DESYNAPTIC MUTANTS IN BETZES BARLEY

by

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ABSTRACT

Ten desynaptic mutants conditioned by single recessive genes were isolated from partially sterile plants collected in commercial fields of *Hordeum vulgare*, cultivar 'Betzes' (CI 6398). The mutants occupy six loci.

A method to characterize desynaptic mutants is proposed. The method is based on the mean number of desynaptic events per cell at metaphase I. Studies of the frequencies of metaphase I configurations suggested that desynapsis in barley is of a random nature. Cytological studies supported previous evidence that in highly desynaptic mutants the number of univalents longitudinally splitting at anaphase I is less than expected. Other cytological abnormalities are described. A positive correlation (.796) was found between the mean number of micronuclei per quartet of desynaptic mutants and their mean degree of desynapsis. Desynaptic plants were found to have high ovule sterility, but no association was found between the average degree of sterility of the mutants and their degree of desynapsis. Aneuploids were frequently found among the light seed produced by desynaptic plants. Studies involving the trisomic progenies were used to locate most of the desynaptic genes.

INTRODUCTION

Desynaptic mutants are those mutants in which pairing between homologous chromosomes occurs in the early stages of prophase but the chromosomes fail to remain paired in the later stages (Li, Pao, and Li, 1945).

Desynapsis has been widely reported throughout the plant kingdom (Katayama, 1964). Most of the reports deal with the behavior of the unpaired chromosomes in metaphase I and with its cytological (e.g., splitting of univalents, micronuclei) and physiological (e.g., ovule and pollen sterility) implications.

Ekstrand (1932) reported the first desynaptic occurrence in barley. Since then, other mutational events have been reported (Burnham, 1946; Moh and Nilan, 1954; Tsuchiya, 1959; and Enns and Larter, 1960).

In 1967 and 1968 ten desynaptic lines were isolated from a collection of partially sterile plants found in commercial fields of Betzes barley. This report deals with the study of these ten lines--allelic relationships, meiotic behavior, fertility, progeny, and location of genes--and their relationship to the previously described genes in barley.

LITERATURE REVIEW

Among the many examples of gene mutations affecting chromosomal behavior reported in the literature (Rees, 1961), a large number condition meiotic abnormalities.

Although genes affecting almost every conceivable aspect of meiotic behavior have been described (Darlington, 1937), genes affecting the meiotic synaptic processes have been by far the most frequently reported. The majority of such genes prevent or reduce synapsis between homologous chromosomes, prevent or reduce chiasma formation, or cause premature terminalization of chiasmata. The action of these genes may involve all of the chromosomes, some of the chromosomes, or parts of all or of some of the chromosomes in a cell. Such genes are generally referred to as asynaptic or desynaptic genes.

The term "asynapsis" was proposed to describe the absence of the normal synaptic association of homologous chromosomes during the first meiotic division (Randolph, 1928). A heritable asynaptic condition was reported for the first time in *Gossypium* (Kearney, 1923). The term "asynaptic" and the symbol *as* were first used to describe asynapsis in *Zea mays* (Beadle, 1930).

The term "desynapsis" was proposed by Li, Pao, and Li (1945) to describe those phenomena in which pairing between homologous chromosomes occurred in the early stages of the meiotic prophase (zygotene and pachytene), but in which the chromosomes failed to remain paired in the later stages, resulting in unpaired chromosomes at metaphase I.

Difficulties in differentiating desynapsis and asynapsis arise in cases where early meiotic stages are not easily identified. In fact, most of the cases originally reported as asynaptic were later found to be desynaptic (Katayama, 1964).

In the literature, the term asynapsis is still used by some authors to describe occurrences in which some pairing was ascertained in the early stages of prophase (e.g., Sjödin, 1970, in *Vicia faba* L.). In this study, the term desynapsis, rather than asynapsis, will be used whenever any pairing in the early stages of prophase has been reported.

Desynapsis has been reported in most plants that have been studied cytologically. It appears to be a common occurrence throughout the plant kingdom. Katayama (1964) made an extensive review of heritable desynapsis in 6 families, 22 genera, and 34 species of Monocotyledoneae and 13 families, 27 genera, and 32 species of Dicotyledoneae. Since then, similar phenomena have been reported in *Glycine max* L. (Hadley and Starnes, 1964), *Vicia faba* L. (Sjödin, 1970), *Citrullus* (Kihara, Saito, and Shimotsuma, 1972), *Collinsia tinctoria* Hartw. (Mehra and Rai, 1972), *Allium sativum* L., *A. consanguineum* Kunth. and *A. tuberosum* Rottler (Gohil and Koul, 1971), *Avena strigosa* Schreb. (Dyck and Rajhathy, 1965), and a tetraploid *Avena* (Thomas and Rajhathy, 1966); new occurrences have been reported in *Pisum sativum* L. (Gottschalk and Villalobos-Pietrini, 1965, and Gottschalk and Baquar, 1971), *Lycopersicum esculentum* Mill. (Moens, 1969), *Triticum durum* Desf. (Martini and Bozzini, 1966), and *Hordeum* (Ramage and Hernandez-Soriano, 1971 and 1972).

Genes for desynapsis have occurred spontaneously in natural populations (e.g., in *Secale*; Prakken, 1943), have been found in the progenies

of plants treated with x-rays (e.g., in *Hordeum*; Burnham, 1946), γ-rays (e.g., in *Citrullus*; Kihara, Saito, and Shimotsuma, 1972), *Colchicine* (e.g., in *Avena*; Dyck and Rajhathy, 1965), chemical mutagens (e.g., in *Brassica campestris* L.; Stringam, 1970) or atomic bomb irradiation (e.g., in *Cassia tora* L.; Katayama, 1953), and have been found in progenies of interspecific crosses (e.g., in *Avena abyssinica* Hochst. × *A. barbata* Pott.; Thomas and Rajhathy, 1966).

A nonheritable desynaptic condition can be due to a loss of a chromosome pair, to mechanical chromosomal conditions, to interspecific hybridization, or to a normal process in apomictic organisms, or can be induced by external conditions (Prakken, 1943).

Mendelian desynapsis is generally controlled by a single recessive gene. Exceptions to this rule were found in *Crepis capillaris* Wallr., where desynapsis was controlled by a dominant gene (Hollingshead, 1930); in *Gossypium* (Kearney, 1923, and Beasley and Brown, 1942) and *Triticum* (Smith, 1936), where it was controlled by two recessive genes; and in *Picea* (Andersson, 1947), where it was controlled by more than one gene.

Very few allele tests have been performed among different desynaptic mutants. In *Collinsia* a single recessive gene for desynapsis was found to be allelic to another single recessive gene that conditioned "chromosomal stickiness" (Mehra and Rai, 1972).

In desynaptic genotypes the presence of chiasmata at diplotene has been widely observed. The lack of terminal affinity combined with strong terminalization of chiasmata will result in unpaired chromosome arms at metaphase I. When one of the two arms of a bivalent fails to remain paired, a "long" bivalent with a rodlike appearance occurs at

metaphase. Lesley and Frost (1927) first described such a condition in *Matthiola*, and Burnham (1946) reported a "long chromosome" recessive gene (really a desynaptic gene) in *Hordeum*. These rod bivalents of desynaptic genotypes have been reported to be more attenuated and less contracted than similar occurrences in normal synaptic genotypes (Koller, 1938; Prakken, 1943; and Thomas and Rajhathy, 1966). Two univalents will result at metaphase I if both arms of a bivalent fail to remain paired.

The degree of unpairing, or desynapsis, at metaphase I is used to characterize different desynaptic mutants. This degree of desynapsis is expressed by most authors in terms of frequency of univalents at metaphase I. The frequencies of ring bivalents and rod bivalents are usually confounded in most of the scientific reports; even though most of the bivalents are of the rod type, both the ring and rod bivalent frequencies are included under a single "bivalent frequency" (e.g., Miller, 1963; Katayama, 1962; and Dyck and Rajhathy, 1965).

The degree of desynapsis observed in microsporocytes is highly variable from cell to cell, and all possible metaphasic configurations are sometimes detected. In a desynaptic mutant in *Avena*, $2n = 14$, the number of univalents per cell was reported to range from 0 to 14 (Dyck and Rajhathy, 1965). In a study of 14 desynaptic mutants in maize, $2n = 20$, the highest desynaptic mutant showed 20 univalents in all cells; the lowest desynaptic mutant showed 95% of the cells with 10 bivalents and 5% with 2 univalents; and the rest of the mutants showed a range in number of univalents per cell varying from 0 to 20 (Miller, 1963).

These variations in the expression of desynapsis of the metaphasic cells within and between desynaptic mutants have been attributed to

environmental fluctuations (Gottschalk and Villalobos-Pietrini, 1965). The events leading up to and including chiasma formation are the most susceptible to change during meiosis (Rees, 1961), and since mutant genes are not buffered against environmental fluctuations (Darlington, 1958), variations in the phenotypic expression of desynapsis can be expected under different environments. For example: Low temperatures were found to cause an increase in the degree of desynapsis in *Triticum* (Li, Pao, and Li, 1945) and were able to induce desynapsis in some otherwise normal barley plants (Tsuchiya, 1959); high temperatures were found to increase the degree of desynapsis in *Oryza* (Wang, Yeh, Lee, and Li, 1965) and in rye-grass (Ahloowalia, 1969); and an insufficient water supply was found to increase the degree of desynapsis in *Secale* (Prakken, 1943).

Owing to the variations in the expression of desynapsis of a mutant, in order to characterize different desynaptic mutants, Prakken (1943) proposed to classify them in three types: a "weak" desynaptic type for mutants that exhibit few univalents, a "medium strong" type for mutants with many univalents, and a "complete" desynaptic type for mutants that exhibit mostly univalents with some rare bivalents.

Desynaptic mutants show normal development up to the early meiotic stages, but the subsequent abnormal desynaptic metaphases lead to several cytological abnormalities in the later gametophytic stages.

In desynaptic metaphases, most of the univalents, when not lying too close to one of the poles, move toward the equatorial plate (Prakken, 1943), and according to Ostergren (1951), only these univalents in an equatorial position will divide equationally after the dyads have reached the poles at anaphase I. This has been found to be true of most desynaptic

mutants (e.g., *Pisum*, Koller, 1938, and corn, Dempsey, 1958). However, in a completely desynaptic mutant in *Hordeum spontaneum* C. Koch. all univalents showed longitudinal splitting regardless of their position in the spindle (Tsuchiya, 1959). With some exceptions (e.g., Miller, 1963, in corn and Tsuchiya, 1959, in *Hordeum*), highly or completely desynaptic mutants have been found to show rare splitting of univalents at anaphase I (e.g., Prakken, 1943, in rye). Johnsson (1944) suggested that in medium strong desynaptic mutants the bivalents retard the meiotic division, thereby giving the univalents time to divide, whereas in highly or completely desynaptic mutants the rate of division is so great that the univalents move to the poles undivided.

The poleward movement of unsplit univalents at anaphase I is in general at random (e.g., Enns and Larter, 1960) and thus the univalents are not always evenly distributed to the poles. For example, in *Pisum sativum* L. (n = 7), Gottschalk and Baquar (1971) found that only 12% of the analyzable telophase I cells had the "normal" 7:7 distribution and all distributions up to 12:2 were observed. The "normal" distribution may not indicate the haploid complement because of the random movement of univalents.

Several other cytological abnormalities have been reported in desynaptic plants: clumped chromosomes in the early stages of prophase (e.g., Moens, 1969, in tomato); spindle abnormalities (e.g., Beadle, 1933, in corn); several meiotic stages, from diakinesis to quartets, within the same anther whereas normal plants have a well synchronized meiotic process (e.g., Putt, 1954, in rye); laggards at anaphase I (e.g., Tsuchiya, 1959, in *Hordeum*) that will end up as micronuclei (e.g., Sjödin, 1970, in *Vicia*

and Burnham, 1946, in barley), if they retain their metabolic activity (Celarier, 1955, in *Tradescantia*); misdivision of univalents at anaphase I and II (e.g., Beadle, 1930, in corn); failure of cytokinesis and nuclear fusion (e.g., Miller, 1963, in corn); and tetraploid sporocytes (Tsuchiya, 1959).

It is important to note that the majority of the cytological reports on desynapsis have been done exclusively on microsporocytes. In corn, where studies were done on both microsporocytes and megasporocytes, no significant differences in desynaptic behavior were detected (Beadle, 1933, and Miller, 1963).

