Theoretical and Applied Genetics 43, 190–195 (1973)

Unstable Mutants of Bronze Induced by Pre-Meiotic X-ray Treatment in Maize[1]

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Summary. A study was made of the effects of pre-meiotic x-irradiation on the bronze locus in chromosome 9 of maize. Plants of *Sh Bz Wx/Sh Bz Wx* constitution were treated with ca. 1000r and pollen from these individuals was applied to silks of *sh bz wx* tester plants. In the F_1 progeny, three *Sh Wx* kernels having a *bz* aleurone or showing *Bz-bz* variegation (the *bz-x3*, *bz-x4* and *bz-x5* mutants) were selected as possible mutations at the *bz* locus. One kernel of *sh bz wx* phenotype as well as one exhibiting *sh*, *bz* and *Wx* (*sh-bz-x3*) were also selected for more intensive study. Progeny tests of the *sh bz wx* individual along with cytological observations indicated that a ring chromosome was the probable cause of the mutant phenotype although an alternative hypothesis is not ruled out. The behavior of *sh-bz-x3* can be interpreted as the result of either a minute deficiency involving the *Sh* and *Bz* loci or a simultaneous suppression of the two dominant alleles. Progeny of the *bz-x* mutants exhibited genetic instability of bronze. It is hypothesized that this behavior is due to the activation or alteration by x-rays of gene control mechanisms which affect the bronze gene.

Introduction

From experiments designed to reveal the nature of x-ray-induced mutations in maize, no data have been obtained indicating that intragenic changes occur. In an extensive study, Stadler (1944) was unable to induce somatic reversions of the a_1 (anthocyanin) allele although it is known that this gene reverts in the presence of *Dt* (Rhoades, 1938). In 1948, Stadler and Roman reported that among 415 x-ray-induced losses of A_1 activity, three were putative intragenic mutations. Extensive analyses indicated that they were, in fact, minute deletions of the *A* locus. Likewise, no x-ray-induced intragenic mutations were found by Emmerling (1955) and Nuffer (1957).

A study of x-ray-induced forward mutations at the closely linked bronze and shrunken loci in maize yielded no evidence of intragenic mutation (Mottinger, 1970a). Two alterations involving only the bronze locus (*bz-x1* and *bz-x2*) and one involving bronze and shrunken (*sh-bz-x1*) proved to be minute deletions. A fourth mutant, *sh-bz-x2*, appears to be due to a gene regulating system since it has reverted to the dominant state (Mottinger, 1970b).

Data from experiments with *Drosophila* indicate that certain alleles of forked, yellow, scute and white are prone to x-ray-induced back mutation when treatment is applied pre-meiotically (Lefevre and Green, 1959; Green, 1961 and 1962). However, conclusive evidence has not been obtained indicating that these are intragenic changes.

Studies with Chinese hamster cell cultures (Chu, 1971) indicate that some forward mutations to azaguanine sensitivity which arise in x-rayed cultures can be induced to revert by treatment with ethyl methanesulfonate (EMS), N-methyl-N'-nitro-N-nitrosoguanidine (MNNG) and 2-methyl-6-chloro-9-[3-(ethyl-2-chloroethyl)aminopropylamino] acridine dichloride (ICR-170). Chu suggests that such revertibility constitutes evidence that the original forward mutations were intragenic changes, either base substitutions or additions and deletions. However, he reserves the possibility that suppressor mutations may have been involved.

In addition to the studies with hamster cells, investigations with *Neurospora* have demonstrated the ability of x-rays to induce intragenic mutations and in fact, the base pair alterations have been partially identified (Malling and deSerres, 1967).

In the original studies on the bronze locus in maize, x-rays were applied to mature pollen or to tassels at a stage prior to anthesis (Mottinger, 1970a). Thus in the F_1 populations, forward mutations included those due to gross chromosomal aberrations as well as those resulting from minute changes. Presumably, the only pollen grains incapable of functioning were those in which mutations were induced affecting gametophyte development after the stage of treatment.

