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# Analysis of chromosome pairing and breakage in pearl millet

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## SUMMARY

The relationship between chromosome pairing and chromosome fragmentation has been studied in a gene controlled mutant of pearl millet ($2n = 14$). Premeiotic mitosis, premeiotic cell development and early prophase I are normal without any fragments, which first appear at pachytene. The extent of fragmentation varies from zero to very extreme with two discrete classes of plants, namely those with partial fragmentation and those with multiple fragmentation. A quantitative analysis of bivalent distribution and the distribution of A I bridges in desynaptic and fragmented cells show all of them to be nonrandom events. We suggest that in cells showing partial fragmentation the bridges and fragments result from U-type exchanges at pachytene. The reduced frequency of A II bridges indicates relatively low sister chromatid reunion at pachytene. In multiple fragmented plants numerous minute fragments were seen from pachytene. Despite these anomalies most PMCs complete meiosis but subsequently abort at the pollen grain stage. The mutant gene also causes disturbances in the sequence of meiotic development in the ear and in the synchronous development of PMCs within an anther. It has no effect on the tapetum or on the physiological development of the anther.

## 1. INTRODUCTION

Since the discovery of the meiotic mutant '*C(3)G*' in *Drosophila* (Gowen & Gowen, 1922) numerous cases of the genetic control of meiosis have been reported (Baker *et al.* 1976). In plants the identification of such mutants depends on (1) the detection of anomalies only in meiotic cells with no effect on the surrounding nutritive cells and with normal flower development and (2) genetic evidence to show that anomalous meiosis follows Mendelian inheritance. Among the numerous meiotic genes identified those controlling synapsis and its maintenance, or else the formation of nonfunctional gametes (male or female steriles), are particularly wide spread (for reviews see Gottschalk & Kaul, 1974; Koduru & Rao, 1981). In this paper we document the cytogenetic features of a gene causing varying meiotic abnormalities in pearl millet, *Pennisetum americanum* (L.) Leake.

## 2. MATERIALS AND METHODS

Several types of trisomics were isolated among the progeny of a spontaneously produced triploid in the line IP 457 (Koduru *et al.* 1981). In the selfed progeny of a primary trisomic ($2n = 14+1$) all the 114 plants grown to maturity were euploids. Twenty six of these showed complete pollen and ovule sterility and on checking were found to carry a syndrome of meiotic anomalies. Material for cytological analysis was fixed in acetic acid alcohol (1:3) mixture for 24 h, stored in 70% alcohol until used and subsequently analysed using acetocarmine squash preparations of PMCs.

Table 1. *Segregation of the mutant in the selfed families*

| Generation | Families studied | | Number of plants | | | $\chi^2$ (d.f. 1) | $P$-value |
|---|---|---|---|---|---|---|---|
| | No. | Segregating | Total | Normal | Mutant | | |
| $S_1$ | 3 | 1 | 108 | 88 | 20 | 2·420 | > 0·1 |
| $S_2$ | 2 | 1 | 42 | 32 | 10 | 0·032 | > 0·7 |
| $S_3$ | 5 | 2 | 129 | 88 | 41 | 2·814 | > 0·005 |
| Total | 10 | 4 | 285 | 208 | 77 | 0·516 | > 0·3 |
| Heterogeneity $\chi^2$ | | 3·18 | | | | 2·579 | > 0·2 |

## 3. OBSERVATIONS

### (*a*) *Inheritance*

The mutant plants did not differ from their normal sibs in vegetative characters, but at flowering the ears on the main culm showed feeble anther emergence and the emerged anthers did not dehisce. The ears of tillers were weak and there was no anther emergence. The emergence and maturity of styles appeared normal. Pollen stainability was almost nil and no seed was set on selfing, on open pollination or on crossing to normals using the mutants as females. Thus it is probable that the syndrome also occurs on the female side. Therefore inheritance of the mutant condition was followed in the selfed progenies of sib heterozygotes. In three successive generations the segregation pattern suggested monogenic recessive inheritance of the mutational event (Table 1). The symbol '*mbr*' (meiotic breakage) is proposed to describe the mutant responsible for this syndrome.

### (*b*) *Disturbance in the sequence of meiotic development in the ear*

An effect associated with the mutant gene is disturbance of the normal sequence of meiotic development in the ear. Normally florets mature basipetally. Meiotic stages are confined to a small segment of the ear while it is inside the sheath of

the flag leaf which encloses the developing ear either in the primary or secondary florets. Meiosis is complete when the ear emerges from the flag leaf sheath. In mutant plants meiosis is found in spikelets all through the length of the spike even after emergence. Further, and in contrast to the initial line, considerable asynchrony in the meiotic development of PMCs within an anther was revealed by the occurrence of PMCs from preleptotene to tetrad stage. Despite these disturbances, a large number of PMCs passed through meiosis and degeneration did not set in until the pollen grain stage. However in plants showing extreme fragmentation of chromosomes the PMCs seemed to degenerate before they completed meiosis. Even in such plants, degeneration began after the completion of meiosis in those PMCs lacking fragments.

