Genetic analysis of a mosaic pericarp in maize, with special reference to the genic change of the color pattern induced by the possibly unequal crossing-over in a small region containing the *P*-locus and its neighbors of chromosome 1

By

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(With 1 plate, 4 text-figures and 27 tables)

Contents

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Introduction

Up to date there have been numerous published studies on the inheritance of the pericarp color character in maize, most of which have been carried out by American geneticists, especially EMERSON and his associates. By their studies it has become well-established that the fundamental gene for pericarp color, *P*, forms the basis of an extensive series of multiple alleles. The colors and color patterns of pericarp are numerous, but they are grouped into relatively a few classes. According to ANDERSON's finding (1924), each of those classes is governed respectively by each member belonging to the *P*-allelic series. Any one of such alleles, without P^{rw} for a basal recessive class of pericarp color, has been generally well-known to be unstable and has changed so as to produce a large series of *P*-alleles. For this reason the unstable nature of such alleles governing the pericarp color character should be of special interest.

The writer has dealt with the genetic nature of pericarp variegation in maize controlled by one of the *P*-allelic members, P^{mo}, which exhibits a high degree of mutability and disturbed segregation in crossing progenies. The present experiments were designed to solve the mechanism of genic changes of the gene, P^{mo}, into some other members of the *P*-allelic series. In connection with this point, STURTEVANT (1925) from his excellent data on a double *Bar* in *Drosophila melanogaster* concluded that the genic change from one member into other one of a supposedly allelic series occurs through duplication of a gene locus itself. Recently, critical evidences on this situation have been furnished by OLIVER (1940), LEWIS (1945), STEPHENS (1948), and LAUGHNAN (1949) in examples of *lozange*, *Star-asteroid* in *Drosophila*, *G-S* pseudo-alleles in *Gossypium* and of some members of an *A*-allelic series in maize, respectively. Such genic changes, other than those which must be regarded as true mutation of the gene, have all been established as proven to be associated with a crossing over between intrachromosomal duplications. Further some data, in entire accord with those previously reported, have now been obtained from the present experiments. Three years ago the writer preseted some results of such a study (T. SUTÔ, 1948)*. Additional data bearing upon new phases of this idea are also now available (SUTÔ, 1951). Those results will be reported in this paper.

* Details were read in meetings of both the Sapporo Branch of the Botanical Society of Japan and the Japanese Journal of Genetics in the autumn of 1947.

Material and method

All of the material used in the present experiments originated in pedigreed cultures from a single ear. It is of a commercial open variety of flint corn which has been known as *"Calico"* having a mosaic pericarp and a mosaic cob. The pericarp of kernels is characterized by either narrow or broad red stripes extending irregularly from the point of attachment of the silk to the base of kernel. This character was highly inconstant. It varies not only in the intensity of the color, but also changes to colorless, to self color of red, and to a number of distinct types of mosaic nature.

The gene, *P*, concerned with the pericarp and cob color has a series of multiple alleles composed of nine members, P^{rr}, P^{or}, P^{wr}, P^{ow}, P^{cw}, P^{cr}, P^{ww}, P^{vv} and P^{mo} (ANDERSON 1924). Each member of the alleles has been indicated in general by the superscripts alone, namely the first letter for pericarp and the second for cob color as follows: *RR* red pericarp and cob, *OR* orange pericarp and red cob, and so on. The pericarp character of the present material, *"Calico"* maize, was controlled by one of such *P*-allelic members designated by the symbol *MO*. For convenience all the allelic members of *P* were symbolized by only the first letter, because the writer in the present paper deals with the genetic behavior of a pericarp character alone. Accordingly, the symbols, *R*, *M* and *W*, refer to the pericarp color, red, mosaic and white, respectively.

The work herein reported was started in the spring of 1938 at the Faculty of Science, Hokkaidô University, with a single ear furnished through the kindness of Mr. H. HARA, horticulturist of our university, from the Yamato Seed Co. Ltd., Sapporo. This ear was of the mosaic nature in pericarp color, and consists therefore of kernels, of which types were found to grade from entirely white to strongly variegation with red and further to nearly self red. Six classes were used in the present paper for the description of kernels according to the intensity of pericap variegation. They are: (1) R, a self-red color pattern, (2) M_h, a heavy striped pattern of variegation, (3) M_m, a medium striped pattern of variegation, (4) M_s, a slight striped pattern of variegation, (5) W_p, a nearly colorless pattern, appearing to be due to the presence of a single fine patch of stripes on the pericarp, and (6) W, a colorless pattern. Plate XI is representative of kernels of each class.

For the first seven years from 1938 to 1944, the work was carried

on in conjunction with the Breeding Department, Snow Brand Seed Co. Ltd., Sapporo, through the kindness of Mr. K. IGARASHI, director of that company. A continuous selection was made for the purpose of isolating pure types of the pericarp color character. The parental ears used were all self-pollinated by using paper bags. The results showed that all ranges of variegation, from mosaic ears of various typs (M_h, M_m, M_s and W_p) to colorless pericarped eârs (W) and also to self-red (R), could be obtained from the original one mosaic ear (M_m). The classes of ears, an illustration of which is showed in Figure 1, are alike in part to the classes of kernels described above and are as follows:

1). R : All of kernels from an ear are always of a self-red color.

2). M_h: Most of kernels from an ear have a heavy variegated pericarp (M_h) and a few of them have either a self-colored (R) or a medium variegated pericarp (M_m) and very rarely a slight-variegated pericarp (M_s).

3). M_m: The ear is composed mostly of medium variegated kernels, but some of them produce often heavy or slight variegation, or rarely become self-red or colorless.

4). M_s: The ear differs from the M_m-ear in degree of pericarp variegation only. Ears have fewer heavy variegated kernels and show an increase of colorless kernels as compared with the above class.

5). W_p: The ear consists mostly of kernels which do not show any prominent stripe on the pericarp; nevertheless only one or two of them are recognized to have a single fine patch belonging to the W_p-class of kernel.

6). W : All of kernels from an ear are always of the colorless nature of pericarp. This class is apparently similar to that of the W_p-class above, but the latter differs from the former in having only one or two W_p-kernels.

It may be realized that no sharp distinction exists amongst the classes of kernel as well as of ear. Especially, all of the classes within the pericarp variegation (M), such as M_h, M_m, M_s and W_p, are gradational into each other since the variegation is of highly inconstant nature. There are also some difficulties in distinguishing between the two classes of ear, W_p and W, because a distinction consists in only one or two kernels with the W_p-variegation of pericarp color, being too fine to be dectectable. In addition to these types of mosaic ears, there were occasionally some ears representing a sectorial chimera

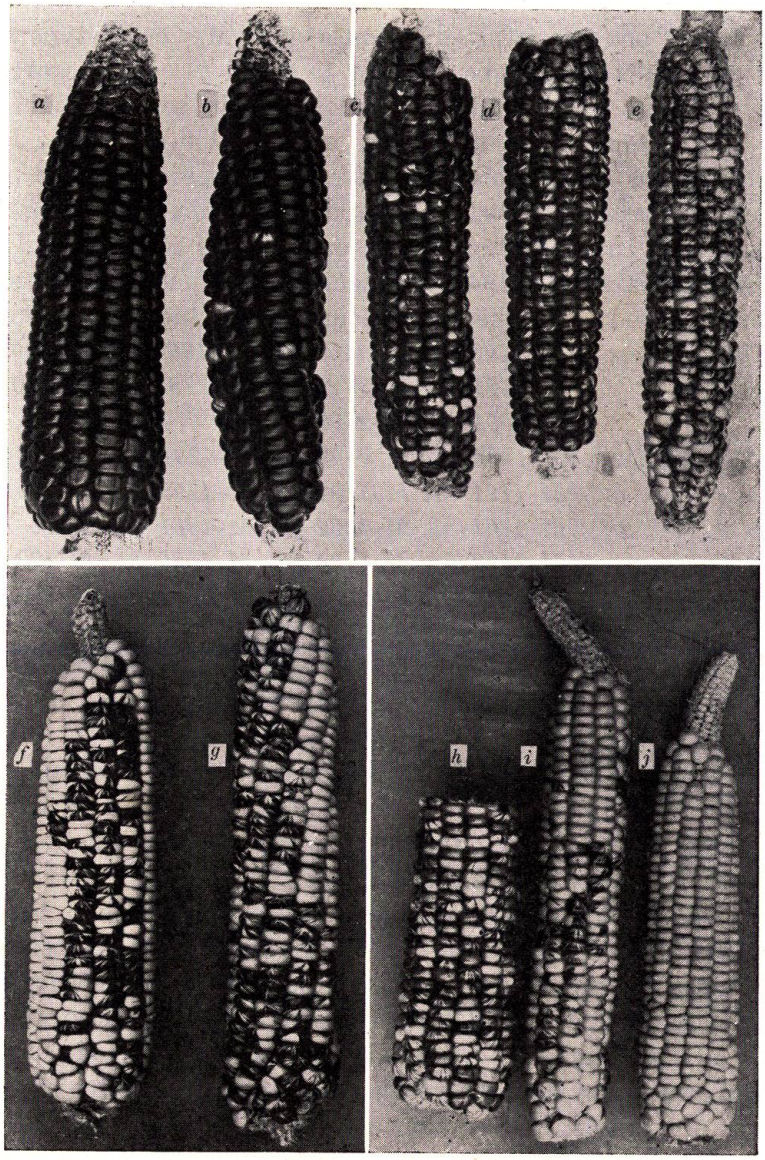

Fig. 1. Various types of mosaic pericarp in open-pollinated ears of *"Calico"* maize dealt with in text. From left to right, they represent respectively ; *a*, self-red (R), *b-d*, heavy mosaic (M_h), *e-h*, medium mosaic (M_m) *i*, slight mosaic (M_s) and *j*, very slight mosaic (W_p). Of these, two ears (*f* and *g*) are of a sectorial type of M_m, which were recognized to be non-heritable, as well as a *"dark-crown"* type of variegation.

of mosaic types. Such the ear was always found to be divided sharply into two parts, one consisting of kernels with M_m- or M_s-type and the other of kernels with W-type (Figure 1, *f* and *g*). The occurrence of such chimera was found to be more common in some certain strains than in the other strains. However, no heritable difference between such two sections of kernels in an ear was observed in the progeny. In the present paper, the writer treated all of such chimera under the above described categories of mosaic ear, such as M_m and M_s.

After six years (1938-1943) of selection, the following 43 pedigreed lines were isolated from the original ear by means of self-pollination:

1). 10 R-lines; *M-1192-R, M-1201-R, M-1361-R, M-1492-R, M-1525-R, M-1721-R, M-1734-R, M-2002-R, M-2004-R, M-2007-R.*

2). 22 M-lines; *M-1-M, M-2-M, M-120-M, M-123-M, M-1121-M, M-1191-M, M-1193-M, M-1201-M, M-1261-M, M-1262-M, M-1361-M, M-1491-M, M-1493-M, M-1494-M, M-1525-M, M-1721-M, M-1723-M, M-2001-M, M-2003-M, M-2004-M, M-2005-M, M-2007-M.*

3). 11 W_p-lines; *M-120-W_p, M-1361-W_p, M-1363-W_p, M-1492-W_p, M-1493-W_p, M-1521-W_p, M-1522-W_p, M-1523-W_p, M-1733-W_p, M-1734-W_p, M-2005-W_p.*

Since 1944 the work has been conducted at the Hokkaidô Forage Plant Institute, Sapporo, where an attempt was made to decide the mode of inheritance in each type of the pericarp variegation and the genic relations of each of them to each of the others. For this purpose the behavior of segregation in progenies has been studied from many inter-crosses between each other of such strains and also between any one of them and other members belonging to the different pericarp character, especially *WW*, *WR* and *RR*. In fact, during the two years, 1944 and 1945, a large number of crosses and of selfings were made, but owing to the bad conditions, the cold weather and the damages by wire worms, the output of kernels was very low and, in addition, the plants growing from those kernels were very poor. Indeed, plants of less than only 10 percent successful growing were observed per plot where about 90 plants were in general planted. This made the material less extensive than was desired, so that most of the pedigreed lines isolated were lost. This publication has therefore been delayed until the further work could bring forth more data. A new series of similar pollinations

was furthermore projected in 1947 by the use of 10 pedigreed lines derived from only 5 lines surviving as follows:

1). 2 R-lines; *M-1363-118-R* and *M-1525-6-14-R*.

2). 5 M-lines; *M-120-17-M*, *M-120-18-M*, *M-1193-6-1-M*, *M-1363-118-M* and *M-2005-5-3-M*.

3). 3 W_p-lines; *M-120-18-*W_p, *M-1193-6-*W_p and *M-2005-5-3-*W_p.

The results on each of these projects obtained during the three years from 1948 to 1950 will be reported here separately for convenience, of which data are listed in appendix Tables, 14 to 17.

Statistical procedure: The segregation data obtained in the present experiments all were treated in accordance with the methods of statistical analysis developed by FISHER (MATHER 1938, and FISHER 1948).

That is to say, the χ^2 for testing deviations between the observed and expected numbers was calculated by the general formula: $\chi^2 = \frac{(a_1 - l \cdot a_2)}{l \cdot n}$, where expected ratio is l:1, the observed numbers are $a_1 : a_2$ and n is its total number. Then the χ^2 for judging the homogeneity among sets of segregation data was computed by the Brand & Snedecor formula: $\chi^2 = \frac{(n_t)^2}{a_{1t} \cdot a_{2t}} \cdot \left[S\left(\frac{a_1^2}{n}\right) - \frac{(a_{1t})^2}{n_t} \right] = \frac{(n_t)^2}{a_{.t} \cdot a_{2t}} \cdot \left[S\left(\frac{a_2^2}{n}\right) - \frac{(a_{2t})^2}{n_t} \right]$, where a_{1t} and a_{2t} are the total sum of a_1 and a_2 respectively, $n_t = a_{1t} + a_{2t}$, and S stands for summation over all classes.

In order to calculate the hypothetical value, the combined method of maximum likelihood was applicable to the estimation, since the present data include always different kinds of crossing in all cases. That is, the individual logarithms of the likelihood expansion were given for every kind of segregation separately. Then, a longarithm of the combined likelihood was obtained by summing of the individual logarithm likelihood expansions. The estimation of a value, such as p (recombination value), was settled by maximizing this summed likelihood expansion with respect to p. This equation of estimation ($dL/dp = 0$, where L represents the logarithm of such expansion) was solved by using arthmetic approximation as an expected method of algebraic approach. Then, the estimation of variance was directly derived from the figures of this arithmatic interpolation, although the precision of value calculated is less than that obtained from the formulae; $I_p = 1/V_p = n \cdot i_p = -nS\left(m \cdot \frac{d^2 \log m}{dp^2}\right) = nS\left[\frac{1}{m} \cdot \left(\frac{dm}{dp}\right)^2\right]$, where i_p denotes an amount of

TABLE 1

Summary of the segregation data on the pericarp colors obtained from all intercrosses between *P* and $E\widehat{\cdot}zl$. For detailed data see appendix Tables 14 to 27

I. Backcrossing (*2*, *3* and *6*) and F_1 (*1*, *4*, *5* and *7*) data

	Genotype	Year observed	No. of pedigrees	Progenies R	M	W	Total
1,	$M\cdot E\widehat{\cdot}zl/M\cdot+\times W\cdot+$	1945	4	0	101	0	*101*
		1948	6	0	364	3	*367*
		1949	8	0	306	0	*306*
		1950	12	0	430	4	*434*
	Total	*4*	*30*	*0*	*1201*	*7*	*1208*
2,	$W\cdot+\times M\cdot E\widehat{\cdot}zl/W\cdot+$	1945	1	0	21	23	*44*
		1949	7	0	69	69	*138*
		1950	7	0	231	238	*469*
	$M\cdot E\widehat{\cdot}zl/W\cdot+\times W\cdot+$	1949	19	4	614	604	*1222*
		1950	26	7	1070	1139	*2216*
	Total	*3*	*63*	*11*	*2005*	*2078*	*4089*
3,	$W\cdot E\widehat{\cdot}zl/M\cdot+\times W\cdot+$	1944	1	0	35	4	*39*
4,	$M\cdot E\widehat{\cdot}zl/R\cdot+\times W\cdot+$	1945	4	72	64	0	*136*
		1949	1	7	7	0	*14*
		1950	1	12	12	0	*24*
	Total	*3*	*6*	*91*	*83*	*0*	*174*
5,	$R\cdot+/R\cdot+\times W\cdot+$	1945	8	237	0	0	*237*
		1948	1	18	0	0	*18*
	Total	*2*	*9*	*255*	*0*	*0*	*255*
6,	$W\cdot+\times R\cdot+/W\cdot+$	1948	2	60	0	59	*119*
	$R\cdot+/W\cdot+\times W\cdot+$	1948	1	10	0	11	*21*
		1949	2	68	1	78	*147*
		1950	3	70	1	71	*142*
	Total	*3*	*8*	*208*	*2*	*219*	*429*
7,	$M\cdot E\widehat{\cdot}zl/W\cdot+\times R\cdot+/W\cdot+$	1944	7	27	64	50	*141*
		1949	6	84	138	92	*314*
		1950	3	32	151	49	*232*
	$R\cdot+/W\cdot+\times M\cdot E\widehat{\cdot}zl/W\cdot+$	1949	5	19	64	32	*115*
		1950	1	38	14	16	*68*
	Total	*3*	*22*	*200*	*431*	*239*	*870*

TABLE 2

Summary of the segregation data on the pericarp colors obtained from all intercrosses between *P* and *E͡zl*. For detailed data see appendix Tables 14 to 27

II. Selfing data (*8–14*)

Genotype	Year observed	No. of pedigrees	Progenies R	Progenies M	Progenies W	Progenies Total
8, $M \cdot E\widehat{\cdot}zl / M \cdot +$	1944	3	0	26	0	*26*
	1945	14	0	332	0	*332*
	1948	4	1	84	2	*87*
	1949	8	0	112	0	*112*
	1950	10	0	339	5	*334*
Total	*5*	*39*	*1*	*893*	*7*	*901*
9, $M \cdot E\widehat{\cdot}zl / W \cdot +$	1945	13	0	192	92	*284*
	1948	4	0	40	17	*57*
	1949	3	0	34	13	*47*
	1950	33	6	1251	636	*1893*
Total	*4*	*53*	*6*	*1517*	*758*	*2281*
10, $W \cdot E \cdot zl / M \cdot +$	1944	6	0	48	119	*167*
	1948	5	0	54	123	*177*
	1950	2	0	39	79	*118*
Total	*3*	*13*	*0*	*141*	*321*	*462*
11, $W \cdot E\widehat{\cdot}zl / W \cdot +$	1944	9	2	1	219	*222*
	1948	4	0	2	182	*184*
	1949	4	0	3	213	*216*
	1950	5	0	1	264	*265*
Total	*4*	*22*	*2*	*7*	*878*	*887*
12, $M \cdot E\widehat{\cdot}zl / R \cdot +$	1944	6	32	59	0	*91*
	1945	5	24	51	0	*75*
	1950	8	95	233	0	*328*
Total	*3*	*19*	*151*	*343*	*0*	*494*
13, $R \cdot + / R \cdot +$	1945	1	18	0	0	*18*
14, $R \cdot + / W \cdot +$	1944	11	109	3	42	*154*
	1949	2	53	0	19	*72*
	1950	6	160	0	53	*213*
Total	*3*	*19*	*322*	*3*	*114*	*439*
Grand total	*5*	*305*				*12546*

information concerning p_2, V_p is the variance of $p(=(s_p)^2=1/n\cdot i)$, and m is the expected proportion in any class. Similarly, the heterogeneity χ^2 was easily calculated for each set of data from the formula; $\chi^2 = S[D_p^2/I_p]$ where D_p is the deviation from the estimated value (p) of linkage.

Genetic behavior of *P* and *E·zl* alleles

1). A zygotic lethal (zl) closely linked with M

Since 1938, a continuous inbreeding from a single ear of *"Calico"* maize with mosaic pericarp (M) has been made by means of hand pollination. Nevertheless, isolating any type of homozygous mosaics proved difficult. Most of the inbreeding progenies from the phenotypic M-ears were usually heterozygous so far as the mosaic character is concerned. In the selfing progenies of them, it was observed obviously that the M and W plants were segregating into a 2 : 1 ratio instead of the 3 : 1 expected. For example, appendix Table 15 exhibits a case in point, in which the selfing population is formed of 192 M and 92 W plants.

When an M plant of a strain (*M-1193-6-2*) described above was crossed with an inbred *W* strain, the F_1 population was observed to be composed of 21 M and 23 W plants, considered a 1 : 1 relation of the segregation. It is therefore quite natural that the M strains isolated should be heterozygous for the mosaic pericarp : *M/W*. In the F_2 populations from the M plants chosen out of such F_1 segregants, there was again a 2 : 1 relation of the segregation, of which the actual numbers were 271 M aud 145 W plants. Detailed data are given in appendix Table 20. The same crossing experiments were further repeated on the other two strains; *M-1201-81* and *M-1363-118*, and increased the data to 1325 M and 666 W. All the same data are summerized in second row (*9*) of Table 2-II, where a 2 : 1 relation was confirmed by a total of 2281 plants consisting of 1523 M and 758 W. The deviation-χ^2 from the 2 : 1 relation is 0.0107 in a total of all the data, and the heterogeneity-χ^2 amongst crossing sets is 20,7360 (DF = 16). The former value of χ^2 corresponds with P = 0.95-0.90 and the latter P = 0.20–0.10. This fact means statistically that the difference from an expectation of the 2 : 1 ratio should be not significant both in a total population and in any one of all the crossing strains. The 2 : 1 segregation may be brought out by the complete elimination of the ex-

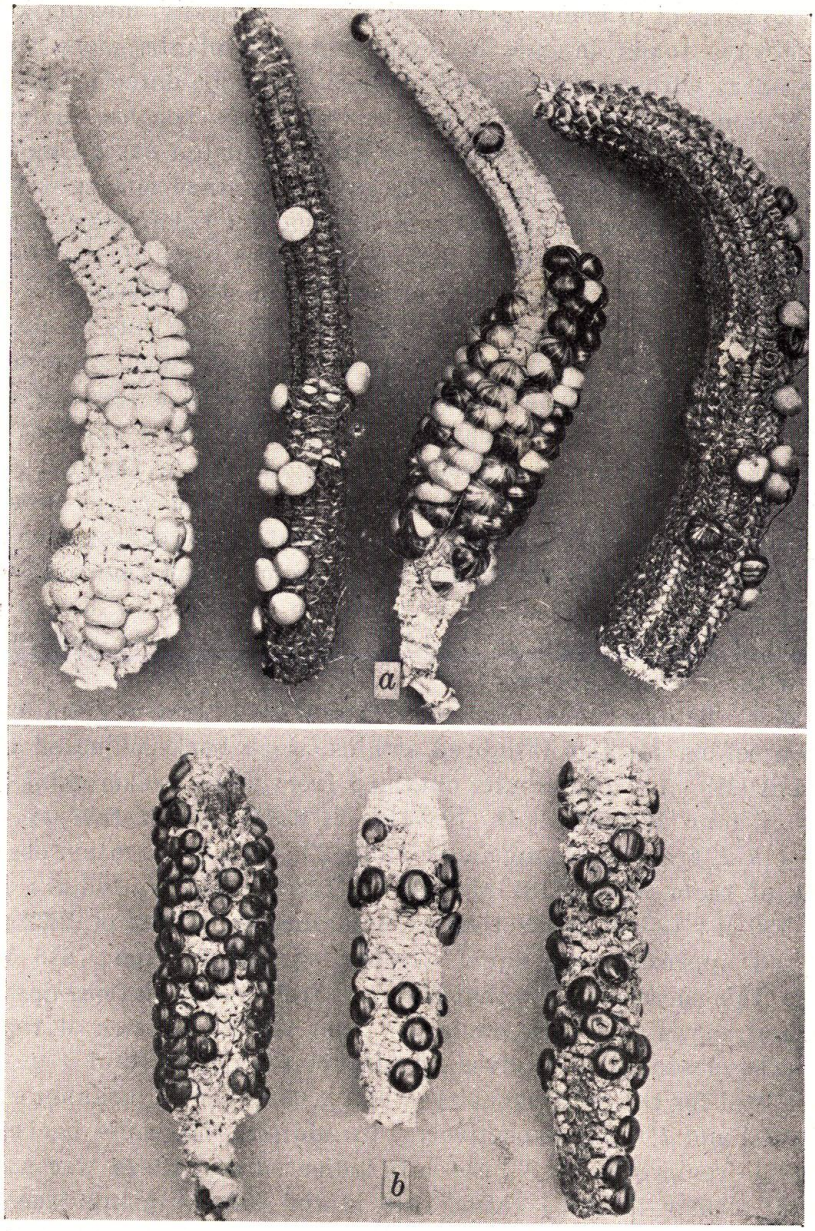

Fig. 2. Types of pericarp color in self-pollinated ears with (*a*) and without (*b*) $E \cdot \widehat{zl}$; (*a*) representing two types, left two W_p and right two M_m, and (*b*) representing self-red type, R.

pected 25 percent of homozygous M-plants. Practically, missing kernels were always found in ears of such selfing M-plants, more than in crossing ears with another inbred strains. But, the detection of the 25 percent of missing kernels was usually impossible, because there were generally many sterile kernels on the hand-pollinated ear owing to the faulty fertilization (see Fig. 2). The result obtained agrees with that reported by EMERSON (1939). It will be concluded that there must be a *zygotic lethal* gene, *zl*, closely linked with one (*M*) of the *P*-allelic members. EMERSON states: "the effect of *zl* is to prevent the homozygosis of genes with which it is closely linked, and thus to change a 3 : 1 to 2 : 1 F_2 ratio when linked with a dominant gene, or to prevent the occurrence of the one class when linked with a recssive gene". This opinion on the subject may be supported by the facts that all of the M strains, having arisen from the "*Calico*" maize through selfing, has a strong tendency to heterozygosity for *M* and *E* (as presented later), and thus the 2 : 1 and the 1 : 1 relation of segregation were always observed in their selfing and backcrossing populations respectively. The present "*Calico*" maize is therefore expected to have a genotype of "*M·zl/W·+*", and so the *zl* homozygote is to be regarded as lethal without an exception.

