (1943) noticed less relational coiling than in normals.

3.4.4 Cytochemical Factors

Ehrenberg (1949) suggested that since crossing-over involves the breaking and reunion of protein chains under the action of proteolytic enzymes, the absence or diminution of any of these enzymes may interfere with crossing-over, leading to the failure of chiasma formation. Apparently the synaptic gene mutation might change the structure and/or function of such macromolecules or reduce the life period of the gene products involved in these functions. Sheridan and Stern (1967) identified a type of meiotic histone (which is not found in somatic cells) whose concentration decreased as meiosis progressed to completion. This indicates that the meiotic histone is produced only once in the meiotic cycle (may be during the premeiotic interphase). Ahokas (1977) reported that the partial desynaptic mutants of barley contained more proteins than the normals. Ahloowalia (1969b), Jauhar and Singh (1969) suggested that the synaptic gene mutation might change the structure and/or function of such macromolecules which effect pairing.

Ansley (1954, 1957, 1958) and Sinha (1959) believed that changes in the relative quantities of histones and DNA, or RNA and DNA might affect chromosome pairing. In *Loxa* and *Scutigera* normal male meiocytes showed 1:1 ratio for histones and DNA while the asynaptic meiocytes showed 3:2 ratio. A comparative study of the relative rates of synthesis of histones and DNA through the onset and progress of meiosis in normal and asynaptic meiocytes revealed that the premeiotic synchronous increase of histones and DNA inhibit one part of the pairing mechanism while asynchronous increase of histones after the onset of meiosis inhibit another part of pairing (presumably resulting in the falling apart of the homologues). When both are present together, asynapsis is complete, i.e. pairing may not even be attempted. In *Scutigera*, wherein there was premeiotic synchronous increase of histones and DNA, there were no traces of prophase I stages while in *Loxa* in which there was asynchronous increase of histones and DNA there was no bivalent formation (desynapsis). The examination of somatic cells, normal spermatogonia and harlequin cells of *Loxa*, revealed that somatic and normal meiocytes contained a mean histone value of 1.79 ± 0.04 while those of desynaptic harlequin meiocytes had 2.70 ± 0.04 (Ansley 1957, 1958). In the young ears of ameiotic corn there was more RNA than DNA. Further, these plants differed from normals in the relative quantities of histones to DNA (Sinha 1959). Mackenzie et al. (1967) observed a variation in the content of RNA in meiotic prophase indicating that relatively rapidly metabolizing RNA molecules were also involved in the mechanism of chromosome pairing.

It is now well established that all events of meiosis, including chiasma formation and crossing-over, are under genetic control (Rees 1961; Riley and Law 1965; Riley 1966; Smith 1966; Baker et al. 1976). Since gene action is enzyme mediated, it can be supposed that several specific enzymes might be essential for the onset and normal progress of meiosis and chiasma formation. Jauhar (1969), Jauhar and Singh (1969) and Ahloowalia (1969b) suggested that in the synaptic mutants the concentration of enzymes necessary for pairing and its maintenance may be enough at the onset of meiosis and as meiosis progresses to metaphase I, a scarcity or inactivation of the enzymes may develop so that the homologues may fall apart rapidly.

4 The Process and Control of Synapsis

A review of the studies on synaptic mutants reveals that while important information concerning the genetic control of synapsis and recombination processes has been obtained little insight has been gained into the actual mechanisms and the nature of events involved in the control of chromosome synapsis. However, remarkable progress has been made in our understanding of the processes that control synapsis and in their time of occurrence, by combined biochemical, genetic and experimental analyses of normal genotypes, especially those of lily, wheat and maize.

Experimental evidence from several organisms is available and shows that the pattern of DNA synthesis in the S phase of cells destined to undergo meiosis and mitosis is

different and that the S phase is longer in the former than in the latter. Further, DNA replication in the meiocytes was not complete and in *Lilium* 0.3% of the DNA remained unreplicated. This part of the DNA was replicated during zygotene (Z-DNA) (Stern and Hotta 1973, 1974, 1977) in transient association with the lipoprotein complex (Hecht and Stern 1971) and is located in *Chlamydomonas* in the hypothetical synaptic regions of the chromosomes (Chiu and Hastings 1973). Hotta et al. (1966). Ito et al. (1967) and Stern and Hotta (1969) showed that the inhibition of Z-DNA synthesis at the beginning inhibits chromosome synapsis and the cells did not enter zygotene while the inhibition of the DNA synthesis during zygotene inhibits the further development of synaptinemal complexes (Roth and Ito 1967). On the other hand, P-DNA (DNA synthesised during pachytene) was found to have no role in chromosome synapsis (Hecht and Stern 1971) but was involved in the repair of DNA (Hotta and Stern 1971b).

