MUTABLE a_1 OF THE En SYSTEM IN MAIZE[1]*

PETER A. PETERSON

Agronomy Department, Iowa State University, Ames, Iowa

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MUTABLE genes in maize are under the control of identifiable components that exhibit an unusual behavior. These components control the loci with which they are associated in that the action of a particular locus becomes altered when the component is intimately associated with it.

Characteristics of mutable genes include variable rates of mutability which are heritable and can be recognized as distinct pattern types, specificity between members of a mutable system, diverse pathways of mutation and the transposition of mutable gene components from one position to another in the genome. It is hypothesized that normal gene action is inhibited by controlling elements (*Ds*, McCLINTOCK 1951; *Mp*, BRINK and NILAN 1952; or *I*, PETERSON 1960a) when they are located in the vicinity of the affected locus. In certain cases the presence of a second element (such as *En*) is necessary before any expression of mutability can be discerned; i.e., in the absence of the second element, these inhibited loci appear to be stable recessives (Figure 2h).

The components of a mutable system exhibit a high degree of specificity. This is evident in the relationship between *Ac* and *Ds* in one system and *En* and *I* in another system. In addition to these, there are other two-element systems of mutability control such as a_1^{dt}–*Dt* (RHOADES 1941; NUFFER 1955) and a_1^{m-1}–*Spm* (McCLINTOCK 1956). The activators (*Ac*, *Dt*, *Spm*, *En*) do not affect any but their own specific inhibiting component (except in the case noted below) even though the same locus may be involved, such as the a_1 locus on chromosome 3. Each system is highly specific. For example, in tests of the interrelations of systems it was found that *En* does not cause the instability of McCLINTOCK's *Ds* component. There is, however, an interrelationship between *Mp* and *Ds; Mp* affects *Ds* instability (BARCLAY and BRINK 1954) and *Ac* affects P^{vv} mutability (BRINK 1958a).

Another characteristic of mutable gene systems is that the mutable components may move from one location to another, a feature which McCLINTOCK termed transposition. This is recognized in mutable gene stocks where stable standard dominant alleles at other loci have been observed to have become mutable (McCLINTOCK 1951, 1953). In such a case, the problem is to determine if these new mutables respond to the components of the specific system in whose presence

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they arose. If this proves to be the case, such evidence would strongly suggest transposition—the transfer of mutable gene components from a mutating locus to a stable one.

In this paper, a new mutable which arose in a pale green mutable stock (*I–En* system) is described. In addition, it will be demonstrated that it responds to the components of the Enhancer system. Abstracts of this material have been reported (PETERSON 1957, 1960b).

MATERIALS AND METHODS

The new mutable, a_1^m, arose in a pale green mutable stock. The mutability of a_1^m is recognized by the mutation of the a_1 allele to A_1 (colorless to colored—purple or red depending on the *pr* locus) and appears in both sporophyte and endosperm tissue. The a_1^m allele may also change to a nonmutating colorless allele. It was originally recognized in the field in 1952 as a sectored tassel. One fourth of the tassel was fully colored (reddish-purple) and the rest of the tassel had anthers with a colorless background and small reddish-purple stripes (Figure 1). Outcrosses were made with pollen from this tassel.

The genetic constitution of this original plant with reference to the a_1 locus was A_1/a_1^{dt}. A_1 is a full color allele and a_1^{dt} mutates to A_1 in the presence of *Dt*. *Dt*, however, was absent. It was verified by further tests that the A_1 allele and not the a_1^{dt} had mutated to a_1^m. The source of this particular A_1 allele was an unidentified midwestern inbred line.

The original mutable pattern was designated $a_1^{m(dense)}$ due to the dense appearance of the pattern of mutability (Figures 2b and 3a). Patterns of mutability are determined by the time (manifest in size of mutant area) and frequency (manifest in number of mutant areas) of mutation events (Table 1). The dense pattern type is intermediate in timing and possesses a high rate of mutability. From this original dense pattern other patterns showing a change in frequency (high or low) and timing (late or early) of the mutation events were recovered (Figure 3). Some of the kernels showing the diverse expression possible with a_1^m are shown in Figure 2. These patterns are based on inherent differences in the allele itself and are not due to modifiers, since in successive outcrosses (5–6 generations), a particular pattern shows a consistency in pattern type (Figure 3).