Table 2. *Distribution of fragments at diakinesis in PMCs with partial fragmentation*

| Plant | Number of PMCs with fragments | | | | | | | | Total cells | % of cells with fragmentation |
|---|---|---|---|---|---|---|---|---|---|---|
| | 0 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | | |
| 1 P | 195 | 5 | 15 | 30 | 60 | 50 | 30 | 32 | 417 | 53·24 |
| S | 150 | 3 | 10 | 20 | 15 | 20 | 45 | 60 | 323 | 53·56 |
| 2 P | 119 | 10 | 6 | 11 | 15 | 40 | 65 | 7 | 273 | 56·41 |
| S | 175 | 0 | 10 | 10 | 60 | 45 | 45 | 40 | 385 | 54·55 |
| 3 P | 163 | 5 | 10 | 25 | 15 | 50 | 30 | 60 | 358 | 54·47 |
| S | 52 | 2 | 4 | 10 | 15 | 5 | 25 | 20 | 133 | 60·90 |
| 4 P | 43 | 4 | 11 | 20 | 16 | 6 | 7 | 15 | 122 | 64·75 |
| S | 95 | 2 | 5 | 42 | 10 | 12 | 20 | 43 | 229 | 58·52 |
| 5 P | 56 | 0 | 0 | 2 | 15 | 13 | 35 | 20 | 141 | 60·28 |
| S | 90 | 0 | 0 | 4 | 20 | 20 | 40 | 50 | 224 | 59·82 |

P, Primary floret; S, Secondary floret.

### (*c*) *Partial fragmentation*

These plants carried PMCs with two to several fragments and one to six bivalents, together with PMCs with no fragments but with a strong reduction in chiasma frequency.

Premeiotic mitosis was normal without fragments (Fig. 1). There were no visible changes in the nuclear phenotype of the developing PMCs (Fig. 2). Leptotene and zygotene chromosomes were normal with no evidence of breakage (Figs. 3, 4). Chromosome pairing at pachytene is complete and fragments first appeared at this stage. The paired condition of the fragments shows that chromosome breakage occurred at pachytene (Fig. 5). During subsequent stages of prophase I more than 50% of the PMCs carried two to several fragments of large, small or minute size (Table 2). In PMCs with a lesser number of fragments the other chromosomes usually formed bivalents, some of which were clearly heteromorphic, with a

corresponding number of large fragments (Fig. 6). In some cases there is considerable attenuation of the centric region in one of the homologues within a bivalent or else between the two arms of a univalent (Fig. 7). As the mean number of fragments per cell increased, the PMCs usually contained both small and large fragments. The origin of such small fragments could be explained on the basis of simultaneous breakage at two points (intra-arm) in a paired bivalent and the reunion of the proximal and distal segments, eliminating the interstitial segment as an acentric (paired) fragment. Such paired fragments, if not bound by a chiasmate connection, will fall apart into individual chromosome fragments during post-pachytene.

In order to ascertain whether all bivalents contribute equally to the formation of fragments, the distribution pattern of normal bivalents (in cells with partial fragmentation) in the primary and secondary florets of each of the five plants studied has been compared with a random and Poisson series. In all cases the observed values deviated widely from the expected (Table 3) indicating that the probability of undergoing breakage is not the same for all chromosomes. In an asynaptic mutant of pearl millet, Krishna Rao & Koduru (1978) also found evidence for a differential involvement of bivalents in breakage. The mean frequency of bivalents per cell in the primary florets was 1·77 while in the secondary florets it was 2·62; this difference is significant ($t = 4{\cdot}58$, $P - < 0{\cdot}001$). In respect of the frequency of normal cells, secondary florets were deficient by some 5·76% compared to primary florets. Thus it appears that the action of the mutant gene varies between flowers of the same spike.

Metaphase I behaviour of the fragments varied according to whether they were centric or acentric. Centric fragments oriented but it was impossible to decide whether a centric fragment divided at AI because of the occurrence of unoriented acentric fragments. The presence of more than 7 elements at prophase II in dyads suggests that some fragments were included in telophase I nuclei.

