Genetic Evidence of Mutator-Induced Deletions in the Short Arm of Chromosome 9 of Maize

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ABSTRACT

Evidence is presented that at least 12 of the Mu-induced yg2 mutants found in an extensive mutation analysis of this locus are the result of deletions in the region of the yg2 locus on the short arm of chromosome 9. Twelve of these putative deletions were characterized genetically, and in every instance, they were confirmed to be deletions involving chromosomal segments that include the yg2 and wd loci as well as additional portions of the short arm of chromosome 9.

The Mutator (Mu) system of maize was first described by Robertson (1978) as being responsible for a 50-fold increase in mutation frequencies. Strommer et al. (1982) and Benetzen et al. (1984) established that a Mu-induced mutant at the Adh1 locus had a DNA insertion (Mul) in the first intron of this gene. This insert was later sequenced and found to have characteristics associated with transposable DNA systems of other organisms (Barker et al. 1984).

One characteristic of transposable DNA systems is that they frequently induce deletion as well as other aberrations in the genomes of their host organisms. [See Shapiro (1983) for a recent review of transposable DNA systems in various organisms. In many instances, the role of these systems in inducing chromosomal aberrations is considered.] Dooner (1985) established that this was true for the maize Ac transposable system by demonstrating the presence of a deletion adjacent to the site of insertion of this transposon in a stable mutant derivative of the bz-m2 mutant.

In 1982, crosses were undertaken that were designed to determine, by genetic means, if the Mu system could induce deletions. Since this work was begun, Taylor and Walbot (1985) have demonstrated that a derivative stable null allele (Adh1-S3034a) of the Mu-induced Adh1 mutant (Adh1-S3034a) involves a deletion. The DNA of this null allele (i.e., Adh1-S3034a) was shown to have a deletion that started at the Mul insertion site in the first intron and extended 74 bp leftward including the intron-exon junction and 2 bp of the first exon.

The events responsible for the Mu associated deletion at the Adh1 locus and the one found at the bz locus by Dooner (1985) are unknown. For the Adh1 deletion Taylor and Walbot (1985) proposed a mechanism similar to that suggested for deletion found adjacent to IS elements (Reif and Saedler 1977; Saedler et al. 1980). Such a deletion also could result from crossing over between Mul elements on homologous chromosomes. The possibility for generating deletions by exchanges between two inserts of a transposable DNA system was suggested by Nevers, Shepard and Saedler (1986) and is particularly attractive for the Mutator system because of the large number of Mul elements in active lines. This possibility will be considered in more detail in the discussion.

The chromosomal region chosen for study was the tip of the short arm of chromosome 9 involving the yellow-green-2 locus (yellow-green plant, gene symbol, yg2). This region was selected because of McClintock's (1942, 1944) extensive study of deletions in this chromosomal segment. The major findings of McClintock's work, which bear on the studies reported here, are summarized in Figure 1.

The evidence reported here suggests that deletions of varying lengths can be generated by Mutator and that their production may be a common Mutator-induced event.

MATERIALS AND METHODS

To generate the putative deletion plants, an isolation plot was set up in the summer of 1982 in which the female (detasseled) rows were Mu stocks. The Mu parents of these Mu lines had been tested and had been shown to have an active Mu system. The male rows were homozygous yellow-green-2 plants (generation 1, Figure 2). Because the female gametophyte will tolerate longer deficiencies than the male gametophyte, this procedure ensures the isolation of the longest possible deletions, some of which might be too long for successful male transmission. McClintock (1942) has shown that deletions in this region, which extend much beyond the midpoint of the first chromomere, are not transmitted through the pollen. Using Mu plants as females has the further advantage of eliminating the problem of contamination giving false positives (i.e., yg2 contaminants).