High sterility results from the numerous cytological abnormalities present in desynaptic plants. The sterility depends on the frequency of cytological abnormalities in the mutant plant and on the degree of tolerance to those abnormalities by that plant (e.g., tolerance to aneuploidy). In general, there is a positive correlation between sterility and univalent frequency (e.g., in *Avena*; Thomas and Rajhathy, 1966), and usually completely desynaptic mutants are also completely sterile (e.g., in *Pisum*; Gottschalk and Baquar, 1971, and in *Vicia*; Sjödin, 1970). Because desynapsis is environmentally affected, so is the fertility of the plant. For example, variations in fertility have been detected in spikes maturing at different times (e.g., in wheat; Li, Pao, and Li, 1945, and in rice, Katayama, 1961a). The high sterility of desynaptic mutants has been proposed as an alternative method to obtain seedless fruits in watermelon (Kihara, Saito, and Shimotsuma, 1972).

Few studies have been done to explain the mode of action of desynaptic genes. The synaptinemal complexes seemed to be normal in desynaptic

mutants of tomato (Moens, 1969). A relationship may exist between the lack of proline in the anthers of a desynaptic wild *Cassia tora* and its sterility (Katayama, 1961b).

From experiments with desynaptic rye grass where high temperatures increased the degree of desynapsis and treatment with barbiturates decreased desynapsis, Ahloowalia (1969) suggested that desynaptic plants are defective for a thermosensitive compound that participates in maintaining the hydrogen bonds in the coiled structure of the paired chromosomes. The barbiturates form specific hydrogen bonds with adenine derivatives, while temperature variations easily disrupt these hydrogen bonds that may be in some form involved in keeping the chromosomes paired after the initial synapsis.

A positive correlation found between the number of bivalents per cell and the mean number of chiasmata per bivalent in desynaptic maize (Beadle, 1933) and a similar relationship in desynaptic *Avena* (Thomas and Rajhathy, 1966) was explained as being due to the random nature of chiasmata formation in desynaptic plants. This correlation suggests that, since the mode of action of desynaptic genes is by preventing, reducing, or causing premature terminalization of chiasmata, the distribution of the possible desynaptic configurations at metaphase I may also be at random.

Because of the abnormal distribution of univalent chromosomes during meiosis in desynaptic plants, various kinds of aneuploids are expected in their progeny. Aneuploids, the majority of them trisomics and occasionally some double trisomics, were reported in the progenies of desynaptic *Zea* (Beadle, 1930), *Cassia tora* (Katayama, 1953 and 1962),

Hordeum (McLennan, 1947), *Brassica* (Stringam, 1970), and *Oryza* (Katayama, 1963). In fact, desynaptic plants have been proposed as a better source of primary trisomics than the progenies of triploids (e.g., Katayama, 1963, in rice). By contrast, in *Hordeum* a higher frequency of trisomics was obtained from progenies of autotriploid plants than from desynaptic plants (Tsuchiya, 1958 and 1960b).

Trisomics from desynaptic plants were found not to be correlated with seed weight in rice (Katayama, 1966) although in *Avena* (Dyck and Rajhathy, 1965) the light and shriveled seed yielded more trisomics than did the heavy seed.

In *Hordeum distichum nutans*, Ekstrand (1932) reported a single recessive mutant of spontaneous origin with a low degree of desynapsis (two to four univalents at metaphase I), longitudinal splitting of univalents at anaphase I, lagging chromosomes at anaphase II, and very high sterility (70% to 90%).

A single recessive "long" chromosome mutant was found among the progeny of an x-ray-treated plant (cultivar 'Mars'; CI 7015) (Burnham, 1946). It was assigned the symbol *lc* for "long" chromosome because of the frequent occurrence of rodlike bivalents at metaphase I. Plants with this mutant gene, located on chromosome 1, have a low degree of desynapsis, with many rod bivalents and few univalents at metaphase I, have high sterility, and show some trisomic progeny (McLennan, 1947; McLennan and Burnham, 1948). Because the chromosomes are paired at pachytene, this *lc* mutant is really a desynaptic one, and under the new rules for genetic symbolization adopted by the Eighth American Barley Research Workers Conference, the symbol *lc* was changed to *des1a* (Ramage and Hernandez-Soriano, 1972).

A "short" chromosome mutant was found among the progeny of a plant (*Hordeum vulgare* L.) subjected to atomic bomb irradiation. In this single recessive mutant, synapsis occurs at early prophase. At diakinesis the chromosomes are more condensed or shorter than in normal plants and undergo precocious terminalization of all chiasmata, resulting in 14 univalents at metaphase I. Many other abnormalities are also present in this completely sterile mutant, which has to be maintained in a heterozygous condition (Moh and Nilan, 1954).

From the progeny of x-ray-treated wild barley *Hordeum spontaneum* C. Koch var. *Transcaspicum*, a completely desynaptic mutant was obtained. The mutant was completely sterile and was lost for future studies (Tsuchiya, 1959 and 1960a).

A single recessive desynaptic mutant was found among the progeny of a semisterile plant obtained through x-ray-treated seed of *Hordeum vulgare* (cultivar 'Husky'; CI 9537) (Enns and Larter, 1960). This mutant with medium strong desynapsis and high sterility was located on chromosome 3 and assigned the symbol *ds* (Enns and Larter, 1962). The symbol *ds* was later changed to *des2b* (Ramage and Hernandez-Soriano, 1972).

MATERIALS AND METHODS

Partially sterile plants were collected in commercial fields of *Hordeum vulgare* cultivar Betzes (CI 6398) in 1967 and 1968 (cf. Hockett and Eslick, 1969). Progenies of the partially sterile plants were examined and both cytological (e.g., aneuploids, translocations) and genetic (e.g., male sterility, desynapsis) causes of partial sterility were found. From these plants ten desynaptic lines were isolated and assigned genetic symbols according to the rules for genetic symbolization adopted by the Eighth American Barley Research Workers Conference as reported in *Barley Genetic Newsletter* (Ramage and Hernandez-Soriano, 1971 and 1972). Mutant designations of the ten desynaptic lines and the sterility of their parental plants are given in Table 1.

Table 1. Sterility of Ten Plants Selected for Partial Sterility that Proved to Be Desynaptics.

Mutant designation	Plant number	No. of spikes	No. of flowers	No. of seeds	Percent sterility
des,,c	B-67-N-5	3	60	13	78
des,,d	B-67-N-12	2	44	10	77
des,,e	B-67-F-32	7	172	41	76
des,,f	B-67-F-44	12	286	48	83
des,,g	B-67-F-84	13	324	71	78
des,,h	B-68-N-5	4	71	18	75
des,,i	B-68-N-57	3	61	12	80
des,,j	B-68-N-85	6	117	31	74
des,,k	B-68-N-146	8	184	64	65
des,,l	B-68-N-154	5	92	7	92

The previously described genes for desynapsis *des1a* and *des2b* were obtained from the Barley Genetic Stock Center at Fort Collins, Colorado.

The procedures used in the different studies are described below. Numbers correspond to the numbers of the subheadings in the chapter entitled "Results and Discussion."

1. Inheritance of Desynapsis. Progenies of the ten desynaptic mutants in Betzes were examined cytologically to confirm desynapsis and were crossed as females with normal Betzes. The F_1's were examined cytologically and grown to maturity. F_2 plants were classified as normal or desynaptic based on ovule sterility; highly sterile plants were classified as homozygous for the desynaptic gene, and plants with normal fertility were classified as either homozygous or heterozygous for the normal allele.
2. Allele Test. A complete diallele cross was attempted among the ten Betzes mutants and the two previously described mutants *des1a* and *des2b*. All plants involved in crosses were examined cytologically. In some cases pollen from certain desynaptic plants was difficult to obtain, so pollen from heterozygous (*Des des*) plants was used. All F_1's were examined cytologically at metaphase I and classified as either normal or desynaptic.
3. Cytological Studies. All cytological observations were made on microsporocytes. Spikes were collected in a solution of 3 parts absolute ethanol to 1 part glacial acetic acid and were either immediately analyzed or preserved in 70% ethanol at approximately

5°C for future studies. All material was studied using the acetocarmine smear technique. Besides the cytological examination of all meiotic stages of all mutants, the following analyses were carried out:

Early Stages of Prophase: All Betzes mutants were examined at the early stages of prophase to determine the extent of pairing.

MI Configurations: The exact metaphase I configurations, ring bivalents, rod bivalents, and univalents per cell of analyzable cells were recorded for all desynaptic mutants in 1971. In 1972 the analysis was repeated but only on six mutants representing the six different desynaptic loci found among the Betzes mutants.

Anaphase I: The number of chromosomes at each pole and the number of univalents undergoing precocious separation in the equatorial plate of the analyzable anaphase I cells were recorded in three of the mutants grown in 1972.

Micronuclei per Quartet: The total number of micronuclei per analyzable quartet was recorded for all the desynaptic mutants grown in 1971.

4. Fertility. Ovule fertility was determined by counting all seeds set and dividing by the total number of fully developed spikelets, expressing the result as percentages. The averages of the fertilities of two plants per mutant for the 1971 data and three plants per mutant for the 1972 data were used as the final expression of fertility per mutant.

5. Correlation of Seed Weight with Trisomic Frequency. Spikes from two mutants (*des,,i* and *des,,j*) were harvested and all seed was recovered (the light seed is often lost in the mechanical threshing). Two hundred seeds from *des,,j* and 100 seeds from *des,,i* were randomly selected and individually weighed. Seeds were grown to maturity and all plants were classified for chromosome number and trisomic type based on cytology and plant morphology.

6. Location of Genes by Trisomic Inheritance. Two methods were employed to obtain trisomic F_1 plants: (1) Trisomics were isolated from selfed progenies of each desynaptic line and pollinated with normal *Des* pollen. The F_1's were grown and examined cytologically, and normal trisomic F_1 plants were selected. (2) Diploid desynaptic plants were pollinated with normal *Des* pollen. The F_1's were grown and examined cytologically, and normal trisomic F_1 plants were selected. With the first method it was easy to obtain trisomic desynaptic plants (only the light seed was used), but very few crossed seeds were obtained owing to trisomic and desynaptic sterilities, and the F_1 trisomics obtained were in many cases different from the maternal desynaptic trisomic (the abnormal distribution of megasporocytic chromosomes in the trisomic desynaptic plant gave rise to new trisomic types). The second method is more practical since desynaptic plants set more seeds when pollinated with normal pollen. Also, this method requires one less generation. The F_1 normal trisomics obtained by either method were morphologically

classified according to a modification of Tsuchiya's description (1958 and 1960b) by Eslick and Ramage (1969) using the standard nomenclature (Hagberg and Tjio, 1951) for trisomic types. These F_1 trisomics were either *Des des*, if the locus was not carried on the chromosomes involved in the trisome, or *Des des des* if the locus was carried on the chromosomes involved in the trisome. F_2 seeds from all the trisomics obtained were grown in 12-oz. aluminum cans, three to four plants per can. Under these conditions trisomic plants in the F_2 were crowded out and seldom reached maturity. The remaining F_2 diploid plants were classified as normal or desynaptic based on ovule sterility, and the F_2's were classified as being in a disomic ratio (3 *Des*:1 *des*) or trisomic ratio (5 *Des*:4 *des*).

RESULTS AND DISCUSSION

1. Inheritance of Desynapsis

Desynaptic plants from the ten Betzes mutants were crossed with normal Betzes. The F_1's were examined cytologically, and all were found to have normal metaphase I cells and all plants were completely fertile. The F_2's were classified as either normal or desynaptic on the basis of ovule sterility. The results of the F_2 segregation (Table 2) show that desynapsis appears to be controlled by a single recessive gene in all of the ten Betzes mutants.

Table 2. F_2 Segregation of Crosses Between Desynaptic Mutants and Normal Betzes.

Mutant	F_2 segregation		χ^2 fit to a 3:1	P (1 d.f.)
	Normal	Desynaptic		
des,,c	29	11	.100	.75 - .90
des,,d	53	21	.450	.50 - .75
des,,e	106	36	.007	.90 - .95
des,,f	59	11	2.414	.10 - .25
des,,g	51	22	1.027	.25 - .50
des,,h	41	18	.716	.25 - .50
des,,i	39	14	.057	.75 - .90
des,,j	45	19	.750	.25 - .50
des,,k	60	22	.146	.50 - .75
des,,l	38	11	.463	.25 - .50

2. Allele Test

F_1 plants from intercrosses among desynaptic mutants were examined cytologically at metaphase I and classified as normal or desynaptic. The resulting classifications are given in Table 3. In Table 3, the top line in each cell gives the cytological classification (*D* for normal and *d* for desynapsis) when the parents were as indicated, and the bottom line of each cell gives the results of reciprocal crosses. Results with an * sign indicate that the paternal line in the cross was heterozygous for desynapsis and thus if the parents were allelic a 1 *Des*:1 *des* ratio is expected. The number found is indicated in Table 3 by an a:b ratio (for normal vs. desynaptic). For example, the result of the allele test between *des,,d* and *des,,h* is as follows: The F_1 of *des,,d* × *des,,h* contained 3 desynaptic plants (top line) and the F_1 of *des,,h* × *des,,d* × Betzes contained 3 normal and 2 desynaptic plants (bottom line). Both results indicate that *des,,d* and *des,,h* are allelic.