Bivalents underwent normal disjunction at AI. Bridges were seen in all plants and varied in number from 0 to 4 (Table 4, Figs. 9, 10). In no case was an equational division of univalents seen at AI. Thus it is safe to assume that all AI bridges were formed between homologues (half bivalent bridges). These result from the union of nonsister chromatids (NSU) following chromatid breakage. In view of the non-random fragmentation observed above, the distribution frequency of AI bridges per cell was compared with the binomial series $(p+q)^7$ where $p$, the average probability for a bivalent to form a bridge, is computed by dividing the total number of bridges by the product of the potential number of bivalents (7) and the total number of AI cells studied. The results, presented in Table 5, show no fit between the observed and expected frequencies. Thus, as with fragmentation, the probability for any one bivalent to contribute to bridge formation is not the same for all bivalents. At anaphase II less than 2% of the dyads showed bridges (Table 4, Fig. 11). These would have resulted from sister chromatid union (SU) following chromatid breakage at pachytene. Other abnormalities included weak neocentric activity (Fig. 8) and the suppression of dyad wall development in some PMCs.

Table 3. *Distribution pattern of bivalents in cells with partial fragmentation (Cells with zero fragments were not included)*

| Bivalents | Floret | Plants | | | | | Total | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | 1 | 2 | 3 | 4 | 5 | Observed | Expected[a] | Expected[b] | $\chi^{2b}$ |
| 0 | P | 35 | 60 | 60 | 47 | 60 | 262 | 128·71 | 185·98 | 31·07 |
| | S | 27 | 17 | 35 | 7 | 10 | 96 | 42·44 | 96·31 | 0·001 |
| 1 | P | 26 | 22 | 55 | 69 | 70 | 242 | 330·96 | 328·20 | 22·64 |
| | S | 25 | 30 | 20 | 10 | 30 | 115 | 200·06 | 275·20 | 93·26 |
| 2 | P | 75 | 25 | 90 | 47 | 35 | 272 | 354·60 | 295·38 | 1·85 |
| | S | 45 | 90 | 100 | 105 | 90 | 430 | 392·98 | 344·00 | 21·5 |
| 3 | P | 30 | 90 | 10 | 17 | 30 | 177 | 202·63 | 175·04 | 0·02 |
| | S | 67 | 60 | 90 | 88 | 150 | 455 | 411·69 | 302·76 | 76·55 |
| 4 | P | 20 | 32 | 12 | 27 | 4 | 95 | 65·13 | 76·58 | 4·43 |
| | S | 40 | 40 | 23 | 45 | 16 | 164 | 242·60 | 192·63 | 4·26 |
| 5 | P | 8 | 5 | 8 | 5 | 6 | 32 | 11·17 | 21·88 | 4·68 |
| | S | 32 | 20 | 17 | 10 | 6 | 85 | 76·25 | 110·07 | 5·71 |
| 6 | P | 5 | 5 | 2 | 1 | 1 | 14 | 0·8 | 10·94 | 0·86 |
| | S | 15 | 3 | 5 | 4 | 4 | 31 | 9·98 | 55·03 | 10·49 |
| Total | P | 199 | 239 | 237 | 213 | 206 | 1094 | 1094·0 | 1094·0 | 65·55 |
| | S | 251 | 260 | 290 | 269 | 306 | 1376 | 1376·0 | 1376·0 | 211·77 |
| Mean bivalents per cell | P | 2·09 ±0·10 | 2·20 ±0·10 | 1·54 ±0·08 | 1·84 ±0·04 | 1·37 ±0·09 | 1·77 ±0·04 | — | — | — |
| | S | 2·89 ±0·09 | 2·57 ±0·08 | 2·40 ±0·08 | 2·74 ±0·07 | 2·54 ±0·06 | 2·62 ± 0·04 | — | — | — |
| Total $\chi^{2b}$ | P | 28·73 | 131·4 | 41·05 | 30·32 | 17·04 | 66·70 | — | — | — |
| | S | 29·25 | 40·99 | 22·03 | 92·68 | 158·2 | 230·38 | | | |

$\chi^2$ table value at 5% is 11·07[b]

P, primary floret; S, secondary floret; [a], random series; [b], Poisson series.

### (*d*) *Partial desynapsis*

In both the primary and secondary florets around 50% of the PMCs had no fragments but showed reduced chiasma formation ($3{\cdot}55 \pm 0{\cdot}021$ to $4{\cdot}05 \pm 0{\cdot}032$ per cell). The mean bivalent frequency for five plants showing medium to strong desynapsis is $2{\cdot}66 \pm 0{\cdot}059$ in the primary florets and $3{\cdot}40 \pm 0{\cdot}169$ in the secondary florets; these values are not significantly different ($t = 1{\cdot}73$, $P > 0{\cdot}10$). The observed distribution pattern of the bivalents in the PMCs of each of the five plants was compared with the random and Poisson series and these results showed non-random bivalent formation (Table 6). Similarly the distribution of chiasma frequency is also non-random in the one plant studied. This could have been due to preferential localisation of chiasmata in particular bivalents.