In the present study, plants were irradiated prior to meiosis for two reasons. Firstly, the meiotic divisions and the male gametophyte stage would act as efficient screens eliminating mutations in which genetic information necessary for vital processes from meiosis through fertilization was destroyed or removed. Since genes affecting gametophyte development are widespread in the maize chromosomes, any substantial deletions or gross aberrations would result in pollen abortion and only those grains containing minute alterations would be able to accomplish fertilization. Secondly, it was felt that mutations induced

[1] Dedicated with appreciation and affection to Dr. M. M. Rhoades on the occasion of his 70th birthday.

at that stage might be of a different nature from those produced in the mature pollen. The *Drosophila* experiments indicate that reverse mutations are recovered primarily from pre-meiotic rather than post-meiotic irradiation (Green, 1962).

While no evidence of x-ray-induced intragenic changes was found in the present work, some of the mutants recovered are not deletions but involve gene regulating mechanisms. This paper constitutes a preliminary report on the nature of these mutations.

Materials and Methods

Bronze-1 is located in the short arm of chromosome 9, approximately two map units proximal to shrunken-1 and 18 units distal to the waxy locus. In the proper genetic background, the dominant allele of bronze, *Bz*, produces a purple color in the plant and aleurone while the recessive, *bz*, results in a bronze pigment in those tissues. Kernels with the dominant *Sh* are plump and those with the recessive *sh* are collapsed or shrunken. The dominant allele of waxy (*Wx*) produces starch in the endosperm, female gametophyte, and pollen which stains blue with iodine potassium iodide (IKI) while that produced by the recessive (*wx*) stains red.

Plants homozygous for *Sh, Bz, Wx* and *Pr* were treated with ca. 1000r at a stage prior to meiosis. Microsporocytes of the two largest plants in the stock to be irradiated were examined to insure that they had not entered the meiotic divisions. It was assumed that the smaller plants were at the same stage or an earlier one. The x-ray source was a Picker console therapy unit operating at 280 kV and 20 ma. No filter was employed. Pollen from the irradiated plants was applied to silks of a *sh bz wx pr* stock. In the presence of *Bz*, the *Pr* allele produces a purple aleurone color while the *pr* phenotype is red. *bz Pr* and *bz pr* kernels can be distinguished on the basis of color shade but positive confirmation of the phenotype is best made by crossing to a *Bz pr* tester and noting the red or purple color.

Results

In the F_1 progeny of the irradiated stock, ten *Sh Wx Pr* kernels having a *bz* aleurone or showing *Bz-bz* variegation were selected as possible mutations at the *Bz* locus. Of these, two did not germinate and in testcrosses of five, the *bz* phenotype was not transmitted. The remaining three have been designated *bz-x3*, *bz-x4* and *bz-x5*. The original *bz-x3* and *bz-x5* kernels had a few small sectors of *Bz* tissue on a *bz* background. These mutants will be considered further in a later section.

In addition to the *bz* mutations, four kernels of *sh bz Wx Pr* phenotype were obtained. Of these, one did not germinate and a second did not transmit the phenotype to its progeny. The third case, although *sh bz Wx Pr* in phenotype, was considered to be a product of contaminating pollen because it lacked an aleurone color factor present in the irradiated stock which produces different levels of pigmentation when passed through the male and female gametophytes. The fourth case has been designated *sh-bz-x3*.

Nine kernels of *sh bz wx Pr* phenotype were recovered. Of these, six did not germinate and two did not transmit the phenotype. The remaining case has exhibited an aberrant behavior in progeny tests which will be described more fully in a later section.

The constitution of the exceptional kernels which did not germinate cannot be determined. Those which did not transmit the mutant phenotypes may have arisen following some coincidental event affecting only the sperm uniting with the polar nuclei or by a spontaneous loss of chromatin in the endosperm tissue.