In addition to the above mentioned exceptional pattern types, a number of colorless kernels also appeared. Although all colorless kernels appear similar, genetic differences were demonstrated with appropriate tests. The origin of these colorless kernels as well as those showing pattern changes is not necessarily associated with a meiotic event since frequent somatic changes are observed (Figure 4). Germinal mutations to purple and intermediate pigmentation levels (pales) also occur.

The pales arising from a_1^m are generally lighter in color than the A^d pale alleles described by LAUGHNAN (1952). Although they are members of the same allelic series at the A_1 locus, they seem to have a different origin. The A^d pale allele

FIGURE 1.—Colored stripes on a colorless background of anthers of mutable plants showing the mutation from a_1^m to A_1.

arises as a result of a crossover event (LAUGHNAN 1960). The pales of the a_1^m series are observed as somatic mutation and the germinal mutations are not necessarily associated with crossover events. Studies on the interaction of these allelic forms are in progress.

In order to expedite the detection and isolation of changes in the parent pattern, the mutable allele was almost universally carried in the heterozygous form with $a_1^{dt}sh_2$ and testcrossed by this parent. These two genes, a_1^{dt}, which is colorless and mutates in the presence of *Dt* and sh_2, which results in a shrunken kernel, are .25 units apart. Thus, the a_1^{dt} allele is inherited with the sh_2 allele except for crossovers (1/800) appearing as colorless nonshrunken ($a_1^{dt}Sh_2$).

In the following crosses, reference to mutable or colorless kernels denotes only the nonshrunken (Sh_2) kernels unless otherwise specified.

TABLE 1

Designation of symbols

$a_1^{m(dense)}$ = a very dense mutable pattern: mutates from colorless to color (purple or red, depending on *pr*)

Derivative patterns

first letter designates the timing of the mutation event

f = a *fine* pattern, late occurring mutation

e = early occurring

i = intermediate

second letter designates rate

h = high

m = medium

l = low

for example: $a_1^{m(f.m.)}$—late occurring, medium frequency mutation

$a_1^{m(r)}$ = colorless form that responds to *En*

$a_1^{m(nr)}$ = colorless form that does not respond to *En*

En = mutable component necessary for mutability to occur: appears at a_1 locus and by transposition becomes located at an independent position

I = mutable component adjacent to mutable locus which is hypothesized to inhibit gene activity

I–En = two components of a specific system (contrast to other systems such as a_1–*Dt*, *Spm*, *Ac–Ds*)

Independent *En*—*En* in a chromosome position independent of the a_1 *locus* (with the $a_1^{m(r)}$ allele gives typical one mutable to one nonmutable when in heterozygous state)

Autonomous *En*—*En* at a_1 locus—(in backcross tests gives almost all mutables due to its presence adjacent to locus)

a_1^{dt}—colorless aleurone mutates to A_1 (colored aleurone) in presence of *Dt*; in text, referred to as a_1

Testcross—cross of the heterozygous type by the homozygous recessive

RESULTS AND DISCUSSION

The mutable allele here described has a dense pattern which is characterized by a very high rate of mutability (Figures 2b and 3a). Three types of changes are recovered in the nonshrunken (Sh_2) progeny of testcrosses of this allele—$a_1^{m(dense)}Sh_2/a_1^{dt}sh_2 \times a_1^{dt}sh_2/a_1^{dt}sh_2$. These include kernel changes to fully colored and colorless, in addition to many permanent and heritable pattern alterations. An array of pattern types is recognized; these are due to differences in timing and frequency of mutation events (Figures 2 and 3 and Table 1). In addition to these pattern exceptions, colorless types appeared in a high frequency (Figure 3a). Two representative families that arose from testcrosses of this dense allele (out of more than 300 families) tested over a 5-year period are shown in Table 2.

Differences in the stability of individually isolated dense-mutable alleles can be observed between families 1958 492 and 1958 500 listed in Table 2. Although these two families represent pattern types that are phenotypically similar and