The F_1 heterozygous M-plants (*M·zl/W·+*), grown from crosses between selfing M- and *W*-inbred strains, were back-pollinated by the *W* parent (*W·+*). The results obtained from those backcrossing populations of four M-strains, *M-1193-6-1*, *M-2005-5-3*, *M-1363-18-1* and *M-120-118-2*, are given in appendix Table 23. The segregation observed in each of them was of 181 M : 190 W, 158 M : 155 W, 143 M : 124 W and of 136 M : 135 W, respectively. Summing up, a total of 1222 plants comprised approximately equal number on the average; 616 M and 604 W. This corresponds to that obtained from F_1 populations described above, and shows very little deviation from the expectation of the 1 : 1 ratio. The closeness of fit gives a value for the deviation of χ^2 of 0.1614 (DF = 1) and for the heterogeneity-χ^2 of 1.2305 (DF = 3), meaning a value for $P = 0.7$ and $P = 0.8$ respectively. In addition, the same backcrosses were made reciprocally. All the data indicated that there was a segregation of clearly the 1 : 1 ratio; 2016 M and 2078 W plants (see Table 1-*2*). Any ear of those backcrossed plants has usually a full set of kernels, this indicating that the *zygotic lethal* (*zl*) has no concern with the gametic lethality.

2). An Enhancer of P-alleles, "E"

A heterozygous M-plant of an M-strain (*M-1193-6-1*) with an "*M·zl/W·+*" genotype was crossed with a self-red homozygous plant of the *M-1363-118-R* strain with an "*R·+/R·+*" genotype, which had originated as a mutant from a pedigree culture of an M-strain (*M-1363*). The F_1 segregation of this cross was of the 1 : 1 relation of R to M; the actual number was 7 R : 7 M in a total of 14. In the F_2 populations from the F_1 R-parents there were 213 R and 72 W plants while the F_2 populations from the F_1 M-parents were composed of 233 M and 95 R plants, not far from the 3 : 1 relation in the former and the 2 : 1 in the latter. Such F_2 segregation was further examined by crosses between the R or M plant of the F_1 population and the W inbred plant. The resultant plants were found to be segregating into equal numbers; 51 R and 71 W from R parents and 12 M : 12 R from M parents. The same crossing was also made between other strains; "*M-120-18-1-M* × *M-1525-6-14-R*", "*M-1636-118-M* × *R*-inbred" and so on. The data of those crosses are arranged in appendix Table 26. The results indicate clearly that the *M* gene belonging to a *P*-allelic series is dominant to *R*. This fact is in conflict with the long-established and well-supported opinion that a regular order of dominancy within *P* allelic members has, since ANDERSON's finding (1924), been adopted as the "*R*>*M*>*W*" sequence (the symbol, >, means "is dominant over").

An "*M*>*R*" relation instead of the "*R*>*M*" expected was also confirmed by a double crossing data, which are given in appendix Table 25. At first, *W* inbred strains (*W·+*) were crossed with both the heterozygous M (*M·zl/W·+*) and the R strains (*R·+/W·+* or *R·+/R·+*) independently. Next, the F_1 M-plants (*M·zl/W·+*) obtained from the first cross were further pollinated reciprocally by the F_1 R-plants (*R·+/W·+*) from the second cross. In those double-crossed populations, no cases were found to be without segregating three types of pericarp colors (R, M and W) into a 1 : 2 : 1 relation. A total of 729 plants included 173 R, 367 M and 189 W, this indicating a ratio of approximetely 1 : 2 : 1. A summary of the data with respect to the R to M relation has been arranged in Tables, 1 (in rows *4-7*) and 2 (in rows *12* to *14*). The results indicated clearly that the *M* gene is completely dominant to the *R* gene. Once, HAYES (1917) demonstrated a phenomenon of the same reversible relation of dominancy as that described here. But, his paper has already been discussed by EYSTER (1924), who questioned

HAYES's conclusion according to the well-known general opinion proposed by himself and EMERSON's school. Judging from all the data, an opposite relation, "$M>R$", in this case, can now be explained by the existence of a newly recognizable gene in the present "*Calico*" strains of maize. This gene, which is considered as an *Enhancer* of *P* alleles, was designated here as "*E*".

Anticipating the data to be present later, it will be said that the *Enhancer*, "*E*", is a spontaneous dominant mutant and gives a specific modifying effect to the so-called *P* multiple alleles controlling the pericarp color. That is, whenever any one of the *P* alleles has its locus in close sequence on the same chromosome as that possessing "*E*", this *P* allele acts always as a top dominant, having essentially nothing to do with the dominant nature in itself. It is natural beyond reasonable doubt that the *M* gene originating from "*Calico*" maize should be closely linked with not only a zygotic lethal factor (*zl*) but also with an *Enhancer* of *P* (*E*) without any consideration of the crossing over. One may assume from such an idea about the disturbed segregation in the selfing progenies, that the heterozygous M-strains used should have a pair of chromosome 1, in which one possesses a genic constitution of "$M\cdot E\cdot\widehat{zl}$" and the other of "$W\cdot E^{+}\cdot\widehat{zl^{+}}$". Similary, the R strain should contain a genotype with the chromosmal constitution of either "$R\cdot\widehat{E^{+}\cdot zl^{+}}/R\cdot\widehat{E^{+}\cdot zl^{+}}$" or "$R\cdot\widehat{E^{+}\cdot zl^{+}}/W\cdot\widehat{E^{+}\cdot zl^{+}}$" and if so it be, there will always be induced a normal segregation as to the self-red character of pericarp, because $\widehat{E\cdot zl}$ are not located on an *R*-bearing chromosome nor on an *W*-one at all. Actually the segregation in the F_2 and backcrossed populations occurred in a 3 : 1 and 1 : 1 relation of R to W. When heterozygous M-strain ($M\cdot\widehat{E\cdot zl}/W\cdot\widehat{E^{+}\cdot zl^{+}}$) was crossed reciprocally with the homozygous R-strain, there was also occur a segregation of the 1 : 1 relation of M to R, of whch M plant must have a genotype of "$M\cdot\widehat{E\cdot zl}/R\cdot\widehat{E^{+}\cdot zl^{+}}$" while R of "$R\cdot\widehat{E^{+}\cdot zl^{+}}/W\cdot\widehat{E^{+}\cdot zl^{+}}$". Also, the F_1 population from reciprocal crosses between the heterozygous M-strain ($M\cdot E\cdot zl/W\cdot\widehat{E^{+}\cdot zl^{+}}$) and the heterozygous R-strain ($R\cdot\widehat{E^{+}\cdot zl^{+}}/W\cdot\widehat{E^{+}\cdot zl^{+}}$) must be composed of R, M and W plants in the proportion of 1 : 2 : 1, because such three phenotypes are to be determined by the following gentoypes; (1) R="$R\cdot\widehat{E^{+}\cdot zl^{+}}/W\cdot\widehat{E^{+}\cdot zl^{+}}$", (2) M="$M\cdot\widehat{E\cdot zl}/R\cdot\widehat{E^{+}\cdot zl^{+}}$" and "$M\cdot\widehat{E\cdot zl}/W\cdot\widehat{E^{+}\cdot zl^{+}}$", and (3) W="$W\cdot\widehat{E^{+}\cdot zl^{+}}/W\cdot\widehat{E^{+}\cdot zl^{+}}$".

3). *Further evidence on the genic relation of* $E\widehat{\cdot}zl$ *to P-alleles*

As mentioned later, the "*E*" gene is closely located on the right side of *M* in chromosome 1, where its locus shows 1.5 percent units of map distance to the *M* locus, corresponding with the *zl* locus. The genes, *E* and *zl* have their loci in too close sequence to be detected between them. Actually there was no any crossover plant in a total 12546 plants observed in all of the present experiments. It is therefore impossible to ascertain in the present state of experiment whether the genes, *E* and *zl*, are to be very closely linked together or fallen into a single locus (as a gene). For convenience in this paper, its designation is "$E\widehat{\cdot}zl$" and its normal type simply "+".

An occurrence of the phenotypic change of R into M, when the plant possessed an *M*/*R* genotype with respect to *P*-alleles, could be explicable clearly from a speculation mentioned already on the ground that the "$M\cdot E\widehat{\cdot}zl$" loci are represented to lie in chromosome 1 while the *R* locus is represented to be adjacent to the $E^{+}\widehat{\cdot}zl^{+}$ locus in other homologous one. If so, when any one of the *P*-allelic members, other than *M*, is adjacent to the $E\widehat{\cdot}zl$ locus, what kind of reversible change of phenotype occurs? In addition, has the *lethal Enhancer* of *P* ($E\widehat{\cdot}zl$) nothing to do with the phenotypic effect on the pericarp color? A possibly certain answer was given by only one of other crossing experiments.

About 200 heterozygous M ($M\cdot E\widehat{\cdot}zl/W\cdot+$) and 100 homozygous M ($M\cdot E\widehat{\cdot}zl/M.+$) strains were cultured in an ear-to-row field, in which six open pollinated colorless ears occurred in each row of the following strains; *M-120*, *M-136*, *M-152*, *M-149*, *M-173* and *M-200*. In 1943, the fifteen plants grown from such open-pollinated ears were self-pollinated. The data obtained from fifteen selfing populations are given in appendix Table 18. Of them, nine populations were composed of 219 W or W_p, 2 R and 1 M plants, This seems to indicate probably that the colorless parent is of a phenotype of the genic constitution, "$W\cdot E\widehat{\cdot}zl/W\cdot+$", of which the "$W\cdot E\widehat{\cdot}zl$" region of chromosome 1 might have arisen from the "$M\cdot E\widehat{\cdot}zl/W\cdot+$" plant through a crossing over. If so, the selfing colorless progenies will be segregating "$W\cdot E\widehat{\cdot}zl/W\cdot+$" and "$W\cdot+/W\cdot+$" plants into the 2:1 ratio. Actually, there were always found to be existing of such two phenotypes, W and W_p, although it is difficult to

draw a clear line between them. Of course, W_p is a type of very slight pattern of pericarp variegation which appers to be due to the presence of a faint stripe in only one or rarely a few kernels per ear. It is evident from the present and next data that the W_p phenotype is revealed by a genotype of "$W \cdot \widehat{E \cdot zl}/W \cdot +$". Consequently, the $\widehat{E \cdot zl}$ behaves as not only a homozygotic lethal and a dominant intensifier of *P*, but also by itself as a dominant mosaic mutant. But the mosaic appearance caused by $\widehat{E \cdot zl}$ is so very slight that it is usually inseparable from the colorlsss pericarp (W) without exact examination under the microscope.

Another six populations grown from fifteen selfing ears consisted of 48 M and 119 W_p in a total of 167 plants, indicating a 1 : 2 relation of the segregation. This may be considered to certainly be due to this W_p phenotype with a genic constitution of "$W \cdot \widehat{E \cdot zl}/M \cdot +$", one chromosome being of a crossover ($W \cdot \widehat{Ezl}$) while the other being of a non-crossover ($M \cdot +$) from the parent strains. When one of 119 W_p plants was pollinated by the colorless inbred strain (*W*), there were 35 M and 4 W_p plants in its F_1 population. This ratio of segregation is significantly far apart from the 1 : 1 relation ($\chi^2 = 26.4610$, see Table 6), but the data are too scanty to be considered, contrary to expectation. Two types ("$W \cdot \widehat{E \cdot zl}/M+$" and "$W \cdot \widehat{E \cdot zl}/W+$") of W_p segregants from each of such selfing populations were used again to make certain the 1 : 2 and 0 : 1 relation of M to W_p or W. For that purpose twenty W_p segregants of three W_p strains were self-pollinated; *M-120-18-*W_p, *M-1193-6-*W_p and *M-2005-5-*W_p. The results obtained are arranged in appendix Table 27. They are in accordance quite well with the previously described results on this parent populations. That is, there were two distinct classes in respect to the segregation. Of the two, a class is of the 1 : 2 relation of M to W_p, containing 7 W_p-parents which might has originated from W_p segregants with a "$W \cdot \widehat{E \cdot zl}/M \cdot +$" genotype: viz. 93 M and 202 W_p. The other class is of the 0 : 1 relation, containing 13 W_p-parents orignated from the "$W \cdot \widehat{E \cdot zl}/W \cdot +$" segregants; viz. 5 M and 659 W_p or W.

In summary, a lethal enhancer of *P*, "$\widehat{E \cdot zl}$", was first found in the heterozygous M-strains of "*Calico*" maize to lie in a chromosome with *M*, and next in the W_p strains, which might have arisen from the M strain through a crossing over between the "*M-*$\widehat{E \cdot zl}$" and "$W-+$" region, to be close to the right of the *W* locus. A survey of segregations

which have been observed in all of crossing populations from such an $\widehat{E \cdot zl}$ involving strain is presented in Table 3. As can be seen in this table, it is evident that all the segregating ratios observed are quite in agreement with the theoretical ratios based on the genic nature of $\widehat{E \cdot zl}$. Such results with respect to the genic behavior of mosaic pericarp indicated that the mode of segregation is more complicated than that of another mosaics which have hitherto been reported. Nevertheless, there is no actual proof that the results obtained could not be explained by assuming the existence of $\widehat{E \cdot zl}$.

4). *Linear sequence* of *P and* $\widehat{E \cdot zl}$ *loci*

The *zygotic lethal* (*zl*) was first announced by EMERSON (1939) in his study on the disturbed segregation of a mosaic-pericarp character. He found that there is a group of four genes having their loci in close sequence; *Pericarp-color* (*P*), *male-sterile 17* (ms_{17}), *tassel-seed 2* (ts_2) and *zygotic lethal* (*zl*). Those gene-loci were given by him to be lie on the middle region of the short arm of chromosome 1 in the following order: ms_{17}—1.7—ts_2—1.3—*P*—1.5—*zl*. The region from the ms_{17} to *zl* locus has therefore a map distance of approximately 4.5 units. EMERSON found a double crossover plant in only one case of his many crossing experiments and advanced a possible speculation as to the minimum length of double crossing-over which may be able to occur in a region of about 4.5 crossing-over units, shorter than the five unit length well-known in *Drosophila*.

In order to determine precisely whether or not the unit distance of this region is correct at 4.5, the crossover values between each other of such gene loci were estimated by developing the joint method of FISHER's maximum likelihood (MATHER, 1938). For that puprose, all the data which have hitherto been reported were got together from the two papers (EMERSON et al, 1935 and EMERSON, 1939). A survey of data is given in Table 4 wherein ANDERSON's data obtained from reciprocal translocations are excluded on account of being usually a remarkable decrease of the crossing over in the neighborhood of translocated point on the chromosome. As is seen in Table 4, the calculated values of the six distances between each other of the possible combinations of four gene loci in a close sequence are given; such as linkage value (p), deviation from zero of maximum likelihood expansion (D_p), amount of information per a total of populations (I_p), heterogeneity-χ^2,

TABLE 3
Summary of data from Tables, 1 and 2 on the color segregation in progenies from

			Parent character	Self	
				R	M
M	1,	Observed	M	1	893
		Exp. ratio	—	*0*	*1*
		Exp. genotype	$M\cdot\widehat{E\cdot zl_1}/M\cdot+$	—	$M\cdot+/M\cdot+$ or $M\cdot\widehat{E\cdot zl}/M\cdot+$
	2,	Observed	M	6	1517
		Exp. ratio	—	*0*	*2*
		Exp. genotype	$M\cdot\widehat{E\cdot zl}/W\cdot+$	—	$M\cdot\widehat{E\cdot zl}/W\cdot+$
	3,	Observed	M	151	343
		Exp. ratio	—	*1*	*2*
		Exp. genotype	$M\cdot\widehat{E\cdot zl}/R\cdot+$	$R\cdot+/R\cdot+$	$M\cdot\widehat{E\cdot zl}/R\cdot+$
R	1,	Observed	R	0	18
		Exp. ratio	—	*0*	*1*
		Exp. genotype	$R\cdot+/R\cdot+$	—	$R\cdot+/R\cdot+$
	2,	Observed	R	322	3
		Exp. ratio	—	*3*	*0*
		Exp. genotype	$R\cdot+/W\cdot+$	$R\cdot+/R\cdot+$ or $R\cdot+/W\cdot+$	—
	3,	Observed	R	—	—
		Exp. ratio	—	*3*	*1*
		Exp. genotype	$R\cdot+/M\cdot+$	$R\cdot+/R\cdot+$ or $R\cdot+/M\cdot+$	$M\cdot+/M\cdot+$
W_p	1,	Observed	W_p	2	7
		Exp. ratio	—	*0*	*0*
		Exp. genotype	$W\cdot\widehat{E\cdot zl}/W\cdot+$	—	—
	2,	Observed	W_p	0	141
		Exp. ratio	—	*0*	*1*
		Exp. genotype	$W\cdot\widehat{E\cdot zl}/M\cdot+$	—	$M\cdot+/M\cdot+$
	3,	Observed	W_p	—	—
		Exp. ratio	—	*1*	*0*
		Exp. genotype	$W\cdot\widehat{E\cdot zl}/R\cdot+$	$R\cdot+/R\cdot+$	—

observed and expected ratio of the pericarp
various combinations between P and $E\cdot zl$

		× $W\cdot+/W\cdot+$	
W	R	M	W
7	0	1201	7
0	*0*	*1*	*0*
—	—	$M\cdot+/W\cdot+$ or $M\cdot E\cdot zl/W\cdot+$	—
758	11	2005	2078
1	*0*	*1*	*1*
$W\cdot+/W\cdot+$	—	$M\cdot E\cdot zl/W\cdot+$	$W\cdot+/W\cdot+$
0	91	83	0
0	*1*	*1*	*0*
—	$R\cdot+/W\cdot+$	$M\cdot E\cdot zl/W\cdot+$	—
0	255	0	0
0	*1*	*0*	*0*
—	$R\cdot+/W\cdot+$	—	—
114	208	2	219
1	*1*	*0*	*1*
$W\cdot+/W\cdot+$	$R\cdot+/W\cdot+$	—	$W\cdot+/W\cdot+$
—	—	—	—
0	*1*	*1*	*0*
—	$R\cdot+/W\cdot+$	$M\cdot+/W\cdot+$	—
878	—	—	—
1	*0*	*0*	*1*
$W\cdot E\cdot zl_1/W\cdot+$ or $W\cdot+/W\cdot+$	—	—	$W\cdot E\cdot zl/W\cdot+$ or $W\cdot+/W\cdot+$
321	0	35	4
2	*0*	*1*	*1*
$W\cdot E\cdot zl/M\cdot+$	—	$M\cdot+/W\cdot+$	$W\cdot E\cdot zl/W\cdot+$
—	—	—	—
2	*1*	*0*	*1*
$R\cdot+/W\cdot E\cdot zl$	$R\cdot+/W\cdot+$	—	$W\cdot E\cdot zl/W\cdot+$

TABLE 4

Summary of the linkage data on *P* and its neighboring estimation of the recombination value (p) from

Genes X Y	Linkage phase	Observed data						Total
		XXY	*XxY*	*XXy*	*Xxy*	*xxY*	*xxy*	
P–zl	R·S	69	4474	—	—	2359	—	*6902*
	R·S	1362		—	—	677	—	*2039*
	C·S	705		—	—	7	—	*712*
	Total							*9653*
P–ts₂	C·B	1558		19		21	1510	*3108*
	R·B	0		94		92	2	*188*
	C·S	56	88	1	0	6	49	*200*
	R·S	0	15	6	0	4	0	*25*
	Total							*3521*
P–ms₁₇	C·B	1307		36		45	1318	*2706*
	C·SB	108	153	9	55	11	104	*440*
	C·S	105	163	0	12	16	85	*382*
	C·S	184		4		1	58	*247*
	Total							*3775*
ms₁₇–zl	R·BS	537		—	—	919	—	*1450*
	C·S	1155		—	—	39	—	*1194*
	R·S	821		—	—	439	—	*1260*
	Total							*3904*
ms₁₇–ts₂	R·S	428		196		161		*785*
ts₂–zl	R·S	1073		—	—	521	—	*1594*
	R·BS	81		—	—	147	—	*229*
	Total							*1823*

1). Cited from EMERSON, Beadle and Fraser in Cornell Univ., Agr. Exp. Sta. Mem., **180** (1935) : 34, and EMERSON in Genetics, 24 (1939) : 370–382.
2). D_p = deviation from zero of maximum likelihood expression of p.
3). $I_p = 1/V_p$ = amount of information concerning p.
4). V_p = variance of $p = (S_p)^2 = 1/n \cdot i_p$.
5). S_p = standard error of p.

alleles reported by EMERSON et al[1], and the combined various segregations and linkage phases

Estimation of recombination value					
p	D_p [2]	I_p [3]	$D^2/I\,(\chi^2)$	V_p [4]	S_p [5]
	+ 11,21904	296686,5	0,0004		
	+ 0,42000	26,6	0,0066		
	− 12,35934	29936,8	0,0051		
0,01526	− 0,72030	326649,9	0,+ 0,0121	0,00000306	±0,00175
			(DF=2, P>0,99)		
	− 95,80398	103670,3	0,0885		
	− 89,06388	5269,5	1,5056		
	+ 184,30425	32425,6	1,0475		
0,01986	− 0,56361	141365,4	0,+ 2,6416	0,00000707	±0,00266
			(DF=2, P=0.3−0,2)		
	− 578,70003	59870,0	5,5937*		
	+ 310,34150	14312,0	6,7277**		
	+ 380,34911	20037,0	7,2256**		
	− 111,45718	3603,0	3,4567		
0,03769	+ 0,53340	97822,0	0,+ 23,0037**	0,0000102	±0,00319
			(DF=3, P<0,01)		
	+ 38,18134	729,9	2,0054		
	− 35,24627	1329,5	0,9346		
	− 3,19276	67,7	0,1522		
0,05270	− 0,25769	2124,1	0,+ 3,0922	0,00047081	±0,02169
			(DF=2, P=0,3−0,2)		
0,00500	+ 0,18000	35,9	0,0009	0,02787068	±0,16694
	+ 1,66925	15,6	0,1790		
	− 1,56250	108,5	0,0023		
0,08000	+ 0,10675	224,1	0,+ 0,1813	0,00446286	±0,06682
			(DF=1, P=0,7−0,5)		

* significant (at the level of 0.05).

