THE TIMING OF *Mu* ACTIVITY IN MAIZE*

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Manuscript received July 11, 1979
Revised copy received November 1, 1979

ABSTRACT

The timing of mutator activity of *Mu* in maize was tested in three ways: (1) by allelism tests of phenotypically similar male-transmitted mutants, (2) by studying the clustering of phenotypically similar mutants as demonstrated by ear maps and the subsequent allelism tests of these mutants, and (3) by the induction of somatic sectors in *Mu* plants heterozygous for plant and endosperm marker genes. Allelism tests of phenotypically similar mutants in outcrosses of *Mu* plants as males established that 18.6% were allelic and that premeiotic mutants are induced. This conclusion was supported by ear maps of *Mu*-bearing plants, which revealed sectors of seeds that produced plants bearing phenotypically similar allelic mutants. The smallness of these sectors indicated that the premeiotic activity of *Mu* that gave rise to them occurred very late. The lack of visible sectors in mature sporophytic, endosperm and aleurone tissue in plants carrying *Mu* supports the conclusion that the mutator activity of *Mu* does not occur throughout the ontogeny of the plant and seems to be restricted to a time shortly before and/or during meiosis.

ROBERTSON (1978) described a mutator system (*Mu*) in maize that increased the forward mutation rate approximately 30-fold. Plants in outcross progenies of *Mu*-bearing plants segregate more frequently for new mutants when self-pollinated (about 6%) than do outcrosses of non-*Mu* plants (about 0.2%). Approximately 90% of outcross plants also exhibit mutator activity when tested by further outcrossing. This high transmission of *Mu* is repeated in succeeding outcross generations and is not expected if mutator activity were due to a single gene. Tests (to date) indicate that neither cytoplasmic transfer nor segregation disorder activity will account for the unusual pattern of *Mu* transmission. Other possible explanations for the unusual inheritance pattern of *Mu* are currently under investigation.

In a given outcross test family, frequently more than one plant will segregate for a mutant of a given phenotype. Such phenotypic clusters could result from a somatic mutation or from independent meiotic mutants at different loci that happened to have the same phenotype. To be on the conservative side, such mutants were assumed by ROBERTSON (1978) to have a mitotic origin; thus, the putative clusters were counted as resulting from one mutational event. The mutation rate reported above was based on this assumption. If this assumption is incorrect for

* Journal Paper No. J-9461 of the Iowa Agriculture and Home Economics Experiment Station, Ames, Project No. 2300, supported in part by a grant from the National Science Foundation, PCM 76-18758.

all or some putative sectors, the mutation rate will be underestimated. Thus, the timing of *Mu* activity has an important bearing on the determination of the mutation rate and is of interest *per se* as one of the properties of this mutator system. This paper describes studies designed to provide insight into the timing of *Mu*-induced mutations.

MATERIALS AND METHODS

Mu was present in a stock of the white endosperm mutant, *y9*, which originated in genetic stocks at the University of Wisconsin. Seeds of this mutant (*y9*) were supplied by J. K. KERMICLE.

In brief, the standard procedure of screening for mutations is to self-pollinate and outcross a *Mu*-bearing stock to a standard line. Fifty or more seeds from the outcross are sown, the resulting plants are self-pollinated and the progeny scored for seedling mutations. Most seedling mutants observed are pigment deficient (*e.g.*, albinos, luteus, yellow-green, pale-green, virescent, etc.). However, other seedling mutants are also included in the scoring (*e.g.*, dwarfs, glossies, blue fluorescence, necrotics, etc.). The mutation frequency is calculated by dividing the number of plants segregating mutants by the total number of plants selfed. If appropriate, the mutation frequency is adjusted to accommodate putative allelic mutants (See ROBERTSON 1978 for more details on isolating and scoring mutations.)

A description of the three tests utilized in these experiments follows.