Studies of *sh-bz-x3*

Since the *sh-bz-x3* mutation was induced before the meiotic divisions and was transmitted through the male gametophyte stage, it was assumed that if it involved a loss of chromatin, the loss was minute. Any extensive deficiency in the bronze region would include genetic information necessary for the survival of the pollen. To assess the nature of this mutant, studies were made of male and female gametophyte transmission frequencies and of crossing over in the *sh-bz* segment (region 1) and the *bz-wx* interval (region 2) in both the microspore and megaspore mother cells. The results from these tests and from a control series are listed in Table 1. In the recombination and transmission studies, control plants were first cousins of the mutant heterozygotes except in the case of family #2288. In this instance, the control and experimental plants were full sibs.

The absence of crossing over in region 1 suggests that the *Sh* and *Bz* loci may be deleted. However, in the case of *sh-bz-x2* where the mutant phenotype is probably due to a gene regulating element, no crossovers have been recovered between the two loci (Mottinger, 1970a). Hence, no conclusions regarding the nature of *sh-bz-x3* can be made on the basis of this observation.

If a deletion in the heterozygous condition disrupts homologous pairing, a reduction in crossing over is expected between the deletion site and flanking markers. The mean recombination frequency in megaspore mother cells for the *bz-wx* segment is slightly less in mutant heterozygotes than in control plants but the difference is not statistically significant. Also, there is considerable overlap in values from the experimental and control series. It appears therefore that *sh-bz-x3* has no effect on *bz-wx* crossing over in the female.

Recombination for region 2 in pollen mother cells of *sh-bz-x3 Wx/Sh Bz wx* heterozygotes appears to be greater than in the control heterozygotes. The mean recombination frequencies are 24.1% and 19.3% respectively. The difference is significant at the 1% level. However, in family #2288, which includes full sib plants in the experimental and control groups, the figures for the two classes overlap. The recombination value for plant #2288-4, a normal heterozygote is 24.5% while the values for the three mutant heterozygotes, #2288-1, #2288-2 and #2288-3, are 22.0%,

Table 1. *Recombination and transmission data from sh-bz-x3 Wx/Sh Bz wx individuals and sh bz wx/Sh Bz Wx control plants. Transmission values represent # sh bz* kernels/# *Sh Bz* kernels

Plant #	% recombination in microspore mother cells		% recombination in megaspore mother cells		% transmission of *bz* by female gametophyte	% transmission of *bz* by male gametophyte	population totals	
sh-bz-x3	*sh-bz*	*bz-wx*	*sh-bz*	*bz-wx*			male testcross	female testcross
2285-3	0	27.3	0	16.1	102.0	54.0	1604	422
2285-4	0	28.2	—	—	—	70.7	1581	—
2287-2	0	27.9	—	—	—	73.8	1562	—
2287-3	0	21.5	0	11.0	97.8	75.0	1073	263
2287-4	0	19.1	0	13.9	108.0	86.3	1062	281
2288-1	0	22.0	0	16.0	98.0	64.7	1400	299
2288-2	0	28.3	0	21.7	99.9	69.8	1489	406
2288-3	0	18.6	—	—	—	72.7	1635	—
Mean	0	24.1	0	15.7	100.1	71.0		
Control								
2261-1	3.1	17.7	—	—	—	92.7	744	—
2261-5	—	—	1.6	19.3	86.3	—	—	257
2261-6	—	—	1.6	16.2	79.8	—	—	257
2281-3	2.1	16.2	—	—	—	89.5	1321	—
2281-4	1.6	19.1	1.7	14.1	112.0	98.1	802	235
2288-4	1.9	24.5	—	—	—	102.6	1711	—
Mean	2.2	19.3	1.6	16.5	95.8	92.7		

28.3% and 18.6% respectively. In order to determine if *sh-bz-x3* increases crossing over in the *bz-wx* interval, an extensive analysis of recombination in full sib plants of *sh bz wx/Sh Bz Wx* and *sh-bz-x3 Wx/Sh Bz Wx* constitution must be completed.