### (*e*) *Multiple fragmentation*

As in plants with partial fragmentation, premeiotic cell development and the early prophase I stages lacked fragments. Pachytene pairing is normal in the small percentage of cells without fragments. The appearance of fragments in these cells is associated with the appearance of unpaired segments. In cells classified as extreme the nuclei had a shattered appearance with numerous small pieces of chromatin (Figs. 14, 15). Such cells constituted more than 95% of the total. The remainder were without fragments but with reduced chromosome pairing. In most of the PMCs several chromatin segments fused to form large masses which oriented at MI, indicating that the fused elements are centric. Anaphase I pulling of the centromeres of these masses resulted in the development of chains with perpendicularly spreading arms (Fig. 16). Other fragments were distributed to the poles irregularly and were also present during second division. In spite of these irregularities involving a complete disruption of chromosome integrity, the PMCs passed through all stages of meiosis and formed tetrads of polyads of different sizes which also contained fragments (Fig. 17). Compared to cells of the same anthers which lacked fragments, the meiotic development in the fragmented cells is much delayed and at the time of formation of tetrads in the normal cells, fragmented

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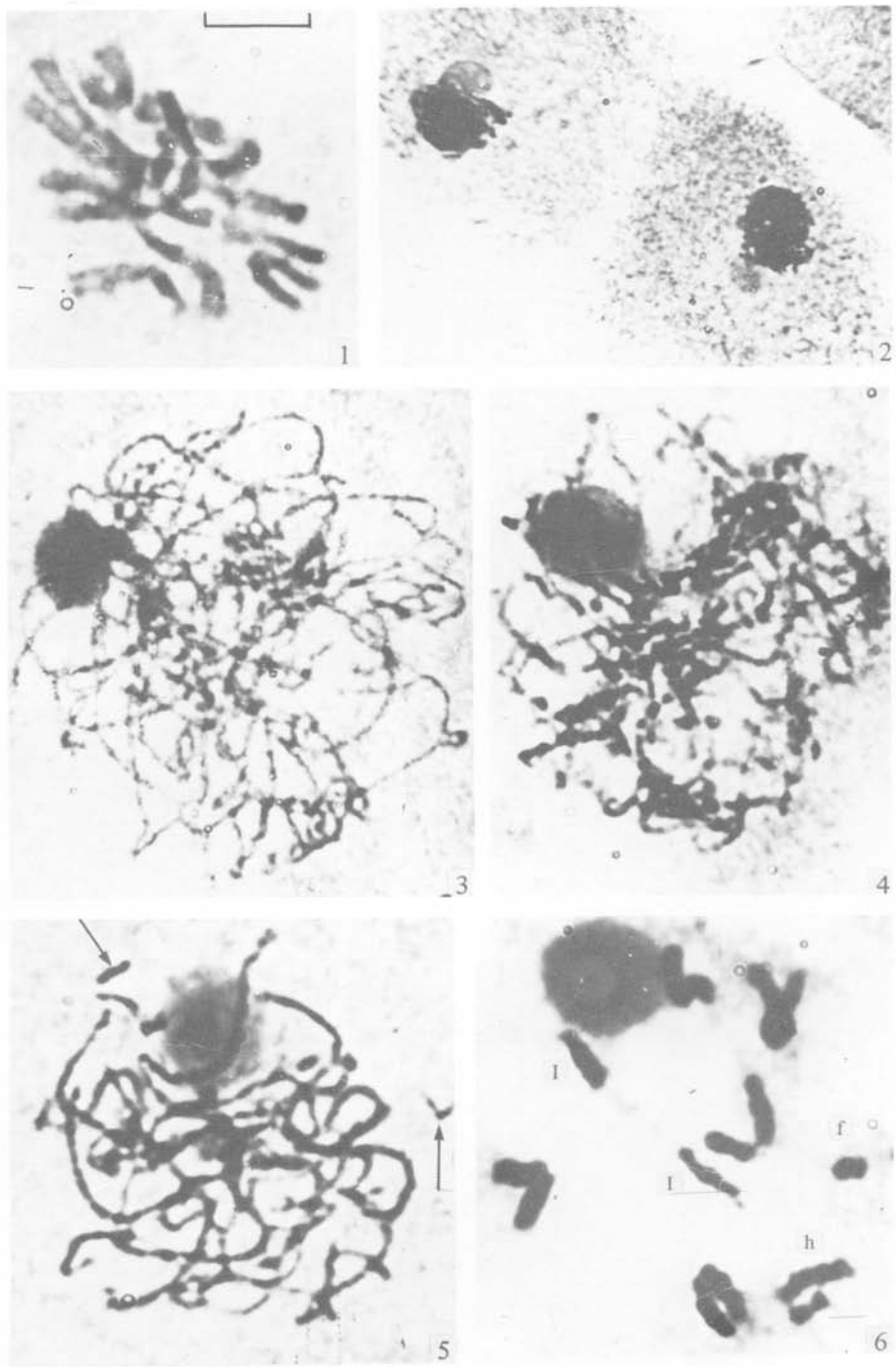
PLATES I AND II