A diallele cross among the 10 Betzes desynaptic mutants plus the two previously described mutants *des1a* and *des2b* was not completed, but sufficient information was obtained to assign locus numbers to all of the Betzes desynaptic mutants.

The allele test showed that *des,,c* is not allelic to any of the other mutants. It was assigned locus number 3 and symbolized *des3c*. The mutants *des,,d* and *des,,h* were found to be allelic to each other and not allelic to the other mutants. They were assigned locus number 4 and symbolized *des4d* and *des4h*. The mutants *des,,e*, *des,,f*, and *des,,g* are allelic to each other and not allelic to the other mutants. They were assigned locus number 5 and symbolized *des5e*, *des5f*, and *des5g*.

Table 3. Results of the Allele Test.

Top line in each cell is the cross as indicated in the table; bottom line is the reciprocal cross. Asterisk (*) indicates that a heterozygous (Des des) male parent was used; D = normal, d = desynaptic, a:b = ratio of Des:des plants (a heterozygous male parent was used).

♀ \ ♂	des2b	des,,c	des,,d	des,,e	des,,f	des,,g	des,,h	des,,i	des,,j	des,,k	des,,l
des1a	2D 2D	1D 6D	6D -	4D -	4D 1D	5D -	9D -	5D -	- -	7D 8D	9D 6D
des2b		5D 4D	7D -	3D -	5D 3D	6D -	5D -	3D -	4D -	7D 9D	6D 1D
des,,c			3D 3D	3D 3D	3D 5D*	3D 3D	2D 5D*	1D -	3D 3D	- 5D	- 4D
des,,d				3D 2D	2D 3D	3D 2D	3d 3:2*	5D -	3D 3D	- 10D	- 8D
des,,e					2d 1:4*	1d 2d	2D 4D	1D 4D	2D 3D	- 10D	- 6D
des,,f						3:2* 2:2*	5D* 5D*	5D* 1D	3D 2D	- 3D	- -
des,,g							5D* 2D	4D 1D	3D 3D	- 10D	- 5D
des,,h								3D 3D	2D 3D	- 9D	- 9D
des,,i									- 6D	- 7D	- 9D
des,,j										- 10D	- 10D
des,,k											10d 8d

All F_1's between nonallelic mutants showed normal MI configurations except where the *des5* mutants were involved. When *des5* mutants were crossed with all other nonallelic mutants, the F_1's always showed a low degree of desynapsis (up to 3 rod bivalents) and a very low sterility (up to 10% sterility). These F_1's were classified as normal in Table 3 although both the cytological and ovule sterility observations were different from those of normal plants or from those of plants homozygous for *des5*.

The mutant *des,,i* is not allelic to any of the other mutants and was assigned locus number 6 and symbolized *des6i*. The mutant *des,,j* was not crossed with *des1a*, but the latter mutant has been reported to be located on chromosome 1 (McLennan, 1947) whereas *des,,j* was found in the present study to be located on chromosome 2. Because *des,,j* was found to be nonallelic with the remaining mutants, it was assigned locus number 7 and symbolized *des7j*. The mutants *des,,k* and *des,,l* are allelic to each other and not allelic to the other mutants. They were assigned locus number 8 and symbolized *des8k* and *des8l*.

The previous and new symbolizations for all barley desynaptic mutants are shown in Table 4.

Table 4. New and Previous Symbolization for the Barley Desynaptic Mutants.

New	Previous	New	Previous
des1a	1c	des5f	B-67-F-44
des2b	ds	des5g	B-67-F-84
des3c	B-67-N-5	des6i	B-68-N-57
des4d	B-67-N-12	des7j	B-68-N-85
des4h	B-68-N-5	des8k	B-68-N-146
des5e	B-67-F-32	des81	B-68-N-154

3. Cytological Studies

3-a. Prophases

The early stages of prophase up to diplotene, in all the Betzes mutant lines, were found to be indistinguishable from those of normal barley plants as illustrated in Figs. 1-A to 1-D. Then, whereas in normal plants the homologous chromosomes remain paired up to anaphase I (Figs. 1-E to 1-H), in all of the Betzes lines some unpaired chromosomes can be detected in late diplotene and early diakinesis (Figs. 1-I and 1-J) although not as frequently or as easily as in late diakinesis or metaphase I (Figs. 1-K and 1-L). Because of this cytological behavior, all of the Betzes lines were classified as being desynaptic mutants.

All the mutants showed well synchronized stages throughout prophase.

Fig. 1. Prophase of Normal and Desynaptic Cells.

A. Desynaptic leptotene.

B. Desynaptic early pachytene.

C. Desynaptic late pachytene.

D. Desynaptic early diplotene showing all chromosomes paired.

E. Normal late diplotene.

F. Normal early diakinesis.

G. Normal late diakinesis.

H. Normal metaphase I.

I. Desynaptic late diplotene showing some unpaired chromosome arms.

J. Desynaptic early diakinesis showing one rod bivalent.

K. Desynaptic late diakinesis showing three ring bivalents, three rod bivalents, and two univalents.

L. Desynaptic metaphase I showing one ring bivalent and six rod bivalents.

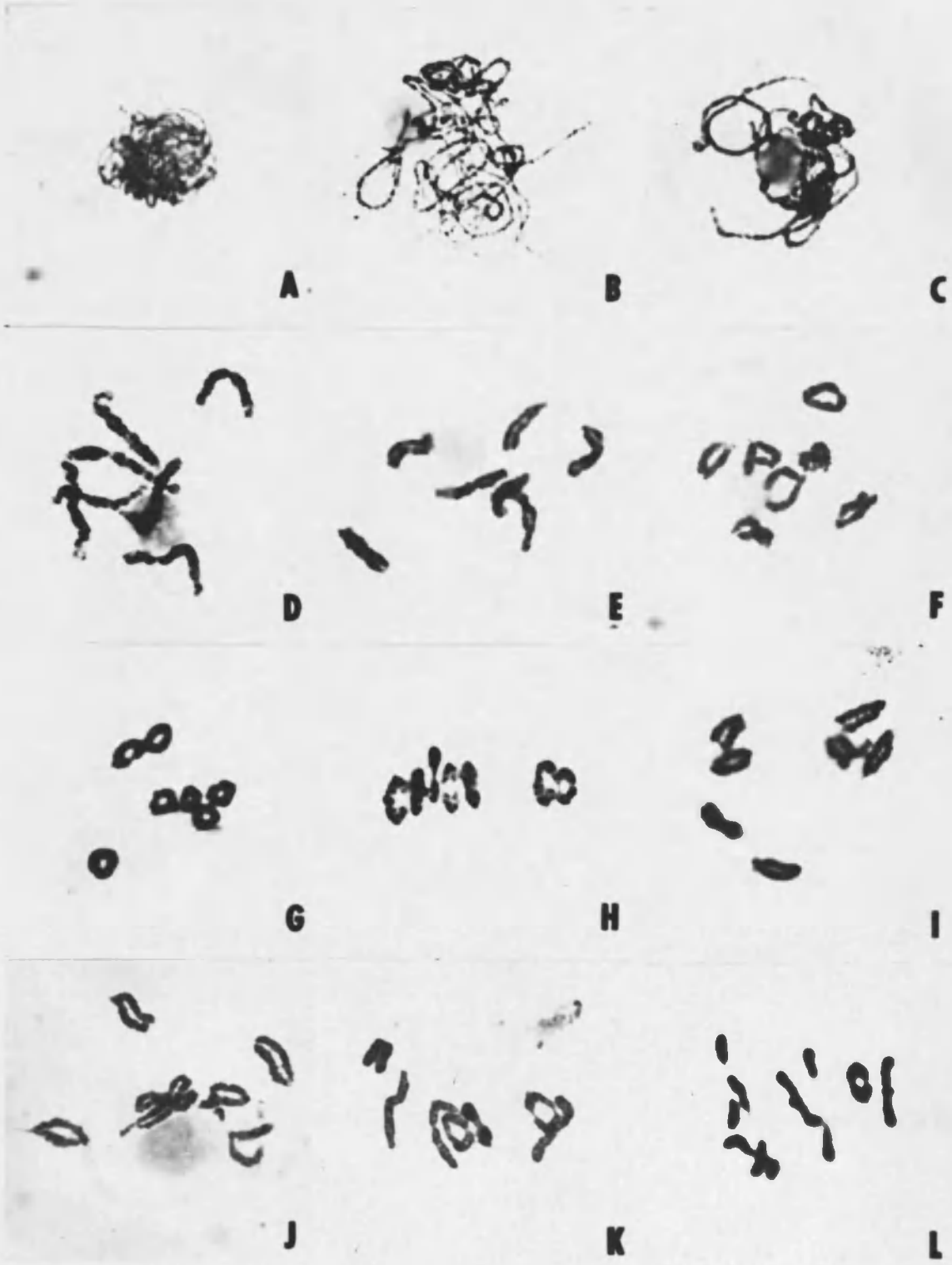

Fig. 1. Prophase of Normal and Desynaptic Cells.

3-b. Metaphase Configurations

Metaphase I cells of desynaptic plants have a variable number of unpaired chromosome arms. This number depends on the degree of desynapsis of the mutant plants.

The metaphase I configuration of a desynaptic cell depends on the number of unpairing events and on where these events occur. If one of the two arms of a chromosome fails to remain paired with its homologue, a long rodlike bivalent is formed. If both arms fail to remain paired, two univalents are formed. Thus, metaphase I cells of desynaptic plants have different combinations of ring bivalents, rod bivalents, and univalents (see Figs. 2 and 3).

In most of the previous reports, the degree of desynapsis of a mutant has been expressed in terms of univalent frequency at metaphase I. This equivalence can be very misleading. For example, in a $2n = 14$ organism, metaphase I cells with 6II + 2I and cells with 6R + 2I (where II = ring bivalent, R = rod bivalent, and I = univalent) have the same degree of desynapsis if classified according to the number of univalents per cell, but the 6II + 2I cells have only two pairs of chromosome arms unpaired while the 6R + 2I cells have eight.

For clarity in the present study the degree of desynapsis of a cell is expressed in terms of the number of unpaired chromosome arms at metaphase I. That is, the degree of desynapsis (d) of a cell is mathematically expressed as $d = r + i$, where r and i are the numbers of rod bivalents and univalents, respectively.

The total number N of metaphase I configurations made up of all possible combinations of ring bivalents, rod bivalents, and univalents

Fig. 2. Metaphase I Configurations of Cells with Desynapsis of 1 to 7.