** highly significant (at the level of 0.01).

variance (V_p) and standard error (S_p) of D_p. The best fitting value of p was calculated on each of six distances; they are, P-zl=1.526±0.175%, P-ts_2 = 1.986±0.266%, P-ms_{17} = 3.769±0.319 %, ms_{17}-zl = 5.270±2.169%, ms_{17}-ts_2 = 0.5±16.694% and ts_2-zl = 8.0±6.682%. The heterogeneity-χ^2 for each of those values was smaller than the 5 percent level of probability obtained by chance. Thus, all the data from various linkage phases used may be considered to be homogeneous in all cases, showing the expected good segregations from the estimated values of linkage. There is, however, an exception to those values—that of the P-ms_{17} distance which will be described later as to its nature.

TABLE 5

Linear sequence of four genes, *zl*, *P*, ts_2 and ms_{17}
(A summary of data from Table 4)

Gene loci	Genic distance (%)	Fiducial limits * at 5% level
zl–*P*	*1.526*	*1.183—1.869*
P–ts_2	*1.986*	*1.465—2.507*
ts_2–ms_{17}	*1.783*	*1.488—2.078*
zl–ms_{17}	*5.295*	*5.035—5.555*

* Both values are of the upper and lower limits in both tails jontly.

Of standard errors of p when the p value is fixed to be good fitting, the first three's in Table 4 are so small that those values are highly worthy of confidence, while the remaining three's are too large to trust. Without consideration on the linkage values of the latter, a map distance from the ms_{17} to *zl* locus may be therefore estimated as given in Table 5, and so the locus of each gene may be arranged in the following order;

$$sr \longleftarrow ms_{17} \overset{1.8}{\text{——}} ts_2 \overset{2.0}{\text{——}} P \overset{1.5}{\text{——}} zl \longrightarrow br$$

The genic distance of about 5.3 units differs from EMERSON's estimation of 4.5 units. With the object of ascertaining whether thus its difference is significant, a statistical comparision was made between two. Firstly, the present estimation may be considered to be more reliable than EMERSON's, because the value was reached by maximizing the sum of the individual logarithm likelihood expansions with respect to p. Secondly, EMERSON's value of 4.5 units is beyond the five percent fiducial limit of the present case; from 5.035 to 5.555 units. Thirdly,

the standard error for the best value of 5.295 units was computed as approximately 0.133 units ($S_p = \sqrt{V_p}$), and the difference (D_p) between the two, 5.3 and 4.5, is 0.8. The calculated value of t^0 (D/S) is therefore 3.0769, greater than that of the one percent fiducial limit ($t=$ 1.95996), and hence the probability of deviation is about 0.001. Accordingly, the genic distance given by EMERSON, 4.5 units, should be revised to be about 5.3 units owing to such three reasons. If so, the limit distance of double crossing-over in maize will evidently correspond with that in *Drosophila*. It is quite natural that double crossover should be expected to occur rarely within this region.

The *P–zl* region has a distance of 1.5 percent units according to the estimation made by EMERSON, which is in strict accord with that by the writer. An attempt was made to ascertain whether this value of the *P–zl* region corresponds with that of the *P-Ezl* region in the present case or not.

TABLE 6

Heterogeneity-test on three F_2-segregations from different types of crossing, when an average crossing-over value between *P* and *E·zl* is expected to be $p = 1.5\%$

Genotype		Observed data				χ^2-analysis		
		R	M	W	Total	D_p	I_p	$D^2/I\,(\chi^2)$
M·E·zl/W·+		0	192	92	*284*			
		6	1325	666	*1997*			
Subtotal		*6*	*1517*	*758*	*2281*	*−0,79353*	*99,380*	*0,0063*
M·E·zl/R·+		56	110	0	*166*			
		95	233	0	*328*			
Subtotal		*151*	*343*	*0*	*494*	*−0,61660*	*698,232*	*0,0005*
W·E·zl/M·+		0	48	119	*167*			
		0	93	202	*295*			
Subtotal		*0*	*141*	*321*	*462*	*−0,44990*	*35,440*	*0,0057*
Total	Deviation-χ^2					*−1,86003*	*833,052*	*0,0041*
	Heterogeneity-χ^2							*0,0081*
	Sum of χ^2's							*0,0125*

Results are given in Table 6 showing the segregotion-data from three different genotypes. The deviation-χ^2 for the expectation of 1.5 percent units in this region is of a value 0.0041 corresponding to the

probability of about 0.3. The heterogeneity-χ^2 testing the agreement between three different genotypes used is of 0.0081 ($DF=2$), having a probability of about 0.4. This indicates that the data are all in agreement with the expectation. Thus, the P–$\widehat{E\cdot zl}$ distance may be considered as agreeing sufficiently well with the P–zl distance computed from EMERSON's data. The conclusion may be justified that $\widehat{E\cdot zl}$ has its locus in the same point as EMERSON's zl on chromosome 1, so that the present $\widehat{E\cdot zl}$ and zl may be considered to belong to a so-called multiple allelic series.

Disturbed segregation of pericarp color

The mosaic pericarp character is well-known to be consisting of various types of kernels in an ear, ranging from colorless to self-red. According to the amount of red stripe per kernel, mosaic patterns can be classified in both the kernel and ear into six types: W, W_p, M_s, M_m, M_h and R (p. 69–70). The mosaic character has also been well-established to be of inconstant nature, and so occasionally it is known to be changing from one type to another. Nevertheless, it is established that the segregation observed is usually in accords with that expected from a hypothesis as to the existence of P and $\widehat{E\cdot zl}$ alleles. In rare cases, there was found what appeared as disturbed segregation. If the disturbance of segregation is significant statistically, it would be due to either the incomplete manifestation of dominance or to the instability of gene itself. In order to testing this point, ears were firstly grouped into each type with respect to the mosaic patterns in the population from each of the M strains, and next, all of kernels in each ear were carefully classed in each type. And then, the kernels were planted separately by type.

The analysis of segregations was made by use of the χ^2 disturbution. χ^2 was evaluted for every type of mosaics according to the method of Brandt and Fisher, best adapted to the statistical analysis of data. A sum of those χ^2's is also itself a χ^2 for DF obtained by adding number of intial χ^2's. Results are summarized in Tables, 7 to 11, wherein each χ^2 are arranged for every one of the different genotypes. As can be seen in Table 7, it may be evident that the deviation-χ^2's all agree in showing good single factor segregation as expected, although there was an exception in which only one backcrossed population from an "$W\cdot\widehat{E\cdot zl}/M\cdot+$" plant did not show significaty the theoretical 1 : 1

relation of segregation, since $\chi^2 = 24{,}6410$ (DF=1) corresponding to a probability of less than 0.001. In this case, the heterogeneity-χ^2 was impossible to calculate for each component, because the data were of only one segregation obtained from a single ear, and in addition the number observed was very small, viz. actually 35 plants in total. This case was therefore omitted from the consideration.

The heterogeneity-χ^2 was further analysed into various components in every different genotype, such as kernel and ear type of mosaic intensity, crossing phase, observed generation, strain used and population from every ear. If the calculated heterogeneity-χ^2 is of a value, as large as or larger than that obtained from the χ^2 distribution owing to random sampling fluctuation, it is to be expected that the present material should be not strictly homogeneous with reference to the phenotypic variability in the mosaic segregation. To make the significant heterogeneity still more obvious, a comparison of χ^2's from such components was then made in coming to a conclusion as to the source of disturbed segregation as follows.

1). *Between kernel types*

Since the M strains used in the present study have originated all from a single ear of the *"Calico"* maize through inbreeding, they are expected to have the same gene concerning the mosaic pericarp, and so, to have a certain uniformity on the mosaic intensity excepting mutants. This expectation was however not fulfilled. In every progeny from different types of kernels in an ear, ears grown from each type were observed as producing all types of the mosaic nature, varying all the way from self red (R) to colorless (W).

Plants from the various types of mosaic ears in different M-strains were back-pollinated by the colorless inbred plants (*"M·Ẽ·zl/W·+"* × *"W·+/W·+"*). Plantings were made separately from kernels of various mosaic types. Segregations in the next generation were grouped according to the mosaic type of parent kernel. A complete analysis of the segregation into various components is recorded in Table 7, proving that the probability is always more than five percent fiducial limit in every component. The segregation of present mosaic character may be concluded to occur always in exactly equal number in every type of parent kernels or ears as well as in all other components. These data indicate therefore that both the parent kernel and ear

TABLE 7

Statistical analysis of the pericarp color segregation by means of the χ^2-test. I, Backcrossing data ("$M\cdot E\cdot zl/W\cdot +$"×"$W\cdot +/W\cdot +$") consisting of a total of 1222 plants (Summary of data from appendix Table 23)

	Heterogeneity between				Deviation	Total
	Kernel types	Individuals	Ear types	Pedigrees		
M^2/n	*318,8042*	*315,0712*	*313,0161*	*312,9007*	*312,5401*	
D	*3,7330*	*2,0551*	*0,1154*	*0,3066*	—	
χ^2	*14,9824*	*8,2482*	*0,4632*	*1,2805*	*0,1604*	*25,0847*
DF	26	11	4	3	1	45
P	*0,98–0,95*	*0,7–0,5*	*0,98–0,95*	*0,8–0,7*	*0,7–0,5*	

types have no relation to the disturbed segregation of mosaic character, and hence prove that all of mosaic types have behaved strictly according to the expectation of a single unit inheritance.

Further, two M_s and three M_m ears were obtained from backcrosses between two heterozygous M-strains (*M-1193-6-1-*M_m and *M-120-118-2-*M_m) and colorless inbred strains: "$M\cdot E\cdot zl/W\cdot +$"×"$W\cdot +/W\cdot +$". All the kernels in those five ears were also grouped into each type of mosaic and then were planted separately. Results are given in Table 8, wherein those populations as the whole consisted of M and W plants

TABLE 8

Variation of the mosaic intensity in backcrossed ears, "$M\cdot E\cdot zl/W\cdot +$"× "$W\cdot +/W\cdot +$", grown from various parent ear and kernel types

Types of mosaic		Mosaic grades R(1)[1]	M_h(2)	M_m(3)	M_s(4)	W_p(5)	Total of M	W	m[2]	S_m[3]	Range of 5% fiducial limits
Kernel	M_h	0	2	13	6	0	*21*	16	*3,1904*	*±0,3447*	*2,4739–3,9074*
	M_m	1	20	160	7	0	*188*	200	*2,9202*	*±0,2277*	*2,4698–3,3711*
	M_s	2	3	13	7	0	*25*	34	*3,0000*	*±0,7200*	*1,5168–4,4832*
	W_p	0	0	9	3	0	*12*	17	*3,2500*	*±0,1875*	*2,8412–3,6588*
	W	0	2	35	22	2	*61*	73	*3,3934*	*±0,3699*	*2,6536–4,1332*
Total		*3*	*27*	*230*	*45*	*2*	*307*	340	*3,0050*	*±0,2969*	*2,4231–3,5869*
Ear	M_m	3	23	189	15	0	*230*	245	*2,9391*	*±0,2006*	*2,5459–3,3323*
	M_s	0	4	41	30	2	*77*	95	*3,3896*	*±0,3936*	*2,6024–4,1768*

1). The number within brackets represents the arbitrary index of mosaic intensity.
2). Mean grade of mosaic intensity.
3). Standard error of mean.

in approximately equal number (307 M and 340 W in a total of 647 plants). The χ^2 value for the deviation from the 1 : 1 ratio is 1.6832, producing $0.3>P>0.2$, so that the difference is not significant. In order to form a statistical basis for the mosaic intensity, an arbitrary number was given for each of all six types, garding from R=1 to W=6 (Pl. XI). All the ears grown from each of the typed kernels were similarly classified into the arbitrary mosaic-intensity classes. The frequency of ears obtained are given in the same table for every type of parent kernels and ears respectively. As the whole, the frequency curve was recognized to approach that of the binomial distribution, the modal class being of the M_m type and the mean being of 3.05 in index number. This average mean value compared favorably with each of individual means of mosaic types. Column 8 of Table 8 represents such mean value, of which calculation was made for all excluding colorless ears on the genetic ground that the colorless ears should have been derived from the colorless inbred other than mosaic strain, through the back-crossing. Column 9 gives the upper and lower five percent fiducial limits of the respective mean value. Little difference in frequencies of ears from the parent types was observed in appearance. But, the average mean value was found always to be lie between the upper and lower fiducial limits of the individual mean. Thus, the observed difference in the mean could be never said to be true statistically in all cases. From those data, a conclusion may be set up with a reasonable degree of certainty that the variability of mosaic intensity in the present case is not due to the inheritance but is due to the simple fluctuation of unit character controlled by a single gene.

Such a conclusion may be further established by a continuous selection for various types of mosaics, which has been made during about ten years. It was recognized as the result that the phenotypic variability of mosaic intensity in parent kernel or ear did not produce any shift in the manner of inheritance of mosaic intensity in the progeney. That is to say, neither a selection for plus type nor that for minus one was able to change the frequency distribution of the mosaic classes. Both the crossing and selection experiment can be said to lead to the same conclusion.

Since the studies by EMERSON (1914 and 1917) and HAYES (1917), it has been well-supported and well-established respecting the mosaic nature of maize pericarp that the mosaic gene is one of the *P*-multiple alleles, and in addition, itself forms further a multiple series in which

the dominant relation is proportional to the amount of mosaic intensisty on the kernel or ear, viz. $R>M_h>M_m>M_s>W_p>W$, in the present abbreviation. This finding has been confirmed by EYSTER (1924 and 1925) through his study of the genic analysis of color intensisty in an unstable orange pericarp, of which the result is in entire accord with that of the general mosaic pericarp. However, the present finding may be considered to not necessarily correspond with that obtained by those students. So far as experiments were made, it may be said that the present material is more stable than that used by them in spite of the existence of the fact that in the pattern variation of mosaic pericarp the studies have yielded similar results. However, it seems reasonable to recognize that a difference tends to appear between the two parent ear types, M_s and M_m, although it was insignificant statistically. If such a difference does appear, it may be possible to demonstrate that the plant has a tendency to produce the heavy type of mosaic classes proportional to the mosaic intensity of the parent ear. Concerned with this viewpoint, detailed data is now in progress of preparation, and results will be published later.

2). *Between individuals* (*ears*) *and between strains* (*pedigrees*)

A summary of selfing and backcrossing data from four different genotypic populations is recorded in appendix Tables, 14 to 27. Table 9 represents the χ^2 analyses of those data into various components, in which an attempt was made to asscertain whether or not the heterogeneity in the fraction of segregation observed occurs in every one of the components, giving data in each column of the table separately. It was recognized from the χ^2's in Table 9 that the significant heterogeneity sometimes occurs amongst only three components; individual, ear type and pedigree, and not in any other three at all; such as year, phase and deviation. This is of much interest because the following two conclusions may be drawn: (1) There is nothing in the genetic evidence that the environmental components, such as the observed years and the crossing phases, appear to affect the theoretical segregation of the present mosaic character. (2) The significant disturbance of segregation occurs in the genetic components other than environmental ones, such as individuals, ear types and pedigrees. In consequence, the total χ^2 value must be revised with the χ^2 value of such heterogeneity taken under consideration. For example, the value of a backcrossed

TABLE 9
Statistical analysis of the pericarp color segregation by means of the χ^2-test. II, Selfing and backcrossing data obtained from the four different genotypes (A summary of data from appendix Tables 14 to 27)

Parent		Total plants	Phase		Heterogeneity between					Deviation	Total
Pheno-type	Geno-type				Individuals	Ear types	Pedigrees	Years	Phases		
M	$\frac{M\cdot E\cdot zl}{W\cdot +}$	2281	Self	χ^2	*21,3570*	*17,3950**	*20,7360*	*0,0545*	*1,1047*	*0,0107*	*61,1487*
				DF	26	7	16	1	2	1	53
		4159	Back	χ^2	*151,5779***	*19,7443**	*48,8109***	*1,2727*	*0,0036*	*0,8298*	*222,2392*
				DF	40	9	9	2	1	1	62
W_p	$\frac{W\cdot E\cdot zl}{M\cdot +}$	462	Self	χ^2	*28,6167***	—	—	*0,6050*	—	*1,6461*	*30,8678*
				DF	10			2		1	13
		39	Back	χ^2	—	—	—	—	—	*24,6410***	*24,6410*
				DF						1	1
M	$\frac{M\cdot E\cdot zl}{R\cdot +}$	494	Self	χ^2	*1,2815*	—	—	*0,1026*		*0,3678*	*1,7519*
				DF	3			2		1	6
		174	Back	χ^2	*7,3189*	—	—	*1,3757*		*3,4028*	*12,0974*
				DF	16			2		1	19
R	$\frac{R\cdot +}{W\cdot +}$	439	Self	χ^2	*7,5840*	—	—	*0,2736*		*0,2194*	*8,0770*
				DF	16			2		1	19
		429	Back	χ^2	*2,3024*	—	—	*0,4622*		*0,1888*	*3,4534*
				DF	6			1		1	8

χ^2 in row 2 of Table 9 can not be revised because it is based upon a calculation of a single population from only one ear, so that this case must be treated as an exception in spite of the highly remarkable value. It is probable therefore that the significant heterogeneity in the present mosaics should not be due to the external condition but due to the genetic factors existing in the plant itself.

The heterogeneity-χ^2 between individuals is highly significant in only two cases out of a total of seven. Of the two, one is not revised with another components, such as ear type and pedigree, and hence in such a significant heterogeneity it is impossible to ascertain which of those components is in connexion with it. Since the other significant χ^2 is of a value revised with other components of heterogeneity, it is of a highly fiducial value. In such case, the pedigree component is found to be of the same meaning as the individual one. Both results were obtained from a genotype of "$M\cdot E\cdot zl/W\cdot +$". This fact may indicate that both the pedigrees and its individuals used in the back-

crossing experiment involve some of the genetic factors modifying the theoretical segregation of mosaic character. If so, it may be considered, as can be seen in appendix Tables 23 and 24, that, of four M-strains, two (*M-1193* and *M-2005*) are of the normal 1 : 1 segregation, while the other two are of the disturbed segregation; *M-120* showing a M<W relation and *M-1363* showing a reversible relation of M>W, and also that such relation of segregation is not always corresponding to all of the parent individuals within the strain.

3). Between ear types

Column 6 in Table 9 presents the data regarding the heterogeneity-χ^2 between ear types. The difference between them was always found to be significant, which is highly credible on account the revision with all of the other χ^2's of components, although this was computed in only

TABLE 10.

Summary of selfing and backcrossing data, which were gathered from populations of the three heterozygous M-strains ($M \cdot E \cdot \widehat{zl}/W \cdot +$) in appendix Tables 20 and 24, on the estimation of the disturbed rates (f) from their normal segregations

Ear type	Phase	Observed segregation M	W	Total	χ^2 for expectation	dL/df $f=0{,}060$	$f=0{,}064$	I_f	χ^2 for f Individual	Ear-type	Deviation
M_h	Self	243	105	*348*		*− 4,69554*	*− 6,22111*	*381,39*	*0,1015*	*0,0033*	
	Back	445	392	*847*		*+12,22401*	*+ 8,32816*	*973,96*	*0,0712*		
	Subtotal	—	—	*1195*	*1,7711*	*+ 7,52847*	*+ 2,10705*	*1355,36*	—	—	*0+*
M_m	Self	406	186	*592*		*−19,85421*	*−22,51328*	*664,77*	*0,7622*	*0,0032*	
	Back	230	183	*413*		*+22,30028*	*+20,65259*	*411,92*	*1,0352*		
	Subtotal	—	—	*1005*	*2,9468*	*+ 2,44607*	*− 1,86069*	*1076,69*	—	—	
Total		—	—	*2200*		*+ 9,97454*	*+ 0,24636*	*2432,05*	—	—	
Heterogeneity-χ^2									*1,9636−*	*0,0065*	*0+*
						f=−0,090	*f=−0,091*				
M_s	Self	608	345	*953*		*+41,69305*	*+43,04830*	*1355,25*	*1,3675*	—	*0+*
	Back	462	601	*1063*		*−43,68384*	*−42,61993*	*1063,91*	*1,7064*	—	
	Subtotal	—	—	*2016*	*5,2168**	*− 1,99079*	*+ 0,42837*	*2429,16*			
Heterogenetiy-χ^2					*9,8414**				*2,0740−*		
Deviation-χ^2					*0,0933*						*0+*

one case of genotypes in the present experiment. To solve the cause of such the difference, a survey of segregation data obtained in 1950 was prepared from the two appendix Tables 20 and 24 (see Table 10). So far as the ear type is concerned, it is clear that there is a definite tendency in the degree of disturbance of segregation: the greater the mosaic intensity of the ear, the more likely is its progeny to produce an excess of M segregants, and correspondingly, the less likely it is to produce W segregants. A strong support for this finding was derived from the estimation of disturbed rates (denoted here as "f") made by the two statistical procedures. Firstly, a detection of the heterogeneity, between selfing and backcrossing sets of data, to be recognized as the same parameter of disturbance, was made*. In this case, a sum of individual χ^2's calculating from data of each of three sets (M_h, M_m and M_s) was considered to be itself a heterogeneity-χ^2. In consequence, the observed deviation was not significant, this meaning that both types of cross in the three sets entirely agree in showing a disturbed segregation. The calculated heterogeneity-χ^2 is significant, this indicating that the three ear types are different from each other in having different amounts of disturbed segregation.

In the next step, the combined estimation of a disturbed rate (f) can be made for each of three sets as classified by the ear type, according to the joint method of maximum likelihood. Before it, the plausible interpretations must be given by assuming a genetic mechanism in such disturbed segregation, which will be considered under two different categories. One of them is that the disturbance may result in the incomplete manifestation of a mosaic character. Another possible explanation may be that there is a genetic modifier to produce two types of gametes in unequal numbers. Genetical evidence for the latter possibility does not justify that; (1) Such a genetic factor, if it exists, must be responsible differently for every type of mosaic ears, because the distinct line can be drawn between each other of ear types in connection to the disturbed amount of segregation as will be mentioned later. (2) Concerning the occurrence of such disturbance as related to a modifier, there is always a significant discrepancy be-

* In the present case, a joint deviation from the normal segregation was expected as $D=-\frac{a_1}{1/2}+\frac{a_2}{1/2}+\frac{a_3}{2/3}-\frac{a_4}{1/3}$, and then an amount of information (I) was given by a sum of both backcrossing ($I=n_1\cdot i=4n_1$) and selfing ($I=n_2\cdot i=9/2\times n_2$). Thus, a heterogeneity χ^2 was obtained by calculating the formula: $\chi^2=D^2/I=\frac{[2(a_2-a_1)+3(2a_4-a_3)]^2}{4n_1\times 9/2\times n_2}$.

tween the selfing and backcrossing populations if this hypothesis is applied in calculation of gametic frequency. Accordingly, it may never be difficult to believe that such disturbance is attributed to the former possibility.