At planting time in the spring of 1983, all available greenhouse space was used to plant seeds from the isolation plot. The seeds were sown in rows, and all yellow-green...
of the short arm of chromosome 9. Numbers in parentheses are the minimal deletions of the short arm of chromosome 9. I: Linkage map of the short arm of chromosome 9. These deletions do not include the yg2 locus. B. These deletions extend from just proximal to the yg2 locus to about half of the way through the first chromatome. The homozygous phenotype is white seedling (wd). The shorter of these deletions are readily transmissible through both the male and female. The longest deletions in this class have reduced male transmission but normal female transmission. C. These deletions involving breakpoints from about the midpoint of the first chromatome through the sixth chromatome are not male transmissible but are transmissible through the female. Homozygotes for this region, when generated by selected breakage-fusion-bridge events, result in inviable embryos. Lyg2 (yellow-green), this class have reduced male transmission but normal female transmission. Plants carrying the standard yg2 allele, when reciprocally crossed with yg2 plants, will segregate for yellow-green seedling in a 1:1 ratio in both male- and female-outcross progeny (Figure 2), pattern (I). Plants with the chromosome carrying the Mu-induced alteration also may segregate for yellow-green seedlings in a 1:1 ratio in both male- and female-outcross progeny, if a yg2 mutation was induced by Mu or if a deletion involving only the yg2 locus has occurred or if a terminal deletion is present that does not extend much beyond the yg2 locus. However, if long deletions were induced, pollen grains carrying the deleted chromosome are expected to be completely empty or are partly devoid of starch, or they could be small completely filled pollen grains (McCLINTOCK 1942). Plants carrying such deletions would be expected to show about 50% of such abnormal pollen grains. When possible, each plant was crossed as a female with standard (Yg2 Yg2) lines (generation II, Figure 2).

To determine if some of the original yellow-green mutant plants carried deletions, the progency from the ears of 43 yellow-green plants of generation II (Figure 2) were reciprocally crossed to yg2 yg2 plants in the winter of 1983-1984 and the summer of 1984 (generation III, Figure 2). Results expected from such crosses, with and without deletions being involved are shown in Figure 2 as patterns of transmission (1), (2), (3a), (3b), (4) and (5). Because the original yellow-green plants were heterozygous for the standard yg2 allele and the Mu-induced yg2 allele or a deleted chromosome, half of the seeds from the ears on the yellow-green plants will be expected to carry the chromosome with standard yg2 allele, and the other half will carry the homolog that has the chromosome with the Mu-induced mutation or deletion. Plants carrying the standard yg2 allele, when reciprocally crossed with yg2 plants, will segregate for yellow-green seedling in a 1:1 ratio in both their male- and female-outcross progeny (Figure 2, pattern (I)). Plants with the chromosome carrying the Mu-induced alterations may also segregate for yellow-green seedlings in a 1:1 ratio in both male- and female-outcross progeny, if a yg2 mutation was induced by Mu or if a deletion involving only the yg2 locus has occurred or if a terminal deletion is present that does not extend much beyond the yg2 locus [Figure 2, pattern (2)]. This latter type of deletion would be similar to the yd and some wd deletions described by McCLINTOCK (1944). Most deletions extending to the region of the midpoint of the first chromatome will be expected to show reduced male transmission but normal female transmission [Figure 2, patterns (3a) and (3b)]. If the deletion extends beyond the middle of first chromatome, male transmission may be eliminated completely [Figure 2, pattern (4)], and the more extensive deletions with four or more chromosomes missing might even show reduced female transmission as well (McCLINTOCK 1942) [Figure 2, pattern (5)]. Deletions with reduced male transmission will result in reciprocal crosses with 1:1 green:yellow-green ratios when the heterozygous plants are used as the male parent. Some plants with a deletion that is not transmitted through
the male will be expected to have a 1:1 ratio when heterozygous with $wd$. A. The cytological configuration of the end of the short arm of chromosome 9. B. The cytological configuration of the end of the short arm of chromosome 9 with the $wd$ deletion. C and D. Cytological configurations and expected phenotypes of two $Mu$-del/$wd$ heterozygotes in which the $Mu$-del is longer than that in the $wd$ chromosome. (There are many other possible $Mu$-del configurations that would also result in the albino phenotype.) Note: The $Mu$-del deletions diagrammed in Figures 3 and 4 are indicated as having terminal deletions. Terminal deletions were used for ease of understanding. It should be noted, however, that it has not been demonstrated that any of the $Mu$-del chromosomes have terminal deletions. The cytology of these aberrations have not been studied as yet.

Figure 3.—Results expected if Mutator deletion chromosomes ($Mu$-del) are made heterozygous with $wd$. A. The cytological configuration of the end of the short arm of chromosome 9. B. The cytological configuration of the end of the short arm of chromosome 9 with the $wd$ deletion. C and D. Cytological configurations and expected phenotype of a $Mu$-del/A-B heterozygote in which the $Mu$-del is the size of a $wd$ deletion. D. One cytological configuration and expected phenotype of a $Mu$-del/A-B heterozygote in which the $Mu$-del is larger than a $wd$ deletion.