A. MI configuration: 6II + 1R, degree of desynapsis = 1
B. MI configuration: 5II + 2R, degree of desynapsis = 2
C. MI configuration: 4II + 3R, degree of desynapsis = 3
D. MI configuration: 3II + 4R, degree of desynapsis = 4
E. MI configuration: 5II + 4I, degree of desynapsis = 4
F. MI configuration: 4II + 2R + 2I, degree of desynapsis = 4
G. MI configuration: 2II + 5R, degree of desynapsis = 5
H. MI configuration: 4II + 1R + 4I, degree of desynapsis = 5
I. MI configuration: 3II + 2R + 4I, degree of desynapsis = 6
J. MI configuration: 2II + 4R + 2I, degree of desynapsis = 6
K. MI configuration: 7R, degree of desynapsis = 7
L. MI configuration: 1II + 5R + 2I, degree of desynapsis = 7

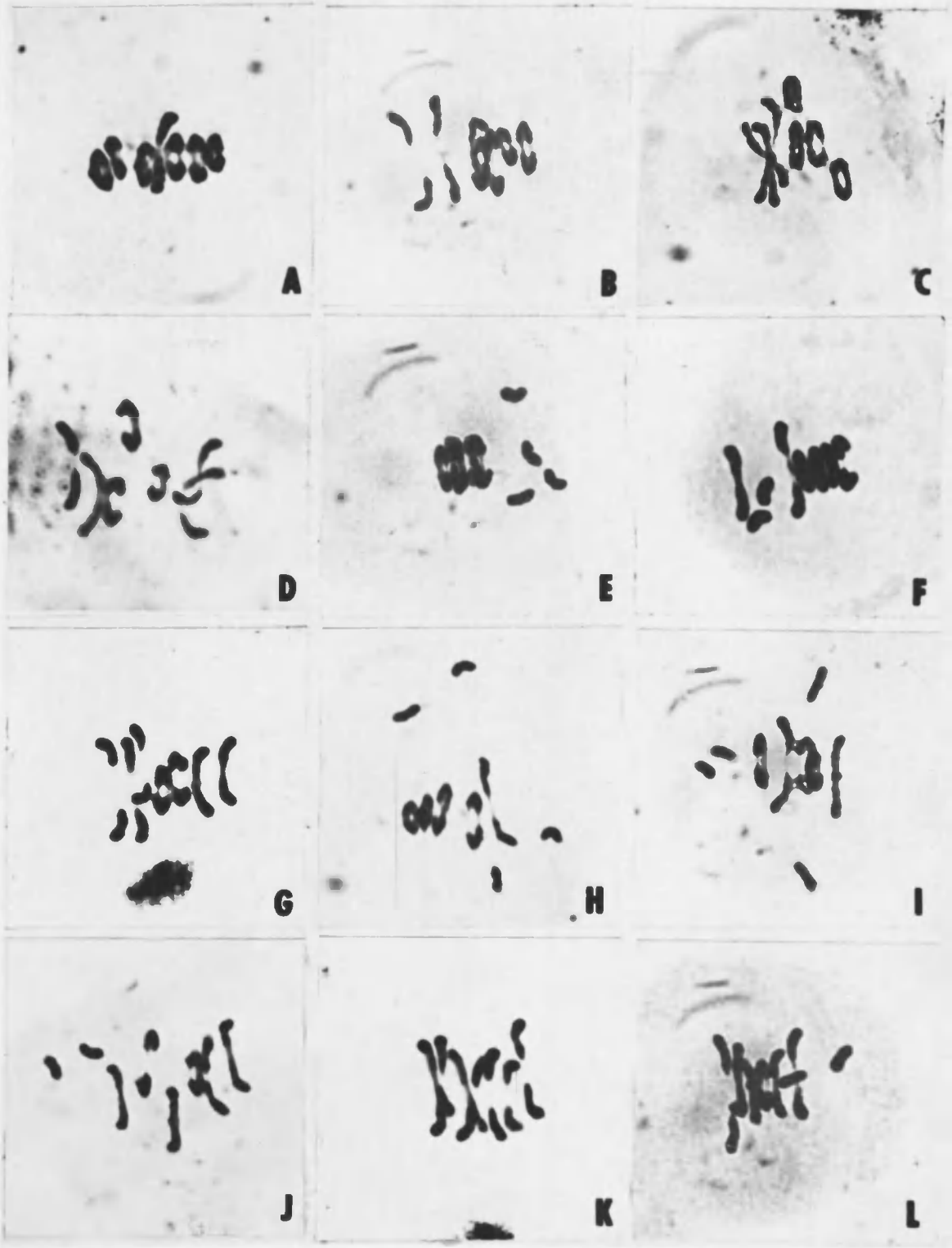

Fig. 2. Metaphase I Configurations of Cells with Desynapsis of 1 to 7.

Fig. 3. Metaphase I Configurations of Cells with Desynapsis of 7 to 14.

A. MI configuration: 2II + 3R + 4I, degree of desynapsis = 7
B. MI configuration: 3II + 1R + 6I, degree of desynapsis = 7
C. MI configuration: 2II + 2R + 6I, degree of desynapsis = 8
D. MI configuration: 6R + 2I, degree of desynapsis = 8
E. MI configuration: 5R + 4I, degree of desynapsis = 9
F. MI configuration: 2II + 10I, degree of desynapsis = 10
G. MI configuration: 1II + 2R + 8I, degree of desynapsis = 10
H. MI configuration: 4R + 6I, degree of desynapsis = 10
I. MI configuration: 1II + 2R + 8I, degree of desynapsis = 10
J. MI configuration: 2R + 10I, degree of desynapsis = 12
K. MI configuration: 1II + 12I, degree of desynapsis = 12
L. MI configuration: 14I, degree of desynapsis = 14

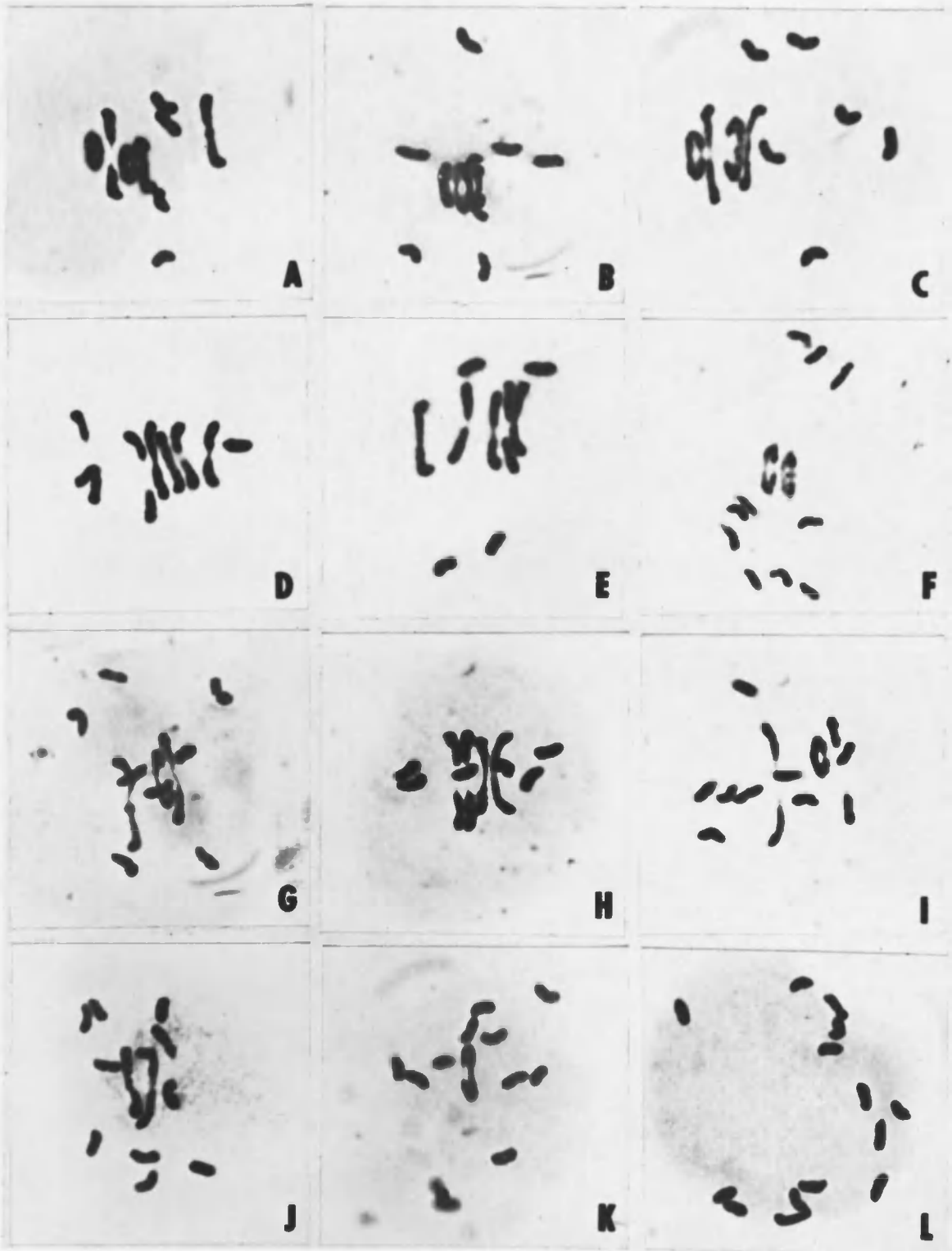

Fig. 3. Metaphase I Configurations of Cells with Desynapsis of 7 to 14.

in a $2n$ organism is equal to $[(n+1)^2 + (n+1)]/2$. These N configurations can be classified according to their degree of desynapsis. Since the degree of desynapsis can range from 0 (all ring bivalents) to $2n$ (all univalents), there are $2n+1$ possible desynaptic classes. In barley ($2n = 14$) there are 36 possible metaphasic configurations and 15 desynaptic classes, as shown in Table 5. Most of these configurations are illustrated in Figs. 2 and 3.

All barley desynaptic mutants were cytologically analyzed in 1971 and the number of cells for each metaphase I configuration was recorded (Table 6).

The following conclusions can be drawn from Table 6: (a) Since the cytological analysis was done prior to the completion of the allele test, mutants that were later found to be allelic showed very similar frequency distributions, e.g., *des5e*, *des5f*, and *des5g*. (b) Mutants that later proved to be nonallelic showed highly different frequency distributions, e.g., *des3c* and *des5*. (c) In each mutant the frequencies of metaphase I configurations having the same degree of desynapsis were in many cases quite different. These differences seem to follow similar patterns for all mutants; e.g., 6II + 2I cells were observed much less frequently in all mutants than cells with 5II + 2R, and both types of cells have a degree of desynapsis of 2; the same was true for metaphase I cells with a degree of desynapsis of 3, where 5II + 1R + 2I cells were observed less frequently than cells with 4II + 3R, and so on.

When it was realized that the recorded metaphase I frequencies could be altered by a misclassification of the proper metaphase I configurations or the identification of only the easily analyzable cells

Table 5. Theoretical Possible Metaphase I Configurations Classified According to Degree of Desynapsis in a $2n = 14$ Desynaptic Organism.

Abbreviations: d = degree of desynapsis, II = ring bivalent, R = rod bivalent, I = univalent.

	Metaphase configuration											
d	II	R	I	II	R	I	II	R	I	II	R	I
0	7	-	-									
1	6	1	-									
2	5	2	-	6	-	2						
3	4	3	-	5	1	2						
4	3	4	-	4	2	2	5	-	4			
5	2	5	-	3	3	2	4	1	4			
6	1	6	-	2	4	2	3	2	4	4	-	6
7	-	7	-	1	5	2	2	3	4	3	1	6
8	-	6	2	1	4	4	2	2	6	3	-	8
9	-	5	4	1	3	6	2	1	8			
10	-	4	6	1	2	8	2	-	10			
11	-	3	8	1	1	10						
12	1	-	12	-	2	10						
13	-	1	12									
14	-	-	14									

Table 6. Number of Cells per Metaphase I Configuration Observed in Microsporocytes of the Mutants Grown in 1971.

	M I			Desynaptic mutants											
d	II	R	I	des1a	des2b	des3c	des4d	des4h	des5e	des5f	des5g	des6i	des7j	des8k	des81
0	7	-	-	19	13	254	45	35	-	5	2	-	25	-	-
1	6	1	-	85	19	86	39	33	2	3	2	2	34	-	-
2	5	2	-	60	27	24	45	26	-	5	2	2	64	-	-
2	6	-	2	17	3	7	6	6	2	-	1	-	5	-	-
3	4	3	-	52	22	2	34	24	-	6	3	1	44	-	-
3	5	1	2	20	14	1	11	15	-	-	2	1	18	-	-
4	3	4	-	40	17	-	25	9	2	4	-	1	31	6	-
4	4	2	2	16	27	-	21	11	-	5	4	6	47	-	1
4	5	-	4	3	5	-	2	4	8	3	-	-	7	-	5
5	2	5	-	13	6	-	5	3	2	2	-	1	14	5	-
5	3	3	2	9	20	-	16	9	-	5	1	6	45	9	1
5	4	1	4	2	4	-	21	4	2	4	2	1	17	1	-
6	1	6	-	4	3	38	11	28	-	2	6	1	22	2	1
6	2	4	2	5	12	-	13	4	3	1	2	13	29	11	2
6	3	2	4	4	12	-	8	9	1	11	5	6	43	14	1
6	4	-	6	1	4	-	1	-	14	3	-	-	14	1	-
7	-	7	-	-	2	-	8	-	-	4	-	2	-	1	-
7	1	5	2	2	10	-	2	-	4	1	-	10	20	8	2
7	2	3	4	-	14	-	3	2	-	4	4	24	38	20	12
7	3	1	6	-	4	-	9	-	-	7	3	3	17	7	6
8	-	6	2	-	2	-	6	2	3	5	-	8	16	5	2
8	1	4	4	1	10	-	2	-	10	4	3	29	24	20	11
8	2	2	6	1	13	-	4	1	5	11	12	28	37	32	14
8	3	-	8	-	2	-	1	1	17	1	2	4	6	1	-
9	-	5	4	1	9	-	3	1	6	13	3	31	17	14	5
9	1	3	6	-	22	-	5	-	4	16	8	41	27	28	37
9	2	1	8	-	10	-	-	-	1	13	10	13	13	4	4
10	-	4	6	-	11	-	5	-	20	54	6	39	15	29	25
10	1	2	8	-	10	-	1	-	41	19	25	47	20	28	30
10	2	-	10	-	-	-	-	-	12	8	1	6	1	-	-
11	-	3	8	-	18	-	2	-	37	102	36	55	14	35	48
11	1	1	10	-	10	-	-	-	1	6	50	25	9	7	4
12	1	-	12	-	1	-	-	-	26	15	26	9	1	-	-
12	-	2	10	-	14	-	-	-	101	125	75	43	7	21	37
13	-	1	12	-	-	-	-	-	128	92	141	23	3	3	9
14	-	-	14	-	-	-	-	-	99	5	160	3	1	-	1
Total cells				355	370	412	354	227	551	564	597	484	745	312	258

(cells with many univalents are more easily identified than cells with combinations of ring bivalents, rod bivalents, and univalents), a much more thorough analysis was carried out in 1972 with six different desynaptic mutants representing the six loci found in Betzes. The data (Table 7) showed that, as in the 1971 data, there were similar differences in magnitude in all mutants between the different metaphase I configurations of equal degree of desynapsis.