Allelism tests: If a mitotic mutation occurs in a *Mu*-bearing plant, a sector of sporophytic tissue would be produced that possesses the new mutant. Depending on the size of the sector, various numbers of the newly induced mutant genes will be transmitted to the outcross progeny. These will result, therefore, in several plants segregating for mutants of the same phenotype. Two or more mutants of a similar phenotype in an outcross progeny does not necessarily indicate allelism because independent mutants frequently share a common phenotype. Thus, when sibling plants of outcross progeny segregated for similar mutants, it was necessary to establish whether or not they were allelic. To test for allelism, crosses were made between similar mutant lines. Because all mutants were recessive lethal seedling traits, crosses were made by using putative heterozygous plants from self-pollinated heterozygous plants. Two-thirds of the plants from selfed ears are expected to be the heterozygotes that will provide useful information. The standard test procedure involved the self-pollination of a plant from one mutant line and outcrossing it to at least 3 plants of the other mutant line being tested for allelism. Several such selfs and outcross series were made for each allelism test. Only outcrosses from selfed plants that segregated were scored. Because the probability of selecting a homozygous normal plant in a single outcross is 1/3, the probability of missing a heterozygous plant in four outcrosses is $(1/3)^4$ ($P = 0.0123$), and $(1/3)^5$ ($P = 0.0041$) in five outcrosses. The criterion to establish nonallelism was the occurrence of four or more nonsegregating outcrosses of known heterozygotes.

Ear maps: In planting ear maps, only well-filled ears were used. Seeds were taken for a given row starting at the butt end, from which any irregularly aligned seeds had been removed. The seeds from the first 25 ovules of regularly aligned rows are taken in order and stuck to a piece of masking tape. Missing seeds were marked by an "X" on the tape, and seeds were planted at regular intervals in the same order in which they were removed from the ear. Stakes were used to mark missing seeds, and seeds that failed to germinate were noted. Six consecutive rows per ear were planted. This procedure insured that each plant tested could be related back to the position of a given seed on the ear.

Sectoring tests: To determine if *Mu* induces mitotic mutants that might be observed as somatic sectors, *Mu* plants with dominant alleles were crossed as males to lines homozygous for one or more recessive traits. Homozygous purple-aleurone *Mu* lines were crossed to stocks with the following combination of seed markers: *a sh2*, *a2 bt*, *c sh bz wx*. Control (non-*Mu*) purple-aleurone lines were crossed to the same stocks. The resulting seeds were scored under an illuminated magnifying glass for the presence of sectors showing one or more of the recessive traits. Two-by-

two χ^2 contingency tables were used to determine if the sectoring frequency of the *Mu* and control crosses differed significantly. One additional sectoring test was made involving *yg2*. *Yg2 Yg2 Mu* and *Yg2 Yg2* non-*Mu* plants were crossed to homozygous *yg2* plants and the progeny scored for the frequency of yellow-green sectors during the ontogeny of the plants. (See Table 1 for descriptions of the genes used.)

RESULTS

Allelism tests: The occurrence of mutants with a similar phenotype, which are also allelic, in a given outcross progeny is indicative of mitotic mutation. But, because many instances are known in corn in which two or more different (nonallelic) mutants can share the same phenotype, phenotypic similarity does not necessarily indicate allelism. It is, therefore, necessary that all instances of putative alleles (based on phenotype only) be checked by testing for allelism.

Table 2 summarizes the results of allelism tests made to date from outcross families when *Mu*-bearing plants are crossed as pollen parents. Eighty-one percent of the putative allelic situations were nonallelic. Among families that had only two mutants of similar phenotype tested, 23.81% (5/21) were allelic, whereas in families with more than two similar mutants tested, 42.86% of the families had allelic mutants.