Although no conclusions can be drawn regarding the effect of *sh-bz-x3* on crossing over in the *bz-wx* region, it is clear from the data that crossing over is higher in the male than in the female. In the experimental and control series where data were obtained from both male and female flowers of the same plant, the differences in recombination frequencies were significant. Numerous studies comparing crossing over in the mega- and microsporocytes of maize have been made. (For a review, see Phillips, 1969.) In the short arm of chromosome 9, Stadler (1926) found recombination values for the *sh-wx* region significantly higher in male than in female flowers while Collins and Kempton (1927) reported significant differences in both directions for the *c-wx* interval (*c* is distal to *sh*). In some plants, crossing over was higher in the males while in others, the opposite was true. Although the intervals considered in these two reports and the experiments described here are not precisely the same, the *bz-wx* segment is common to all of them. The variable behavior of this region of chromosome 9 is probably due to genetic modifiers in the various stocks.

Transmission through the female gametophyte of a chromosome 9 carrying *sh-bz-x3* is unaffected by the presence of the mutant allele. Deviations of *Sh Bz* and *sh bz* phenotypes from a 1:1 ratio in testcross progenies of mutant heterozygotes used as females occurred in both directions and did not approach significance. On the other hand, in progenies of *sh-bz-x3/Sh Bz* plants used as male parents, deficiencies of the *sh bz* class were significant at the 1% level in all but one case (plant #2287-4) where the deviation was significant at the 5% level. The transmission frequencies of the mutant allele through the pollen ranged from 54.0% to 86.3% and averaged 71.0%. Hence, genes in the *sh-bz* region which affect pollen function are either deleted or repressed in the homolog containing *sh-bz-x3*.

In addition to the tests on recombination and gametophyte transmission, crosses were made to determine the viability of *sh-bz-x3* homozygotes. In the progeny of three self-pollinated *sh-bz-x3/Sh Bz* individuals, the percentages of *sh bz* kernels were 14.3, 18.8 and 20.9 (Table 2). Thus, the mutation is at least partially viable in the homozygous condition in the mature kernel. The observed frequencies are less than the 25% expected in an F_2 population; but part if not all of these deficiencies can be explained by the reduced transmission of the mutant allele

Table 2. *Homozygote viability and male gametophyte transmission of sh-bz-x3 from three sh-bz-x3/Sh Bz plants*

Plant #	% transmission of *bz* on testcross ears				% *bz* kernels in selfed progeny
	1	2	3	4	
2285-4	54.8	72.5	77.3	86.0	14.3
2287-2	64.0	71.2	78.4	81.3	18.8
2288-3	55.5	70.7	76.7	93.2	20.9

through the male gametophyte. Table 2 lists the transmission values when pollen from each of the three self-fertilized individuals was applied to four tester plants. In the case of #2285-4, the homozygote frequency of 14.3% would require a pollen transmission percentage of 40.1 if the homozygous mutant condition is not deleterious. This figure is below the range of values in the four testcrosses. From plant #2287-2, the homozygote frequency of 18.8% indicates a transmission of 60.3% while in #2288-3, the transmission percentage must be 71.8. The last figure falls within the range of values from the four testcross ears of #2288-3. In the case of #2287-2, the required percentage is slightly below those from the testcrosses. Thus, if *sh-bz-x3* homozygotes are not 100% viable in the mature kernels, they are very nearly so.

The wide range of transmission percentages obtained when pollen from identical samples was applied to four female testers suggests that a compatibility factor may be operating. The ear parents may vary in their production of substances differing qualitatively or quantitatively which affect the growth of *sh-bz-x3* pollen tubes in their silks. Competition between *sh-bz-x3* and normal grains would result in different transmission rates of the mutant on different female parents.

Sporophytes derived from homozygous *sh-bz-x3* kernels are able to complete the life cycle and produce well filled ears. The size of the plants, however, is substantially reduced in comparison to normal sibs. Thus, the *sh-bz-x3* alteration must include genes involved with sporophyte development.