Now, if the incomplete manifestation is represented as f, then backcrossed M-population will be composed of $\frac{"1+f"}{1/2}$ plants expected to be genotypic M-types and $\frac{"1-f"}{1/2}$ to be W-types, while selfing M-population is composed of $\frac{"1+2f"}{2/3}$ =M and $\frac{"2(1-f)"}{1/3}$ =W. From such two sets of data, a combined f value was derived for each of the ear types. In consequence, two different f values were found to be significant; one being of 6.4±2.0 percent in both M_h and M_m sets and the other of −9.1±2.0 percent in an M_s set. The difference between such two values, 15.5 percent, is highly significant since its t^0 value (D/S) is 5.4044 corresponding to $P<0.001$. The heterogeneity-χ^2 for each f value was evaluated to be 1.9636 and 2.0740 respectively showing repsective $0.5<p<0.3$ and $0.2>p>0.1$. It is therefore clear that all the data are in agreement with a hypothesis as to the incomplete manifestation of a mosaic character.

In the light of these data, a conclusion may be made that the disturbance of segregation is not controlled by the presence of a genetic modifier but is in relation to the degree of the mosaic intensity of parent ears. In the same genetic population, plants grown from the heavy mosaic ears, such as M_h and M_m, were characterized by having an excess of M segregants, and plants from the slight mosaic ear, such as M_s, by having reversely an excess of W segregants. Thus, some (about 9%) of the phenotypically W plants will be expected to be genotypic M, and similarly some (about 6%) of M plants to be genotypic W in respective cases from heavy and light parent ears. In other words, it may be stated with certainty that the amount and type of incomplete manifestation in the present mosaic character should change phenotypically proportional to the mosaic intensity of the parent ears. It is further a very interesting feature that such an excess of one side segregants is in a parallel relationship to the occurrence of fluctuately heavy mosaic tendency as mentioned already (p. 94), in respect to both sides being roughly proportional to the degree of mosaic intensity of parent ears.

Lastly, a crossing combination between two differnt genotypic strains with respect to the pericarp color, both arisen from the same *M* gene throughout a continuous inbreeding, was always regarded as encountered with the highly significant heterogeneity for all of the components in the segregation. Such a case was met with in F_2 populations of the reciprocal crosses; "*M·E·zl*/*W·+*" × "*R·+*/*W+*", record of which are given in Table 11. This F_1 segregation was most unstable in all segregations from possible combinations of the *P*-allelic members, and hence all of the components always gave rise to the disturbance of segregation

TABLE 11

Statistical analysis of the pericarp color segregation by means of the χ^2 test. III, F_1 data ("*M·E·zl*/*W·+*"×"*R·+*/*W·+*") from the reciprocal crosses consisting of a total of 729 plants (A summary of data from appendix Table 25)

Phenotype	Genotype	Plants observed		Heterogeneity between Individuals	Ratios	Phases	Years	Deviation	Total
R	*R·+*/*W·+*	173	χ^2	*32,2844***	*48,9556***	*7,4251***	*0,8929*	*0,6260*	*90,2840*
			DF	11	1	1	1	1	15
M	*M·E·zl*/*R·+* or *M·E·zl*/*W·+*	367	χ^2	*25,4360***	*44,8617***	*5,8251**	*4,5258**	*0,0345*	*80,6831*
			DF	11	1	1	1	1	15
W	*W·+*/*W·+*	189	χ^2	*39,2352***	*3,5003**	*0,0131*	*4,9938**	*0,3333*	*48,0757*
			DF	11	1	1	1	1	15
Total		729		*97,0556*	*97,3176*	*13,2633*	*10,4125*	*0,9928*	*219,0428*

in their genetic components as well as in their environmental ones. However, it may be premature to offer a conclusion from this data, that regarding of such a unit factor as the present mosaics, the three class segregation is to shift its expected frequency in a more irregular manner than all of the two class segregations. A plausible support for this finding was also found in an another case of the same segregation, (appendix Ttable 16–selfing *1*). Those two cases were found to have a strong resemblance to each other in the manner and mode of disturbance of the segregation, but the former was different from the latter in the crossing mode, comming of selfing populations from different genotypes: "*M·+*/*W·+*". The selfing progeny from such genotypes on the whole consisted of the same R, M and W plants in about 1 : 2 : 1 relation as that of F_1 from the hybrid: "*M·E·zl*/*W·+*" × "*R·+*/*W·+*", their heterogeneity analysis into each of the components

being quite in accord with each other in both cases (p 102–103).

Concerned with a certain relationship of the disturbance of segregation to the change of amount of the stripe in single parent ear, possibile interpetation will be given in discussion.

Genic change of pericarp color

1). Occurrence of crossing over

The present mosaic of pericarp variegation is a typical one of the so-called mutable characters as well as the pericarp mosaics hitherto well-known. Since 1938, a continuous inbreeding from an original mosaic ear with the "$M\cdot E\cdot zl/W\cdot +$" genotype has been carried on for the purpose of isolating the type as to the mosaic nature. For the first six seasons (1938–1943) selection experiments have succeeded in isolating the following types which are separable from the original M-type in consequence of the progeny test about the genetic behavior of segregation:

a). A self red (R); $R\cdot +/W\cdot +$.

b). A heterozygous mosaic (M); $M\cdot +/W\cdot +$.

c). A special type of heterozygous mosaic (M); $M\cdot E\cdot zl/R\cdot +$.

d). A homozygous mosaic (M); $M\cdot E\cdot zl/M\cdot +$.

e). A very slight mosaic (W_p); $W\cdot E\cdot zl/W\cdot +$ and $W\cdot E\cdot zl/M\cdot +$.

Of those types, only the self-red (R) may be considered as the so-called "*spontaneous mutant*". All of the others will be expected to be crossovers, taking place between two loci, M and $E\cdot zl$, in course of the inbreeding program of heterozygous M-strains.

Every population from the selfed M-strains ($M\cdot E\cdot zl/W\cdot +$) must be expected theoretically to consist of non-crossnver plants, M and W, and very rarely of crossover plants of the following five genotypes: "$M\cdot +/M\cdot E\cdot zl$", "$M\cdot +/W\cdot +$", "$M\cdot +/M\cdot +$", "$W\cdot E\cdot zl/M+$" and "$W\cdot E\cdot zl/W\cdot +$". Such crossovers must phenotypically fall under both classes of non-crossover, M and W, the former of which without fail comprises a crossover chromosome, "$M+$", and the other an opposite one, "$W\cdot E\cdot zl$". Thus, all of the plants will always be found having a 2 : 1 relation of segregation. Since such crossing over is, however, infrequent between genes so closely linked, it is very difficult to hand pollinate such crossover plants even if they do occur. When the crossover plant was detected, it had already been open-pollinated. But the crossover chromosome may be relatively stable for furthermore crossing-over because

of the close linkage, the map distance of a *M–E·zl* region being of ca. 1.5 percent units as described already (p. 89). If such a crossover was met with, it might easily be identified by the progeny test. The continuous selection method of the present experiments was successful in obtaining several plants of crossovers. The genetical evidence that might have arisen from the original mosaics throughout the crossing over is as follows.

"W·E·zl"-crossover:

During more than ten generations, W_p ears have been chosen from the selfing progenies of the heterozygous M-strains (*M·E·zl/W·+*). In this case, it is of course reasonable that most of the W_p ears should comprehend the same genetic composition as the M parent, as a result of the minus selection of frequency distribution in the M population, but a few should be of the crossover : *"W·E·zl/W·+"* or *"W·E·zl/M·+"*. Actually, each of such the crossover W_p-plants could be obtained respectively by such minus selection from the six M-strains as *M-120, M–136, M–152, M-149, M–173,* and *M–200.* Kernels of those ears were detected to have a genotype of either *"W·E·zl/W·+"* or *"W·E·zl/M·+"* as expected (see appendix Table 18), of which the *W·E·zl* chromosome may be derivative from the crossing over, while both the *"M·+"* and *"W·+"* chromosomes may be of the non-crossover nature, since there were two types of M strains (*M·E·zl/M·+* and *M·E·zl/W·+*) planted in this experimental field and since the simultaneous combination of both crossover chromosomes is all but impossible to meet with owing to their closely linked sequence (see p. 89).

The important features of *E·zl*, as pointed out already, are well in keeping with the additional data on the mode of inheritance in the present W_p-strain. In summary, the main findings obtained are set forth as follows:

a). This allele changes any one of *P*-allelic members completely to a top dominance whenever located together on the same chromosome, viz. the *M·E·zl* chromosome always gives rise to the M type of mosaic pericarp and similarly the *W·E·zl* chromosome results in the W_p type.

b). Then, any one of *P*-allelic members located on the opposite chromosome of its homologoue is not related to the phenotypic effect to reveal itself. Actually, the genotypes, such as *"W·E·zl/M·+"*, *"W·E·zl/W·+"* and *"W·E·zl/R+"*, all were found as the W_p type in phenotypic expression. Any distinction can not be drawn among them.

c). The $\widehat{E \cdot zl}$ homozygote is lethal, even if it associates with every one of *P*-allelic members. Further, by itself, this factor behahes as a dominant mutant of mosaic nature (as W_p type), but its phenotype is usually inseparable from the colorless type (W) (see p. 70).

"M·+"-crossover:

On the other hand, the plus selection in connexion with the mosaic intensity has been carried on. As a result, five pure M-plants, which breed true for mosaic character, were isolated respectively from each of five original M-strains; *M-119*, *M-120*, *M-152*, *M-172* and *M-200*. The mosaic intensity of homozygous M-strains obtained was similar to that of the original M-strains, representative of all types of mosaics ranging from M_h to W_p. In addition, there was a striking resemblance between the two's of strains in the mode of inheritance, which has nothing to do with the mosaic intensity. For example, an extreme minus typed ear (such as M_s or W_p) within a given strain did not tended to give a progeny containing more ears of the minus type than that obtained from the extreme plus type (such as M_h or M_m ear) within the same strain. The genetic behavior of mosaic character was recognized to be quite similar in each progeny of both types without the consideration of heterozygosity.

As none of the M ears in this strain were found to segregate any W-ear in its progeny, it is possible to conclude that the gene, *M*, should be available as homozygous. Such homozygous M-strain will presumedly contain one of crossover chromosomes, "*M·+*", so that its genotype may be expected to be "$M \cdot \widehat{E \cdot zl}/M+$". If so it be, its selfing progeny will give rise to a new $\widehat{E \cdot zl}$-absent M-type with a probability of one in three, and similarly, to F_1 progeny from cross with colorless plant (W) in equal numbers. Actually the heterozygous M-plant without $\widehat{E \cdot zl}$ could be obtained from such F_1 population, having a genotype of "$M \cdot + / W \cdot +$". Selfing data from seven selfed M-ears obtained is reported in appendix Table 16. There was a total of 141 plants consisting of 27 R, 64 M and 50 W. This may be considered as a 1 : 2 : 1 relation of segregation because of the non-significant difference between the observation and the expectation; $\chi^2 = 1.1986$ $(0.3 > P > 0.2)$. The *M* gene, which might have been separable from $\widehat{E \cdot zl}$ through the crossing over, revealed the mosaic color in the heterozygous condition for the *W* gene ($M \cdot + / W \cdot +$) while the self-red color is revealed in the homozygous condition ($M \cdot + / M \cdot +$), in spite of a fact that the phenotype is always of the mosaic

type when one of *M* is adjacent to $\widehat{E \cdot zl}$ ($M \cdot \widehat{E \cdot zl}/M \cdot +$). This may suggest that this *M* gene is responsible for the self-red color and is so incompletely dominant over the *W* gene that the mosaic color is specially conditioned by a heterozygosity in this case. So far as the mosaic and self-color relation in the *P*-allelic series is concerned, the present data observed are too preliminary and too fragmentary to justify putting forward this suggestion because no experiments have been made to gain further support. At any rate, the fact is that a crossover chromosome ($M \cdot +$) was derived from a crossing over between two loci, *M* and $\widehat{E \cdot zl}$, giving rise to the homozygous *M*-type without the $\widehat{E \cdot zl}$ allele; "$M \cdot +/M \cdot +$".

2). Genetical features of the so-called "mutant"

Of an inbreeding program, the segregation data on homozygous M-strains ($M \cdot \widehat{E \cdot zl}/M+$) which have originated from heterozygous M-strains ($M \cdot E \cdot zl/W+$) through the crossing over, showed that most ears obtained from their selfing progenies were of the same range of M types as parent ears, usually comprising ears of all types; M_h, M_m, M_s and W_p. But, a very few ears occurred rarely in rare cases of inbreeding, of which the type was recognized as R or W_p. Both these types, R and W_p, may be therefore attributable to the "*Mutation*" of a gene, *M*, because they can not be expected as the usual Mendelian mode of segregation in the homozygous M-strains.

The occurrence of such genic changes, in various combinations of different genotypes as to *P* and $\widehat{E \cdot zl}$, into unexpected types of pericarp color is summarized in Table 12, where data are presented on a total of 12546 plants. Details of data can be seen in Tables, 1 and 2, also. The main generalizations obtained from a careful study on such genic changes were as follows.

1). The R mutant is characterized by showing always a normal Mendelian segregation for the W type with a "$W \cdot +/W \cdot +$" genotype, instead of the disturbed segregation as observed in its original M-type, and by being recessive to the original M-character. As pointed out already, the selfing segregation is of the 3 : 1 relation of R to W when heterozygous for the "$W \cdot +$" chromosome, and of the 2 : 1 of M to R when heterozygous for "$M \cdot \widehat{E \cdot zl}$". A cross between the M plant with "$M \cdot \widehat{E \cdot zl}/R \cdot +$" and the W plant with "$W \cdot +/W \cdot +$" gives rise to a progeny consisting of M and R plants in equal numbers. These results in detail

are recorded in appendix Tables, 17 and 26, and therefore seem reasonable, to lead to a conclusion that the R mutant changed from the M plant has either a genotype of "$R\cdot+/R\cdot+$" or "$R\cdot+/W\cdot+$", neither of which is ever related to $\widehat{E\cdot zl}$.

2). All ears from the selfing progeny of the R type were carefully examined for the color type. It was demonstrated that, of ears, there occur usually only an R type, and hence no M type was observed in most of ears, nevertheless there was a rare reverse change from R to M or W in a very few of ears. It is very interesting to note that such reverse change represents not merely a fluctuation of M character but always a genic shift in so far as observations have been made as described later.

3). The W_p mutant can not be distinguished in appearance from W_p types which have originated from the original M-type through crossing over as well as through fluctuation. Such W_p ears obtained were not all examined to detect whether they had genotype according to the progeny-test. In fact, plants belonging to the W_p type were found to possess all of those various genotypes in their progenies.

4). Although none of such W_p ears which has been observed during the last three years (1948–1950) was tested for the genetic behavior of its progeny, the frequency of phenotypic change from original M to W_p is always greater than to R in frequency. For example, selfing data of homozygous M-strains showed that eight ears of unexpected types, which have been found in a total of 901 plants, consisted of 1 R and 7 W_p, and of course, all of the remainders were of the original M types. Similarly, of 1208 F_1 plants (M) of a cross between two homozygous strains, W ($W\cdot+/W\cdot+$) and M ($M\cdot\widehat{E\cdot zl}/M\cdot+$), seven plants all were found as the W_p type and no single R ear was observed, and so on. This fact may suggest the great probability that the excess of W_p type may be ascribed to a contamination of various sources of genotypes in a progeny, and that the non-excess of R type may be ascribed to only a certain association with only such genic change. Thus, excepting the excess of W_p ears over R ears, both types, to which a change occurs from an original M-type, should be recogniged to occur in the same frequency.

5). The W_p mutant is characterized by always associating with $\widehat{E\cdot zl}$, like the W_p plants derived by crossing over; its genotype is composed both of "$W\cdot\widehat{E\cdot zl}$" on the one chromosome and any one of either

"*M*·+" or "*R*·+" or "*W*·+" on the opposite homologue. The phenotypic effect of such W_p plants is always determined by one half of a duplex gene constitution, "*W*·$\widehat{E\cdot zl}$", which is dominant over every one of *P*-allelic members. Thus, the segregation of selfing progeny is of the same abnormal ratio as observed in original M-plants with the *M*·$\widehat{E\cdot zl}$ chromosome. This characteristic is in remarkable contrast with that of the R mutant on the genic ground that the changed *R* gene is certainly adjacent to the wild mate of $\widehat{E\cdot zl}$ on the same chromosome.

6). Both the types, W_p and R, which should have arisen equally from the *M* gene through a genic change, give rise to a reversible change from W_p to R or M and from R to M or W in a few rare cases. Actually, the total segregation of selfing W_p ears with "*W*·$\widehat{E\cdot zl}$/*W*+" is the following: 878 W or W_p, 7 M and 2 R, this making in a total of 887 plants to 9 reversible mutants. In the selfing W_p-ears with "*W*·$\widehat{E\cdot zl}$/*M*·+", there occurs no genic change; all showing only the expected segregation which is of 141 M and 321 W_p in total of 462 plants. While, of 813 ears expected as the R type in a total of 1141 plants, 5 ears were of the M type.

3). *Possible interpretations of the genic change*

Any interpretation of the genic change must account for all of the foregoing features. Characteristics which are common to the genic change in the present mosaic character are probably similar to those in all of the another types of mosaic pericarp in maize, judging from the statements reported by American geneticists (Emerson, 1914, 1917 and 1929; Hayes, 1917; Eyster, 1924, 1925 and 1929; Anderson & Eyster, 1928 and others). According to their speculation, such genic change has been supposed as a gene mutation, and hence the mosaic pericarp in maize has been considered as a typical one of the so-called mutable characters controlled by the mutable gene. In order to explain the mechanism of mutation with respect to such mosaic character, various working hypotheses have hitherto been advocated, five of them being the following:

1). Eyster's *genomeric hypothesis*. Eyster (1924, 1925, and 1928) to account for the mutual mutability of *P*-allelic members postulated that each gene is made up of numerous subunits, "*genomers*", belonging to two types; one having the color producing ability and the other lacking such ability, and that the gene is conditioned by the relative number

of two-typed genomeres involved in itself. According to this speculation, various ranges of a mosaic pericarp in an ear result in the somatic segregation of two-typed genomeres in the division course of ontogeny, and in addition, germinal cells resulting from the random assortment of genomeres give rise to the gene mutaion. There must always be the two stable genes consisting of only the one-typed genomeres because the genomere is permanently constant in its nature. Such case is of both the colorless and self-red pericarp. While, all of genes without *W* and *R* must change from one to other in the manner of frequency distribution as a mode of the original gene components.

In fact, this hypothesis is not in agreement with the obtained data in respect to the following facts: (1) the *R* gene, as well as the other genes, gives rise to genic change in considerable frequency, (2) the actual genic change in every gene is not so frequent as to be expected from the random assortment of genomeres and, (3) the genic change of a gene is usually limited to the other two instead of all of other genes, for example, from M to R and W_p and so on. DEMEREC (1935) reported that the gene mutation can be explained by the genomere theory only when such self-contradiction is corrected by the use of additional facts: "(1) that certain genes are stable at one stage of ontogeny and unstable at another, (2) that certain genes change with different rates at various stages of ontogeny, and (3) that various modifieres may influence the mutation rate."

2). EMERSON'S *hypothesis of modifying genes.* EMERSON (1929) put forward a working hypothesis that the mutability of pericarp color-patterns is ascribable to the other gene which has an ability to change from the one *P*-allele into another mate, in its result giving rise to a multiple series of genes. Strong supports for this hypothesis were found in various organisms; for example, modifiers for *miniature-3* in *Drosophila virilis* (DEMEREC, 1929, 1935), and for a gene, *a*, in maize (RHOADES, 1936). EMERSON supposed from his data on the pericarp variegation that the mutator genes exist, perhaps more than one, and their loci are always adjacent to *P*-locus, and also that they differ from one another in having the different ability of modifying its mutability. Accordingly, the mutation rate of mosaic pericarp is recorded to be different in material from different sources owing to the existence of different combinations of various modifying genes. This was also confirmed by the present data indicating that the $E\text{-}zl$ is capable of increasing the mutability of every one of *P*-allelic members. Nevertheless, this hypothesis, as well

as the genomere hypothesis, indicates nothing in the genetic sense to account for the mechanism of gene mutation.

3). HUTCHINSON's *hypothesis of "episomes"*. In order to explain the gene mutation and the complementary interaction of any two within multiple alleles, HUTCHINSON (1932) adopted a formal speculation based on the data obtained from *Gossypium*, in which the gene itself was supposed to be subdivided into a number of adjacent *"gene centers"*, arranged in linear order along the axis of chromosome, each of which carries one or more *"episomes"*. The dominant relation between allelic members is so in proportion to the numbers of episomes that the basal recessive gene of allelic members is lacking of the episome and is permanently stable, while each of the other members is capable of changing to another one, according to the shift of numbers of episomes in each gene center.

This hypothesis involves THOMPSON's side-chain hypothesis (1931) and AGOL & DUBININ's theory of step allelomrphism (SEREBROWSKY 1938, RAFFEL & MULLER 1940, and STADLER & FOGEL 1945) on the supposition that the gene units, or *"protosomes"* to use THOMPSON's term, are of a chain-arranged nature. It is worthy of note that HUTCHINSON recognized the gene locus as a region of the chromosome instead of a point, because the gene is composed of adjacent centers, each having a different effect on the character expression. This speculation is however so similar to the hypothesis of unequal crossing-over, best known to interprete the mechanism of gene differentiation, that his working hypothesis is no longer useful.

4). MATSUURA & SUTÔ's (*1948*) *hypothesis of chromatid segregation*. In the present status of cytology, it is a well-known fact that a chromosome has two sister chromatids, each of which is formed of at least two (half chromadits), certainly 4 or more chromonemata. Of recent years, most cytologists have come to the impression that the chromonemata in a chromosome may be very numerous and variable in different tissues, and that they seem to correspond with the *"lamellae"* of simple protein molecule. On the other hand, it has become well-established and long-suppoted by the genetic evidence that the gene is a unit of such characteristics as the following: the discontinuity of heritable character, the crossing over and the mutation, and also that the chromosome, as well as the gene, behaves as a four-stranded unit in meiosis.

HUSKINS (1947) postulated the lamellae hypothesis of gene structure

to fit both the genetical and cytological data jointly. His speculation is to the effect that the gene as well as the chromosome is not a structural unit but obviously of the two or many more distinct units corresponding to the lamellae (chromonemata). Such a many stranded units are usually found dividing into two equal numbers in course of the chromosome splitting of cell division mechanism, owing to the existence of "a weakest point between the two central laminations" but occasionaly into three or more units. If some one of such gene-units is different from the others in the phenotypic expression, then obviously the daughter chromosomes produced through random assortments of such different units in the mother chromosome in the division mechanism must become of various types in relation to the combination of gene units. If such segregation of gene units occurs in course of ontogeny of pericarp tissue and of germinal tissue, then there must result in the mosaic pericarp and in the gene mutation, respectively.