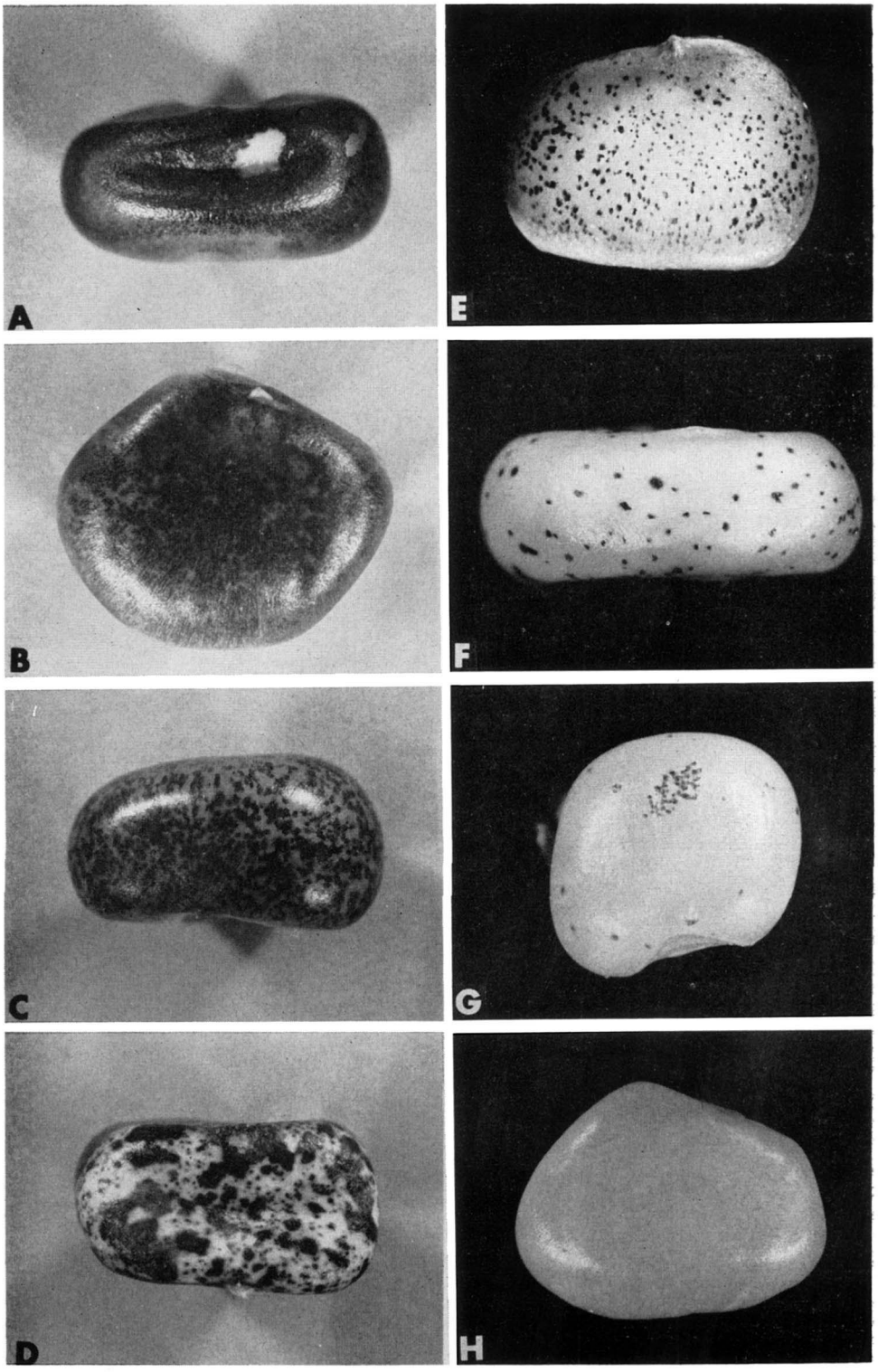

FIGURE 2.—Various mutable patterns of a_1^m (see Table 1). (A) very dense—universally recognized by the colorless sector; (B) dense; (C) f.h.; (D) i.m.; (E) f.m.; (F) f.l.; (G) low type with a reversion to f.h.; (H) colorless—$a_1^{m(r)}$ or $a_1^{m(nr)}$ depending on its response to *En*.