Ear maps: The ear maps for two ears segregating for *Mu*-induced mutants are given in Figure 1. Ear number 1 has three putative mutant mitotic sectors, and ear number 2 has five. In sector I, two plants segregated for both yellow-green and pale-yellow mutants. The same was true for one of the plants in sector VIII. One plant in sector VII segregated for both pale-green and pale-yellow mutants. Another plant in sector VII segregated for a luteus and pale-yellow mutants. The same mutant, grown in different genetic backgrounds or under different environmental conditions, can vary considerably in phenotype. For example, some white (albino) mutants in some situations can produce considerable yellow pigment. A luteus mutant, under certain conditions, can produce some chlorophyll that might change the classification from luteus to yellow-green. Because this variation in phenotype was known to exist, mutants with similar, but not necessarily identical, phenotypes were included in putative sectors (*e.g.*, *py* and *w* in sector

TABLE 1

Genes used in sectoring tests

a — colorless aleurone when other genes necessary for color are present	(3L)*
a2 — colorless aleurone when other genes necessary for color are present	(5S)
c — colorless aleurone when other genes necessary for color are present	(9S)
sh2 — very collapsed and wrinkled endosperm	(3L)
bt — very collapsed and wrinkled endosperm	(5L)
sh — endosperm collapsed from crown or sides	(9S)
bz — with other aleurone genes present, seeds are lightly colored	(9S)
wx — seeds appear dull (waxy) rather than vitreous	(9S)
yg2 — yellow-green plant color	(9S)

* The chromosomal location of the respective genes is indicated in ( ).

TABLE 2

Allele tests of mutants with similar phenotypes occurring in a given outcross progeny of Mu *plants*

	Number of putative allelic mutants per outcross family tested					
	Two		More than two			
	Positive (allelic)	Negative* (nonallelic)	Positive (allelic)	Negative (nonallelic)	Mixed	allelic (+), nonallelic (—)
	5	16	1(3)† + 1(5)	1(3) + 1(3) + 1(3) + 1(3)	1(7—,	1+)
Total	5	16	2	12	7—	1+

Total positive allele tests = 8
Total negative allele tests = 35
Percent allelic 8/43 = 18.60

* Negative test => 0.01 probability of allelism.
† Numbers in ( ) represent the number of different combinations tested.

III, *yq* and *l* in sector III). If these are indeed sectors, the mutants within a sector should be allelic. Allelism tests established that the *py* and *w* mutants within sectors I were allelic. The sector II mutants were also allelic, as were the *py* mutants in sector VII. Mutants within sectors III, IV, VI, and VIII were nonallelic. The mutants of sector V did not segregate in the test progeny. The expression of the pale-green phenotype frequently is sensitive to temperature and light intensity. It may be that conditions in the seedling bench did not permit the expression of these mutants.

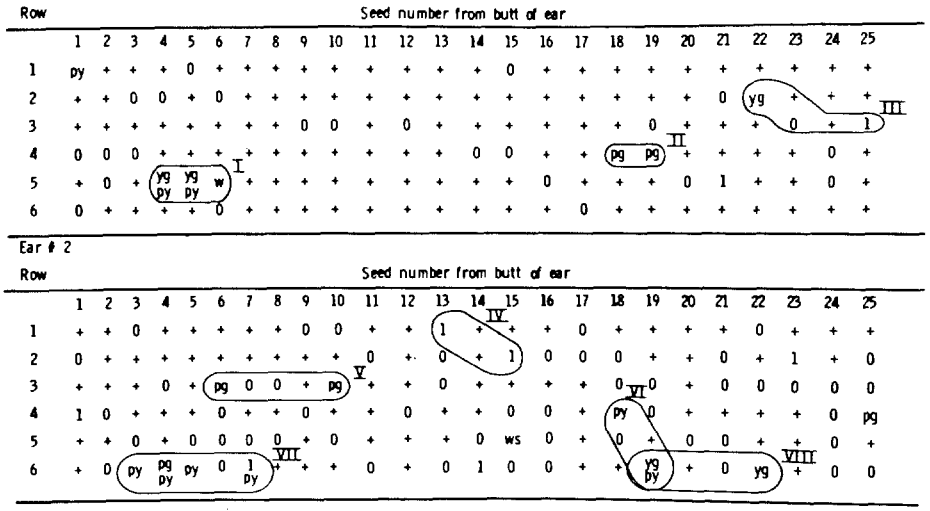

FIGURE 1.—Ear maps of mutants induced by *Mu*. [+ = ear with no mutants segregating, 0 = no ear (seed missing or seed failed to germinate or self did not take. Since regions of the ears with good seed set were chosen for sampling, most 0's are due to poor germination or loss of ears due to plant breakage, poor pollination and/or seed set or disease.) Letter(s) = mutants segregating (l = luteus, w = albino, yg = yellow-green, py = pale-yellow, pg = pale-green, ws = white stripe). Circled seeds represent putative sectors. (Each is numbered for convenience of reference in the text.)]