Figs. 1–13. Cytology of plants with partial fragmentation. 1, Last premeiotic metaphase in sporogenous cells, note the absence of fragments. 2, Premeiotic nuclear phenotype of PMCs. 3, Leptotene. 4, Zygotene. 5, Pachytene – note the paired small fragments (↑). 6, Diakinesis showing 5 bivalents, 2 univalents (I), one heteromorphic bivalent (h) and the corresponding fragment (f) (1–6, same magnification, bar represents 10 $\mu$m). 7, Diakinesis showing centric exaggeration (↑), heteromorphic bivalent (h) and seven fragments (f). Note the only normal bivalent (nb). 8, MI showing weak neocentric activity. 9, AI bridge (NSU) together with one small (↑f) and one large fragment (f). 10, AI with two single bridges (NSU), 11, AII one chromatid bridge (SU) in one of the dyad cells (↑). Note the persisting AI (NSU) bridge (↕). 12, Synchronous tapetal development. 13, Late prophase of tapetal mitosis showing 14 chromosomes and no fragments (Figs. 7–10, 13 same magnification, bar represents 10 $\mu$m).




P. R. K. KODURU AND OTHERS

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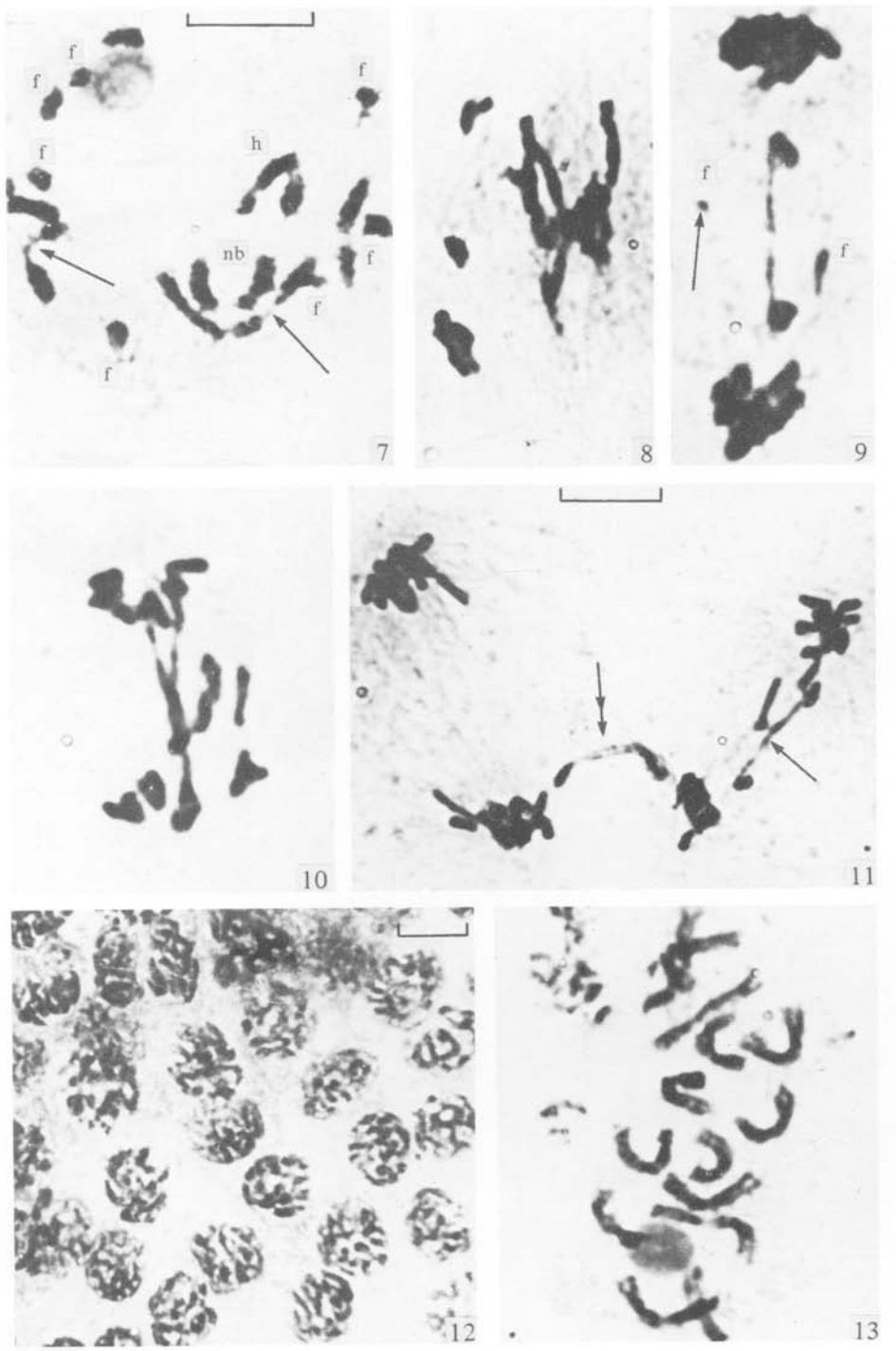