Mutator-induced deletions of Maize

Mutator plants and hemizygous for the portion of chromosome 9 missing from the B-9 chromosome but present in the deleted $Mu$-del chromosome. If the region missing in the $Mu$-del plant is more extensive than in the $wd$ deletions, some of the resulting plants will be albinos. More extensive deletions in the $Mu$-del chromosome will result in inviable embryos if it is combined with the hypoploid A-B chromosome in the zygote. (See Figure 4 for a schematic explanation of the expected results.)

In summary, the results expected from the $wd$ and TB-9Sb tests are the following: (1) If the deletions in the $Mu$-del plants are small, albino seedlings should be observed in both the $wd$ and TB-9Sb tests. (2) If the deletions are somewhat larger than those in (1) above, albino seedlings will be observed in the $wd$ test but no albino seedlings will be seen in the TB-9Sb crosses.

Two types of $Mu$-del plants were tested with $wd$. In the 1984–1985 winter nursery a few generation II plants (Figure 2) which, when reciprocally crossed to yg2, gave no yellow-green progeny when crossed as males (Figure 2, class 4 type of cross) were pollinated by heterozygous $wd$ plants. In summer of 1985 individual yellow-green plants from crosses of types (3a), (3b), (4) and (5), as illustrated in Figure 2, in which the $Mu$-del/yg2 plants were used as females, were pollinated by pollen from either heterozygous $wd$ or heterozygous TB-9Sb plants.

RESULTS AND DISCUSSION

Of 779,213 seedlings screened, 125 good yellow-green seedlings were found. The frequency with which Mutator-induced events occurred on chromosome 9 involving the yg2 locus was $1.60 \times 10^{-4}$. These not only included mutations at the yg2 locus, but the putative deletions that were also induced. Unlike Mutator-induced mutants at other loci, very few of Mutator-induced alterations involving the yg2 locus were mutable. Only one clearly mutable mutant has been found to date. A second mutant when first observed appeared to be mutable but this one has not been confirmed as yet by further crossing. It is possible that some of the other Mutator-induced yg2 mutants that are yet to be tested
by additional crossing will turn out to be mutable. However, we have studied a large enough sample of the original yellow-green isolates to know that the frequency of mutable mutants is well below that expected for Mu-induced mutants. (The percentage of mutable mutants varies from one Mutator cross to another. The last determination of the frequency of mutable mutants was made in 1984. Of 395 seedling mutants scored, 193 or 48.86% were mutable.) The reason for the paucity of mutable alleles induced by Mu at the yg2 locus is not known. It is possible that the yg genes be heterozygous for deletions (see Table 2). Those plants that have been demonstrated to have putative deletions was about 42% (18 of 43) (see the next paragraph). The conclusion that most deletions are short is supported by the observation that pollen determination was normal for all except one of those plants that have been demonstrated to have been heterozygous for deletions (see Table 2).

A total of 43 F1's (plants from generation II, Figure 2) were reciprocally test crossed with yg2yg2. Eighteen F1's showed distorted ratios when crossed as males or, as females with a standard line and thus two types of heterozygotes result: Mu-del/YgZ+ and Yg2/Yg2. Both types were reciprocally test crossed. This table gives only the results from the reciprocal test crosses of Mu-del/Yg2 with yg2/yg2.

### Table 1

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### Table 2

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The heterozygotes were the result of crossing the original yg2 mutant isolate (genotype Mu-del/yg2) as a female with a standard line and thus two types of heterozygotes result: Mu-del/YgZ+ and Yg2/Yg2. Both types were reciprocally testcrossed. This table gives only the results from the reciprocal testcrosses of Mu-del/Yg2 with yg2/yg2.
in one instance, in both the male and female crosses (Table 2). No conclusions can be made as to the nature of those F1's that did not show distorted ratios or the F1's that have not yet been tested by reciprocal crossing. Among those that gave no distorted ratios when reciprocally crossed, undoubtedly, are point mutations and smaller deletions that are fully male transmissible. These are presently being tested for the presence of small deletions. The F1's with distorted testcross ratios were the most likely candidates for being deletions and these were the first to be singled out for further testing. They are the ones considered in this report.