Sectoring tests: The frequency of sectoring in crosses involving purple aleurone *Mu* and non-*Mu* (control) stocks are recorded in Table 3. There is no significant difference in the amount of sectoring in *Mu* and non-*Mu* crosses. In a sectoring test involving 142 plants of the genotype *Mu* +/+ *yg2*, no sectors were found in mature plants.

DISCUSSION

Allelism tests: The results from these tests indicate that premeiotic mutants can occur, but the bulk of the mutants seem to be post-premeiotic S (or perhaps very late mitotic). The probability of a given testcross family having mitotic mutants increases if more than two similar mutants occur. Even in instances in which mitotic mutants occur, they must be relatively late events giving rise to small tassel sectors, inasmuch as none of the outcrosses to date with mutants of similar phenotype approaches the 50% mutant frequency expected if a mutation had occurred in the progenitor cell of the tassel.

Because of the transmission pattern of *Mu* (ROBERTSON 1978), most plants used in these allele tests carried *Mu* and, thus, new *Mu*-induced mutants similar in phenotype to the original mutants could be carried by both the male and female parents. On the male side of the allele tests, this might mean that a plant thought to be segregating for the mutant under test was in reality segregating for an entirely different mutant (a new *Mu*-induced mutant). If the two mutants being tested were indeed allelic, this would result in a negative test. However, in all but one of the negative tests, outcrosses from two or more segregating male parents were used; thus, the probability of false negative tests is reduced. If the two lines being tested were not allelic, there is the possibility that a new mutation that happened to be allelic to the female parent mutant might occur in the male line. In the positive tests, however, three or more segregating males were involved, resulting in four or more segregating outcrosses. On the female side in an allelic situation, the induction of a new nonallelic mutant would not complicate the tests. In a situation of nonallelism, the induction of a new mutant in the female allelic to the mutant in the male parent could give one positive test.

TABLE 3

Test for endosperm and aleurone sectoring in Mu *and control crosses*

Gene combinations tested	*Mu*				Control			
	Purple	Sector*	Total	% Sector	Purple	Sector*	Total	% Sector
a sh2	4,116	32	4,148	0.77	7,421	39	7,460	0.52
a2 bt	10,164	43	10,207	0.42	8,067	43	8,110	0.53
c sh bz wx	11,979	63	12,042	0.52	6,318	27	6,345	0.43

* Sector for one or more of genes under test:

Test	χ^2	*P*
a sh2	2.3180	0.20–0.10
a2 bt	0.9263	0.50–0.30
c sh bz wx	0.6252	0.50–0.30

As was indicated above, however, in instances of positive tests, more than one segregating cross was observed in each pair of mutants tested.

The frequency of total mutants induced by *Mu* is 11% (Table 4). These are distributed over a range of 18 or more phenotypes. From many genetic studies, it is known that any given phenotype can be expressed by mutants at many different loci. Thus, although the mutation rate in *Mu* stocks is high, the likelihood of any particular mutant occurring is still quite low. This is true unless a given locus is particularly susceptible to the activity of *Mu*. Tests to localize more than 40 *mu*-induced mutants to 17 the chromosome arms that can be tested using B-A translocations have revealed one or more mutants in 16 of the chromosome arms (unpublished results). Although the number of mutants located to date is inadequate for a definitive statement, there certainly is no indication that any particular arm has an unusually high number of mutants of a given phenotype as would be expected if there are loci highly sensitive to *Mu*. The possibility of a newly induced *Mu* mutant giving a false reading in an allele test cannot be ruled out. Such an event, however, would be expected very infrequently and probably would not alter the results significantly.