This hypothesis of chromatid segregation may be *a priori* recognized as a revised speculation of EYSTER's genomere hypothesis on a modern cytogenetical basis. EYSTER's speculation may be brought into line with the present idea in replacing his "*genomeres*" by the chromosomal constituting units, "*chromonemata* or *half chromatids*". The process as to how to the gene mutation and the mosaic expression have occurred in course of ontogeny can be easily understood by both the hypotheses. But, neither the present idea nor any one of the foregoing hypotheses gives an explanation enough for the nature of gene mutation. The gene mutation must first begin with a change of a genomere or a chromonema; there is no touching upon this changing mechanism in all of the above hypotheses.

5). STURTEVANT's *hypothesis of unequal crossing-over*. Recently, some of the genes, which have been known as belonging to so-called a "*multiple allelic series*", are re-established to not lie in a definite locus of a chromosome but obviously to arrange in closely linear order. They are therefore formed of a series of respective loci, and certainly are separable, though rarely, by the same mechanism of crossing over as in the other independent genes. Such case has recently been found as common in animals as well as in plants. Such a gene group, for which the name "*pseudo-allelic*" was given by LEWIS (1945), can be distinguished from the true multiple alleles in its having special features. A survey of pseudo-allelic genes was recently made by KOMAI (1950).

The critical evidence on the origin of pseudo-allelic genes has been

given from both the cytological and genetical data. Namely, the genetical data showed that the loci have arisen from the duplication of a single locus through the unequal crossing-over, and thus each of the loci takes its place in a side-by-side sequence in a chromosome (STURTEVANT 1925, OLIVER 1940, OLIVER & GREEN 1944, LEWIS 1945, STEPHENS 1948 and LAUGHNAN 1949). The same cases of such pseudo-allelic nature have recently been suggested from genetic respects to consist of many loci (HOSHINO 1943, STADLER & FOGEL 1945, DUNN & CASPARI 1945, and KOMAI & TAKAKU 1949). The studies of salivary chromosomes in *Drosophila* have cytologically demonstrated the presence of serial duplication of the band, *"repeat"*, for the pseudo-allelic genes (BRIDGES 1936 and LEWIS 1945). In spite of such duplication as "repeat", there is always observed to exist the difference of phenotypic expression between pseudo-allelic loci in a slight degree. This difference has been recognized as the phenotypic effect owing to a chromatin rearrangement, *"position effect"*. GOLDSCHMIDT (1946) pointed out in his review of position effect that all cases of the position effect parallel in every respect the behavior of so-called gene mutation and the gene mutation should be termed as the *"rearrangement effect of chromatin"* rather than the position effect.

It is a well-known fact that the crossing over occurs between any two of four chromatids in meiosis, and the resulting crossover chromosome is of the same phenotypic effect on the gene in question as the non-crossover, because there is no any rearrangement of chromatids. Such crossing over will be also expected to occur between sister chromatids of a somatic chromosome in the same frequency as that of meiotic chromosome. Similarly, if an unequal crossing-over takes place infrequently in meiosis, as well as in mitosis, then the resulting crossover chromosomes should be grouped to the three types concerned with the rearranged chromatid ; they are, (1) having only a duplication of its gene locus in close sequence, (2) with only a deficiency of its locus and (3) invlving both (1) and (2). Each of those three typed cells will segregate in course of ontogeny, according to the random assortment of chromatid combination at the time of chromosome division (based on chromatid segregation hypothesis). Cells having such rearranged chromosomes will in some case give rise to the genic change, which distinguishes them from cells having the non-crossover chromosome in the phenotypic expression (the position effect of gene). Possible mechanism on the occurrence of both the mutation and mosaicism can

thereby be interpretated easily. The present case has no factual evidence in conflict with this simple and rational hypothesis; but there is certain evidence to support it, as well as all of the other cases of pseudo-allelic genes, as discussed later.

4). A comparison of the various rates of genic change

The present study deals with the genic change of five members in a *P*-allelic series, all of which have arisen from a single ear with a mosaic pericarp ($M \cdot \widehat{E \cdot zl}/W \cdot +$) through a continuous selection in our inbreeding program: viz. W, M, R and W_p. In populations from possible combinations of them, the type of pericarp colors, which can not be expected from the basis of Mendelian segregation, was infrequently observed, and it was recorded as a gene change. Results obtained are given in Table 12, showing a summary of Tables, 1 and 2. An assumption, in accordance with a hypothesis of unequal crossing-over, was applied to the present data to get the rate of genic change. Namely, a single gene shifts simultaneously to two genes of different effect in the phenotypic expression—an original chromosome, that is, two sister chromatids, shifts simultaneously to two rearranged chromatids of different types—when a genic change occurs as the result of an unequal crossing-over, and thereby the producing frequency of such two types is always equal. Actually, both mates of such paired change did not give rise to so equal frequency as expected. There was always an excess of the W_p mate. There was also found to occur only in one of mates within an expected pair explicable on the ground that the other mate is of either the same phenotypic expression as the original type, or is masked by the dominant character of others.

The combined method of maximum likelihood, suggested by MATHER (1938), was adopted to estimate the value of changing rate. The value (α) was indicated by the percentage of producing gametes, as well as the recombination value of normal crossing over, at this evalution, no correction was given for the above mentioned actual frequency to obtain a logical value, because the frequency of genic change is too small to treat statistically. It is a fact therefore that the calculated value is always much larger than that occurring in fact.

As can be seen in Table 12, it seems that the genic change is different in relative frequency with each of the rows. Actually, some of them showed nothing in the occurrence of genic change, in others it

TABLE 12

Rates (α) of the genic change in various chromosomal constitutions, which are summarized from Tables, 1 and 2, estimated from occurring of the unequal crossing-over in a *P* containing region of chromosome 1

Genotype	Phase	Observed		Calculated			
		Plants	Mutants	α	I_α	V_α	S_α
1, $M\cdot\widehat{E\cdot zl}/M\cdot+\times W\cdot+$	F_1	1208	7	*0,0058*	*234547,65*	*0,00000426*	*±0,00206*
2, $M\cdot\widehat{E\cdot zl}/W\cdot+\times W\cdot+$	Back	4089	11	*0,0055*	*368693,91*	*0,00000271*	*±0,00165*
3, $W\cdot\widehat{E\cdot zl}/M\cdot+\times W\cdot+$	"	39	0	*0*	—	—	—
4, $M\cdot\widehat{E\cdot zl}/R\cdot+\times W\cdot+$	F_1	174	0	*0*	—	—	—
5, $R\cdot+/R\cdot+\times W\cdot+$	"	255	0	*0*	—	—	—
6, $R\cdot+/W\cdot+\times W\cdot+$	Back	429	2	*0,0140*	*16629,22*	*0,00006013*	*±0,00818*
7, $M\cdot\widehat{E\cdot zl}/W\cdot+\times R\cdot+/W\cdot+$	F_1	870	+	*0,0160*	*463,62*	*0,002157*	*±0,04644*
8, $M\cdot\widehat{E\cdot zl}/M\cdot+$	Self	901	8	*0,0069*	*141497,12*	*0,00000707*	*±0,00265*
9, $M\cdot\widehat{E\cdot zl}/W\cdot+$	"	2281	6	*0,0039*	*500013,22*	*0,000002*	*±0,00141*
10, $W\cdot\widehat{E\cdot zl}/M\cdot+$	"	462	0	*0*	—	—	—
11, $W\cdot\widehat{E\cdot zl}/W\cdot+$	"	887	9	*0,0152*	*37898,42*	*0,00002638*	*±0,00514*
12, $M\cdot\widehat{E\cdot zl}/R\cdot+$	"	494	0	*0*	—	—	—
13, $R\cdot+/R\cdot+$	"	18	0	*0*	—	—	—
14, $R\cdot+/W\cdot+$	"	439	3	*0,0095*	*21064,76*	*0,00004747*	*±0,00689*
Total		12546	46+	*0,0057*	*1544842,61*	*0,000000647*	*±0,000804*

occurred rarely and in still others many times, its value ranging from zero to 1.6 percent. An average mean of changing rate was of the value of 0.57 ± 0.08 percent in a total of 12546 plants. Of this value, the upper and lower fiducial limit at a five percent level is at 0.73 and 0.41 percent ($0.57\pm2\times0.08$). Seemingly, most of the calculated α values of mean in each row are beyond both limits. If the α value in each row were to be compared with that of a total average (0.57) according to the t° test, then the difference between them would be always significant. To make such a comparison is against the statistical rule when a value of "$n\times\alpha$" is at least less than five. Therefore, it is impossible to apply the t° test to the present case.

The χ^2 method of heterogeneity analysis was applied to test an expectation that the obtained values all agree in showing one value (0.57) calculated on the basis of the best combined estimation of α. This method will not suffer from the same serious disadvantage as that obtained from the t° test according to the basis of the separate esti-

TABLE 13

Summary of data from Table 12 on a best combined

	Genotype	Phase	dL/dx (D)		
			$\alpha = 0{,}006$	$\alpha = 0{,}005$	$\alpha = 0{,}0057$
1,	$M \cdot \widehat{E \cdot zl}/M \cdot + \times W \cdot +$	F_1	− 41,58282	192,96483	20,18523
2,	$M \cdot \widehat{E \cdot zl}/W \cdot + \times W \cdot +$	Back	−183,76928	184,92463	− 86,66945
3,	$W \cdot \widehat{E \cdot zl}/M \cdot + \times W \cdot +$	"	− 35,21127	− 35,17588	− 35,20064
4,	$M \cdot \widehat{E \cdot zl}/R \cdot + \times W \cdot +$	F_1	− 83,50101	− 83,41709	− 83,47581
5,	$R \cdot +/R \cdot + \times W \cdot +$	"	−226,35815	−226,13065	−226,28985
6,	$R \cdot +/W \cdot + \times W \cdot +$	Back	124,07780	190,95477	141,68479
7,	$M \cdot \widehat{E \cdot zl}/W \cdot + \times R \cdot +/W \cdot +$	F_1	5,26266	5,72528	5,42528
8,	$M \cdot \widehat{E \cdot zl}/M \cdot +$	Self	137,19811	404,94043	213,65643
9,	$M \cdot \widehat{E \cdot zl}/W \cdot +$	"	−520,04021	−314,03580	−477,98776
10,	$W \cdot \widehat{E \cdot zl}/M \cdot +$	"	−322,68625	−322,40601	−322,60215
11,	$W \cdot \widehat{E \cdot zl}/W \cdot +$	"	912,31593	1212,70904	991,38129
12,	$M \cdot \widehat{E \cdot zl}/R \cdot +$	"	−345,07042	−344,72362	−344,96631
13,	$R \cdot + \cdot /R \cdot +$	"	− 0,02160	− 0,01800	− 0,02052
14,	$R \cdot +/W \cdot +$	"	284,47122	384,61538	310,83027
	Total		−523,41680	1250,92732	105,95080

mates, although the precision for the χ^2 method is less, differing from the precision for the t° method. Results calculated are shown in Table 13 where a total χ^2 of heterogeneity, 1063,2736, corresponding to fourteen separate estimates was further analysed into three components: individual sets (DF=7), genotypes (DF=4) and crossing phases (DF=2). Of those components, the highly significant χ^2 can be seen in both ones, individual and genotype. It is evident from this table that the significant deviation in those two components is certainly ascribable to non-occurrance of the genic change, because all sets exhibiting the genic change are characterized by having non-significant χ^2. The occurrence of genic change as actually observed is therefore too infrequent for a solution of the interesting problems in accordance with any statistical analysis, such as: is significant difference found among different genotypes or phases of crossing combinations? and what kind of relationship

estimate and on the its testing heterogeneity

I_α	χ^2 (D^2/I_α) between			
	Individuals	Genotypes	Phases	Deviations
233547,65	*0,0017*			
368698,91	*0,0204*	*0,0172*		
35,39	*35,0110***			
83,92	*83,0423***	*83,0423***	*0,1087*	
227,50	*225,1063***	*0,1067*		
66879,97	*0,3002*			
462,62	*0,0636*	*0,0636*	*0,0636*	*0,0073*
267742,32	*0,1706*			
206004,41	*0,9743*			
280,24	*371,3629***	*1,0532*		
300393,11	*3,2339*			
346,80	*343,0104***	*343,0104***	*0,1567*	
3,60	*0,0114*	*0,9645*		
100144,16	*0,9646*			
	1063,2736	*428,2579*	*0,3290*	*0,0073*
1544842,61	*0,0073*			
χ^2:	*635,0157***	*427,9289***	*0,3227*	*0,0073*
DF:	7	4	2	1

exists between the relative value of occurrence and the genetic component?

At any rate, it may be possible to show that such genic change (so-called mutation) occurs, through rarely, in most of both the various genotypes and crossing phases, and that the rate of its occurrence is seemingly different between each other of different genotypes. All of the genotypes examined may be arranged according to the size of changing rate in the following series: "$W\cdot+/W\cdot+$" (W) = "$W\cdot E\widehat{\cdot zl}/M\cdot+$" ($W_p$) = "$M\cdot E\widehat{\cdot zl}/R\cdot+$" (M) = "$R\cdot+/R\cdot+$" (R) < "$M\cdot E\widehat{\cdot zl}/M\cdot+$" (M) < "$M\cdot E\widehat{\cdot zl}/W\cdot+$" (M) < "$R\cdot+/W\cdot+$" (R) < "$W\cdot E\widehat{\cdot zl}/W\cdot+$" ($W_p$).* Of these, four genotypes, "$W\cdot+/W\cdot+$ (W)", "$W\cdot E\widehat{\cdot zl}/M\cdot+$ (W_p)", "$M\cdot E\widehat{\cdot zl}/R\cdot+$ (M)" and

* The symbol, >, means "has a larger and less rate than", while the sumbol, =, means "is approximately equivalent to"; and also the abbreviation in parenthesis indicates a phenotype which is determined by a genotype outside of parenthesis.

"$R\cdot+/R\cdot+$(R)", cannot be compared with each other owing to the non-occurrence of genic change, but it may be possible to say that its frequency is very small in every one, probably near to zero even if it does occur. The sequence of another one, "$R\cdot+/W\cdot+$", may be uncertain in its ordinal position, on the ground that its observed plants, as compared with the others, are few in number. Accepting a possible supposition, this genotype would better be rearranged in a position between the genotypes, "$R\cdot+/R\cdot+$" and "$M\cdot\widehat{E\cdot zl}/M\cdot+$", rather than in its above position, and similarly, the other genotype, "$M\cdot+/M\cdot+$", between "$R\cdot+/R\cdot+$" and "$R\cdot+/W\cdot+$" although there is no data with respect to the genic change of this genotype. Revised data based upon such a supposition may bring about the following seriation;

"$W\cdot+/W\cdot+$" (W)$\leqq$? "$W\cdot\widehat{E\cdot zl}/M\cdot+$" ($W_p$) = "$M\cdot\widehat{E\cdot zl}/R\cdot+$" (M)$\leqq$? "$R\cdot+/R\cdot+$" (R)$<$"$M\cdot+/M\cdot+$" (M)?$<$"$R\cdot+/W\cdot+$" (R)?$<$"$M\cdot\widehat{E\cdot zl}/M\cdot+$" (M)$<$"$M\cdot\widehat{E\cdot zl}/W\cdot+$" (M)$<$"$W\cdot\widehat{E\cdot zl}/W\cdot+$" ($W_p$)

From this seriation and from Table 12, remarkable features concerned with the relative frequency of genic change may be pointed out as follows:

a). *The changing rates of P–allelic members.* Taking no notice of the combination effect of mutual genes in a heterozygous condition, the genic change may be stated to have the following characteristics: The *W* gene is so highly stable in respect to genic change that it does not give rise to any genic change at all. While all of the other genes are so unstable that they are found to change from one to another in different degrees of considerable frequency. Namely, the rate of original genic change, from the *M* gene to the another *R* or *W*, is different from the changing rate in the opposite direction, from *R* and *W* to another; the changing rate of *M* is less than that of W_p and more than that of *R*. In other words, the reversible change does by no means occur in the same frequency as the original change. According to those findings, the frequency order of genic change with respect to ear type may be written as "$W\cdot+<R\cdot+<M\cdot+<+\cdot\widehat{E\cdot zl}$".

Good cases of such a reversible change, in keeping with the present data although its occurring rate does not always correspond, have been described by several geneticists, Emerson (1917), Eyster (1924 and 1925) and Anderson & Ter Louw (1928) in other mosaic pericarps of maize, where the changes occur in both directions.

b). *The heterozygosity relation of genic change.* Since it is a fact that

the *W* gene never changes itself to any another member of *P*-alleles in a homozygous condition ("*W*·+/*W*·+"), of a heterozygous genotype (such as "*W*·+/*M*·+" and "*W*·+*R*·+"), a genic change should affect only one (either R or M) of duplex genes. Accordingly, the genic change would occur about twice as frequently in homozygosity for every one of the other *P*-allelic members as in heterozygosity for the *W* gene, if the changing ability of each of *P*-alleles without *W* were not influenced by its opposite mate, *W*, in a heterozygous condition.

In order to ascertain this expectation, a comparison of the relative changing ability was made between homozygous and heterozygous ears obtained from the selfing and backcrossing progeny, both of which have descended from the same genotype. It is confirmed from Tables 1 and 2 that, contrary to expectation, the heterozygous ear, such as *R*/*W* and *M*/*W*, changes more frequently than the homozygous ear, such as *R*/*R* and *M*/*M*, in all cases. This is specially noticeable in the heterozygous condition for *W*. In fact, a genic change, from *M* to *R*, coming from backcrossing was not observed in a total of 1208 plants in homozygous condition (*M*/*M*) while it was observed in 11 ears in a total of 2016 plants in heterozygous condition (*M*/*W*). Selfing data on the similar genic change showed that there is one ear in a total of 901 plants, and 6 ears in 1523 plants for respective hom- and heterozygosity. Similarly, in the heterogosity of *R* or *M* for another one of the *P* alleles, the changing ability of its opposite allelic mate may be not, or probably in small degree, recognized to increase its combining effect, as seen in Table 12.

Summing up, the modifying ability on the occurrence of a genic change, when heterozygous, is not uniform amongst P-alleles. Namely, the *W* gene increases more the changing ability of its opposite mate than do the other genes, *R* and *M*: the order of influence may be arranged as "$R \leqq ? M \ll W$". This seriation is in an opposite direction against the order of dominance represented by the gene itself. Especially, a heterozygous combination, *M*/*R*, did not show any type of genic changes at all, instead of occasional occurrences of genic change in all of other combinations. An important feature of this data is that each of the *P*-allelic members not only itself contributes to differential effect on a pericarp color expression on the one hand, but also behave itself as responsible differently for an acceleration of the genic change of its opposite allele, when heterozygous, on the other hand.

A strong support for the present finding can be found in another mosaic character of maize reported by EMERSON (1929). He advocated

a working hypothesis to account for his results on the mutability of pericarp variegation, suggesting a possiblity that there exists one or more genes at loci, other than the *P* locus, always closely linked with the *W* gene on the same choromosome, and capable of modifying the mutability of *P*-allelic member.

c). *The modifying effect of* $E\widehat{\cdot}zl$ *on the genic change of pericarp color.* Of six possible types of the gamete which is expected from all simplex combinations of *P* and $E\widehat{\cdot}zl$ alleles used, all but one ("$R\ E\widehat{\cdot}zl$"), could be obtained, viz. "$W\cdot+$", "$M\cdot+$", "$R\cdot+$", "$W\cdot E\widehat{\cdot}zl$", and "$M\cdot E\widehat{\cdot}zl$", through a continuous selection of an original duplex combination, "$M\cdot E\widehat{\cdot}zl/W\cdot+$". In fact, the nine viable, duplex combinations of the genotype have been synthesized by using such five types of the gamete. They may be divided into the following two groups, according to the degree of the relative rate of the genic change:

Group 1		Group 2	
$W\cdot+/W\cdot+$	(W)	$[M\cdot+/W\cdot+$	(M)]*
$R\cdot+/R\cdot+$	(R)	$R\cdot+/W\cdot+$	(R)
$M\cdot E\widehat{\cdot}zl/R\cdot+$	(W_p) ?	$M\cdot E\widehat{\cdot}zl/M\cdot+$	(M)
$W\cdot E\widehat{\cdot}zl/M\cdot+$	(W_p) ?	$M\cdot E\widehat{\cdot}zl/W\cdot+$	(M)
		$W\cdot E\widehat{\cdot}zl/W\cdot+$	(W_p)

Group 1 may be characterized by giving rise to no genic changes, or probably a very few even if it occurs, while group 2 by occasional occurrence of genic change. A careful comparison of each genotype within a group may show that the changing ability is different in each of the *P*-allelic members, depending on whether any one of them is adjacent to $E\widehat{\cdot}zl$ or to its wild-mate ($E\widehat{\cdot}zl^+$). Of genotypes belonging to group 2 all but one ("$M\cdot E\widehat{\cdot}zl/M\cdot+$") are not only heterozygous for W, but further three of the most effective genotypes are associated with $E\widehat{\cdot}zl$, viz. "$M\cdot E\widehat{\cdot}zl/M\cdot+$", "$M\cdot E\widehat{\cdot}zl/W\cdot+$" and "$W\cdot E\widehat{\cdot}zl/W\cdot+$". This fact may suggest that $E\widehat{\cdot}zl$ behaves to increase the accelerating effect of the *W* gene on the genic change of its opposite mate of *P*-alleles. An extreme case can be seen in comparison of "$W\cdot+$" with "$W\cdot E\widehat{\cdot}zl$"; the

* It is not feasible to estimate the actual rate of the genic change on the following two grounds: that (1) genic changes, if they occur, are masked by any one of segregants owing to its consisting in three class segregation, R, M and W, and (2) the difference between three classes from expectation is so highly significant that it is impossible to rationally distinguish genic changes from segregants. Thus, the position was of a presumed one, but it is sure that this genotype belongs to this group because the genic change is found to occur occasionally, although its rate is uncertain.

W gene being highly stable when associated with $E\widehat{\cdot zl}^{+}$, "$W\cdot+/W\cdot+$", while most mutable when adjacent to $E\widehat{\cdot zl}$, "$W\cdot+/W\cdot E\widehat{\cdot zl}$".

On the other hand, group 1 may show that the modifying effect of $E\widehat{\cdot zl}$ on the genic change is in an opposite direction as compared with group 2. In this case, $E\widehat{\cdot zl}$ is always conditioned by combining with "$M\cdot+$" or "$R\cdot+$" as its opposite mate in duplex, viz. "$M\cdot E\widehat{\cdot zl}/R\cdot+$" and "$W\cdot E\widehat{\cdot zl}/M\cdot+$". A conclusion may be thereby reached that the modifying effect of $E\widehat{\cdot zl}$ exists in both directions, depending upon the mode of duplex combination as to whether an $E\widehat{\cdot zl}$ containing gamate is fertilized with the "$W\cdot+$" gamete or with the "*non*–$W\cdot+$" one; one resulting in an acceleration of the genic change (group 2) while the other results in a suppression (group 1). It is of much interest to note that $E\widehat{\cdot zl}$ is not only contributed itself to a phenotyic expression on the pericarp color as mentioned already (p 101), but also is responsible for a reversible, modifying effect on the genic change of each *P* allele, to which $E\widehat{\cdot zl}$ is adjacent.

A further evidence on the present finding can be found in EMERSON' data (1929) about the modifying effect on the genic change of pericarp variegation. EMERSON stated that differential effects on the modifying ability were observed in various *W* stocks from different sources when they were crossed with a mosaic strain.