3-c. Desynaptic Mode of Action

The phenotypic expression of a particular desynaptic mutant can be characterized (as done in the previous section) by the frequencies of each one of all the possible cell configurations at metaphase I. This method, although accurate, can be elaborate because of the high number of different cell types. The higher the haploid number the more elaborate is this method. For example, in a desynaptic corn ($n = 10$) there are 66 possible metaphase I configurations. In general, most authors have simplified the task of characterizing a desynaptic mutant by giving only the frequencies of cells with different numbers of univalents, a simplification that was criticized in the previous section. Another procedure to characterize a mutant can be by giving the frequency of each desynaptic class (as defined in the present study). With this method there are $2n + 1$ possible types of cells. The problem arises in whether it is or is not permissible to pool cells of different metaphase I configurations but equal degree of desynapsis under a single desynaptic class frequency. As seen in the previous section, these configurations were not equally distributed within each desynaptic class although in

Table 7. Number of Cells per Metaphase I Configuration Observed in Microsporocytes of the Betzes Mutants Grown in 1972.

	M I			Desynaptic mutants					
d	II	R	I	des3c	des4d	des5e	des6i	des7j	des8h
0	7	-	-	480	50	-	5	4	5
1	6	1	-	136	91	-	4	5	20
2	5	2	-	21	126	-	8	12	28
2	6	-	2	2	12	-	1	4	2
3	4	3	-	5	113	-	15	29	35
3	5	1	2	-	24	-	4	5	18
4	3	4	-	-	47	1	25	27	43
4	4	2	2	1	24	1	15	15	28
4	5	-	4	-	2	-	1	-	5
5	-	5	-	-	21	5	26	25	23
5	3	3	2	-	28	1	31	35	43
5	4	1	4	-	5	2	5	12	12
6	1	6	-	-	6	-	15	18	12
6	2	4	2	-	8	9	35	46	56
6	3	2	4	-	7	3	19	26	31
6	4	-	6	-	-	-	1	1	7
7	-	7	-	-	-	1	7	10	4
7	1	5	2	-	1	7	42	34	23
7	2	3	4	-	12	10	35	40	41
7	3	1	6	-	1	2	8	12	5
8	-	6	2	-	-	11	17	12	5
8	1	4	4	-	4	15	44	30	27
8	2	2	6	-	1	15	17	22	29
8	3	-	8	-	-	2	3	3	6
9	-	5	4	-	1	17	21	21	20
9	1	3	6	-	2	39	17	25	25
9	2	1	8	-	-	4	4	8	7
10	-	4	6	-	-	54	18	21	19
10	1	2	8	-	1	51	7	12	12
10	2	-	10	-	-	1	1	3	3
11	-	3	8	-	-	103	8	17	18
11	1	1	10	-	-	30	1	3	3
12	1	-	12	-	-	17	-	-	-
12	-	2	10	-	-	179	4	3	9
13	-	1	12	-	-	191	1	2	1
14	-	-	14	-	-	180	-	-	2
Total cells				645	587	951	465	542	627

all mutants similar distributions were observed. If a rule is established governing the distribution of all subclasses (or metaphase I configurations) within each desynaptic class, then it will be permissible to pool the subclasses' frequencies under a single class frequency.

Previous reports, based mainly on chiasmata frequency studies, suggested that desynapsis may be of a random nature (e.g., Beadle, 1933, and Thomas and Rajhathy, 1966). This implies that in a desynaptic cell the chromosome arms separate at random during prophase. The subsequent metaphasic configuration of that cell will then be a function of which ones of the total $2n$ pairs of chromosome arms did separate. For example, if two unpairing events occur at random in a desynaptic cell, it is more likely that these events occur in two different ring bivalents than within the same ring bivalent. That is, in a $2n = 14$ organism, the final metaphase configuration of that cell will more likely be 5II + 2R than 6II + 2I. If within each desynaptic class the metaphasic configurations occur in a random pattern, then a desynaptic mutant can be characterized by giving the different desynaptic class frequencies.

The expected probability P of a particular metaphase I cell with a d degree of desynapsis if the unpairing mechanism is of a random nature is

$$P\left[n - (r + i/2)II + rR + iI\right] = \frac{\binom{n - i/2}{r} 2^r \binom{n}{i/2}}{\binom{2n}{d}}$$

where n = haploid number, r = number of rod bivalents, i = number of univalents, and d = degree of desynapsis = $r + i$. The sum of all probabilities within each desynaptic class is equal to one.

The probabilities of all the metaphase I configurations in a $2n = 14$ organism, when d varies from 0 to 14 and the unpairing mechanism is random, are shown in Table 8. For example, in Table 8, P(6II + 2I) = .0769 and P(5II + 2R) = .9231; both have a degree of desynapsis of 2, but as mentioned before, 5II + 2R cells are more likely to occur than 6II + 2I cells, an occurrence that was also observed in the data of the previous section.

The observed data on metaphase I configurations were tested with the expected probabilities by means of χ^2 tests (with the appropriate number of degrees of freedom for each case). Every desynaptic class except classes 0, 1, 13, and 14 (where there is only one possible configuration) was independently tested.

When the 1971 data (where the frequencies of metaphase I configurations of allelic mutants were pooled together) were tested, the results (Table 9) showed a poor approximation to a random model. Of 74 desynaptic classes tested, only 43 (58%) were not significantly different at the .01 level. Because of this poor agreement, the χ^2's for the total numbers showed a poor fit. Only the total for an 11 degree of desynapsis was not significantly different at the .01 level.

Some of the mutants behaved in the test much better than others. Of these, *des1* showed an unusually good fit; only one desynaptic class, $d = 2$, deviated significantly from the expected random model. This mutant was reported to express an unusual number of rod bivalents at metaphase (Burnham, 1946), but the χ^2 tests indicate that the observed metaphase I configurations seem to follow a random pattern.

Table 8. Probabilities of Every Metaphase I Configuration in a $2n = 14$ Desynaptic Organism if the Unpairing Mechanism Is of a Random Nature.

Abbreviations: d = degree of desynapsis, II = ring bivalent, R = rod bivalent, I = univalent. Configurations in the same line have the same probability.

	M I				M I			
d	II	R	I	Probability	II	R	I	d
0	7	-	-	1	-	-	14	14
1	6	1	-	1	-	1	12	13
2	5	2	-	.9231	-	2	10	12
2	6	-	2	.0769	1	-	12	12
3	4	3	-	.7692	-	3	8	11
3	5	1	2	.2308	1	1	10	11
4	3	4	-	.5594	-	4	6	10
4	4	2	2	.4196	1	2	8	10
4	5	-	4	.0210	2	-	10	10
5	2	5	-	.3357	-	5	4	9
5	3	3	2	.5594	1	3	6	9
5	4	1	4	.1049	2	1	8	9
6	1	6	-	.1492	-	6	2	8
6	2	4	2	.5594	1	4	4	8
6	3	2	4	.2797	2	2	6	8
6	4	-	6	.0117	3	-	8	8
7	-	7	-	.0373				
7	1	5	2	.3916				
7	2	3	4	.4895				
7	3	1	6	.0816				

Table 9. Results of χ^2 for the 1971 Data.

The observed metaphase configurations in each desynaptic class of the mutants grown in 1971 (from Table 6 with pooled data for allelic mutants) were tested with the expected probabilities of a random model (Table 8). Note: ** = probability of a larger $\chi^2 < .01$, and Total $\chi^2 = \chi^2$ for the total number of observations.

Des. class	des1	des2	des3	des4	des5	des6	des7	des8	Total χ^2
2	22.45**	.23	9.68**	5.36	7.01**	.71	.02		26.37**
3	.90	5.07	.18	2.93	.15	.82	1.24		8.08**
4	7.37	21.29**		14.34**	205.50**	5.51	24.59**	92.76**	215.03**
5	4.61	2.49		67.27**	22.12**	1.61	15.56**	.42	56.91**
6	7.04	39.97**	236.00**	85.86**	498.40**	1.88	152.28**	7.14	373.03**
7	3.11	1.97		91.41**	41.48**	3.30	25.17**	23.37	82.88**
8	.68	15.65**		34.02**	446.70**	19.45**	48.42**	8.31	299.30**
9	1.99	10.93**		1.26	38.62**	2.99	9.23**	8.42	27.81**
10		.65		1.85	80.99**	13.41**	2.98	6.12	46.91**
11		2.52		.60	.29	3.01	3.34	6.85	.56
12		.02			57.33**	6.78**	.26	4.83	43.80**

It should be noted that the low number of cells recorded, combined with the low values of some of the expected probabilities (e.g., P(4II + 6I) = .0117) will give a poor approximation in a χ^2 test.

The 1972 data (Table 7) when tested showed a better agreement with the random model (Table 10). Of 54 desynaptic classes tested, 12 (23%) deviated significantly at the .05 level but only 5 (10%) at the .01 level.

The results of Table 10 when analyzed are as follows:

(A) For the different desynaptic classes (rows on Table 10), all classes tested except d = 7, 6, and 8 (save for minor deviations) did not deviate significantly from the expected model. A reason for these three desynaptic classes to depart from the expected model may lie, as mentioned before, in the fact that the classes have a high number of subclasses (or metaphase I configurations), some with very low expected probabilities and the total number of observations made is far below the minimum required for a normal approximation. Another explanation could be that these classes do not behave as in the expected model.

(B) When the behavior of the different desynaptic mutants is compared (columns in Table 10), the following conclusions can be made: Mutants *des3c*, *des4h*, *des5e*, and *des6i* (save for minor deviations) showed a random unpairing mechanism for desynapsis, but mutants *des7j* and *des8k* showed two classes deviating (at the .01 level) from the expected model.

The greatest departures from the expected model were found, as mentioned before, in classes that have metaphase I configurations with very low probabilities. For example, in the case of *des7j* and desynaptic

Table 10. Results of χ^2 for the 1972 Data.

The observed metaphase configurations in each desynaptic class of the mutants grown in 1972 (Table 7) were tested with the expected probabilities of a random model (Table 8). Note: * = probability of a larger $\chi^2 < .05$, ** = probability of a larger $\chi^2 < .01$, Homo. χ^2 = χ^2 for homogeneity, Total χ^2 = χ^2 for the total number of observations.

Des. class	des3	des4	des5	des6	des7	des8	$\sum \chi^2$	Homo. χ^2	Total χ^2
2	.07	.20		.15	6.75**	.04	7.21	5.95	1.26
3	1.50	2.39		.04	1.34	3.54	8.81	7.94	.87
4	1.38	2.37	.09	.49	1.80	7.74*	13.87	8.79	5.08
5		.69	6.30*	2.03	3.34	2.18	14.54	10.49	4.05
6		4.28	5.32	2.49	1.93	27.92**	41.93**	31.28**	10.65*
7		8.07*	.21	6.97	15.11**	2.62	32.98**	20.44	12.54**
8		1.44	11.87**	7.89*	8.73*	45.75**	75.68**	29.58**	46.10**
9		.38	2.21	5.21	2.32	1.38	11.49	9.42	2.07
10		1.38	2.11	2.60	7.34*	7.68*	21.10*	17.75*	3.35
11			.02	.73	.74	.92	2.40	1.55	.84
12			.27	.33	.25	.75	1.60	1.57	.03

class $d = 7$, the contribution to the χ^2 (= 15.11) by the subclass with the lowest expected probability (P(7R) = .0373) was very high (11.51 or 76% of the total χ^2). Another reason for *des7j* and *des8k* to deviate from the expected model could be that in these two mutants almost all of the possible kinds of metaphase I configurations were observed, and it is possible that, in cases where a metaphasic cell could have any one of the possible 36 configurations, some of them were misclassified. A last reason of course could be due to the possibility that these two mutants do not behave in a random pattern (mainly *des8k*, which showed 4 desynaptic classes deviating at the .05 level from the expected model).