The data from Table 2 establish that the bulk of putative allelic mutations proved not to be. Since many of the putative allelic mutants were eliminated from previous calculations of the mutation rate, based on the total number of different mutants, the true mutation rate was underestimated. In order to obtain a more accurate approximation of the mutation rates, a sizeable proportion of the mutants that previously were not included in the "different mutant" class, because of their putative allelic status, must now be included in this class.

Table 4 summarizes the result of all *Mu* tests reported to date. In column 6 ("Percent total different mutants"), the mutation rate is estimated at 6.4%. This does not reflect the true mutation rate, however, because the occurrence of two or more mutants with similar phenotypes in a given outcross was counted as being the result of only one mutational event. From the raw data, it was determined that 34 of the total number of putative allelic mutants were included in column 5 ("Total different mutants"). This number represents one mutant for each putative allelic series. If 34 is subtracted from the column 5 total, we have

TABLE 4

Mutation rate from previous Mu *tests**

Column 1	Column 2 No. of plants	Column 3 Total mutants	Column 4 % Total mutants	Column 5 Total different mutants	Column 6 % Total different mutants
Totals	1,541	171	11.1	98	6.4
Controls	1,265	3	0.2	3	0.2

$$\frac{\text{Mutators}}{\text{Controls}} = \frac{0.064}{0.002} = 32.0$$

Comparison between the mutator and control populations reveals a contingency $\chi^2 = 97.9355$ ($\chi^2 = 6.635, p = 0.01$).

* Data pooled from Tables 2, 3 and 4 of ROBERTSON (1978).

the total of known nonallelic mutants (= 64). The difference between column 3 ("Total mutants") (= 171) and the total number of known nonallelic mutants (= 64) will give an estimate of the putative allelic mutants (= 107). We know that the majority of these 107 mutants are not involved in allelic interactions. A count of the total number of mutants involved in the tests reported in Table 2 reveals that there were 69. Of these, 19 mutants were involved in positive allelism tests. Thus, 72.46% of the mutants in potentially allelic situations proved to be nonallelic. Thus, of the 107 putative allelic mutants included in column 3 of Table 4, 72.46%, or 78, would be expected to be nonallelic and 29 to represent allelic sets. If these 78 are now added to the nonallelic mutants (= 64) of column 5, there is a total of 142 nonallelic mutations. To get an estimate of the total number of different mutants, one each of the sets of allelic mutants must be included. In the next paragraph, it is estimated that there is an average of 2.4 mutants per premeiotic event. Because there are 29 allelic mutants, this means that 29/2.4 = 12 allelic mutants should be added to the 142 nonallelic mutants to get the number of different mutants, giving a total of 154. This new total now gives mutation rate of 10.0% (154/1541), which is 3.6% more than our previous estimate and some 50-fold higher than our best estimate of the spontaneous rate.

The bulk of *Mu*-induced mutations seems to be induced after premeiotic S or in a very late mitotic division. Of situations that could have resulted from events preceding premeiotic S, only 27.54 percent turned out to be. The percent of premeiotic mutants is, thus, 1.9% (171 − 64 = 107; 107 × .2754 = 29, 29/1541 = 1.9%). The 19 mutants in positive tests (Table 2) involved eight different sets of putative allelic mutants; thus, there is an average of 2.4 mutants per premeiotic event. There are 29/2.4 = 12.08 premeiotic S events in Table 4.

As indicated above, 1.9% of the tested plants have a mutant resulting from a premeiotic event. This figure may be very misleading, however, inasmuch as there is evidence that most mitotic events occur late. If this is the case, the sporophytic sector produced will be small; thus, when *Mu* plants are crossed as males, the likelihood of two or more pollen grains from a small sector functioning in producing seeds for the next generation is reduced because of the large surplus of nonsector pollen. The chances of including seed carrying mutations from a single sector is further diminished because half the sporocytes of the sector will receive the nonmutant allele. That only 50 seeds usually were chosen for testing in each test further reduces chance of including two or more mutants from a given sector. Thus, many of the mutants scored as postpremeiotic S may, in actuality, be descended from late mitotic events. Ear maps will give a better estimate of the frequency of mitotic sectors.