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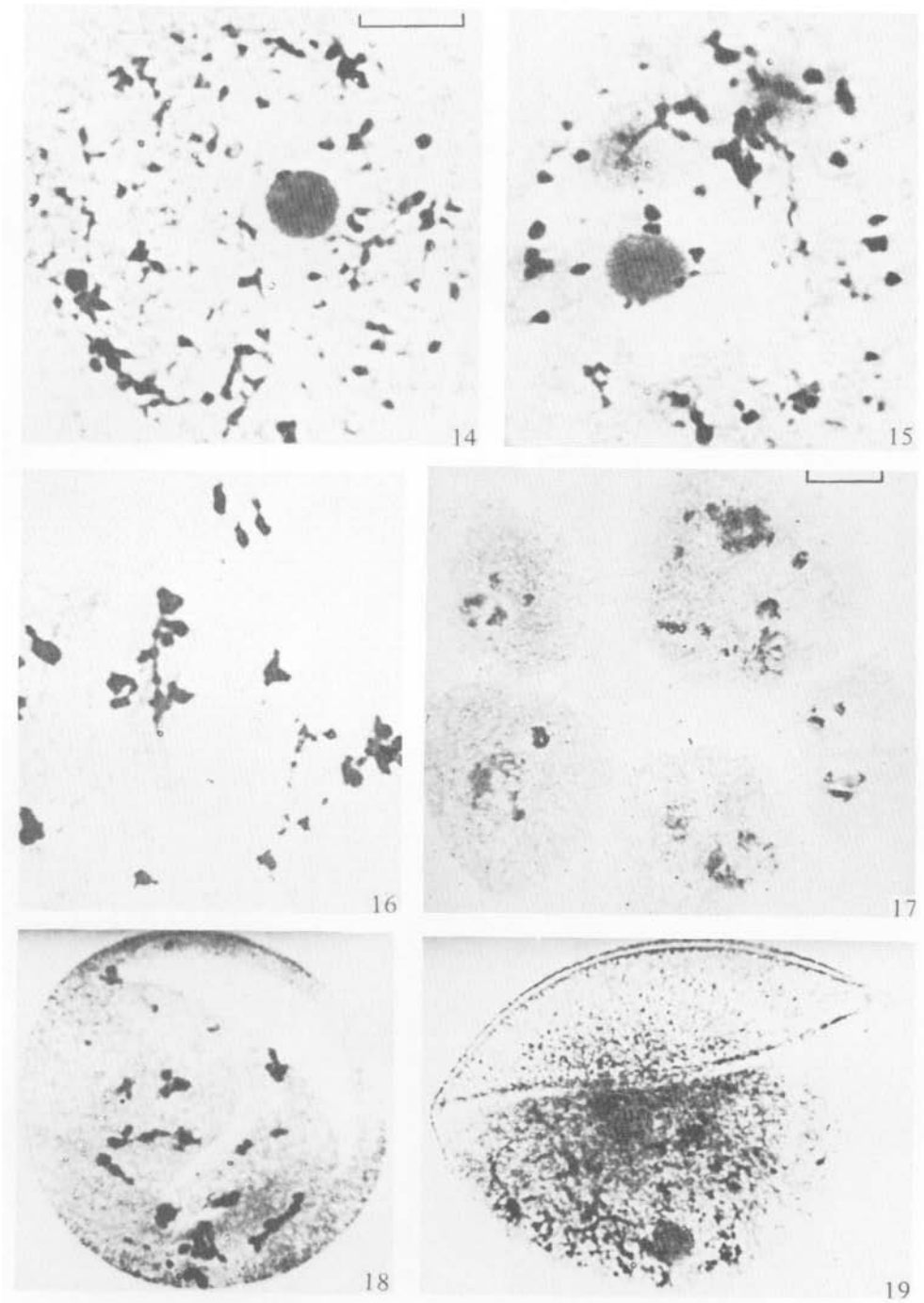


PLATE III

Figs. 14–19. Cytology of plants with complete fragmentation. 14, A stage in PMC comparable to pachytene. 15, Development comparable to diakinesis. Note the shattered appearance of the nucleus in both 14 & 15. 16, AI sticky chains. 17, Hexad at the end of TII showing a variable number of chromatin elements. 18, 19, Wall deposition and suppression of meiotic development in delayed PMCs. 18, PMC at metaphase I. 19, 'Giant pollen grain' developed from the meiocyte arrested at pachytene. (14–16, 17–19, same magnification, bar represents 10 $\mu$m).

cells showed a broad range of variation in developmental stages. Soon after the formation of tetrad cells in normal PMCs, pollen grain wall development was triggered. Interestingly, irrespective of whether it is a pollen grain or a delayed meiocyte, wall deposition started around all the meiotic cells of an anther.