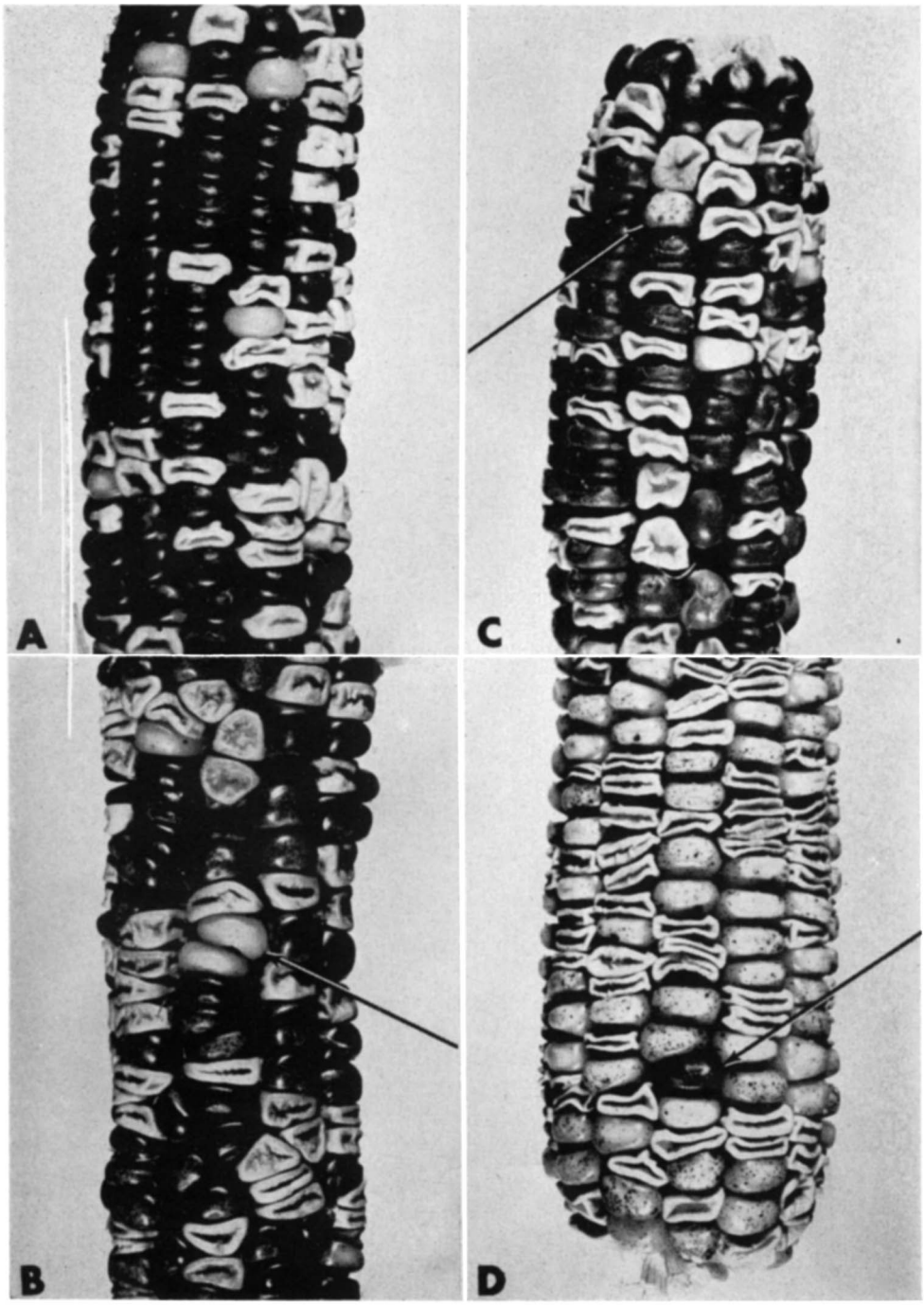

FIGURE 3.—The origin of pattern differences. These exceptions to the parental pattern are heritable; arrows indicate some of the exceptions. (A) dense with colorless exceptions. These exceptions are almost universally $a_1^{m(nr)}$ type. (B) $a_1^{m(f.h.)}$ with 4 $a_1^{m(f.l.)}$ exceptions (C) dense with a coarse exception. (D) a dense derivative in $a_1^{m(f.m.)}$.