Discussion

Like all pericarp variegations hitherto studied, the present mosaic pericarp offers a material for studies of the genic change, because the changing rate is so very high as compared with that of other characters in maize that pertinent data can readily be obtained. The present discussion is offered as to the nature of genetic behavior, which is difficult to explain from the Mendelian basis of segregation. Genetic abnormalities, unexpected from the parent genotypes in course of making segregation-tests in progenies, comprises two different categories; (a) the disturbance of normal segregation and (b) the occurrence of genic change. Each of the cases, as explained already in the foregoing description, was considered as a rearrenged effect of the chromatid derived by an unequal crossing-over; the former case resulting from the somatic segregation of such a rearranged chromatid in the ontogenetic course of somatic tissue, and the latter case resulting from the germinal tissue,

both of which contain such a rearranegd chromosome through the divison mechanism.

There is rational evidence to support the present idea about the unequal crossing-over. A careful study was made to determine a linear relation between the $E\cdot\widehat{zl}$ allele and the so-called mutant allele of *P*, both closely linked together (its crossover value = ca. 1.5 percent units). An original genotype of duplex combinations, *P* and $E\cdot\widehat{zl}$, used was constituted of "$M\cdot E\cdot\widehat{zl}/W\cdot+$". One half of the duplex, "$W\cdot+$" is extremely stable, and therefore never gives rise to any genic change into another member of *P*-alleles, but it is responsible for an increase in the changing ability of its opposite mate (*M* in this case). The other half of the duplex, "$M\cdot E\cdot\widehat{zl}$", is highly unstable, occasionally changing to other two types, R or W_p. The same types of the genic change, R and W_p, occur also in progenies of homozygous M-strains with "$M\cdot E\cdot\widehat{zl}/M\cdot+$". Eight plants of such genic changes, five R and three W_p, which had been obtained from those M-strains during seveen years from 1938 till I944 in open pollinated condition, were studied to find what genotypes they possessed by making tests of their progeny. Genotypes of plants grown from kernels of five R plants were made up as follows: "$R\cdot+/W\cdot+$", "$R\cdot+/R\cdot+$", $R\cdot+/M\cdot+$" and "$R\cdot+/M\cdot E\cdot\widehat{zl}$" (appendix Tables, 17 and 18). Genotypes of selfing W_p plants grown from kernels of open-pollinated W_p ears were identified as either "$W\cdot E\cdot\widehat{zl}/M\cdot+$" or "$W\cdot E\cdot\widehat{zl}/W\cdot+$". In both cases, all of opposite mates of "$R\cdot+$" in R plants or of "$W\cdot E\cdot\widehat{zl}$" in W_p plants must be regarded as originating from non-crossover pollen of both parent homozygous and heterozygous M-plants, because all of changed plants were gathered in open-pollinated condition from their inbreeding field.

It is clear from this data that a genic change from "$M\cdot E\cdot\widehat{zl}$" to R type is lacking in $E\cdot\widehat{zl}$, the changing chromosome having a genic constitution of "$R\cdot+$", while an other change from "$M\cdot E\cdot\widehat{zl}$" to W_p type is certainly associated with $E\cdot\widehat{zl}$, it being of "$W\cdot E\cdot\widehat{zl}$". This fact will strongly support a plausible possibility that an unequal crossing-over must occur in the region between two alleles, *M* and $E\cdot\widehat{zl}$. On that supposition, it is quite natural that resulting crossovers should be expected as dividing into two classes in connection with the linear rearrangement of *P* locus, viz. (1) "$MW\cdot+$" (from "$M\cdot E\cdot\widehat{zl}/W\cdot+$") and "$MM\cdot+$" (from "$M\cdot E\cdot\widehat{zl}/M\cdot+$") as a duplicate form and (2) "$-\cdot E\cdot\widehat{zl}$"

as a deficient one. Of those crossovers, the duplicate form will become a R type in phenotypic expression, and was symbolized as "*R*·+". However, the deficient one will become a W_p type, and was termed as "W·$\widehat{E \cdot zl}$". If the possibility of such unequal crossing-over is admitted in the present case, then a genic change to R should result in a rearrangement of the linear order of two alleles, *P* and $\widehat{E \cdot zl}$;—there is always a duplication of *P* and a lacking of $\widehat{E \cdot zl}$ in the rearranged chromosome—, while a genic change to W_p should be directly opposite to the former change in manner of the rearrangement of alleles. This means itself that an unequal crossing-over should occur at the right side of the original *M*-locus and never occur at the left side of *M*.

There are further available reasons to support the present idea, as follows: (1), The genic change of "$M \cdot \widehat{E \cdot zl}$" is characterized by consisting in certain two types, R and W_p, in appoximately equal frequency (p. 105). (2), If the present change were the so-called "gene mutation (a point mutation)", then the original two alleles, *M* and $\widehat{E \cdot zl}$, would simultaneously mutate into other two, *R* and $\widehat{E \cdot zl^+}$, to get R. This is very difficult to prove from the present knowledge of genetics. (3), The fact that two changed plants, R and W_p, are always conditioned by a genic constitution of "*R*·+" and "$W \cdot \widehat{E \cdot zl}$" respectively, unrelated to either "$R \cdot \widehat{E \cdot zl}$" or "*W*·+", can be supported on a genetic basis that the occurrence of double crossing-over is impossible to expect for a short distance of this region (about 1.5 percent units).

The gene is merely a unit of the crossing over and of the so-called "mutation". There is nothing in any cytogenetical evidence to validate the idea that the gene is an undividing point (or locus) on the chromosome. According to GOLDSCHMIDT'S review on "position effect" (1946), it has been demonstrated by many *Drosophila*-cytogeneticists that the gene corresponds to a very small segment of a chromosome, (about 5–10 bands of salivary chromosome), of which each part is differentially responsible for quantity of a character expression when the linear order within a segment was shifted—a rearrangment whithin a segment occurs—by causing a breakage and reunion between two uncorresponding (non-homologous) loci of two chromosomes, or chromatids, or chromonemata. Goldschmidt strongly asserted that a visible change of linear order in such a segment is of position effect while an invisible change is of the gene (point) mutation. In the present case, a breakage occurring just to the right side of the original *M*-locus must result in

a rearrangment of the linear sequence and such a rearranged effect (a position effect) may reveal itself as R or W_p type of pericarp color instead of the original M-type.

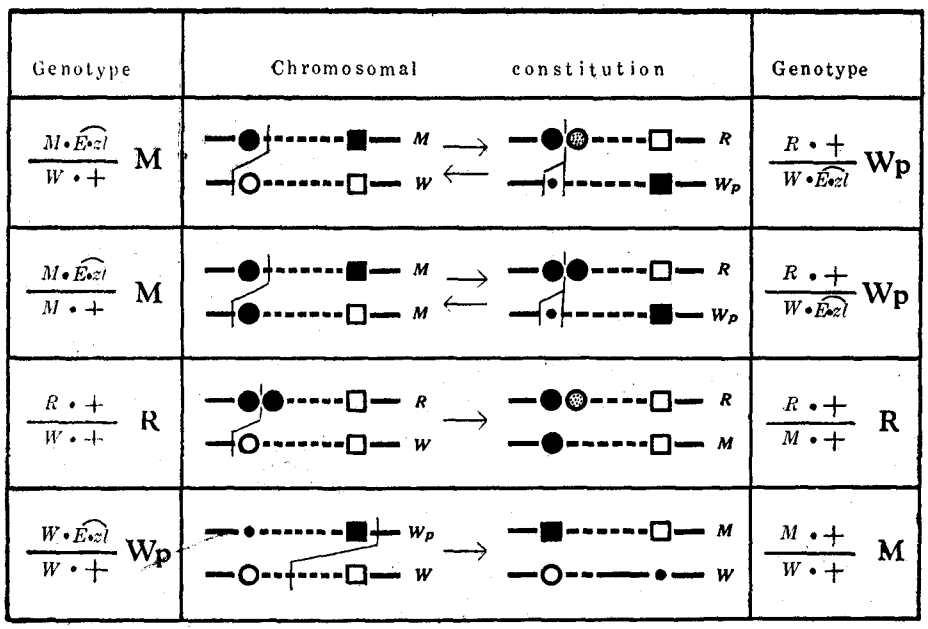

Fig. 3. Diagram showing the suspected shifts of intrachromosomal composition, based on the unequal crossing-over, which accompany the genic change of pericarp color. In diagram, the genotypic constitution used is shown in the right and left columns, and its chromosomal composition in the intervals. The arrow shows the genic changes observed in fact; the top two are found to be reversible in the opposite directions, while the remaining two are found to occur in one side direction but the latter may be also expected to be reversible as well as the former. Two types of Gothic letters indicate the phenotype observed; the Roman type represents that of zygote with its squared genotype, and the Italic type represents that in which the appropriate phenotype appeared when a gamete with a chromosome squared is fertilized with the colorless gamete with "*W*• +".

Explanation of symbols of the chromosomal constitution tabulated: The solid and dotted lines show supposedly euchromatic and heterochromatic parts respectively present near the *P*-locus of chromosome 1. Of *P*-alleles, the original ones used, *M* and *W*, are shown respectively by the symbols, ● and ○. Crossovers induced by unequal crossing-over are indicated by the symbols, ●● (or ●◉) as the duplicated form of an allele and • as the deficient form, the former acting as *R* and the latter as *W* of *P*-allelic members. $\hat{E}$•*zl* is represented by the symbol, ■ and its wild-typed allele by □.

In Fig. 3, the suspected events of intrachromosmal rearrangement, based upon the unequal crossing-over, and their accmpanying genic changes are given in an attempt to illustrate the nature of the pseudo-allelism of gene in the concept of gene-differentiation mentioned above.

Critical evidence, as regards the idea that some members of the so-called "*multiple allelic*" series (termed "*pseudo alellic*") consist in a series of subunits, "loci", certainly arranging in a linear order and that they are separable rarely by the normal crossing-over, has recently been provided by LEWIS (1945), STEPHENS (1948) and LAUGHNAN (1949). They supposed that such a series of loci have arisen by duplication of a single "*ancestral*" locus through the unequal crossing-over. Careful study of salivary chromosomes in *Drosophila melanogaster* has established the existence of such duplications, "*repeat*", in two cases: *Bar* (Bridges 1935) and *Star-asteroid* (LEWIS 1945). Good examples of this situation are probably ones described by MCCLINTOCK (1941-a and 1944) in two cases of pigment characters in maize. All of mutants in both cases are associated with loss of specific part within a minute segment of chromosome, and the specificity of mutant character is developed according to the size and position of the deficient part. A *bm* region of the short arm of chromosome 5 is characterized by having seven sensitive centers showing the following characters; *brown cell walls* (*bm*), *pink*, *blothch*, *blotch-dries*, *pale green*, *striate* and *white*. Of them, the former five have their loci within the limits of the proximal four chromomeres and the remaining three within the next five chromomeres. All of them may be probably of the "*pseudo allelic*" nature. The other case is of a py_2 region containing a heterochiromatic knob and a next adjacent chromomere located in the distal end of the short arm of chromosome 9. Three "*pseudo-allelic*" centers in this region were cytologically established to be arranged in the following order; *yellow green* (yg_2)–*pale yellows* (pyd_1–pyd_7)–*white seedlings* (wd_1–wd_7). MCCLINTOCK (1941–b) reported that a long series of duplications may sometimes be accumulated by particular mechanisms in maize, although there is no any direct evidence of the unequal crossing-over to account for their origin.

According to American geneticists' statements, all of *P*-alleles, except both top dominant and basal recessive ones (*R* and *W*), have progressively mutated in both directions, dominant and recessive. The present *M* changes also in reversible directions, *R* and *W*. The changed *R* is not stable, but further gives rise occasionally to a genic change to *M* or *W* in an opposite direction (Tables, 1 and 2). Good cases of a

reversible change have been described already in pericarp variegations by EMERSON (1917), EYSTER (1924, and 1925), ANDERSON & TER LOUW (1928) and others. All of these data indicate that the genic change of pericarp variegation occurs in reversible directions. A linear arrangement of doublets in *R* may be therefore of a *"direct repeat"* nature. If doublets were arranged in a *"reverse repeat"* condition, the further rearrangement of this segment to be induced by once more crossing over should result in only a dicentric chromatid, which will be lost from the producing gametes. The fact, that a reversible change occurs in this case, is in contradiction to expectation from the *"reverse repeat"* concept but can readily be interpretatetd by the *"direct repeat"* concept. There is however no information to make clear directly whether doublets are "direct" or "reverse".

The genetic behavior of a *P–E·zl* relation in maize seems to be in many respects similar to that of a *S–ast* relation in *Drosophila* reported by LEWIS (1945). That is to say, each of both alleles is associated with a single character, but merely different to each other in quantity of its phenotypic expression; its effectiveness is much larger in one than in the other. Further a less effective allele, such as *S* in *Drosophila* and *E·zl* in this case, not only behaves itself as an enhancer of the more effective allele, such as *ast* and *P*, but also is lethal in homozygosity. However, *E·zl* differs from *S* in regard to the nature of genic change. This can be indicated by the following features; (1), W_p type, *"W·E·zl/W·+"*, gives rise to two types of reverse changes, R. (*R·+/W·+*) and M (*M·+/W·+*). (2), This information means that *E·zl* may shift itself to *R* or *M* belonging to a *P* allelic series. And (3), When such genic change of *E·zl* occurred, all characteristics of *E·zl* as mentioned are found to be lacking. From those features it may be supposed that *E·zl* has arisen from duplication of a *P*-locus through an unequal crossing-over and has newly differentiated its specificity owing to a rearranged effect of chromatid. Such mechanism about origin of *E·zl* may be of the same nature as that of some (R, M and W) of *P*–allelic members. In the case of *Star* in *Drosophila* there is no genic change from *S* to *ast*. LEWIS (1945) concluded thereby that *"S–ast"* doublets are a *"tandem reverse repeat"*. In the present case, *P–E·zl* doublets may represent a *"tandem direct repeat"* (see Fig. 3).

Many of the gametic combinations of various *P*– and *E·zl*– members,

which have been derived by using an original M-ear ($M\cdot\widehat{E\cdot zl}/W\cdot+$), will be expected to be produced. A comparison of them should shed further light on the phenotypic effect of various rearrangements within a P-$\widehat{E\cdot zl}$ segment. In fact, only five gametic combinations could be obtained in the present experiments; "$W\cdot+$", "$M\cdot+$", "$R\cdot+$", "$M\cdot\widehat{E\cdot zl}$" and "$W\cdot\widehat{E\cdot zl}$". By making hybrids of possible combinations between plants with the following nine genotypes; "$M\cdot\widehat{E\cdot zl}/W\cdot+$(M)", "$M\cdot\widehat{E\cdot zl}/M\cdot+$ (M)", "$M\cdot\widehat{E\cdot zl}/R\cdot+$ (M)", "$M\cdot+/W\cdot+$ (M)", "$R\cdot+/R\cdot+$ (R), "$W\cdot\widehat{E\cdot zl}/W\cdot+$ (W_p)", "$W\cdot\widehat{E\cdot zl}/M\cdot+$ (W_p) and "$W\cdot+/W\cdot+$ (W), of 25 possible types of duplex combinations, all of 21 viable duplexs have been successfully synthesized. Their phenotypic expressions are compared to each other in Fig. 4 where "$WM\cdot+$" was neglected owing to entire correspondence with "$MM\cdot+$" in its effect. It is apparent that the data as seen in Fig. 4 are in keeping with the idea of the rearranged effect based on the mechanism of unequal crossing-over.

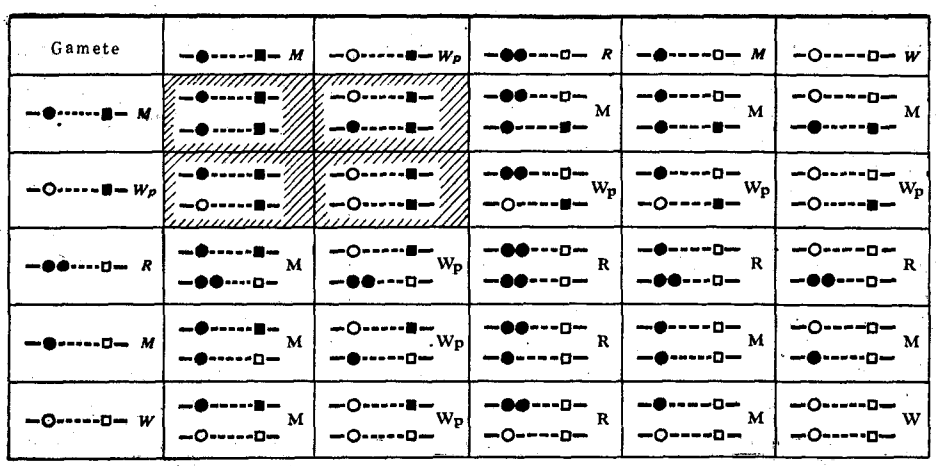

Fig. 4. Diagram showing the phenotypic effects upon the pericarp color of various types of zygotes, obtained from various gametic combinations between some of P-alleles and $\widehat{E\cdot zl}$. Of chromosomal compositions in table, those shown at the top and left are of the gamete, and the others shown at the entries are of the zygote resulting from the fertilization of gametes tabulated. Of such zygotes, in four types within squint-lined squares their phenotypic effects can not be detected because $\widehat{E\cdot zl}$ homozygote is lethal. Abbreviations and symbols used are the same as those in Fig. 3.

The genic distance from P to $E\cdot\widehat{zl}$ is of about 1.5 percent units. May it be possible that such a large section of a chromosome is derived as *"repeated loci"* by duplication of a single *"ancestral"* locus? A possible answer may be obtained in a heterochromatic knob which might have its locus on the middle region of the short arm of chromosome 1, approximately agreeing with that of the P–$E\cdot\widehat{zl}$ region. Because it is a well-known fact in *Drosophila melanogaster* that the heterochromatin is attributed to overlapping a sensitive section on a character and further is in some relation to the unstable mosaic nature (DUBININ, 1936 and DEMEREC 1940).

The mosaic pericarp composed of contrasting colors, usually red and white, may result in a somatic segregation of the genic change which produced it in mitotic tissues as well as in germinal tissues. Many investigators have reported most somatic segregations as originating from various chromosomal rearrangements in much more frequency than at first generally supposed (from JONES's review, 1941). Actually such an interpretation has been cytologically demonstrated by studies of various mosaics in maize (STADLER 1933, MCCLINTOCK 1938, 1941 a, b and c, CLARK & COPELAND 1940 and others). The specificity of mosaic pericarp, like other mosaics, will be controlled by the occurring stage and frequency of genic change in the course of ontogenesis. If the change occurs very early in development, the cell with a rearranged chromosome will have an opportunity to divide many times, and thereby, to produce the special self-colored types (non-mosaics) which can not be expected from its having genotype (see the hypothesis of chromatid segregation, p. 107). Thus, there must result in a disturbed segregation in crossing progenies. Such an unexpected type can be distinguished from the gametic change in its having nonheritable nature. In fact, the excess of W, W_p and M types observed (p. 96–100) may have had such an origin. However, all of R changes are of no importance in this view. If the rearranged chromatid segregates, to produce a genic change and so a mosaic, in latter stage of tissue development, there will arise various types of mosaic nature, viz. M_h M_m, M_s and W_s. Generally concluding, genes controlling mosaicism are merely responsible for the chromosomal rearrangement of a given gene in the section concerned. Thus, size of changed tissue in a mosaic indicates the occurring time in ontogenic course, and number of changed tissues shows frequency of genic changes.

In the present case, the changed R-plant ($R\cdot+$) is similar to that

with P^{rr} in pericarp color, but the fromer is different from the latter in having an orange cob which is recessive to P^{rr} (Fig. 2–b). The present R, for self-red pericarp and orange cob, can be newly symbolized here as "P^{ro}" to discriminate from P^{rr}, for the top dominance of *P*–allelic members. A covering effect was seen in a heterozygous combination, "P^{wr}/P^{ro}", of which the phenotype is in entire accord with that of the top dominant P^{rr}. This is a common feature of the so-called "pseudo-allelic" genes. It was supposed already that *R* should have resulted from a "direct" duplication of an original *M*-locus. There is a possibility to be considered that duplex loci (P^{ro}) will change by once more unequal crossing-overs to further different forms; an original locus in a chromatid and a complementary triplicated loci in the other chromatid. Then, the fromer will give an original mosaic-pericarp (P^{mo}), while the latter may become P^{rr}. The existance of such a "direct" triplicate form was cytogenetically demonstrated in "*Double Bar*" by STURTEVANT (1929) and BRIDGES (1936). Another possibility may be noted to explain the phenotypic difference between P^{ro} and P^{rr}. Namely, if breaks of a chromatid occur in different positions within an M–sensitive section, the produced chromatids will result in various phenotypes according to only the position difference of breakage. Consequently a direction of genic change is not "$P^{mo}\rightarrow P^{ro}\rightarrow P^{rr}$" as considered from the former possibility, but becomes either "$P^{mo}\rightarrow P^{ro}$" or "$P^{mo}\rightarrow P^{rr}$". In fact, all of 26 R-plants obtained are of P^{ro} in the phenotypic expression and thus P^{rr} change does not find in present experiments. But there is no reason in the point that R types, other than P^{or}, do not occur directly from P^{mo} at all.

It has been well-known that the rate of genic change is influenced by modifying genes; some of the modifiers change the rate in only somatic tissues (DEMEREC 1929), others in only germinal tissue (DEMEREC 1930), and still others in both tissues (EMERSON 1929, RHOADES 1941). The present data showed that modifying factors, probably genes, were closely linked with *P*, and are capable of increasing the rate of genic changes of *P*-allales, other than P^{ww}, when adjacent to P^{ww} on a chromosome in the heterozygous condition, while they are responsible for decrease of the changing rate when associated with P^{mo} or P^{ro}. This finding is similar to that of EMERSON's (1929). He reported that many modifiers closely linked with P^{ww} influence differently the mutability of P^{vv} in a heterozygote, P^{vv}/P^{ww}. He reported in 1939 further "*zl*" closely linked with *P*, which agrees with $\widehat{E\text{-}zl}$ in the present paper in its linkage

relation. $E\widehat{\cdot}zl$, like modifiers, is responsible for the genic change of P alleles too. Accordingly, all of them, such as modifiers, zl and $E\widehat{\cdot}zl$, may be of so-called "pseudo-allelic", to P as well as in some within the P alleles. RHOADES (1941) asserted a situation from a study of mosaic endosperm in maize, where an extremely stable gene, a_1 (basal recessive), becomes highly unstable when associated with a modifying gene *Dt* which is located in the heterochromatin knob terminating the short arm of chromosome 9, probably similar to the present $E\widehat{\cdot}zl$ in genetic respects. Genic change of a_1 in presence of *Dt* occurs in both germinal and somatic tissues. Germinal changes of a_1 to five higher dominant alleles were detected by him; A_1, A^{b_r}, A^{r_b}, a^{b_r} and a^s, of a which four, excepting A_1, were new genes. All of them may probably be recognized as "*pseudo-allelic*", which might have had a similar origin to P–$E\widehat{\cdot}zl$.

Summary

1. The present mosaic of pericarp variegations is controlled by two alleles, P^{mo} and $E\widehat{\cdot}zl$. $E\widehat{\cdot}zl$ is a dominant enhancer on *P*-allelic members, and thus a given allele of *P*-members when associated with $E\widehat{\cdot}zl$ on the same chromosome acts as a top dominance, independent of the dominant relation of itself. The $E\widehat{\cdot}zl$ homozygote is lethal, and further by itself, behaves as a top dominant W_p-mutant of mosaic characters when adjacent to P^{ww}.