Table 4. *Bridge formation at* AI *and* AII *in primary florets*

| | Plants | | | | |
|---|---|---|---|---|---|
| | 1 | 2 | 3 | 4 | 5 |
| Anaphase I | | | | | |
| Mean/Cell | 0·287 ±0·030 | 0·283 ±0·05 | 0·213 ±0·04 | 0·421 ±0·05 | 0·281 ±0·04 |
| % of cells | 21·55 | 21·39 | 21·35 | 23·25 | 21·91 |
| Cells scored | 348 | 187 | 267 | 228 | 315 |
| Anaphase II | | | | | |
| % of cells | 1·60 | 1·58 | 0·91 | 0·77 | 1·41 |
| Cells scored | 125 | 190 | 110 | 130 | 142 |

Table 5. *Distribution of* AI *bridges in primary florets*

| No. of Bridges | Number of PMCs in plants | | | | | Total cells observed | Expected | $\chi^2$ |
|---|---|---|---|---|---|---|---|---|
| | 1 | 2 | 3 | 4 | 5 | | | |
| 0 Obs. | 273 | 147 | 210 | 175 | 246 | 1051 | 1010·70 | 1·61 |
| Exp. | 261·50 | 140·52 | 200·64 | 171·34 | 236·71 | | | |
| 1 Obs. | 56 | 30 | 45 | 42 | 52 | 225 | 294·79 | 16·52 |
| Exp. | 76·27 | 40·99 | 58·52 | 49·97 | 69·04 | | | |
| 2 Obs. | 14 | 7 | 8 | 9 | 14 | 52 | 36·85 | 33·51 |
| Exp. | 9·53 | 5·13 | 7·32 | 6·25 | 8·63 | | | |
| 3 Obs. | 4 | 3 | 4 | 2 | 2 | 15 | 2·56 | 60·45 |
| Exp. | 0·66 | 0·36 | 0·51 | 0·43 | 0·60 | | | |
| 4 Obs. | 1 | 0 | 0 | 0 | 1 | 2 | 0·10 | 36·10 |
| Exp. | 0·33 | — | — | — | 0·03 | | | |
| Total | 348 | 187 | 267 | 228 | 315 | 1345 | 1345 | 148·19 |
| $\chi^2$ | 56·25 | 23·31 | 27·51 | 8·29 | 45·54 | — | — | — |

$\chi^2$ table value at 5% level is 9·49.

Consequently in addition to normal pollen grain development giant cells (Figs. 18, 19) were also formed which, however, then degenerated. Such cells indicate that the mutant gene is ineffective in disturbing the physiological development of the anther. In both types of plants, tapetal mitosis is synchronous (Fig. 12) and there were no fragments in the tapetal mitosis (Fig. 13).