In general it seems correct to assume that, until a larger number of metaphasic configurations can be examined, the unpairing mechanism in these six Betzes desynaptic mutants (and there is a strong evidence that it may also happen in *des1*) is of a random nature.

Having established a repeatable pattern for each subclass within a desynaptic class, it is then possible to express the metaphase I behavior of a mutant by stating only the frequency of each desynaptic class. In this manner for example, Table 7 could be reduced as in Table 11. Thus, there is no further need of properly identifying each M I configuration; it will suffice to record the degree of desynapsis ($d = r + i$) of each metaphasic cell. In this way, the number of possibilities can be reduced from 36 to 15 (in a $2n = 14$ organism).

3-d. Desynaptic Class Frequency Distribution and Degree of Desynapsis

As indicated in the previous section, the phenotype of a desynaptic mutant can be described by the observed number of cells in each

Table 11. Number of Metaphase I Cells per Desynaptic Class for the Betzes Desynaptic Mutants Grown in 1972.

d class	Desynaptic mutants					
	des3	des4	des5	des6	des7	des8
0	480	50		5	4	5
1	136	91		4	5	20
2	23	138		9	16	30
3	5	137		19	34	53
4	1	73	2	41	42	76
5		54	8	62	72	78
6		21	12	70	91	106
7		14	20	92	96	73
8		5	43	81	67	67
9		3	60	42	54	52
10		1	106	26	36	34
11			133	9	20	21
12			196	4	3	9
13			191	1	2	1
14			180			2
Total cells	645	587	951	465	542	627

desynaptic class. In order to make comparisons between mutants, the number of cells in each class was expressed as a percentage of the total number of cells observed. If o_i is the observed number of cells with a di degree of desynapsis, the frequency fi (in percent) of that class will be $fi = o_i / \sum o_i \times 100$. The average degree of desynapsis $\bar{d}$ of the mutant was then defined as $\bar{d} = (\sum fi \times di)/100$.

The desynaptic class frequency distribution and degree of desynapsis of the 1971 and 1972 data (from Tables 6 and 7) are shown in Tables 12 and 13, respectively.

Data in Tables 12 and 13 show that the distribution of the classes around the "mean" class (desynaptic class with the degree of desynapsis of the mutants) tends to decrease in proportion to their "distance" (or difference in desynapsis) to the mean class. That is, the frequencies are approximately normally distributed with the limitation that there are no classes below 0 or above 14. A discrepancy was found for *des3c* in the 1971 data; one of the classes ($d = 6$) was found to have a much higher value than expected. There is no explanation for this behavior although it could be due to experimental error since the 1972 data did not reveal such a deviation.

Differences can be observed between the two years' data. These differences in the degree of desynapsis of a mutant from one year to another have been attributed to environmental fluctuations (Gottschalk and Villalobos-Pietrini, 1965). In general, a decrease in the mean degree of desynapsis of all mutants was observed in 1972. Plants of identical genotype showed only slight variations in degree of desynapsis (see Table 14 for data on the different plants analyzed in 1972).

Table 12. Frequencies of the Different Desynaptic Classes and Mean Degree of Desynapsis in Metaphase Cells of the Mutants Grown in 1971.

Abbreviations: d = desynaptic class, $\bar{d}$ = mean degree of desynapsis of the mutant, s = standard deviation. Note: The mutants are shown in an increasing degree of desynapsis.

	Desynaptic mutants											
d	des3c	des1a	des4h	des4d	des7j	des2b	des8k	des6i	des81	des5f	des5e	des5g
0	61.7	5.4	15.4	12.7	3.4	3.5				.9		.3
1	20.9	23.9	14.5	11.0	4.6	5.1		.4		.5	.4	.3
2	7.5	21.7	14.1	14.4	9.3	8.1		.4		.9	.4	.5
3	.7	20.3	17.2	12.7	8.3	9.7		.4		1.1		.8
4		16.6	10.6	13.6	11.4	13.3	1.9	1.5	2.3	2.1	1.8	.7
5		6.8	7.0	11.9	10.2	8.1	4.8	1.7	.4	1.9	.7	.5
6	9.2	3.9	18.1	9.3	14.5	8.4	9.0	4.1	1.5	3.0	3.3	2.2
7		.6	.9	6.2	10.1	8.1	11.5	8.1	7.8	2.8	.7	1.2
8		.5	1.8	3.7	11.1	7.3	18.6	14.3	10.5	3.7	6.3	2.9
9		.3	.4	2.3	7.6	11.1	14.7	17.5	17.8	7.5	2.0	3.5
10				1.7	4.8	5.7	18.3	26.6	21.3	14.4	13.3	5.4
11				.6	3.1	7.6	13.5	16.5	20.2	19.2	6.9	14.4
12					1.1	4.0	6.7	3.1	14.3	24.8	23.1	16.9
13					.4		1.0	4.8	3.5	16.3	23.2	23.6
14					.1			.6	.4	.9	17.9	26.8
$\bar{d}$	.93	2.63	3.04	3.64	5.63	5.98	8.72	9.26	9.77	10.35	11.44	11.82
s	1.75	1.63	2.22	2.60	2.92	3.33	2.04	2.04	1.90	2.72	2.49	2.50

Table 13. Frequencies of the Different Desynaptic Classes and Mean Degree of Desynapsis in Metaphase Cells of the Mutants Grown in 1972.

Abbreviations: d = desynaptic class, $\bar{d}$ = mean degree of desynapsis, s = standard deviation. Note: The mutants are shown in an increasing degree of desynapsis. The 1971 mean degree of desynapsis (d-71) is shown for comparison.

	Desynaptic mutants					
d	des3c	des4d	des8k	des7j	des6i	des5e
0	74.4	8.5	.8	.7	1.1	
1	21.1	15.5	3.2	.9	.9	
2	3.5	23.5	4.8	3.0	1.9	
3	.8	23.3	8.5	6.3	4.1	
4	.2	12.4	12.1	7.7	8.8	.2
5		9.2	12.4	13.3	13.3	.8
6		3.6	16.9	16.8	15.1	1.3
7		2.4	11.6	17.7	19.8	2.1
8		.9	10.7	12.4	17.4	4.5
9		.5	8.3	10.0	9.0	6.3
10		.2	5.4	6.6	5.6	11.2
11			3.4	3.7	1.9	14.0
12			1.4	.5	.9	20.6
13			.2	.4	.2	20.1
14			.3			18.9
$\bar{d}$	.31	2.80	6.07	6.55	6.59	11.59
s	.60	1.81	2.66	2.38	2.23	2.05
d-71	.93	3.64	8.72	5.63	9.26	11.44

Table 14. Degree of Desynapsis of the Plants Used for the 1972 Data.

Xi = degree of desynapsis of the *i*th plant calculated with #*i* cell counts, d-72 = mean degree of desynapsis of the mutants (as in Table 13), and #*t* = total number of cells recorded.

Mut.	*X*1	#1	*X*2	#2	*X*3	#3	*X*4	#4	*X*5	#5	d-72	#*t*
des3	.32	165	.32	165	.27	157	.34	158			.31	645
des4	3.10	153	2.83	159	2.21	142	3.07	133			2.80	587
des5	12.11	187	11.87	181	11.56	189	10.39	157	11.77	237	11.59	951
des6	6.55	154	7.23	183	5.70	128					6.59	465
des7	6.58	151	7.68	125	5.77	132	6.22	134			6.55	542
des8	6.75	269	5.53	118	5.48	129	5.68	111			6.07	627

It should be noted that not all mutants were studied in the same genetic background; *des1* is in the variety Mars, *des2* in the variety Husky, and the rest of the mutants in the variety Betzes. Thus, since the environment plays a great influence in the expression of desynapsis, comparisons between mutants in different backgrounds should be avoided.

Desynaptic mutants can be roughly classified (similar to Prakken's 1943 classification) according to their degree of desynapsis in the following types:

(a) Very weak desynaptic when the degree of desynapsis is less than 1; for example *des3*. This type of mutant very seldom exhibits univalents, usually only a few rod bivalents, and many ring bivalents.

(b) Weak desynaptic when the degree of desynapsis is approximately $n/2$ (n = haploid number); for example *des1* and *des4*. In these types of mutants the metaphase I cells will predominantly exhibit rod

bivalents regardless of the haploid number. Because of this high number of rod bivalents, the mutant *des1* was previously reported as a "long chromosome" mutant (Burnham, 1946).

(c) Medium desynaptic when the degree of desynapsis is approximately n; for example *des6*, *des7*, and *des8*. In this type of mutant the metaphase cells will exhibit different combinations of rod bivalents and univalents.

(d) Medium strong desynaptic when the degree of desynapsis is approximately $3n/2$; for example *des5*. This type of mutant will exhibit predominantly univalents and only a few rod bivalents.

(e) Complete desynaptic when the average degree of desynapsis is close to $2n$; for example the mutant reported in barley by Tsuchiya (1959 and 1960a).

3-e. Univalent Behavior at Anaphase I

In more than 400 anaphase I cells investigated for *des3c* none revealed any splitting of univalents. This mutant had previously shown a very low degree of desynapsis.

The rest of the mutants showed a varying number of univalents longitudinally splitting at anaphase I as illustrated in Fig. 4. The split univalents always appeared in an equatorial position.

Three of the mutants grown in 1972, with different degrees of desynapsis, were selected to study the univalent behavior at anaphase I: *des4d*, *des7j*, and *des5e*. The following possibilities were recorded: normal cells (a 7:7 disjunction as in Fig. 4-A), cells having different even numbers of univalents undergoing precocious division and at the same time

Fig. 4. Anaphase I Cells of Desynaptic Mutants.

A. Normal 7:7 disjunction.

B. 2 univalents splitting and normal 6:6 disjunction.

C. 2 micronuclei at early telophase I.

D. 4 univalents splitting and normal 5:5 disjunction.

E. 6 univalents splitting and normal 4:4 disjunction.

F. 8 univalents splitting and normal 3:3 disjunction.

G. 10 univalents splitting and normal 2:2 disjunction.

H. Abnormal 8:6 disjunction.

I. 1 univalent splitting and abnormal 6:7 disjunction.

J. 3 univalents splitting and abnormal 6:5 disjunction.

K. 7 univalents splitting and abnormal 5:2 disjunction.

L. 9 univalents splitting and abnormal 3:2 disjunction.

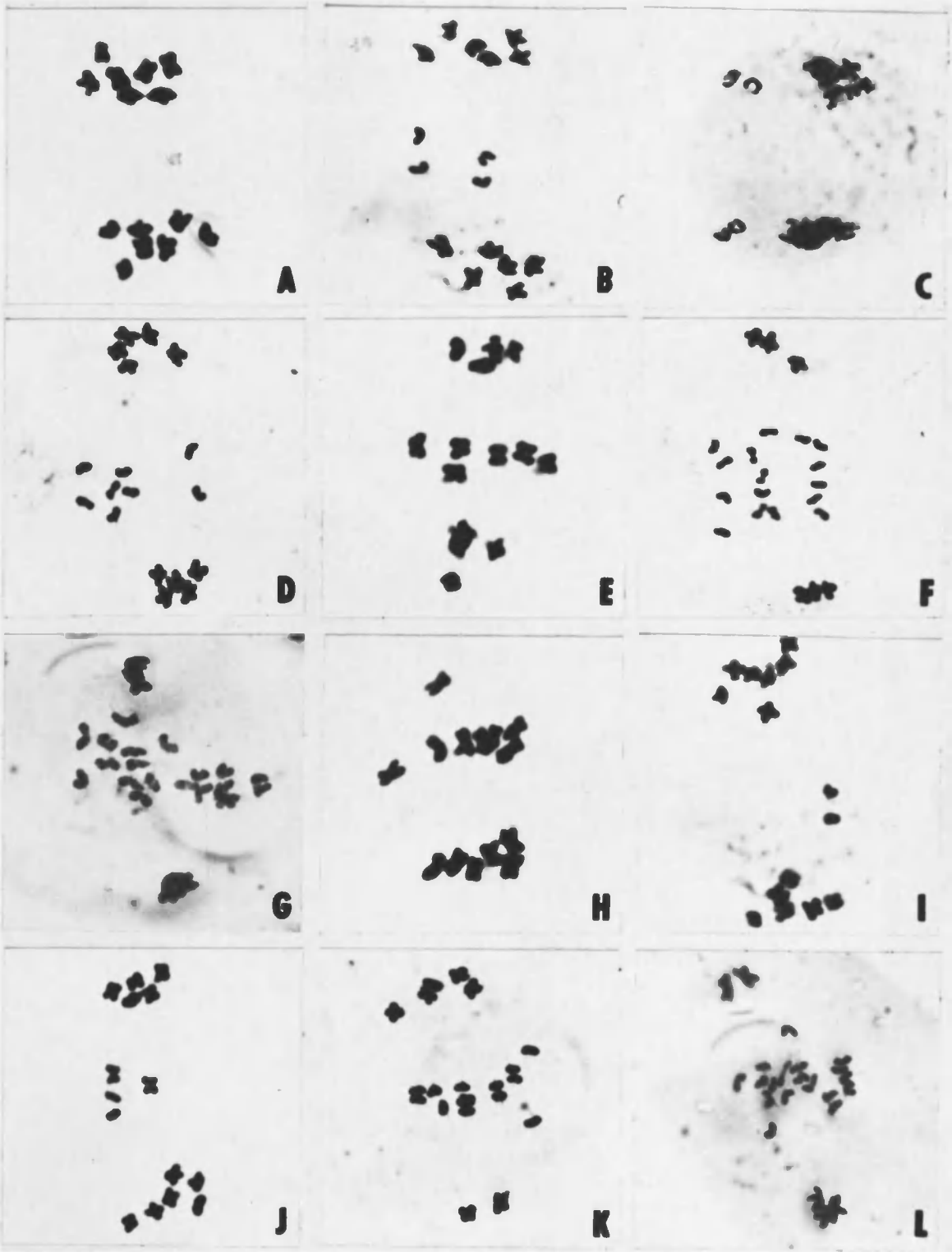

Fig. 4. Anaphase I Cells of Desynaptic Mutants.

a normal distribution of the remaining chromosomes (as illustrated in Figs. 4-B, 4-D, 4-E, 4-F, and 4-G), and finally the abnormal cells as illustrated in Figs. 4-H to 4-L.