A cytological analysis of the pachytene stage from *sh-bz-x3*/normal plants has revealed no abnormalities in the short arm of chromosome 9. However, this does not rule out a deficiency hypothesis. Previous studies (Stadler and Roman, 1948; Mottinger, 1970a) have indicated that minute deletions in maize are not necessarily observable cytologically.

Test of the Mutant Involving *Sh*, *Bz* and *Wx*

Since the loci affected in this mutant occur within a segment comprising approximately 20 map units on the short arm of chromosome 9, and since the pollen grain containing the alteration accomplished fertilization, it was assumed that the cause of the mutant was not a deletion. A deficiency spanning such a long interval could not survive the gametophyte stage. This assumption was confirmed by progeny tests of the plant arising from the exceptional kernel. This kernel yielded a bronze plant which, when used as a female in a testcross, produced a semisterile ear with 21 *sh bz wx* and 5 *Sh Bz Wx* progeny. In the reciprocal cross, a well filled ear contained kernels of the following types: 152 *sh bz wx*; 8 *sh bz Wx*; 1 *Sh Bz wx*; 13 *Sh Bz Wx*; and 11 kernels mosaic for *Bz* and *bz* tissue.

Testcrosses of plants arising from 32 of the 152 *sh bz wx* kernels produced 30 ears yielding only *sh bz wx* progeny and two exceptional ears. The kernels on the first ear included: 1 *Sh Bz Wx*; 3 *Sh Bz wx*; 1 *sh bz Wx*; and 80 *sh bz wx*. On the second ear, 58 *Sh bz wx*, 1 *sh bz Wx* and 46 *sh bz wx* kernels were produced.

The inheritance patterns observed here could be accounted for by the presence of a ring chromosome involving a portion of the short arm of chromosome 9, extending from the centromere to a point beyond the *sh* locus. (For the behavior of ring chromosomes in maize, see McClintock, 1938.) The cases, including the original mutant kernel, in which the endosperm exhibited only recessive phenotypes but progeny from plants arising from such kernels expressed the dominant characters, can be explained by the assumption that a ring containing *Sh*, *Bz* and *Wx* was lost in the endosperm but retained in the embryo. Instability of the ring in subsequent cell generations could give rise to kernels exhibiting various combinations of the dominant phenotypes or to mosaic kernels.

Presumably, the plant arising from the original mutant kernel contained a normal chromosome 9 from the tester parent and a ring possessing most of chromosome 9 from the irradiated parent. High sterility on the ear of this plant would result from ovules which received a ring deficient for enough chromatin to cause abortion. Since pollen grains from this plant would function only if they possessed sufficient genetic information, little or no sterility on the ear of the reciprocal cross is expected. The observed results fulfill this condition.

Examinations of the pachytene stage in sporocytes of plants derived from some of the mosaic kernels have revealed the presence of a small centric fragment. It has not been seen as an open ring but this may be a result of its size. This observation, along with genetic results obtained with *Sh*, *Bz* and *Wx* strongly suggest that a ring chromosome involving the short arm of chromosome 9 is the source of the aberrant behavior.

Studies of *bz-x3*, *bz-x4* and *bz-x5*

Progeny of the original *bz-x* mutants in this study exhibited somatic instability of bronze in the endosperm tissue. Kernels were produced with sectors of *Bz* and *bz* tissue but patterns were not produced with any regularity. In the majority of mosaic kernels, the events leading to expression of the dominant phenotype occurred late in development so that the size of *Bz* sectors was small (Fig. 1a). In some instances, kernels were produced with *Bz* sectors occupying one half or more of the aleurone tissue (Fig. 1c). In these cases, two mechanisms could account for the color patterns. Either *bz* reverted to the dominant state prior to or in the primary endosperm cell with subsequent repression at a later stage; or the reversion event occurred early in endosperm development

producing a large sector with the dominant phenotype. Kernels have also been found having a mixture of early and late events, in which large sectors of *Bz* tissue occupy part of the aleurone while small purple spots are present in the remainder (Fig. 1 b).