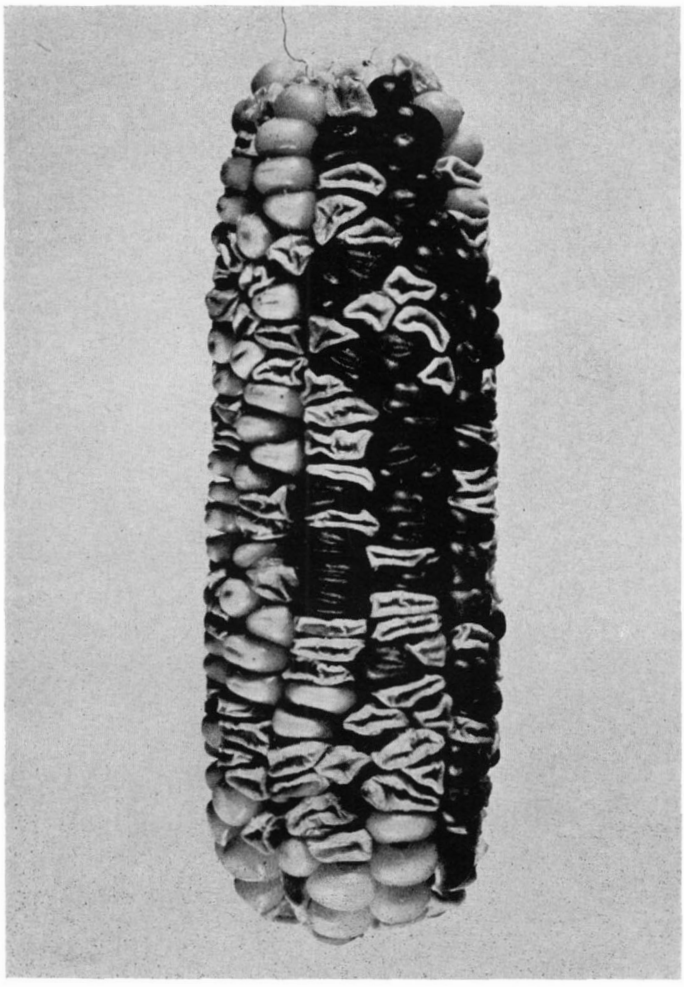

FIGURE 4.—Somatic change from a dense type mutable, $a_1^{m(dense)}$, to a stable, nonmutating allele $a_1^{m(nr)}$. The cross $a_1^{m(dense)}Sh_2/a_1^{dt}sh_2 \times a_1^{dt}sh_2/a_1^{dt}sh_2$. The dots seen on some of the kernels are the result of the action of *Dt* on the a_1^{dt} allele.

have a common origin, differences are nevertheless observed in the frequency of occurrence of colorless types arising from testcrosses. In 1958 492, the frequency of colorless types averaged 14.54 percent, while in 1958 500, it was 8.29 percent. Although there is variation in the frequency of colorless types among individual plants of a family, a common trend within a family does exist. Such a difference between families indicates that a similarity in pattern does not necessarily imply a similarity in mutational behavior.

Mutability is autonomously controlled in these original mutable alleles; i.e., in outcrosses and testcrosses most of the derived nonshrunken (Sh_2) progeny are mutable (Tables 2 and 3). This means that the mutability controller is closely

TABLE 2

The occurrence of colorless types and pattern exceptions in testcross progenies of individual ears from two dense mutable families (1958 492 and 500) arising from the cross* $a_1^{m(dense)}Sh_2/a_1sh_2 \times a_1sh_2/a_1sh_2$

1958 Source	Dense mutable	Colorless exceptions	Pattern exceptions	Total	Percent colorless exceptions
492–1	114	16	1 f.m.	131	12.21
–2	64	11	2 pale	77	14.28
–3	166	15	0	181	8.29
–4	80	18	0	98	18.37
–5	125	33	1 f.m.	159	20.75†
–6	179	43	0	222	19.37
–7	63	7	0	70	10.00
–8	150	21	0	171	12.28
–9	185	38	1 f.m.	224	16.96
–10	130	18	0	148	12.17
–11	133	23	0	156	14.74
–12	176	20	0	196	10.20
–13	76	18	0	94	19.15
–14	81	14	0	95	14.74
Average					14.54
500–1	135	2	0	137	1.46
–2	60	8	0	68	11.76
–3	152	26	1 f.m.	179	14.52
–4	111	10	1 f.m.	122	8.20
–5	98	7	0	106	6.60
–6	39	5	0	44	11.36
–7	149	18	0	167	10.78
–8	139	3	0	143	2.10
–9	81	7	1 f.m.	89	7.86
Average					8.29

* Only nonshrunken (Sh_2) kernels are considered.
† Used as 1959 56 (Table 3).

associated with the a_1 locus. This apparent inherent control of mutability by the locus itself is referred to as autonomous type control (autonomous *En*). From the autonomous type, an independent control of mutability is derived.

In this case the mutability controller is not closely associated with the mutant locus but is on another chromosome (as is true for *Dt* with reference to a_1^{dt} and *En* with *pg*) assorting independently of the mutant locus in question. Kernels lacking this controller would be colorless (designated $a_1^{m(r)}$ and discussed later). This type of control was found in some of the derivatives of the dense allele and is observed in testcrosses where one half of the nonshrunken (Sh_2) kernels are mutable and one half colorless (Table 4). Therefore from a controller that was originally located at or near the a_1 locus, an independently inherited controller arises.