The observed univalent behavior was compared with the expected univalent behavior hypothesizing that all the metaphasic univalents moved to the equatorial plate and underwent precocious division at anaphase I. The expected values can be readily calculated knowing the frequency of each desynaptic class (Table 13) and the probabilities of each metaphase I configuration (Table 8). Note for example that ten splitting univalents could have originated from metaphase I cells with configurations 2II + 10I, 11I + 1R + 10I, or 2R + 10I.

The observed and expected values are indicated in Table 15, and a detailed description of the abnormal anaphase I cells is given in Table 16. The results showed that (a) not all the univalents congressed toward the equatorial plate and thus accounted for the abnormal disjunctions and (b) the observed number of normal disjunctions was higher than expected for *des5* and *des7*, and the higher the degree of desynapsis of the cells in these mutants the greater the difference between the observed and expected values.

These observations tend to agree with previous studies (e.g., Prakken, 1943; Johnsson, 1944; and Ostergren, 1951), reporting that cells that have many univalents have few chiasmata to slow down the rate of division and thus the univalents tend to go undivided to the poles.

Table 15. Frequencies of Observed and Expected Anaphase I Configurations of Three of the Mutants Grown in 1972.

The expected anaphase configurations were calculated as if all the observed metaphasic univalents had undergone longitudinal splitting at anaphase I. N = normal 7:7 disjunction, Abn. = abnormal disjunctions (given in detail in Table 16), $\bar{d}$ = mean degree of desynapsis of the mutant.

Mutants	$\bar{d}$	No. of cells	Univalents longitudinally splitting								
			N	2	4	6	8	10	12	14	Abn.
des4	2.8	239	62.7	15.1	4.6	.4					17.2
Expected			74.4	20.7	3.9	.9	.1				--
des7	6.6	251	47.4	19.9	15.1	5.2	1.2	.4			10.8
Expected			21.3	30.5	25.2	14.3	6.8	1.5	.4		--
des5	11.6	370	3.2	1.1	6.5	8.1	13.2	14.6	11.1	6.8	35.4
Expected			.7	2.8	6.2	11.2	16.1	22.4	21.7	18.9	--

Table 16. Detail of the Abnormal Anaphase I Disjunctions of Table 15.

Listed are the number of chromosomes at each pole (P) and the number of chromosomes longitudinally splitting (S).

Abn. cells			Number of cells		
P	S	P	des4	des7	des5
1	11	2			17
1	10	3			11
1	9	4			5
2	9	3			25
2	8	4			24
2	7	5			3
3	11				1
3	7	4	1	3	19
3	6	5			5
3	5	6			1
4	5	5	3	4	11
4	4	6			3
4	3	7			1
4		10			1
5	3	6	6	7	4
5	2	7	1	1	
5	1	8	2		
5		9	1		
6	1	7	17	8	
6		8	10	4	
Totals			41	27	131

3-f. Other Abnormalities of First Division

The split univalents of anaphase I were found to move to the poles, sometimes joining the rest of the chromosomes or many times forming micronucleilike bodies at telophase I (see Fig. 4-C).

Cells with bridges and fragments were observed (with very low frequencies) in all of the Betzes mutants except *des3c* (see Fig. 5-A).

Some cases of bivalents with abnormal terminalization were observed in mutants *des5*, *des6*, *des7*, and *des8* (see Figs. 5-A, 5-B, and 5-C). Fragmented extra chromosomes were sometimes found among the progenies of these mutants. The abnormal terminalizations can be a possible origin for these fragments as previously reported by Beadle (1930) in corn although Miller (1963), also working with corn, reported that fragmentation of chromosomes was due to misdivision of univalents at anaphase I. In the present study, misdivision of univalents was either overlooked or not present in these mutants.

3-g. Second Meiotic Division

All the Betzes mutants except *des3c* showed: unsynchronization of the late meiotic stages; unsynchronized divisions in both dyads (see Fig. 5-E); various numbers of laggards (see Figs. 5-F and 5-G) that can form micronucleuslike bodies at the end of anaphase II (see Fig. 5-H) and abnormal distributions of chromatids per dyad (e.g., 15 in one dyad and 13 in the other, 12 in one dyad and 16 in the other, etc.) that could have originated from the splitting of univalents at anaphase I and an uneven distribution of the unsplit univalents or that both halves of a split univalent reached the same pole.

Fig. 5. Abnormal Cells of Desynaptic Mutants.

A. Anaphase I cell showing an abnormal bivalent terminalization and a bridge and fragment (arrow).

B. Anaphase I cell with two abnormal bivalent terminalizations.

C. Anaphase I cell with four splitting univalents and an abnormal bivalent terminalization (arrow).

D. Normal anaphase II.

E. Unsynchronized second division.

F. 2 laggards in each anaphase II division.

G. 4 laggards in each anaphase II division.

H. Micronuclei bodies (arrows) at telophase II.

I. Normal tetrad.

J. Abnormal tetrad with four micronuclei.

K. Abnormal tetrad with eight micronuclei.

L. Abnormal tetrad with twelve micronuclei.

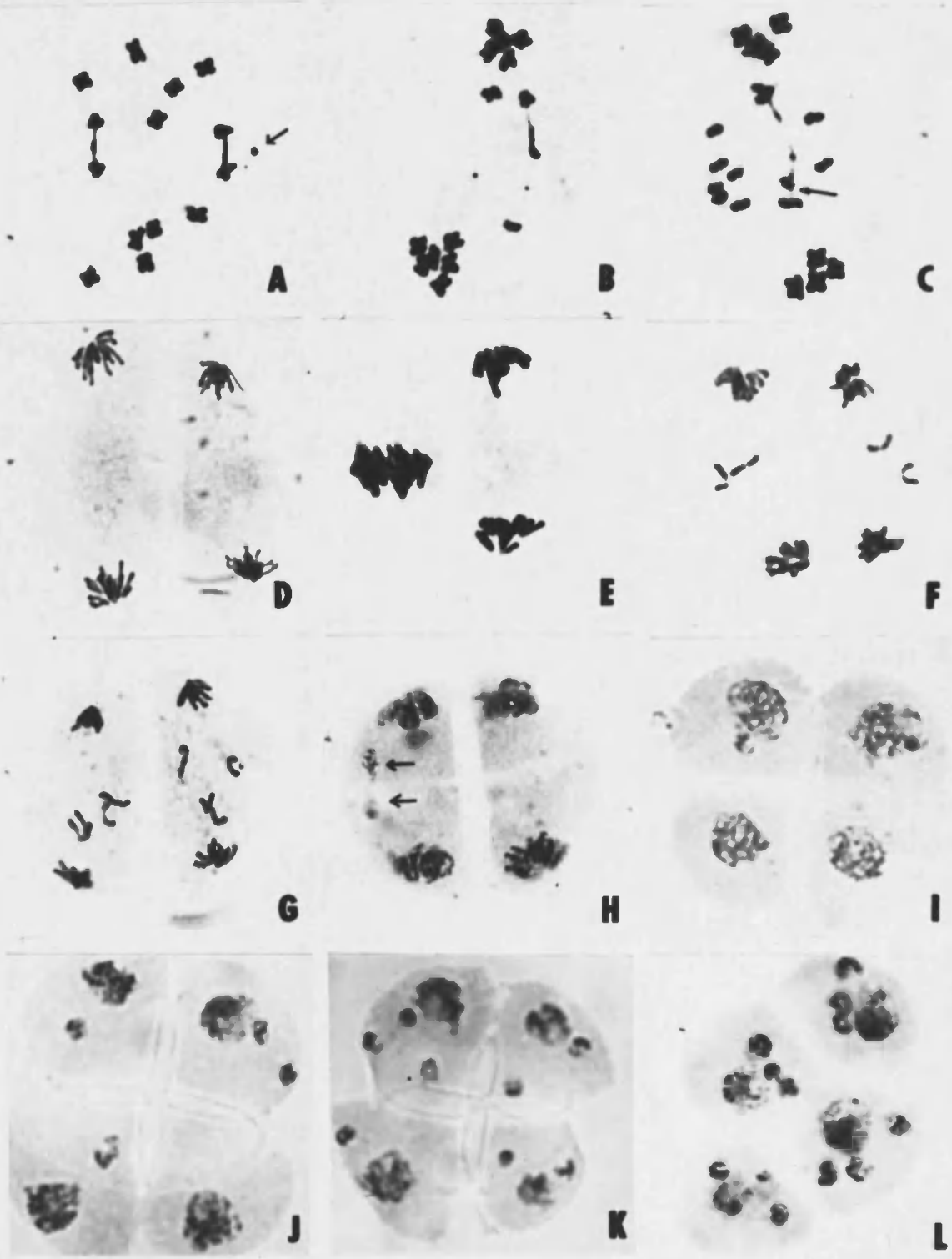

Fig. 5. Abnormal Cells of Desynaptic Mutants.

All of the above-mentioned abnormalities led to very abnormal tetrad stages. The most evident feature was abnormal numbers of micronuclei per quartet (see Figs. 5-J, 5-K, and 5-L). The number of micronuclei per quartet was recorded in all of the mutants grown in 1971. The results (Table 17) showed a highly positive correlation (r = +.796) between the average number of micronuclei per quartet of a mutant and its degree of desynapsis. Only *des8k* and *des8l* were found to deviate from this correlation. If both mutants were not considered when calculating the correlation coefficient, a much higher value was obtained (r = +.945).

4. Fertility

As a result of meiotic abnormalities, desynaptic mutants show a high degree of sterility. In general fertility depends on the number of bivalents at metaphase I; the higher the number of chromosomes remaining paired the higher the fertility (e.g., in *Avena*; Thomas and Rajhathy, 1966).

Data in Table 18 show that fertility was variable for the different mutants and for the two years' data. Betzes used as a control showed 100% fertility. The results indicate that sterility is not a good tool for comparisons among different desynaptic mutants because (a) some mutants are in different cultivars and possibly different cultivars have different degrees of tolerance to cytological abnormalities, (b) the fertility was determined over a great number of spikes per plant that matured at different times and probably the degree of desynapsis of each spike within a plant was variable throughout the growing season, as

Table 17. Frequencies of Micronuclei per Quartet (M/Q) in Microsporocytes of the Mutants Grown in 1971.

Mutants are shown in an increasing degree of desynapsis (d). #Q = total number of quartets recorded, $\bar{x}$ = average number of micronuclei per quartet, s = standard deviation.