Five patterns of transmission are observed with respect to the occurrence of yellow-green seedlings in the male and female progeny of the tested parent: (1) Both male and female progenies segregate in a 1:1 ratio for green and yellow-green seedlings [patterns (1) and (2), Figure 2] (these were not included in Table 2). (2) The female progenies segregate in a 1:1 ratio but, the male progenies, in about a 2 green:1 yellow-green ratio [pattern (3a), Figure 2]. (3) The female progenies segregate in a 1:1 ratio and, the male, in about a 10 green:1 yellow-green ratio [pattern (3b), Figure 2]. (4) The female progenies segregate in a 1:1 ratio while the male progenies have only green seedlings [pattern (4), Figure 2]. (5) The female progenies have less than 50% yellow-green seedlings, while the male crosses have only green seedlings [pattern (5), Figure 2].

The first pattern is expected for point mutations or very short deletions, including terminal deletions through the proximal portion of the yg2 locus. The rest of the patterns are those expected for longer deletions, with the deletions increasing in length in plants with patterns (2) through (5). Genetic phenomena other than deletions could also result in the distorted ratios observed (e.g., a linked pollen lethal mutant). However, the results from the crosses with wd and TB-9Sb establish that deletions are indeed male transmissible. The additional portion that is missing in these mutants is of such a magnitude that plants will not survive that are homozygous for this deleted region in the hypoploid TB-9Sb plants (Figure 4D). This is expected of larger deficiencies because, as McCLINTOCK (1944) has shown, embryos homozygous for a terminal deficiency that are much larger than
TABLE 3
Pollinations of putative heterozygous deletion plants with heterozygous \( wd \) and TB-9Sb plants

<table>
<thead>
<tr>
<th>Deletion no.</th>
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<th>Reciprocal tests of deletion/+ with yg2 as</th>
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<th>( \delta )</th>
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<tr>
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<tr>
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\*In 1:1, 2:1, 3:1, 10:1 ratio; the first number represents green seedling, the second, yellow-green.

McCLINTOCK (1944) showed that short \( yg \) deletions had normal male and female transmission and so did some \( wd \) deletions. Other \( wd \) deletions, however, had a reduced male transmission. The latter were thought to be longer deletions. Because this putative Mu-induced deletion has a reduced male transmission, it probably would have segregated for white seedlings if it had been tested by crossing with \( wd \) bearing plants. The fact that it segregated for white seedlings when crossed with TB-9Lb plants is in agreement with this conclusion.

Deletions 116-10 is of special interest because it is the only deletion so far tested that is not male transmissible but yet when made homozygous by using TB-9Sb the Mu-del/A-B embryos that are produced are viable. Although the embryos can germinate, the albino seedlings they produce are not viable. Thus, the lack of male transmission of a deletion does not necessarily indicate that in the homozygous condition it would be an embryonic lethal. There must be some loci in this region that are essential for the normal functioning of the male gametophyte but are not required for embryonic development or early seedling growth.

Based on the results of the reciprocal \( yg2 \) tests and the crosses with \( wd \) and TB-9Sb, four classes of mutants can be recognized. One of these (Class 1) includes the previously described patterns (2) and (3). Classes 2 and 3 subdivide pattern (4), and class 4 corresponds to pattern (5). The relative sizes of these deletions are illustrated in Figure 5. There is no evidence that any extend into or through the \( sh \) locus (\( C \) cannot be tested because the original Mu stock was \( c^+c^+ \)). One class 3 mutant (107-2) is known not to include \( sh \). In this instance, the Mu-del/yg2 parent carried \( sh \) on its normal chromosome along with \( yg2 \). The heterozygous \( wd \) parent also had \( sh \) on the homolog with

\( wd \), and \( Sh \) on its other chromosome 9. The \( F_1 \) between these two plants segregated for shrunken seeds, and when the seedling test was made, nonshrunken and shrunken seeds were planted separately. All the seedlings from the shrunken seeds were yellow-green, except for one crossover seedling. The nonshrunken seeds gave both green and white seedlings but no yellow-green seedlings. If the deletion had extended through the \( sh \) locus, the seed giving rise to the Mu-del/yg2 plant would have been shrunken, which, phenotypically at least, was not the case, and white seedlings would have been produced by some of the shrunken seeds in the \( wd \) cross. Although \( sh \) was segregating in the \( yg2 \) population, in no instance were shrunken seeds found on the Mu ears from the isolation plot in which the yellow-green seedlings were originally produced. Also, in the crosses of Mu-del/ standard plants with the same \( yg2 \) stocks, no shrunken seeds were produced. These observations would suggest that deficiencies extending from the \( yg2 \) locus through \( sh \) are not transmissible through either pollen or egg, or they are not produced.