In lily, in addition to the Z-DNA, the meiocytes also had a denser (S = 1.2-1.22) lipoprotein fraction in the nuclear membranes. The presence of this lipoprotein fraction in the nuclear membranes spans the critical zygotene and pachytene. The Z-DNA and the lipoprotein form a complex which is unique to meiocytes (Stern and Hotta 1977).

In addition to the role of Z-DNA in chromosome synapsis, Parchman and Stern (1969) and Roth and Parchman (1969) showed that the inhibition of protein synthesis during zygotene inhibits chromosome pairing and destabilizes the S.C. Further, it has been shown that interference with leptotene lipid synthesis inhibits the Z-DNA synthesis which in turn inhibits chromosome synapsis. Thus, protein synthesis and Z-DNA synthesis are linked (Stern and Hotta 1977). This protein, named r-protein was contained in the lipoprotein complex referred to above and has both DNA binding capacity and the ability to catalyse the reassociation of single-stranded DNA (Hotta and Stern 1971a). Therefore, it was presumed that the r-protein had some important role in the alignment of homologues and synapsis (Stern and Hotta 1977). This was supported by its association with Z-DNA replication and also the asynaptic consequence of disrupting the r-protein lipoprotein association (Stern and Hotta 1977). Accordingly, it was observed that the lipoprotein complex appears during leptotene, reaches a maximum at zygotene, begins to disappear by mid-pachytene and has disappeared by the end of pachytene.

The work on wheat has demonstrated that control of chromosome pairing is a multi-stage process, that the pairing of homologous chromosomes is determined by events occurring as early as the last premeiotic mitosis and the subsequent G_1 of the meiotic cycle and that this early determined stage is subsequently maintained by constituents, presumably proteins, that are sensitive to either colchicine, temperature or gene control. Dover and Riley (1977) presented a scheme of the process of homologous chromosome pairing involving the sequential steps of alignment of homologous chromosomes, maintenance of alignment and commitment to synapsis during early stages of meiotic development.

Maguire (1974, 1977b) has found by direct cytological observations that the homologous association in maize extended back to the premeiotic mitotic metaphase. She suggested that homologous chromosome pairing may be accomplished by chance meeting of homologous segments, followed by establishment of invisible elastic connections, at congression for a mitotic metaphase, in many cases perhaps the premeiotic mitosis.

This brief summary of the results on normal genotypes should serve to emphasise the complexity of the problem of chromosome asynapsis and the great need to look at the events of cell development immediately preceeding meiosis, especially in the synaptic mutants.

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4 Consulted abstract only

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Dr. P.R.K. Koduru
Dr. M.K. Rao
Department of Botany
Andhra University
Waltair 530 003 (India)

Note Added in Proof

1. Rao and Rao (1980) reported non-random distribution of chiasmata and univalents between PMCs in a spontaneous desynaptic mutant of *Chrysanthemum coronarium* L.
2. Recently Iwanaga and Peloquin (1979) isolated a gene controlled synaptic mutant *sy* in potato which is effective only on the female side keeping microsporogenesis normal Cytokinesis was so irregular that most MMCs did not generate normal tetrads. The frequency of abnormalities at M I and A I varied from 93 to 99% in various plants. Thus, the expressivity of the *sy* gene is complete.
3. Subsequently, Nel (1979) reported additional data which demonstrated that recombination was reduced between the marked loci even on chromosome 3 in the asynaptic heterozygotes. Further, recombination between the same loci was reduced to a greater extent in asynaptic homozygotes. The effect was attributed to the *as* allele itself or to a closely linked locus.

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Table 1. (continued)

Species	Origin	Genetics	Author(s)
Barley	–	–	Manzyuk et al 1978
Chrysanthemum coronarium	Sp	–	Rao et al 1980*
Corchorus olitorius	Sp, I	1R	Paria et al 1978, 1980*
Crotolaria retusa	Sp	–	Meshram et al 1978*
Fragaria	–	2R	Reighter et al 1979
Potato	Sp	1R	Iwanaga et al 1979 Matsubayashi 1979
Zea mays	–	1R	Nel 1979