In addition to the variation in timing, there was considerable diversity in the frequency of dominant sectors. Among those kernels where reversion events occurred late in development of the endosperm, some possessed but a single spot of dominant tissue while others had 200 or more (Fig. 1 a).

Instability of the *bz-x* alleles is not limited to endosperm tissue. In *bz-x/bz* sporophytes possessing the complementary factors necessary for plant color, pigmented areas are bronze with fine stripes of purple tissue.

The behavior of the *bz-x* mutants suggests that a gene control system has been either induced or activated by x-ray treatment.

In plants containing *bz-x3*, the mutability is autonomous, i.e. the element (or elements) causing instability of the bronze allele is either inseparable from or very tightly linked to the bronze gene itself. In a cross of a *Sh bz-x3/sh bz* plant with a *sh bz* tester, the following progeny were produced: 180 *Sh bz-x3*; 11 *Sh bz(?)*; 2 *sh bz-x3*; and 181 *sh bz(?)*. The classes with the questionable bronze allele exhibited no instability. *Sh bz(?)* kernels probably include region 1 (*sh-bz*) crossovers containing the normal recessive allele of bronze and noncrossovers possessing a stable *bz-x3*. The majority of the *sh bz(?)* kernels are noncrossovers possessing the standard *bz* allele but a few may contain a stable *bz-x3*. The *Sh bz-x3* and *sh bz-x3* are noncrossovers and crossovers respectively. If factors responsible for bronze mutability were not linked to bronze itself, ratios indicating independent assortment would be found; this is not the case.

In populations from the *bz-x4* and *bz-x5* mutants, instability occurs sporadically and it is not possible to determine whether or not the controlling element(s) is linked to bronze. With independent assortment of the controlling element, 25% of the testcross progeny of a *bz-x/bz* individual would exhibit instability if variegation occurs in every kernel containing the necessary factors. In some testcross populations, the frequency of variegated progeny was far less than 25%, indicating that the unstable allele is not always expressed or frequently mutates to a stable form. Plants from kernels having no reversions generally give rise to *bz* progeny which remain stable. A few cases have been recorded, however, in which a kernel void of dominant sectors produced an individual whose offspring exhibited instability.

In an effort to identify the control system induced in these mutants by irradiation, each of the three *bz-x* alleles has been combined with alleles known to respond to the *Ac-Ds* and *Spm* systems reported by McClintock (1956). Crosses were made between

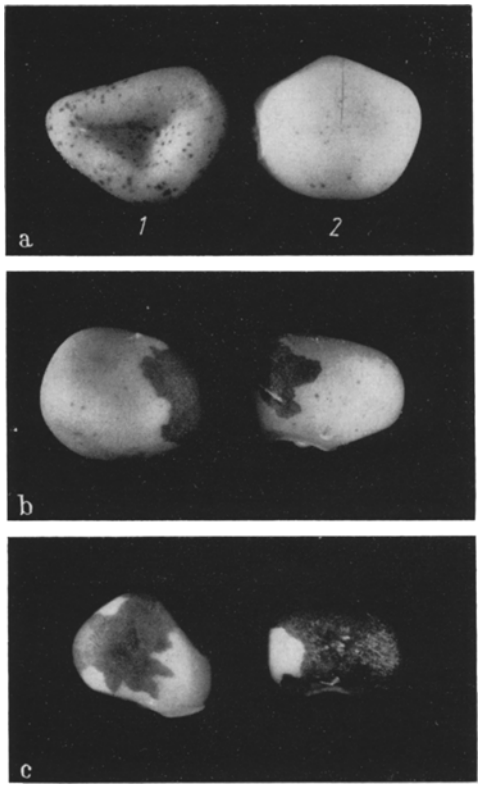

Fig. 1. The kernels in a. and b. contain one dose of *bz-x3* and are progeny of an individual exhibiting only late reversions. The kernels in a. show only late occuring events. Kernel #1 has many small sectors of *Bz* tissuse while #2 has relatively few. The kernels in b. exhibit large deep colored (red) sectors representing early events. The lighter bronze sectors contain small spots of red tissue indicating late reversions. The kernels in c. are progeny of an individual exhibiting both early and late reversions and possess one dose of *bz-x3*. They contain *Bz* sectors covering more than 2/3 of the aleurone tissue. The *bz* sectors could have arisen from a cell in which a reversion event never occurred or one in which *bz* reverted to *Bz* and in a subsequent cell was again repressed