Ear Maps: The ear map results confirm those from the allelism tests of male-derived mutants. *Mu* can induce premeiotic mutations that result in "clusters" of allelic mutants. It can be argued that failure to complement does not necessarily establish that two mutants are identical (*i.e.*, are defective at the same point). However, it is much more reasonable to assume that two or more mutants occurring in close proximity on an ear that give negative complementation are, indeed, the result of a single mutagenic event, rather than assuming that independent mutational events are responsible for producing a cluster of mutants

involving the same locus. It might be argued that some loci are highly sensitive to *Mu* activity and, thus, mutate much more frequently, but evidence to date suggests that this is not the case (see preceding section). Even if this were so, there would still be need to explain the appearance of these mutants in clusters on an ear.

Although results from only two ears are presented, they are sufficient to establish beyond doubt that premeiotic mutants occur. Sectors from many more ear maps will have to be tested to provide more precise data on the timing of the premeiotic event. These tests are now being made. Based on these two ears, it would seem that the mutations responsible for these sectors are extremely late because the sectors seem to involve only a few ovules. The largest sector demonstrated to date consists of five seeds. This is consistent with the tests for somatic sectoring in *Mu*-bearing plants to be discussed later.

If premeiotically induced mutations occur quite late, some single mutations that are normally classified as meiotic in origin might, in reality, be due to a very late premeiotic mutation. This could occur when only one of the line of cells derived from the one in which the mutation occurred gave rise to an ovule. If late induction of premeiotic mutations occurs in the male inflorescence, premeiotic events could be classified as meiotic because of the small number of mutants that result from the very late mitotic mutations.

Since several seeds are involved in these allelic sectors, it seems that the mutation must have occurred before the primary spikelet initials differentiated for that region of the ear. If that is the case, then both spikelet initials that develop from the primary spikelet initials should carry the mutation. Thus, two rows of seeds should be expected to carry the mutant. That was not observed for any of the three positive sectors; the allelic mutants were confined to one row. However, this pattern may have been a chance happening. In sector VII, the longest sector (row 6) was an outside row that may represent only one member of a paired row. If row 5 were the row paired with row 6, there were only one or two plants that could have segregated since most of the seeds in this region of the sector did not produce plants or ears. Sectors II and III are short, three and two seeds, respectively. Since megaspore mother cells are heterozygous for the induced mutation, half of the seeds they give rise to are expected to carry the mutations, and half are not. In these sectors, it would appear that all the members of one of the paired rows received the mutant allele, while all the members of the other paired row received the normal allele. The analysis of additional ear maps, which is underway, will reveal if the distribution of mutants expected on the basis of the paired row structure of corn is realized.

Sectoring tests: The lack of appreciable sectoring could result from the fact that the loci under study are insensitive to *Mu*. However, mutants allelic to *sh*, *sh2* and *bt2* have been found in mutator stocks. This would indicate that at least some of the loci involved in these tests can mutate under the influence of *Mu*. Very large populations of purple aleurone *Mu* lines that would permit the detection of aleurone mutants have not been grown. To date, one *r* locus mutant has occurred. As mentioned previously (the B-A tests in the alleism test section of the DISCUSSION), the evidence suggests that *Mu* is a generalized mutator that

can possibly affect all loci. Thus, there is no reason to think that there are only a limited number of *Mu*-sensitive loci, which do not include the genes involved in the sectoring tests. Tests to measure the *Mu*-induced forward mutation rate at these loci are under way.