How does the Mu system generate these deletions? The deletions McCLINTOCK (1944) analyzed were the result of a breakage-fusion-bridge cycle (BFB cycle). It is possible that Mu insertions could result in chromosome breakage and start a BFB cycle. Perhaps some or all of these deletions were produced via such a mechanism. Although this possibility cannot be ruled out entirely, it seems unlikely. The standard \( yg2 \) stocks used in these tests were segregating for \( sh1 \) (shrunken seed). If BFB cycling was occurring, some plump seeds with shrunken sectors should have been observed. Also, in other tests in which new mutants for aleurone color genes were being sought, involving population sizes exceeding one million, no seeds have been found with the type of sectoring that would indicate a BFB cycle had been induced. These tests involved purple aleurone Mutator male parents and multiply marked aleurone tester stocks (i.e., \( a1 sh2, a2 bml bt1 \) and \( c sh bz wx \)).

Mutator-induced deletions could be produced by unhealed breaks generated by the Mu insertion, resulting in a terminal deletion. TAYLOR and WALBOT (1985) have shown that deletions can occur in association with Mu. In the deletion that they studied, the integrity of the chromosome was maintained, except

\[ sht \\
\[ sht \]
for the deleted portion that extended 74 base pairs proximally from one end of the insertion. The complete Mu1 insert remained in place. The exact mechanism that induced this deletion is not known, but the authors propose that it might be through a system analogous to the deletions mediated by the IS family of elements. Deletions that occur at the termini of these elements, leaving the element intact, have been reported by REIF and SAEDLER (1975, 1977), OHTSUBO and OHTSUBO (1978), SAEDLER et al. (1980), and SOMMER, SCHUMACHER and SAEDLER (1981). Whether the mechanisms proposed for such deletions in prokaryotes (e.g., REIF and SAEDLER 1977 and SAEDLER et al. 1980) are operative in maize is unknown. Whatever the mechanism responsible for inducing deletions associated with Mu1, it is possible that it might result in terminal or subterminal deletions if the insertion occurs near the end of a chromosome arm. Because TAYLOR and WALBOT found the deletion at only one end of the Mu1 insert, the type of deletion (i.e., terminal or subterminal) might depend upon the orientation of the insert. If the insert were oriented so that the deletion occurs distal to the insertion, a terminal deletion might result; if it were oriented in the opposite direction, an internal deletion might occur. These postulated events assume that deletions are associated with only one end of the insert. This, of course, is not established because only one Mu-induced deletion has been analyzed at the molecular level. In prokaryotes, deletions can occur at either end of an insert (SOMMER, SCHUMACHER and SAEDLER 1981).

Another mechanism for the production of Mu1-mediated deletions might involve crossing over between Mu1 elements on homologous chromosomes. Such a mechanism was proposed by NEVERS, SHEPHERD and SAEDLER (1986) as a possible means by which transposable elements could generate deletions. This possibility is particularly attractive because of the high copy number of Mu1 elements present in an active line (i.e., 10–30 or more copies). If in the Mu parent, one chromosome 9 had a Mu1 insert distal to the yg2 locus and the homologous chromosome 9 had a Mu1 inserted proximal to the wd locus, pairing and crossing over between these two Mu1 elements will generate a duplication chromosome and a deletion chromosome including the yg2 and wd loci and various additional chromosomal material proximal to wd (Figure 6), depending upon the position of the most proximally inserted Mu1 element. If this model is a correct explanation for some or all of the deletions in this study, then some or all of these deficiencies would not be terminal, but they would most likely retain the terminal knob of nine and perhaps part of the first chromomere. (Note: In the explanatory diagrams in this paper all Mu-del chromosomes have been shown as terminal deletions. This was done to facilitate the explanation and was not meant to imply that these deletions are indeed terminal.) Cytological analyses of these deletions will determine if they are terminal or subterminal and the extent of the deleted segments. The results of such analyses, however, may or may not provide insight into the mechanism involved in Mu1-mediated deletions. If the terminal knob is missing, terminal deletion can be assumed to have occurred. However, if the knob is present, cytological observations may not provide much insight into the extent of the deletion. Such a determination will have to be made through molecular studies.