bz-x/bz plants and bz^{m-1} testers (the bz^{m-1} allele responds to *Ac*). In the progeny, kernels were found with the phenotypic patterns of the *bz-x* alleles but not with that of bz^{m-1}. Thus, *Ac* was not present in the *bz-x* individuals.

Similarly, in crosses of *bz-x* heterozygotes with an *Spm* tester, wx^{m-8}, no instability of *wx* was observed indicating that *Spm* was not present and therefore is not responsible for the instability of the *bz-x* alleles.

Discussion

The only mutant arising from premeiotic irradiation which has not exhibited instability is *sh-bz-x3* and its behavior is strikingly similar to that of *sh-bz-x2*, a post-meiotic mutant. Although the data from *sh-bz-x3* tests are consistent with its characterization as a deficiency, it is also possible that a controlling element inhibits the activity of all genes in a segment including *Sh* and *Bz*. Distinction between the two

alternatives awaits tests on the ability of *sh-bz-x3* to revert.

Although formation of a ring chromosome is the most likely explanation for the behavior of the mutant involving *sh*, *bz* and *wx* an alternative possibility of an element which causes chromosome breakage and/or genetic instability of the *Sh*, *Bz* and *Wx* alleles cannot be ruled out. Distinction between the two awaits additional data.

In her pioneering studies of gene control systems in maize (1951) McClintock observed that numerous unstable alleles at various loci arose in stocks undergoing the breakage-fusion-bridge cycle in chromosome 9. In her work and in the present study, the mechanisms apparently responsible for induction of mutable alleles are those known to cause disruptions in the physical integrity of the chromosomes. McClintock (1951) suggested that alterations in heterochromatic segments were probably responsible for the occurrence of unstable alleles at various loci. She cited evidence that the mutator gene, *Dt*, which is located in the terminal heterochromatic knob of the short arm of chromosome 9, could be recreated by a breakage-fusion-bridge cycle in that chromosome. This observation was later confirmed by Doerschug (1967). The present results are not inconsistent with the hypothesis that mutability was induced by activation of a controlling system. It is highly unlikely that x-rays induced constructive changes resulting in the creation of elements able to control the action of structural genes. A more tenable hypothesis is that factors indirectly affecting the activity of the bronze gene were already present in the irradiated stocks but in an inactive or suppressed state. The x-rays induced changes in these elements resulting in activity or release of suppression.

In the experiments reported here, the only loci screened for instability were *sh*, *bz* and *wx*. A question which remains unanswered is whether or not instability can occur at other loci in the *bz-x* stocks. Positive results would constitute evidence for transposability of the element or elements involved.

Pre-meiotic irradiation of maize in these experiments has given rise to genetic instability. Is the potential for such a response ubiquitous among stocks of maize or limited to only certain ones? And, is there a difference between cells in the pre-meiotic and post-meiotic stages which allows genetic instability to be induced in the former but not in the latter? The present results support this possibility. The unstable alleles reported here are not the first arising in stocks treated with ionizing radiations. Peterson (1953) reported that his pale green mutable system in maize was recovered from stocks exposed to the radiation of the Bikini atomic bomb tests and Green (1967, 1969a, 1969b) has described a control system affecting the white locus in *Drosophila* which arose in an x-rayed individual. In view of the disruptive nature of ionizing radiations, it is likely that these cases represent alterations in the systems normally present which regulate the amount and timing of structural gene activity.

Literature

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Received November 7, 1972

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