2. Two loci, *P* and $E\widehat{\cdot}zl$, are found to be closely linked together, the map distance between them being estimated as about 1.5 percent units. The linkage sequence of $E\widehat{\cdot}zl$ is entirely in accord with that of *zl* reported by EMERSON (1939).

3. Of the present mosaic character, both the somatic variability and the disturbance of segregation can be interpreted easily by a hypothesis of the chromatid segregation (MATSUURA & SUTÔ, 1948) in course of development of the somatic tissue.

4. This mosaic character is extremely unstable, and genic changes are found to occur occasionally in both directions, from M to R or W_p and *vice versa*. Such genic change may be of the rearranged nature of a gene-locus on a chromosome—the position effect of gene—rather than the so-called point mutation. A plausible possibility of its origin was discussed as being supported by the unequal crossing-over.

5. Four types of pericarp color which have arisen from a single original M-plant; W, W_p, M and R, are different from each other in the frequency of genic change. The rate of genic change is further influenced by the presence of P^{vw} and $\widehat{E \cdot zl}$.

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Post scriptum

(A conclusion concerning the natuure of mutable genes)

While this paper was in press, an opportunity was given the present writer to read the two recently completed volumes of the *"Proc. Nat. Acad. Sci., USA."*; they are vols. 36 and 37 published in 1949 and 1950 respectively. In those volumes, several papers which are focused on the same problem in connection with the induction and occurrence of genic changes within a pseudo-allelic series as discussed in the above text were presented by the following investigators: LAUGHNAN, GREEN & GREEN, DUNN & GLUECKSOHN-SCHOENHEIMER, MCCLINTOCK and GOLDSCHMIDT.*) All of them, but one (LAUGHNAN'S paper), which are not yet cited in the text are worthy of discussion here, as they have connection with the writer's conclusions.

An interesting observation, essentially similar to those in a case

* Literature cited (Papers mentioned under the same title in the text are excluded):

1) DUNN, L. C. and S. GLUECKSOHN-SCHOENHEIMER, 1950. Repeated mutations in one area of a mouse chromosome, **36**: 233–237.

2) GOLDSCHMIDT, R. B., 1950, "Repeats" and the modern theory of the gene, **36**: 365–468.

3) GREEN, M.M. and K. C. GREEN, 1949. Crossing-over between alleles at the lozenge locus in *Drosopoila melanogaster*, **35**: 586–591.

4) MCCLINTOCK, B., 1950. The origin and behavior of mutable loci in maize, **36**: 344–355.

of *Gossypium* (STEPHENS, 1948), of maize (LAUGHNAN, 1949), and also of *Drosophila* (KOMAI & TAKAKU, 1949), was made in case of three recessive lozenge mutants composing a pseudo-allelic series, loci of which were proved to arrange in the order of "←*ct*^{1}–1.0–*sn*^{3}–3.7–[*lz*^{BS}–0.09–*lz*^{46}–0.06–*lz*^{g}] –1.5–*ras*^{1}–0.2–*v*→" on the X-chromosome in *Drosophila melanogaster* (GREEN & GREEN, 1949). Possibility of such pseudo-allelism was also suggested from the genic evidence present in a case of a balanced lethal series comprising the at least four brachyury alleles, T^{1}, t^{0}, t^{t}, t^{2}, in mouse (DUNN & GLUECKSON-SCHOENHEIMER, 1950).

According to the genic proof by GREEN & GREEN, each of three *lz*-alleles located on the one mate of X-chromosomes is always balanced by a wild allele on the other mate of homologous X-chromosomes, and when only one or two of them is located on an X-chromosome, then the remaining wild alleles behave as a dominant enhancer of *lz* located on the same chromosome; consequently the wild-typed alleles change themselves into the opposite direction in their phenotypic nature, becoming recessive to any one of *zl*-alleles. It is worthy of note that this finding is like the writer's one in the relation of *P*-alleles to $E\widehat{\cdot}zl$ as mentioned in the text, although there are some differences in details. Further, a genic change occurs whenever any two of the *lz*-alleles are located together on the same X-chromosome, always giving rise to a characteristic phenotype of the spectacle nature (*lz*^{s}-like) in every combination, clearly distinguishable from each one of the three *lz*-mutants. These *lz*^{s}-like mutants are never obtained from females homozygous for any one of them (lz^{BS}/zl^{BS} or lz^{46}/zl^{46} or zl^{g}/zl^{g}) but they certainly tend to result from heterozygous females for any two of *lz*-alleles (lz^{BS}/lz^{46} or lz^{BS}/lz^{g} or lz^{46}/lz^{g}). It was concluded therefore that the occurrence of such a genic change (a *zl*^{s}-like mutant) is evidently due to a simple association of two pseudo-allelic mutant-loci through the equal crossing-over, rather than due to the duplication of a single locus through the unequal crossing-over as observed in some cases in *Drosophila* (STURTEVANT 1925, OLIVER 1940, OLIVER & GREEN 1944, and LEWIS 1945). This fact may be considered as a genetic evidence to support the stability of gene, because it is merely a genic interaction due to such a gene combination as rarely occurs in a pseudo-allelic series.

As shown in several cases in plants and in animals, as pointed out in the text, it is clear that the position effect of gene resulting from various chromosomal rearrangements should cause a genic change of

the gene itself giving rise to the so-called "mutation of gene". The duplication and deficiency induced by the unequal crossing-over within a given minute region of the chromosome was established to be one of the most reasonable ways to account for the origin of the pseudo-allelism of genes. There is still another case in which a chromosome type of the "breakage-fusion-bridge cycle" in maize causes such a genic change (McCLINTOCK, 1950). The degree of mutability (or instability) of a given gene may be in proportion to the relative frequency of such chromosomal rearrangements. McCLINTOCK concluded that the occurrence of them causes a stickiness of the heterochromatic substance possibly existing as the so-called "chromosomal knob". If such a substance is inserted at a given region of a chromosome, then stable genes adjacent to this inserted heterochromatin become mutable in consequence of its inducing chromosomal rearrangements. On the other hand, removal of the heterochromatin results in restoration of the gene stability. The mutability of a gene is influenced not only by either the presence or the absence of the heterochromatin but also by specific changes either in the state or in the dosage of it. The latter changes are often accmpanied by a specific change of stickiness and consequently are reflected either in an increase or in a decrease in the relative frequency of a gene mutation. Actually, McCLINTOCK has succeeded in the experimental induction of heterochromatic loci on chromosome 9 (designated by her as A_c and D_s) which have a genetic ability to activate the given stable genes (*c*, *wx* and a_1) to their wild-typed alleles.

The same conclusion as pointed out already rests upon the writer's assumption, based on genetical data, as to a mosaic pericarp in maize. That is: (1) The genic change of P^{mo} results from the duplication and deficiency of a P^{mo}–locus depending on an unequal crossing-over which occurs at any point between P^{mo} and $\widehat{E{\cdot}zl}$. (2) One of the changed alleles is designated as P^{ro}, a form of two P^{mo} alleles located together on a chromosome. It seems logical to represent the transferred allele as lying closely to the right side of an original allele. The duplicated loci composing P^{ro} are separated by an equal crossing-over, resulting in recovary of P^{mo}. (3) The other one is of the W_p type of mosaic pericarp conditioned by $\widehat{E{\cdot}zl}$, adjacent to the right side of *P*–locus, and is a deficient form of the *P*–locus. $\widehat{E{\cdot}zl}$ is able to change itself into one of *P*–allelic members if it is translocated to the position of *P*–locus in chromosome 1 by an unequal brossing-over; this suggests that $\widehat{E{\cdot}zl}$

is of the same origin as *P*–pseudo-allelic members. Actually, P^{mo} or P^{ro} is found infrequently as a reversible type of the genic change in selfing populations of the deficient W_p–mutants. (4) Such genic changes of *P*–pseudo-alleles are always activated by the presence of $E\widehat{\cdot}zl$ in a nucleus. It is very interesting to note that the genetical findings obtained from $E\widehat{\cdot}zl$ are very similar to those from McCLINTOCK's A_c. In so far as the present work concerning the genic change of *P* was carried out, it is impossible to answer a suspected subject on the obtained W_p–mutant, whether the wild-typed locus is *de novo* formed at a deficient region of chromosome to be iso-allelic for each of duplicated loci, the same as in a lozenge case observed by GREEN & GREEN, or, if so, whether such new locus is related to the heterochromatin of chromosome.

GOLDSCHMIDT (1950) stated that the real units of the so-called "genes" in the classic genetical concept are sections of different size containing one or more bands in the salivary chromosome, the minimum unit of which is a single band. A larger section is composed of a number of identical bands which might have arisen from a single band through the duplication under the presence of a specific heterochromatin such as D_s, A_c and probably $E\widehat{\cdot}zl$. Further, the repeated reduplication of an ancestral band may be induced by chromosomal events, such as unequal crossing-over, breakage-fusion-bridge cycle and some other structural changes of chromosome, causing formation of the larger section. This phenomenon of the reduplication of a locus is often accompanied by the characteristic phenotype different from the original one, appearing to indicate a genic change. Indeed, this seems reasonable as an interpretation of the occurrence of the pseudo-allelism of genes. Although the large section (the *"repeat"* as termed by BRIDGES) behaves in genic respects as a unit also, the crossing-over occurs at each locus within a section according to the regular MENDELIAN basis. The more the reduplication of an original locus, the larger is the size of a section (LEWIS, 1945), the maximum extent of which is not yet known. Accordingly, there is an increase in number of pseudo-allelic loci in parallel with the size of a section induced. In fact, many loci in a section have been proved as probably belonging to a pseudo-allelic series in several cases, such as over 20 alleles govering elytral pattern in ladybird beetle (HOSHINO, 1943), 22 alleles of *R* in maize (STADIER & FOGEL, 1945), 6 alleles controlling the miniature and dusky character in *Drosophila Virilis* (KOMAI & TAKAKU, 1949), 10 alleles

of *T* in mouse (DUNN & GLUECKSOHN-SCHOENHEIMER, 1950), and so on. If this interpretation is correct, then the new section as a type of the newly differentiated non-allelic gene may be reasonably possible to induce when beyond the maximum limit of section.

The allelic nature of mutant genes may be separable into three categories according to the degree of gene differentiation: in two of them a section in formation is represented as carrying more than one allele composing a pseudo-allelic series; in the remaining one, each of the duplicated loci is *de novo* differentiated to be independent and non-allelic in inherited manner. Consequently, one can understand the relation of different types of sections (different genes) to each other. The pseudo-alleles which fall into the first category are so incomplete in the genic differentiation as to behave as if their phenotypic expressions were iso-allelic when heterozygous for any two of them. Such pseudo-allelic nature might be cited in several cases although their occurrence is very rare, if ever, under natural conditions. They are probably: (1) P^{mo}–P^{ro}–$E\widehat{zl}$ and A^b–A^d in maize, (2) lz^{BS}–lz^{16}–lz^9, m^3–m^4–m^7–m^{10}–dy^1–dy^4 and *S*–*ast* in *Drosophila* and (3) T–t^0–t^1–t^2 in mouse. However, the second category is of an intermediate type in genic respects between the first and third ones. This is characterized by representing the top-dominant nature in the heterozygotic combinations between any two of the pseudo-alleles. Most of them may belong to this category, a detailed review of which was made by KOMAI (1950). The genic nature of P^{ro}–P^{rcr} in a *P* pseudo-allelic series, for example, is a case in point. A series of experiments as described in this paper suggests a type of investigation which may throw light upon genic differentiation as a source of new variation for organic evolution. One of the most reliable mechanisms of genic differentiation will be understandable by assuming the unequal crossing-over to occur between the given loci in a section concerned.

Addendum: From the "FISHER and YATES: *Statistical tables*, 3rd ed. (1949)" the writer has recently been awake to the STEVENS' method of estimating the statistical significance of differences existing among mutution frequencies of a given gene in genetically different stocks, which is very high in statistical accuracy as compared with the customary method of χ^2 analysis as described in the text.

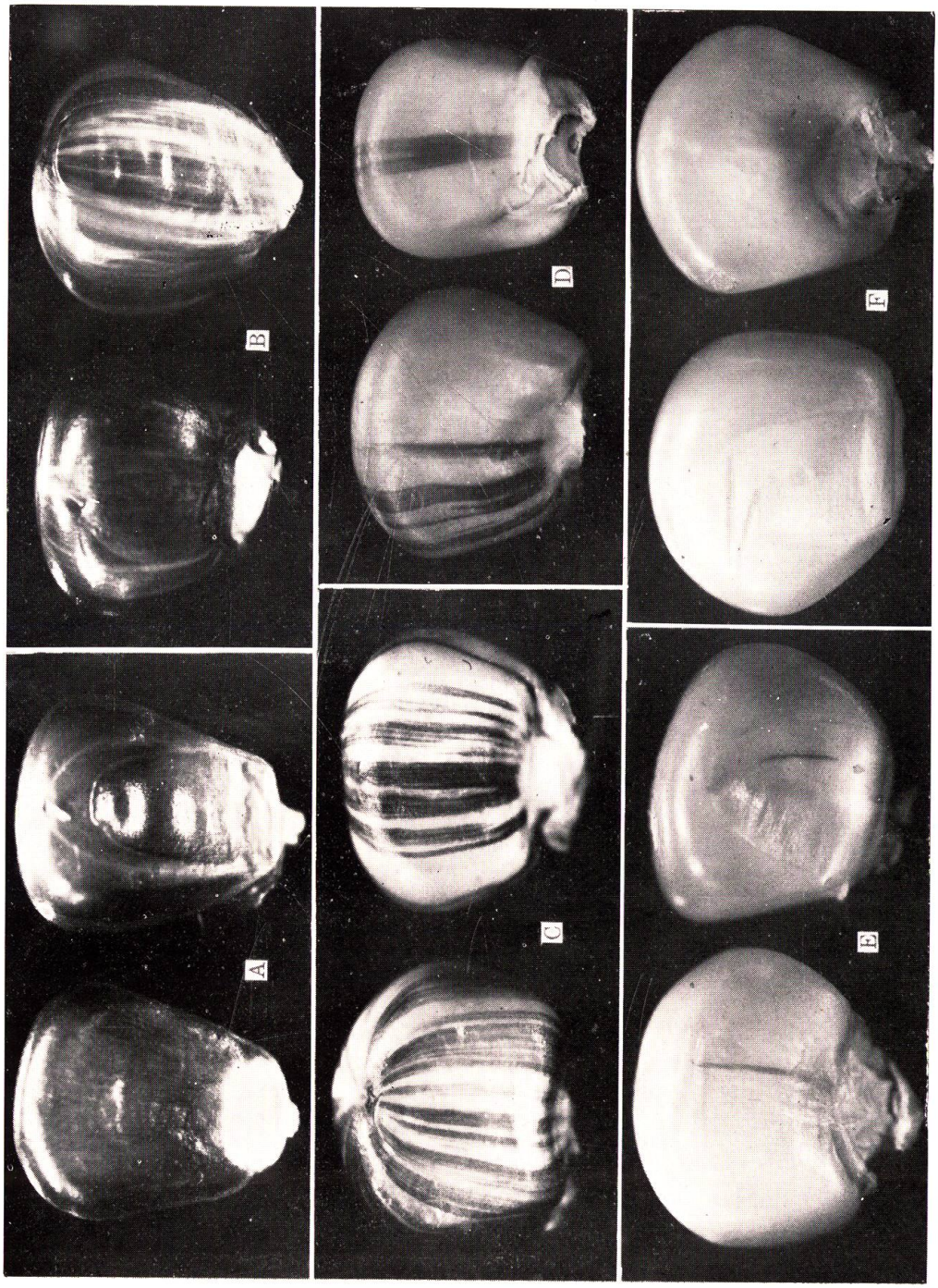
Now, according to an assumption that such the difference between

any two of genotypes resulting from various combiantions of *P*-members and $E\widehat{\cdot zl}$ is without effect upon the mutation concerned, the expected number of mutants in each of the given genotypes can be estimated from an average mutation rate, $0.57\pm0.02\%$. Then, an attempt was made to be determined by the use of STEVENS' table whether the observed number in each genotype is beyond or between the upper and lower limits of the expected number at the 5% level of probability.

The results showed that most of such the differences tested are not-significant statistically. But, only one of them associated with a genotype ($W\cdot E\widehat{\cdot zl}/W\cdot +$) was barely significant. In fact, the observed number is 9, while its expected number is 3.3. The latter is therefore about one-third as high in number as the former, this being beyond both the limits (0.6 and 8.7) of expectation at the P=0.05 level, but not the P=0.01 level. It seems reasonable that this finding is in parallel with that based upon the χ^2 analysis in which, of various genotypes tested for the mutation rate, the "$W\cdot E\widehat{\cdot zl}/W\cdot +$" showed the highest mutation rate, 1.52 ± 0.5 % as a crossover unit, the difference between 1.52 and 0.57 being significant.

Explanation of Plate XI

Types of mosaic kernels selected from variegated ears. They are;
A=self-red (R), B=heavy striped (M_h), C=medium striped (M_m),
D=slight striped (M_s), E=nearly colorless (W_p), and F=colorless (W).
All the photographs were taken by the aid of Zeiss Microplanar. Magnification ca. × 3.



T. Sutô; Genetic analysis of a mosaic pericarp in maize

Appendix-Tables, from 14 to 27

TABLE 14

Selfing populations from seventeen strains breeding true for the mosaic pericarp pattern ($M \cdot \widehat{E \cdot zl}/M \cdot +$), and F_1 populations from such four mosaic ears cross-polinated by colorless inbred plants

Phase	Year observed	Parent		Progenies			
		Pedigrees	Phenotype	R	M	W	Total
Selfing	1944	*M-1193-6*	M_s	—	16	—	*16*
		M-2003-9	M_m	—	2	—	*2*
		M-2005-5	M_m	—	8	—	*8*
		Subtotal	*3*	—	*26*	—	*26*
	1945	*M-120*	M_m	—	57	—	*57*
		M-1191-1	M_s	—	12	—	*12*
		M-1191-2	$W_p(M_s?)$	—	34	—	*34*
		M-1192-3	M_m	—	21	—	*21*
		M-1193-5	M_s	—	23	—	*23*
		M-1193-6	M_m	—	28	—	*28*
		M-1194-3	M_s	—	67	—	*67*
		M-1194-5	M_s	—	5	—	*5*
		M-1525-5-1	M_m	—	23	—	*23*
		M-1721-1	M_s	—	16	—	*16*
		M-1723-4	M_m	—	7	—	*7*
		M-2003-3	M_m	—	3	—	*3*
		M-2004-12	M_m	—	23	—	*23*
		M-2007-17	M_m	—	13	—	*13*
		Subtotal	*14*	—	*332*	—	*332*
		Total	*17*	—	*358*	—	*358*
$F_1(MO \times WR)$	1945	*M-1191-1-1*	M_s	—	28	—	*28*
		M-1192-3-2	M_m	—	26	—	*26*
		M-1193-6-1	M_m	—	19	—	*19*
		M-2003-3-1	M_m	—	28	—	*28*
		Total	*4*	—	*101*	—	*101*

TABLE 15

Selfing populations from thirteen heterozygous mosaic strains ($M \cdot \widehat{E \cdot zl}/W \cdot +$), and an F_1 population from a mosaic ear of the cross between such a mosaic plant and a colorless inbred plant ($M \cdot \widehat{E \cdot zl}/W \cdot + \times W \cdot +/W \cdot +$)

Phase	Year observed	Parent		Progenies			
		Pedigrees	Phenotype	R	M	W	Total
Selfing	1944	*M-120*	M_m	—	32	21	*53*
		M-123	M_m	—	4	4	*8*
		M-1192-2	M_s	—	16	4	*20*
		M-1193-7	M_h	—	12	7	*19*
		M-1201	M_h	—	26	13	*39*
		M-1262	M_h	—	36	18	*54*
		M-1493-6	M_m	—	6	8	*14*
		M-1492-3	M_s	—	19	3	*22*
		M-2001-1	M_m	—	4	2	*6*
		M-2001-4	M_m	—	9	3	*12*
		M-2003-3	M_m	—	7	3	*10*
		M-2003-7	M_m	—	10	5	*15*
		M-2005-13	M_m	—	11	1	*12*
		Total	*13*	—	*192*	*92*	*284*
$F_1(MO \times WR)$*	1945	*M-1193-6-2*	M_m	—	21	23	*44*

* Capital italic letters composed of two spellings tabulated, *MO*, *WR* and *RR*, are an abbreviation for each of *P*-alleles which is P^{mo}, P^{wr} and P^{rr} respectively.