Table 6. *Distribution of bivalents in cells without fragments*

| Number of bivalents per cell | Floret | Plant Number 1 | 2 | 3 | 4 | 5 | Total Observed | Expected[a] | Expected[b] | $\chi^{2b}$ |
|---|---|---|---|---|---|---|---|---|---|---|
| 0 | P | 10 | 5 | 50 | 7 | 29 | 101 | 37·19 | 74·66 | 9·29 |
| | S | 3 | 3 | 3 | 32 | 5 | 46 | 9·42 | 34·61 | 3·75 |
| 1 | P | 5 | 15 | 29 | 20 | 16 | 85 | 159·55 | 197·79 | 64·31 |
| | S | 13 | 5 | 10 | 80 | 10 | 118 | 63·31 | 118·50 | 0·00 |
| 2 | P | 35 | 45 | 102 | 90 | 64 | 336 | 293·27 | 261·89 | 20·97 |
| | S | 40 | 10 | 5 | 60 | 45 | 160 | 182·49 | 209·70 | 11·70 |
| 3 | P | 60 | 90 | 60 | 62 | 20 | 292 | 299·56 | 231·26 | 15·95 |
| | S | 85 | 25 | 25 | 30 | 75 | 240 | 292·22 | 230·80 | 0·37 |
| 4 | P | 45 | 15 | 15 | 32 | 15 | 122 | 183·56 | 153·44 | 6·44 |
| | S | 45 | 60 | 30 | 15 | 25 | 175 | 280·76 | 209·70 | 5·74 |
| 5 | P | 2 | 8 | 22 | 7 | 20 | 59 | 67·55 | 81·31 | 6·12 |
| | S | 5 | 70 | 80 | 23 | 25 | 203 | 161·85 | 136·30 | 32·63 |
| 6 | P | 5 | 13 | 13 | 7 | 5 | 43 | 13·80 | 35·90 | 1·40 |
| | S | 3 | 20 | 20 | 10 | 10 | 63 | 51·83 | 73·41 | 1·48 |
| 7 | P | 1 | 5 | 8 | 2 | 2 | 18 | 1·52 | 19·75 | 0·16 |
| | S | 2 | 10 | 20 | 9 | 3 | 44 | 7·12 | 36·71 | 1·45 |
| Total | P | 163 | 196 | 299 | 227 | 171 | 1056 | 1056 | 1056 | 124·64 |
| | S | 196 | 203 | 193 | 259 | 198 | 1049 | 1049 | 1049 | 57·21 |
| Mean bivalents per cell | P | 2·96 ±0·10 | 3·00 ±0·09 | 2·40 ±0·10 | 2·67 ±0·08 | 2·44 ±0·13 | 2·66 ±0·06 | — | — | — |
| | S | 2·98 ±0·08 | 4·35 ±0·10 | 4·53 ±0·11 | 2·27 ±0·12 | 3·19 ±0·10 | 3·40 ±0·17 | — | — | — |
| Total | P | 57·76 | 79·48 | 73·94 | 35·98 | 76·52 | 140·49 | — | — | — |
| $\chi^{2b}$ | S | 94·04 | 54·09 | 65·16 | 51·82 | 41·30 | 32·84 | — | — | — |

$\chi^2$ table value at 5% is 12·59[b].
P, primary floret; S, secondary floret; [a], random series; [b], Poisson series.

## 4. DISCUSSION

The apparent normal last premeiotic mitosis and normal nuclear phenotype during premeiotic cell development in PMCs show that the effect of the mutant gene '*mbr*' is restricted to the meiotic phases of the PMCs.

Since fragments were absent during the last premeiotic mitosis, and at both leptotene and zygotene, breakage was presumed to have occurred at pachytene. Contrary to McClintock's (1931) inversion bridge hypothesis, meiotic anaphase I bridges have been attributed to chromatid breakage and the subsequent reunion of broken ends in a U-type fashion at pachytene (Rees & Thompson, 1955; Lewis & John, 1966; Jones, 1968, 1969; Jones & Brumpton, 1971; Klein & Baquar, 1972; Giraldez & Lacadena, 1978). Additionally in several cases of synaptic mutants a reduction in chiasma frequency has been found to be associated with nonsister chromatid exchange followed by dicentric bridge formation at AI (see Koduru & Rao, 1981). This widespread association between a reduction in chiasma frequency and the simultaneous appearance of bridges and fragmentation suggests that their formation depends on the events which are regular features of meiosis and which are not found in mitosis. The more specific events of meiosis are pairing, chiasma formation and crossing over which ultimately bring about gene recombination between the pairing partners through chromatid breakage and reunion in X-type fashion (chiasmata). If an error altering the type of reunion of the broken chromatid occurs this will lead to fragmentation and bridge formation.

The relatively high number of fragments per cell and the low number of AI bridges suggests that not all breaks at pachytene are involved in reunion. Most of them must either have restituted or else remained open. In plants with complete fragmentation there was no true bridge formation at AI indicating a total failure of reunion of broken ends in these cells; consequently numerous fragments appeared. These numerous fragments may arise by multiple breaks which may or may not reunite, restitute or remain open. However it is difficult to ascertain the relation between such shattered structures and the breakage hypothesis. If the multiple fragments are the products of U-type exchanges then they might represent the innumerable potential crossover sites present in the genome. There is certainly evidence for the presence of single strand nicks during pachytene of meiosis which furnish subsequent sites for crossing over and chiasma formation (Stern & Hotta, 1978). As in the human chromosome breakage syndrome (Lohman, Bootyma & Bridges, 1977) these might be the consequence of genetic defects of DNA repair mechanism.

Analysis of bivalent distribution in cells of desynaptic type and in the partial fragmented cells and formation of AI bridges in the latter showed that the bivalent and bridge formation are non-random but specific; that is the mutant controls the anomalies on a chromosomal basis. This is in contrast to desynaptic mutants of rye where equivalent meiotic disturbances occurred at random (Giraldez & Lacadena, 1978). Further the mode of deviation of the frequencies of the anomalies from the binomial and Poisson series did not follow the same pattern in the primary

and secondary florets. This suggests the operation of environmental factors in controlling the expression of the mutant gene. Thus the interaction between genic, cytoplasmic and environmental factors may be responsible for the precise relationships obtained.

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