The individual *Mu* plants in the sectoring tests were not checked by the standard test for the presence of *Mu* activity; thus, it cannot be ruled out that, in some crosses, the pollen parents had lost *Mu* (*Mu* loss is known to occur in about 10% of the progeny of an *Mu* outcross). All the *Mu* plants used for these crosses, however, had come from ears that had segregated for *Mu*-induced mutants. Thus, *Mu* was present in the preceding generation and would be expected to be present in at least 90 percent of plants crossed as *Mu* parents. If *Mu* were consistently responsible for somatic sectoring, the occasional non-*Mu* cross would have been obvious.

These results indicate that *Mu* does not induce mutation at any of the loci studied in these sectoring tests early enough to produce observable sectors at an appreciable rate. The results support the conclusion that, when *Mu* induces somatic mutations, it acts quite late in ontogeny.

ROBERTSON (1978) reported that many of the *Mu*-induced mutations are mutable. This observation is suggestive that *Mu* may be a controlling-element system. In these mutable mutants, somatic events that result in visible sectors are occurring. These could be due to the loss of a regulatory element from a controlled locus. If Mu is a controlling-element system its regulatory element, like those of other such systems, could insert at new loci, causing the induction of new mutations. This latter activity could be what was measured in this paper. If so, the data suggest that most insertions, which cause mutations, occur primarily at late mitotic or meiotic stages, while excisions that result in the somatic sectoring of mutable mutants can occur at earlier stages, resulting in visible sectors. In this material, most of these early excisions are not immediately followed by the insertion at functional gene loci that can be recognized in our tests. PETERSON (1970) has reported that when *En* transposes, it is sometimes lost. Such transpositional losses may be characteristic of mutable mutants induced by the *Mu* system. However, it should be emphasized that, at the present stage in our analysis of *Mu*, it has not been established that *Mu* is a controlling-element system. Thus, a better understanding about the relationship between mutable mutants and the induction of mutations will have to wait for further data.

The tests reported in this paper have established that somatic (mitotic) mutations can be induced by *Mu*. The evidence to date indicates that this class of mutants is induced quite late, giving rise to small sectors of sporophytic tissue carrying any given mutant gene. Inasmuch as sectors seem to be small, many of these somatic mutants may occur singly in the samples tested; thus, they would be impossible to distinguish from mutants that are post-premeiotic S. It is not possible at this time to say whether a given mutant observed in a testcross progeny had its origin before or after premeiotic S. Although mitotic mutants have been demonstrated, it is not yet possible to determine whether or not meiotic mutants occur. The presence of many single-phenotype mutants in out-

cross progeny is suggestive of meiotic mutants, but the data presented here emphasize the tentative nature of such evidence.

The production of both premeiotic and meiotic mutants in a mutator system is not unique to maize. GREEN and coworkers found evidence for a similar situation in Drosophila (GREEN 1970; GREEN and LEFEVRE 1972; GOLD and GREEN 1974). This third-chromosome mutator, *mu*, produced both clusters of mutants (premeiotic) and single (meiotic) mutants in outcross progeny. Also, GOLD and GREEN (1973) indicated that this mutator could induce early somatic mutations that did not involve the germ line. These were recognized in the F_1 progeny of crosses of homozygous *mu* females as mutants that did not transmit their mutant phenotype to their offspring. Such early somatic mutants were not observed in the *Mu* system of maize. MINAMORI and ITO (1971) found that the extra chromosomal element, delta, was responsible for inducing premeiotic clusters in a number of instances. Premeiotic mutants have also been reported among those induced by male-recombination strains of Drosophila that have mutator activity (GREEN 1977; GOLUBOVSKY, IVANOV and GREEN 1977; KIDWELL, KIDWELL and IVES 1977; THOMPSON and WOODRUFF 1978). It would seem that mutator systems of both maize and Drosophila have the potential for producing both premeiotic and mitotic mutants.

Note added in proof: Recent work of M. M. JOHRI and E. H. COE (unpublished) on the analysis of clones in maize has established that there does not necessarily have to be correspondence between paired rows with respect to mutant events, as was suggested in the discussion of the ear map results. (Cited by permission.)

I would like to thank J. R. GOLD and M. M. GREEN for permission to publish results that appeared in Drosophila Information Service.

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