The mechanism suggested in the foregoing model might be responsible for the production of the stable Adh1-S3034a derivative of Adh1-S3034, studied by TAYLOR and WALBOT (1985). Adh1-S3034a had a deletion that starts at the Mu1 insertion point and extends leftward through the last two base pairs of the first exon of this gene. Pairing between the Mu1 element in the first intron (Adh1-S3034) and a premeiotic insertion of a Mu1 element in the distal portion of the first exon of the Adh1 followed by crossing over in the region of the insert could have generated the deletion that TAYLOR and WALBOT observed (Figure 7). There is no evidence reported of a second insert at this locus in stocks derived from Adh1-S3034. However, because active Mu lines, and Adh1-S3034 is such a line, will have Mu1 transposition occurring each generation, it is possible that a second insertion had taken place at the Adh1 locus.

The model suggested here for the chromosome 9 deletion could be tested both by genetic and molecular analyses. The genetic consequence of such a mechanism would result in an exchange of outside markers when a deletion is generated. This can be tested by repeating the isolation procedure described in this paper, but instead of using a standard Mu stock as the female parent, stock should be used that would permit the scoring of crossovers in the region between Dt
(distal to yg2 and near the end of the short arm of chromosome 9) and sh (proximal to yg2).

A Mu stock homozygous for the a1-m allele (responds to Df) and heterozygous for Df, sh and a piece of chromosome 3 that has been transposed into chromosome 9 between bz and wx (Tp9) could be used for a test of the crossover hypothesis. RHODES (1968) has shown that plants heterozygous for Tp9 have crossing over reduced in the yg2-sh region from about the standard 21% to about 1-2%. If deletions in the yg2 region are the result of crossing over between Mu1 insertions, three results are possible with respect to new yg2 deletions that result from this cross: (1) The frequency of yg2 mutants would be sharply reduced. This result is expected if most deletions are the result of crossing over, and if the effect of the heterozygous Tp9 condition on reducing crossing over extends to the end of the short arm. However, most deletions would be accompanied by an exchange of outside markers, because double crossovers would be unlikely. (2) The frequency of yellow-green deletions would not be reduced or only slightly, and most of them would be accompanied by the exchange of outside markers. This is expected if the crossing over suppression does not extend to the yg2 locus. (3) There would be no relationship between the exchange of outside markers and the production of yg2 deletions. This result is expected if crossing over is not involved in generating the yg2 deletions.

If Mu1 indeed remains in the deleted chromosome, the series of deletions so far analyzed, and those yet to be, would be useful tools for sequencing the DNA in the region of the yg2 and the wd loci on chromosome 9. Presumably, each Mu1 element will be inserted in a different segment of this region of chromosome 9, and thus, each would have different sequences of host DNA adjacent to the insert. If among these adjacent sequences from different deletions, overlapping sequences are found, it would be possible to “walk” through this region. If such an analysis proves feasible for this region of chromosome 9, it is very likely that series of deletions could be used for sequencing many other regions of the maize genome. A second likely region would include the terminal end of the long arm of nine for which we have already obtained 240 Bf1 mutants, many of which are probably deletions. Tests for deletions in this region are under way at present. There is no reason to assume that terminal regions are the only ones that could be analyzed in this manner. We have well over three hundred Mu-induced mutants at the y1 locus. As yet, only preliminary tests have been undertaken with these to determine if any mutant phenotypes are the result of deletions. The indications are that some do involve small deletions. The presence of small deletions, which have reduced male transmission, can be confirmed in these stocks with another generation of crosses, but it will require two more generations to confirm the presence of larger deletions that are not male transmissible. We have also observed a sector of three a1 a1 sh2 sh2 seeds on a homozygous A1 A1 Sh2 sh2 Mu stock that was pollinated by a1 a1 sh2 sh2. These seeds could be the result of a Mu-generated deletion.

Over half of new Mu-induced mutants are stable (not mutable). In this respect, Mu differs from most other transposable DNA systems. These stable mutants might be deletion mutants generated by crossing over between Mu1 elements inserted at slightly different positions on homologous chromosomes, as suggested by the proposed crossover model.

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