TABLE 16

Selfing populations from seven and eleven ears belonging to two types of the mosaic heterozygosity respectively; one for colorless and the other for self-red (*M*·+/*W*·+ and *M*·$\widehat{E}$·*zl*/*R*·+), and F populations from four mosaic ears of the latter type cross-pollinated by colorless inbred plants (*W*·+)

Phase	Year observed	Parent		Progenies			
		Pedigrees	Phenotype	R	M	W	Total
Selfing-1	1944	*M-1*	Mm	5	9	11	*25*
		M-2	Mm	8	3	6	*17*
		M-1191-1	Ms	4	18	1	*23*
		M-1193-6	Mm	5	14	2	*21*
		M-1261-1	Mm	1	10	7	*18*
		M-1262-5	Mm	2	4	11	*17*
		M-1361-4	Ms	2	6	12	*20*
		Total	*7*	*27*	*64*	*50*	*141*
Selfing-2	1944	*M-1192-3*	Mh	9	11	—	*20*
		M-1525-5	Ms	5	15	—	*20*
		M-1721-1	Ms	4	11	—	*15*
		M-1723-3	Mm	5	8	—	*13*
		M-1723-4	Mm	6	10	—	*16*
		M-2001-2	Mm	3	4	—	*7*
		Subtotal	*6*	*32*	*59*	—	*91*
	1945	*M-1192-3-7*	Mh	1	3	—	*4*
		M-1525-2	Mm	7	11	—	*18*
		M-1525-5	Mm	5	9	—	*14*
		M-1721-1-1	Mm	4	11	—	*15*
		M-1721-1-2	Mm	7	17	—	*24*
		Subtotal	*5*	*24*	*51*	—	*75*
		Total	*11*	*56*	*110*	—	*166*
F_1(*MO*×*WR*)	1945	*M-1721-1-1*	Mm	15	10	—	*25*
		M-1723-3-2	Mm	10	12	—	*22*
		M-1723-4-1	Mh	9	10	—	*19*
		M-1723-4-2	Mh	38	32	—	*70*
		Total	*4*	*72*	*64*	—	*136*

TABLE 17

Three different populations with a self-red character of the pericarp color; selfing data (1) from eleven heterozygous self-red ears ($R\cdot+/W\cdot+$) and (2) from four homozygous self-red ears ($R\cdot+/R\cdot+$), and also (3) F_1 data from the latter eight self-red ears cross-pollinated by colorless inbred plants ($W\cdot+$)

Phase	Year observed	Parent		Progenies			
		Pedigrees	Phenotype	R	M	W	Total
Selfing-1	1944	*M-1201-6*	R	9	2	3	*14*
		M-1363-1	R	10	—	4	*14*
		M-1363-2	R	9	—	6	*15*
		M-1363-3	R	4	—	1	*5*
		M-1492-7	R	13	—	4	*17*
		M-1525-6	R	11	—	3	*14*
		M-1525-7	R	13	1	3	*17*
		M-1734-5	R	13	—	6	*19*
		M-2002-6	R	3	—	1	*4*
		M-2004-11	R	17	—	9	*26*
		M-2007-10	R	7	—	2	*9*
		Total	*11*	*109*	*3*	*42*	*154*
Selfing-2	1945	*M-1172-1-1*	R	6	—	—	*6*
		M-1172-1-2	R	7	—	—	*7*
		M-1192-3-1	R	3	—	—	*3*
		M-1192-3-2	R	2	—	—	*2*
		Total	*4*	*18*	—	—	*18*
F_1 ($RR\times WR$)	1945	*M-1172-1-1-2*	R	13	—	—	*13*
		M-1172-1-1-3	R	10	—	—	*10*
		M-1172-1-3	R	19	—	—	*19*
		M-1122-3-4	R	24	—	—	*24*
		M-1192-3-1-1	R	57	—	—	*57*
		M-1192-3-1-2	R	13	—	—	*13*
		M-1192-3-1-3	R	27	—	—	*27*
		M-1192-3-2	R	74	—	—	*74*
		Total	*8*	*237*	—	—	*237*

TABLE 18

Selfing populations from the two types of heterozygous very light mosaic ears ($W\cdot E\cdot zl/W\cdot +$ and $W\cdot E\cdot zl/M\cdot +$), and an F_1 population from an ear of the latter cross-pollinated by colorless inbred plants ($W\cdot +$)

Phase	Year observed	Parent		Progenies			
		Pedigrees	Phenotype	R	M	W	Total
Selfing-1	1944	*M-120*	W_p	1	—	28	*29*
		M-1361-5	W_p	—	—	14	*14*
		M-1363	W_p	—	—	35	*35*
		M-1492-2	W_p	—	—	17	*17*
		M-1521	W_p	—	—	22	*22*
		M-1522	W_p	—	—	16	*16*
		M-1523	W_p	1	—	54	*55*
		M-1733-2	W_p	—	1	11	*12*
		M-2005	W_p	—	—	22	*22*
		Total	*9*	*2*	*1*	*219*	*222*
Selfing-2	1944	*M-1361-4*	W_p	—	10	11	*21*
		M-1363-5	W_p	—	9	10	*19*
		M-1363-7	W_p	—	4	11	*15*
		M-1493-3	W_p	—	18	28	*46*
		M-1733-6	W_p	—	3	53	*56*
		M-2005-5	W_p	—	4	6	*10*
		Total	*6*	—	*48*	*119*	*167*
$F_1(W_p \times WR)$	1945	*M-1493-3*	W_p	—	35	4	*39*

TABLE 19

Selfing populations from four homozygous mosaic strains ($M \cdot E \cdot \widehat{zl}/M \cdot +$)

Year observed	Parent: Pedigrees	Parent: Phenotype	Progenies: R	Progenies: M	Progenies: W	Progenies: Total
1948	*M-120-17-2-M m*	M n	—	19	—	*19*
	M-120-18-1-M s	Mm	—	29	—	*29*
		Mm	—	7	—	*7*
	M-1363-118-M m	Mm	1	29	2	*32*
	Subtotal *3*	*4*	*1*	*84*	*2*	*87*
1949	*M-120-17-2-M m*	Ms	—	5	—	*5*
	M-120-18-1-M m	Mm	—	4	—	*4*
	M-1363-118-M m	Mh	—	19	—	*19*
		Mm	—	15	—	*15*
		Mm	—	24	—	*24*
		Ms	—	9	—	*9*
		Ms	—	12	—	*12*
	M-1193-6-2-M m	Mh	—	24	—	*24*
	Subtotal *4*	*8*	—	*112*	—	*112*
1950	*M-120-18-1-M m*	Mh	—	26	—	*26*
		Mm	—	30	—	*30*
		Mm	—	8	—	*8*
	M-1363-118-M m	Mm	—	35	—	*35*
		Mm	—	38	1	*39*
	M-120-18-1-M m × *M-1363-118-M m*	Mm	—	37	1	*38*
		Mm	—	39	1	*40*
	M-1193-6-2-M h	Mm	—	40	1	*41*
		Mm	—	23	—	*23*
		Mm	—	63	1	*64*
	Subtotal *3*	*10*	—	*339*	*5*	*344*
Grandtotal	*4*	*22*	*1*	*535*	*7*	*543*

TABLE 20

F_2 and F_3 populations, from seven F_1 mosaic ears ($M \cdot \widehat{E \cdot zl}/W \cdot +$) of the crosses between heterozygous mosaic ($M \cdot \widehat{E \cdot zl}/W \cdot +$) and colorless plants ($W \cdot +$), and from their thirty-three F_2 mosaic ears ($M \cdot \widehat{E \cdot zl}/W \cdot +$) respectively

Phase	Year observed	Parent: Pedigrees	Parent: Phenotypes	Progenies: R	Progenies: M	Progenies: W	Progenies: Total
F_2	1948	*M-120-18-1-M*$_s$	M$_m$	—	6	2	*8*
			M$_m$	—	9	4	*13*
			M$_s$	—	16	8	*24*
		M-1193-6-2-M$_m$	M$_s$	—	9	3	*12*
		Subtotal	*2* *4*	—	*40*	*17*	*57*
	1949	*M-120-18-1-M*$_m$	M$_h$	—	5	2	*7*
			M$_h$	—	9	4	*13*
		M-1363-118-M$_m$	M$_s$	—	20	7	*27*
		Subtotal	*2* *3*	—	*34*	*13*	*47*
	Total		*3* *7*	—	*74*	*30*	*104*
F_3	1950	*M-120-18-1-M*$_h$	M$_h$	—	19	6	*25*
			M$_h$	—	33	14	*47*
			M$_h$	1	18	8	*27*
			M$_h$	1	14	11	*26*
			M$_m$	—	30	16	*46*
			M$_m$	—	43	17	*60*
			M$_m$	—	10	7	*17*
			M$_m$	—	13	9	*22*
			M$_m$	—	10	6	*16*
			M$_m$	—	10	4	*14*
			M$_s$	—	16	8	*24*
			M$_s$	—	43	41	*84*
			M$_s$	—	41	43	*84*
		Subtotal	*1* *13*	*2*	*300*	*190*	*492*

Phase	Year observed	Parent: Pedigrees	Parent: Phenotypes	Progenies: R	M	W	Total
F_3	1950	*M-1193-6-2-M m*	Mh	2	60	34	*96*
			Mm	—	42	22	*64*
			Mm	—	69	29	*98*
			Mm	—	25	13	*38*
			Ms	—	28	18	*46*
			Ms	—	36	26	*62*
		Subtotal	*1*	*6*	*2*	*260*	*142*	*404*
		M-1363-118-M m	Mh	—	24	4	*28*
			Mh	—	3	2	*5*
			Mh	—	15	3	*18*
			Mh	—	53	23	*76*
			Mm	—	20	11	*31*
			Mm	—	38	11	*49*
			Mm	—	44	19	*63*
			Mm	—	52	22	*74*
			Ms	—	8	3	*11*
			Ms	1	122	46	*169*
			Ms	—	84	38	*122*
			Ms	—	77	47	*124*
			Ms	1	81	43	*125*
			Ms	—	70	32	*102*
		Subtotal	*1*	*14*	*2*	*691*	*304*	*997*
	Total		*3*	*33*	*6*	*1251*	*636*	*1893*
Grandtotal	*3*		*3*	*40*	*6*	*1325*	*666*	*1997*

TABLE 21

F_1 populations from twenty-six ears of the reciprocal crosses between homozygous mosaic ($M \cdot E \cdot \widehat{zl}/M \cdot +$) and colorless inbred ($W \cdot +$) palnts

Phase	Year observed	Parent: Pedigrees		Parent: Phenotype	Progenies: R	M	W	Total
MO×WR	1948	*M-1193-6-1*		M_s	—	104	1	*105*
		M-120-17-2		M_m	—	24	—	*24*
				M_m	—	27	—	*27*
				M_m	—	20	—	*20*
		M-120-18-1		M_h	—	89	—	*89*
				M_s	—	100	2	*102*
		Subtotal	*2*	*6*	—	*364*	*3*	*367*
	1949	*M-120-17-2*		M_m	—	8	—	*8*
				M_m	—	5	—	*5*
		M-120-18-1		M_m	—	19	—	*19*
				M_s	—	26	—	*26*
		Subtotal	*2*	*4*	—	*58*	—	*58*
	1950	*M-1363-118*		M_h	—	37	—	*37*
				M_m	—	59	1	*60*
				M_m	—	76	1	*77*
				M_m	—	14	—	*14*
				M_m	—	73	—	*73*
				M_m	—	28	—	*28*
				M_m	—	46	—	*46*
				M_s	—	19	—	*19*
		M-120-18-1		M_h	—	26	—	*26*
				M_m	—	8	—	*8*
		Subtotal	*2*	*10*	—	*386*	*2*	*388*
	Total		*4*	*20*	—	*808*	*5*	*813*
WR×MO	1949	*M-1363-118-*	W_p	W	—	27	—	*27*
			W_p	W	—	90	—	*90*
			M_s	W	—	41	—	*41*
			M_m	W	—	90	—	*90*
	1950	*M-1363-118-*	M_s	W	—	24	1	*25*
			M_m	W	—	20	1	*21*
	Total		*1*	*6*	—	*292*	*2*	*294*
Grandtotal	*2*		*4*	*26*	—	*1100*	*7*	*1107*

TABLE 22

Backcrossed populations from fourteen colorless ears ($W \cdot +$) pollinated by F_1 heterozygous mosaic plants ($M \cdot \widehat{E \cdot zl}/W \cdot +$), which were obtained from the crosses between heterozygous mosaic and colorless inbred plants: $W \cdot + \times M \cdot \widehat{E \cdot zl}/W \cdot +$

Year observed	Parent: Pedigrees	Parent: Phenotype	Progenies: R	Progenies: M	Progenies: W	Progenies: Total
1949	*M-120-18-1-M*$_h$	W	—	3	2	*5*
		W	—	5	6	*11*
		W	—	28	26	*54*
		W	—	9	11	*20*
		Subtotal *4*	—	*45*	*45*	*90*
	M-1363-118-2-M$_m$	W	—	3	4	*7*
		W	—	9	9	*18*
		Subtotal *2*	—	*12*	*13*	*25*
	M-1193-6-2-M$_m$	W	—	12	11	*23*
	Total *3*	*7*	—	*69*	*69*	*138*
1950	*M-120-18-1-M*$_h$	W	—	27	16	*43*
		W	—	55	58	*113*
		W	—	24	42	*66*
		W	—	10	22	*32*
		W	—	22	25	*47*
		Subtotal *5*	—	*138*	*163*	*301*
	M-1363-118-2-M$_m$	W	—	50	35	*85*
		W	—	43	40	*83*
		Subtotal *2*	—	*93*	*75*	*168*
	Total *2*	*7*	—	*231*	*238*	*469*
Grandtotal *2*	*5*	*14*	—	*300*	*307*	*607*

TABLE 23

Backcrossed popultions from different classes of kernels in nineteen mosaic ears of F_1 culture, in which heterozygous mosaic ($M \cdot E \cdot zl/W \cdot +$) and colorless inbred ($W \cdot +$) strains were used as the parent in the cross, further pollinated by colorless inbred plants (1949 data)

Parent		W			W_p			M_s			M_m			M_h			Subtotal			Total
Pedigrees	Phenotype	R	M	W	R	M	W	R	M	W	R	M	W	R	M	W	R	M	W	
M-1193-6-1-M_m	M_s	—	13	15				—	12	8							—	25	23	*48*
	M_m										—	105	97				—	105	97	*202*
	M_w										1	50	70				1	50	70	*121*
	Subtotal *3*	*—*	*13*	*15*				*—*	*12*	*8*	*1*	*155*	*167*				*1*	*180*	*190*	*371*
M-2005-5-3-M_m	M_s	—	47	44	—	12	5	—	16	17							—	75	66	*141*
	M_m	—	7	7				2	8	17	—	20	25	—	21	16	2	56	65	*123*
	M_h										—	6	10	—	19	14	—	25	24	*49*
	Subtotal *3*	*—*	*54*	*51*	*—*	*12*	*5*	*2*	*24*	*34*	*—*	*26*	*35*	*—*	*40*	*30*	*2*	*156*	*155*	*313*
M-1363-18-1-M_h	M_h										—	8	9	—	14	11	—	22	20	*42*
	M_h							—	3	4	—	10	8	—	12	8	—	25	20	*45*
	M_h										—	20	16	—	5	5	—	25	21	*46*
	M_h										—	11	6	—	11	10	—	22	16	*38*
	M_h													—	6	5	—	6	5	*11*
	M_h													1	38	36	1	38	36	*75*
	M_h													—	4	6	—	4	6	*10*
	Subtotal *7*							*—*	*3*	*4*	*—*	*49*	*39*	*1*	*90*	*81*	*1*	*142*	*124*	*267*
M-120-118-2-M_m	M_s	—	9	10	—	8	6										—	17	16	*33*
	M_s	—	1	1	—	5	6	—	3	4							—	9	11	*20*
	M_m	—	4	3	—	2	3	—	5	6	—	12	11	—	5	4	—	28	27	*55*
	M_m	—	5	5				—	5	4	—	4	5	—	5	5	—	19	19	*38*
	M_m				—	5	2	—	16	7	—	12	16	—	13	11	—	46	36	*82*
	M_m										—	9	12	—	8	14	—	17	26	*43*
	Subtotal *6*	*—*	*19*	*19*	*—*	*20*	*17*	*—*	*29*	*21*	*—*	*37*	*44*	*—*	*31*	*34*	*—*	*136*	*135*	*271*
Total	4 *19*	*—*	*86*	*85*	*—*	*32*	*22*	*2*	*68*	*67*	*1*	*267*	*285*	*1*	*161*	*145*	*4*	*614*	*604*	*1222*

TABLE 24

Back crossed populations from twenty-nine heterozygous mosaic ears ($M \cdot E \cdot \widehat{zl}/W \cdot +$) of the F_1 culture, which were crossed heterozygous mosaic strains with the colorless inbred ones, further pollinated by colorless inbred plants (1950 data)

Parent		Progenies			
Pedigrees	Phenotype	R	M	W	Total
M-120-18-1-M_h	M_h	—	16	11	*27*
	M_h	—	24	25	*49*
	M_h	—	6	6	*12*
	M_h	—	25	17	*42*
	M_h	—	35	27	*62*
	M_m	—	30	27	*57*
	M_n	—	11	10	*21*
	M_s	—	17	15	*32*
	M_s	—	45	62	*107*
	M_s	—	28	52	*80*
	M_s	—	16	13	*29*
	M_s	—	59	72	*131*
	M_s	—	41	43	*84*
	M_s	—	31	28	*59*
	M_s	—	19	38	*57*
	M_s	—	9	20	*29*
	M_s	—	19	29	*48*
	M_s	1	17	36	*54*
	M_s	2	33	11	*46*
	Subtotal	*19*	*3*	*481*	*582*	*1026*
M-1363-118-2-M_n	M_h	—	8	8	*16*
	M_h	1	45	50	*96*
	M_h	—	47	48	*95*
	M_h	—	23	32	*55*
	M_h	—	63	56	*119*
	M_h	—	20	8	*28*
	M_h	2	73	37	*112*
	M_h	—	45	49	*94*
	M_m	—	48	60	*108*
	M_m	—	21	23	*44*
	M_m	—	91	26	*117*
	M_n	—	12	8	*20*
	M_m	—	35	49	*84*
	M_m	—	31	63	*94*
	M_m	—	34	50	*84*
	Subtotal	15	*3*	*596*	*567*	*1165*
M-1193-6-2-M_m	M_h	—	16	13	*29*
	M_h	1	5	5	*11*
	M_m	—	9	19	*28*
	M_m	—	20	18	*38*
	M_s	—	16	12	*28*
	Subtotal	5	*1*	*66*	*67*	*134*
Total	3		*29*	*7*	*1143*	*1216*	*2366*

TABLE 25

Double-crossed populations from fifteen ears of reciprocal crosses between the F_1 heterozygous mosaic plants ($M \cdot E \cdot zl/W \cdot +$) from heterozygous mosaics pollinated by colorless and the heterozygous self-red plants ($R \cdot +/W \cdot +$) from homozygous self-reds pollinated by colorless; ($M \cdot E \cdot zl/W \cdot + \times R \cdot +/W \cdot +$)

Year observed	Parent: Crossing pedigrees	Parent: Phenotype	Progenies: R	M	W	Total
1949	(*M-120-18-1-M_h* × *WW*) × (*M-1525-6-14-R* × *WW*)	M_h	4	16	18	*38*
		M_m	25	29	9	*63*
	(*M-120-17-1-M_m* × *WW*) × (*M-1525-6-14-R* × *WW*)	M_m	10	27	9	*46*
	(*M-120 18-1-M_s* × *WW*) × (*M-1525-6-14-R* × *WW*)	M_s	15	32	30	*77*
	(*M-1193-6-23-M_m* × *WW*) × (*M-1525-6-14-R* × *WW*)	M_h	5	7	11	*23*
		M_m	25	27	15	*67*
	Subtotal *4*	*6*	*84*	*138*	*92*	*314*
	(*M-1525-6-14-R* × *WW*) × (*M-2005-5-3-M_s* × *WW*)	R	3	7	7	*14*
	(*M-1525-6-14-R* × *WW*) × (*M-1193-6-2-M_m* × *WW*)	R	7	35	17	*59*
		R	2	12	6	*20*
		R	3	10	—	*13*
		R	4	—	2	*6*
	Subtotal *2*	*5*	*19*	*64*	*32*	*115*
1950	(*M-1363-118-M_m* × *WW*) × (*M-1363-118-R* × *WW*)	M_h	12	80	22	*114*
		M_h	13	27	15	*55*
		M_m	7	44	12	*63*
	Subtotal *1*	*3*	*32*	*151*	*49*	*232*
	(*RR* × *WW*) × (*M-1363-118-M_m* × *WW*)	R	38	14	16	*68*
Total	*8*	*15*	*173*	*367*	*189*	*729*

TABLE 26.

F_1 and F_2 populations from both types of crosses, one between self-red and colorless, and the other between self-red and mosaics, and also populations from back-crossing of such both types of F_1 parents with the colorless inbred strains

Phase	Genotype	Year observed	Parent: Pedigrees	Parent: Phenotype	Progenies: R	Progenies: M	Progenies: W	Progenies: Total
1, F_1*	*W·+/W·+×R·+/W·+*	1948	*WW×M-1525-6-14-R*	W	23	—	21	*44*
				W	37	—	38	*75*
			Subtotal *1*	*2*	*60*	—	*59*	*119*
2, F_1*	*R·+/W·+×W·+/W·+*	1948	*M-1525-6-14-R×WW*	R	11	—	10	*21*
3, F_1	*R·+/R·+×W·+/W·+*	1948	*M-1525-6-14-R×WW*	R	18	—	—	*18*
4, F_1	*M·Ê·zl/W·+×R·+/R·+*	1949	*M-1193-6-1-M_m×M-1363-118-R*	M_{lt}	7	7	—	*14*
5, F_2	*R·+/W·+*	1949	*M-1525-6-14-R/W*	R	41	—	14	*55*
				R	12	—	5	*17*
			Subtotal *1*	*2*	*53*	—	*19*	*72*
		1950	*M-1525-6-14-R/W*	R	24	—	7	*31*
				R	23	—	6	*29*
			M-1193-6-1-M_m/M-1363-118-R	R	33	—	12	*45*
				R	36	—	18	*54*
				R	5	—	1	*6*
				R	39	—	9	*48*
			Subtotal *2*	*6*	*160*	—	*53*	*213*
	Total	*2*	*2*	*8*	*213*	—	*72*	*285*

6, F_2	$M\cdot\widehat{E\cdot zl}/R\cdot+$	1950	*M-120-18-1M$_m$/M-1525-6-14-R*		M_h	4	17	—	*21*
					M_h	15	27	—	*42*
					M_h	9	30	—	*39*
			M-1193-6-1-M$_m$/M-1363-118-R		M_h	13	40	—	*53*
			*M-1363-118-R/RR***		M_h	7	15	—	*22*
					M_h	1	4	—	*5*
					M_h	12	25	—	*37*
					M_m	34	75	—	*109*
	Total	*1*		*3*	*8*	*95*	*233*	—	*328*
7, B*	$R\cdot+/W\cdot+\times W\cdot+/W\cdot+$	1949	*M-1525-14-R/W×WW*		R	64	—	75	*139*
					R	4	1	3	*8*
			Subtota	*1*	*2*	*68*	*1*	*78*	*147*
		1950	$\frac{M\text{-}1193\text{-}6\text{-}1\text{-}M_m}{M\text{-}1363\text{-}118\text{-}R}\times WW$		R	25	—	30	*55*
					R	15	1	10	*26*
					R	30	—	31	*61*
			Subtotal	*1*	*3*	*70*	*1*	*71*	*142*
	Total	*2*		*2*	*5*	*138*	*2*	*149*	*289*
8, B	$M\cdot\widehat{E\cdot zl}/R\cdot+\times W\cdot+/W\cdot+$	1950	$\frac{M\text{-}1193\text{-}6\text{-}1\text{-}M_m}{M\text{-}1363\text{-}118\text{-}R}\times WW$		M_n	12	12	—	*24*

* Concerning the same genotype of those combinations of crossing, an observed $R:M:W$ ratio should be expected to be summed up as 209, 2 and 218 respectively in a total of 429 plants. ** A genic change of R into M.

TABLE 27

Selfing populations from seven and thirteen heterozygous very light mosaic ears having the genic constitution of $W \cdot \widehat{E \cdot zl}/M \cdot +$ and of $W \cdot \widehat{E \cdot zl}/W \cdot +$, respectively

Phase	Year observed	Parent: Pedigrees	Parent: Phenotype	R	Progenies: M	Progenies: W	Progenies: Total
Selfing-1	1948	*M-120-18-1-M_s*	W_p *	—	7	15	*22*
		M-120-18-1-M_m	W_p *	—	21	39	*60*
		M-1193-6-1-M_m	W_p *	—	6	22	*28*
		M-2005-5-3-W_p	W_p	—	10	18	*28*
			W_p	—	10	29	*39*
		Subtotal *4*	*5*	—	*54*	*123*	*177*
	1950	*M-120-18-1-W_p*	W_p	—	17	24	*41*
			W_p	—	22	55	*77*
		Subtotal *1*	*2*	—	*93*	*79*	*118*
	Total	*4*	*7*	—	*39*	*202*	*295*
Selfing-2	1948	*M-2005-5-3-W_p*	W_p	—	1	90	*91*
			W_p	—	1	30	*31*
			W_p	—	—	13	*13*
			W_p	—	—	49	*49*
		Subtotal *1*	*4*	—	*2*	*182*	*184*
	1949	*M-2005-5-3-W_p*	W_p	—	—	71	*71*
			W_p	—	—	23	*23*
			W_p	—	1	89	*90*
			W_p	—	2	30	*82*
		Subtotal *1*	*4*	—	*3*	*213*	*216*
	1950	*M-120-18-1-M_s*	W_p *	—	—	43	*43*
			W_p *	—	—	45	*45*
		M-1193-6-1-M_m	W_p *	—	—	98	*98*
			W_p *	—	—	42	*42*
			W_p *	—	1	36	*37*
		Subtotal *2*	*5*	—	*1*	*264*	*265*
	Total	*3*	*13*	—	*6*	*659*	*665*

* Genic changes of M into W_p.