M/Q	des3c	des1a	des4h	des4d	des7j	des2b	des8k	des6i	des81	des5f	des5e	des5g
0	96.9	86.7	50.0	52.7	25.5	35.6	44.1	17.5	42.5	4.3	11.3	9.0
1	2.9	6.2	23.0	17.4	8.1	11.7	16.3	9.2	21.7	4.6	6.9	4.4
2	.2	2.7	19.9	14.6	13.8	18.7	21.8	15.7	20.0	8.3	14.7	11.9
3		1.5	4.9	8.3	11.7	14.7	8.3	14.3	10.3	10.4	15.4	11.3
4		1.4	2.2	3.7	15.1	13.8	6.7	12.8	4.2	14.0	14.2	16.4
5		.9		2.2	8.9	3.1	1.6	10.8	1.0	12.4	10.5	14.7
6		.3		.6	5.6	1.2	.4	9.3	.3	16.0	14.4	11.3
7				.3	6.6	1.2	.4	5.7		11.7	6.3	8.2
8				.1	2.6			2.5		9.9	4.0	5.7
9					1.2			1.2		4.6	1.4	3.4
10				.1	.4		.4	.5		1.8	.7	1.8
11		.3			.3			.2		1.7	.2	.5
12					.1			.3		.3		.6
13					.1							
>13												.4*
#Q	479	337	226	731	238	326	252	600	360	606	583	732
d	.93	2.63	3.04	3.64	5.63	5.98	8.72	9.26	9.77	10.35	11.44	11.82
$\bar{x}$	.03	.31	.86	1.05	2.97	1.80	1.29	3.26	1.16	5.10	3.75	4.44
s	.19	.52	1.04	1.46	2.58	1.73	1.54	2.46	1.29	2.57	2.40	2.73

*des5g showed .1 quartet with 14 micronuclei, .1 with 15 micronuclei, .1 with 16 micronuclei, and .1 with 18 micronuclei.

Table 18. Percent Ovule Fertility (f%) and Mean Degree of Desynapsis ($\bar{d}$) of the Mutants Grown in 1971 and 1972.

	1971		1972	
Mutant	f%	$\bar{d}$	f%	$\bar{d}$
des1a	45.30	2.6		
des2b	.95	6.0		
des3c	.90	.9	33.12	.6
des4d	12.05	3.8	33.57	2.8
des4h	16.20	3.8		
des5e	2.15	11.4	12.90	11.6
des5f	3.20	10.4		
des5g	4.50	11.8		
des6i	16.35	9.3	21.23	6.6
des7j	27.50	5.6	40.42	6.6
des8k	42.75	8.7	43.09	6.1
des81	.95	9.8		

reported in other studies (e.g., in rice; Katayama, 1961a), and (c) very few plants from each mutant were examined.

Nevertheless, some results were unexpected; for example, *des8l* and *des8k*, two allelic mutants, showed very different fertilities. There is no explanation for this behavior unless some undetected abnormalities are present in *des8l* (this mutant has always shown higher sterility than *des8k*, e.g., see Table 1). Even more abnormal was the behavior of *des3c*, a very low desynaptic mutant. Its fertility varied greatly from one year to the other while the degree of desynapsis remained fairly constant. Sjödin (1970) reported a desynaptic mutant in *Vicia faba* with very low desynapsis and high sterility and attributed this result to possible undetected abnormalities in the later stages of the microgametophyte development, but in the present study the result of the analysis of micronuclei per quartet showed very few abnormalities for the mutant *des3c* (at least detectable abnormalities). Although the possibility of undetected abnormalities should not be ruled out, it is also possible that the expression of desynapsis in *des3c* (and possibly in the other mutants) is different in the megagametophytic stages.

5. Relationship Between Seed Weight and Chromosome Number in the Progeny of Desynaptic Plants

The results of the cytological investigation of plants from randomly selected samples of seed from *des6* and *des7* (from 1971 plants) are shown in Tables 19 and 20. The aneuploids, most of them primary trisomics, were found in the light seed fraction. Similar results were reported in a desynaptic *Avena* (Dyck and Rajhathy, 1965).

Table 19. Numbers of Somatic Chromosomes, by Weight, in 100 Seeds Randomly Selected from des6i.

Abbreviations: $2n$ = 14 chromosomes, $2n+1$ = trisomic, other a. = other aneuploids (fg = fragmented chromosome), N.I. = not identified, N.G. = nongerminated seed, tri. types = trisomic types (morphologically identified). Weights are given in classes of 4-mg intervals. Mean weight of the seeds was 52 mg.

Weight (mg)	Chromosome number					
	$2n$	$2n+1$	Other a.	N.I.	N.G.	Tri. types
28-32	1	2				5,7
32-36		1		1		7
36-40	1	4		1		3,3,4,4
40-44		6		1	1	2,4,4,4,7,7
44-48	6	4	1*			4,4,5,5
48-52	12	2				6,6
52-56	18					
56-60	18					
60-64	13					
64-68	6					
68-72	1					
Total (%)	76	19	1	3	1	

*Type 14+fg

Table 20. Numbers of Somatic Chromosomes, by Weight, in 200 Seeds Randomly Selected from des7j.

Abbreviations: $2n$ = 14 chromosomes, $2n+1$ = trisomic, other a. = other aneuploids (fg = fragmented chromosome), N.I. = not identified, N.G. = nongerminated seed, tri. types = trisomic types (morphologically identified). Weights are given in classes of 4-mg intervals. Mean weight of the seeds was 50.4 mg.

Weight (mg)	Chromosome number					
	$2n$	$2n+1$	Other a.	N.I.	N.G.	Tri. types
20-24		1		1		4
24-28		1				5
28-32	1	2	1*			4,5
32-36	3	4			1	1,3,5,7
36-40		7				1,4,4,4,5,5,7
40-44	7	5				4,4,5,6,7
44-48	35	4				3,4,4,6
48-52	27					
52-56	43					
56-60	37					
60-64	17					
64-68	2				1	
Total	172	24	1	1	2	
%	86	12	.5	.5	1	

*Type 14+1+1

More aneuploids (22% vs. 13%) were found among the progeny of *des6* than among the progeny of *des7*. This may have been due to the different degrees of desynapsis of the parent plants--9.3 for *des6* and 5.6 for *des7*--but it will be hard to prove since the degree of desynapsis of these two mutants was determined on spikes collected at one time in the lifespan of the mutants while the seed was obtained from spikes maturing at very different times and most probably having different degrees of desynapsis (e.g., Li, Pao, and Li, 1945).

All different types of primary trisomics were found in the combined progeny of the two mutants. Of these, 2 were trisomics for chromosome 1, 1 for chromosome 2, 4 for chromosome 3, 16 for chromosome 4, 9 for chromosome 5, 4 for chromosome 6, and 7 for chromosome 7. The different types of trisomics recovered were not distributed at random according to the chromosome (for example, 34% of the trisomics were for chromosome 4) and neither according to weight (for example, trisomics for chromosome 6 were found among the heavier trisomics). Since chromosome number was identified for most seeds, since desynapsis in these mutants is of a random nature (implying a random distribution of extra chromosomes to the gametes), and since the chromosome length is not correlated with the rate of transmission in barley trisomics (Tsuchiya, 1960b), it must be that different extra chromosomes confer different female gamete viability (the extra chromosome is seldom transmitted through the pollen in barley; Tsuchiya, 1960b).

The results of this study suggest a good method to obtain primary trisomics in barley, either to establish a trisomic series (as suggested by Dyck and Rajhathy, 1965, for a desynaptic *Avena*) or for genetic

studies; desynaptic plants can be pollinated with pollen from normal plants (the pollen production of desynaptic plants is very poor, e.g., Katayama, 1964) and the trisomics in the light seed fraction can be recovered. If the normal pollen had a recessive marker, the selfed plants can be recognized cytologically.

6. Location of Genes by Trisomic Inheritance

The F_1 trisomics were cytologically examined and all were normal primary trisomics with 7II + I or 6II + III at metaphase I, being either *Des des des* or *Des des*. In the F_2 the diploid plants grew normally while the trisomics failed in almost every case to reach maturity (only the tri 6 were able to compete with normal plants but were easily identified). Since the classification of the F_2 was done according to sterility, the trisomics would have been hard to classify because of their own sterility. Not all the F_1 primary trisomics were obtained, and in many cases some trisomics yielded very few seeds (very frequently in the case of tri 7). No trisomics were ever obtained for *des3*.

The results of the trisomic analysis (Table 21) indicate that:

(a) *des4* must be located on chromosome 1. The data show that it could also be located in chromosome 2, but the segregation for trisomic 1 highly deviates from a disomic ratio; therefore it was concluded that this mutant is located on chromosome 1.

(b) *des5* is located on chromosome 1.

(c) *des6*, as in the case of *des4*, could be located in either chromosome 2 or 5, but since a higher number of plants could be tested

Table 21. Classification of the Diploid Progeny of the Primary Trisomics Recovered from Crosses Between Desynaptic and Normal Plants.

The χ^2 are calculated for disomic (3 Des:1 des) or trisomic (5 Des: 4 des) ratios.

Mutant	F_1 tri	*2n* plants in F_2 Des	des	Disomic ratio χ^2	P	Trisomic ratio χ^2	P
des4	1	41	29	10.08	.005	.26	.75-.50
	2	45	22	2.19	.25-.10	3.66	.10-.05
	3	41	9	1.31	.50-.25	14.49	.005
	4	104	46	2.57	.25-.10	11.53	.005
	5	67	28	1.01	.50-.25	8.62	.005
	6	165	56	.00	1.0-.99	32.67	.005
	7	78	23	.27	.90-.75	19.21	.005
des5	1	82	51	12.63	.005	2.00	.25-.10
	3	48	19	.40	.75-.50	7.02	.01-.005
	4	82	30	.19	.75-.50	14.15	.005
	5	68	27	.59	.50-.25	9.88	.005
	6	77	26	.00	1.0-.99	15.38	.005
	7	11	7	1.85	.25-.10	.23	.50-.25
des6	2	27	19	6.52	.025-.01	.18	.75-.50
	4	114	48	1.85	.25-.05	14.40	.005
	5	122	75	17.95	.005	3.24	.10-.05
	6	41	15	.09	.90-.75	7.07	.01-.005
des7	2	72	60	29.45	.005	.05	.90-.75
	4	38	14	.10	.90-.75	6.47	.025-.010
	5	78	28	.11	.75-.50	13.95	.005
	6	132	48	.27	.75-.50	23.04	.005
	7	7	4	.76	.50-.25	.29	.75-.50
des8	4	109	32	.40		27.01	.005
	6	35	10	.18		9.00	.005

for trisomic 5, and its segregation highly deviates from a disomic ratio, it was concluded that this mutant is located on chromosome 5.

(d) *des7* is located on chromosome 2.

(e) The only available information obtained for *des8* is that this mutant is not located on either chromosome 4 or 6.

SUMMARY

Desynapsis was found to be controlled by a single recessive gene in ten Betzes mutants. An allele test including the two previously described desynaptic mutants (*des1* and *des2*) revealed six new desynaptic loci: *des3*, *des4*, *des5*, *des6*, *des7*, and *des8*.

All F_1's between nonallelic mutants showed normal metaphase cells except where *des5* mutants were involved. F_1's involving *des5* showed a low degree of desynapsis and some sterility, suggesting the possibility that *des5* is not a completely recessive mutant.

A cytological study of the Betzes mutants revealed that homologous chromosomes are paired in pachytene and early diplotene and subsequently desynapse. The number of unpairing events that occur and where these events occur will determine that the metaphase I cells show one of 36 possible combinations of ring bivalents, rod bivalents, and univalents. The metaphase configurations were classified according to their degree of desynapsis defined as the number of unpaired chromosome arms or number of rod bivalents and univalents per cell. In such a manner, the 36 possible metaphase configurations were classified in 15 desynaptic classes, from 0 to 14 desynapsis. An analysis of the distribution of the metaphase configurations of equal degree of desynapsis revealed that the chromosome arms unpair at random. Thus it was concluded that desynapsis in a mutant can be described much better with the frequencies of each desynaptic class than, as repeatedly done by previous workers, with the frequencies of metaphasic univalents.

A classification of the Betzes mutants according to their mean degree of desynapsis and using Prakken's terminology (1943) is given.

An experiment showed that in mutants with a high degree of desynapsis, not all the univalents longitudinally split at anaphase I. The higher the number of univalents at metaphase I the higher the number of univalents that will go undivided to the poles at anaphase I.

Other cytological abnormalities were found in all mutants except *des3*. The mean number of micronuclei per quartet of a mutant was found to be positively correlated (.796) with the mean degree of desynapsis.

All desynaptic mutants showed a high degree of ovule sterility. No association was found between the degrees of sterility and desynapsis, possibly because the sterility was measured in spikes that matured on different dates while the degree of desynapsis was measured in spikes collected on only one date.

Aneuploids, most of them primary trisomics, were readily obtained from the light seed fraction of all the Betzes mutants except *des3*.

Trisomics recovered from crosses of desynaptic plants with a marker gene can be used to locate that gene. This technique was used to locate *des4* and *des5* on chromosome 1, *des6* on chromosome 5, and *des7* on chromosome 2, and to determine that *des8* is not located on chromosome 4 or 6.

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