The identity of the independent controller of mutability: Since from the auton-

TABLE 3

Tests for En *to verify* $a_1^{m(nr)}$ *types arising from the cross* $a_1^{m}Sh_2/a_1sh_2 \times a_1sh_2/a_1sh_2$*

Crossed × a_1sh_2	Pattern	Segregation a_1^m:cl	Percent cl	Tested as 1959	Carries *En* In a_1sh_2 sibs	Carries *En* In cl	Response of cl types to a known *En*
7 99B–2	dense	39:11	22.0	3	0	+	—
7 101B–3	dense	119:15	11.2	4	0	+	—
7 155B–1	dense	171:18	9.5	13	—	+	0
7 160A–6	dense	125:28	18.3	14	0	+	0
7 166B	dense	150:28	15.7	15	0	+	0
7 171B	dense	69:21	23.3	16	0	0	—
7 177A	dense	89:14	13.6	17	0	+	0
8 410–3	dense	79:15	15.9	30	0	+	0
7 291–1	dense	60:34	36.17	31	—	+	—
7 291–5	dense	78:9	10.3	33	+	+	—
7 292–5	dense	77:39	33.6	34	+	+	0
7 294–13	dense	149:22	12.9	35	0	+	—
8 410–1	dense	166:12	6.7	43	+	+	—
8 418–3	dense sectoring	135:26	9.9	45	0	+	—
8 448–4	dense	75:16	17.6	51	0	+	0
8 470–3	dense	175:20	10.3	55	+	0	0
8 492–5	dense	125:33	20.9	56	0	0	--
8 492–9	dense	185:38	17.0	57	+	0	—
8 501–2	dense	101:30	22.9	58	+	0	—
8 448–6	dense	120:19	13.7	59	+	0	—
8 503–6	dense	107:36	25.2	60	+	0	—
8 502–6	dense	204:31	13.2	60	+	+	0

* Only nonshrunken (Sh_2) kernels considered in ratios. Mutability did not occur in any tests of the a_1sh_2/a_1sh_2 derivatives with the sib colorless types.
0 This test not completed.
— Negative test.
+ Positive test.
cl Colorless as in Figure 2h and 3a.

TABLE 4

Progeny of typical testcrosses of mutable plants carrying independent En *showing the segregation of mutable to colorless types: the cross* $a_1^{m(r)}Sh_2/a_1sh_2$ En/+ × a_1sh_2/a_1sh_2*

1959	No. of mutable	No. of colorless	Exceptions	Total	Percent cl
297–1	59	68	0	127	53.54
–2	55	53	0	108	49.07
–3	98	102	0	200	51.00
–4	110	103	0	213	48.36
306–1	64	59	0	123	47.97
–2	67	80	0	147	54.42
–3	60	69	0	129	45.73
–4	75	59	0	134	44.03
–5	65	59	0	124	47.58
–6	73	75	0	148	50.67
–7	58	63	0	121	52.07

* Only nonshrunken (Sh_2) kernels considered.

omous control of mutability an independent control is derived, the factor controlling mutability must be transposed from one location to another position in the genome. The independent controller-type is recognized by a testcross ($a_1^m Sh_2/a_1 sh_2 \times a_1 sh_2/a_1 sh_2$) ratio of 1 mutable nonshrunken:1 colorless nonshrunken (Table 4). The independent factor controlling mutability is present in the mutable (a_1^m) kernels and absent in the colorless ones. It would then follow that among the $a_1 sh_2/a_1 sh_2$ segregants from the above cross, one half possess the independent factor following random assortment. When a number of these $a_1 sh_2/a_1 sh_2$ segregants from testcrosses of independent types are tested against colorless kernels—$a_1^{m(r)}$—that are known to respond in the presence of the independent factor, mutability results on the ears in approximately half of the crosses. This indicates the existence in these $a_1 sh_2/a_1 sh_2$ segregants of a controlling factor assorting independently.

Is this independent controller identical to the Enhancer (*En*) of the *pg* system (PETERSON 1960a)? It may be recalled that *En* causes the pale green stable allele to become mutable. The following evidence demonstrates that the controller of a_1^m mutability is *En*.

To demonstrate the relationship between a_1^m and pg^m, it is necessary to prove that the independent controller which causes the derived colorless form to become mutable also causes the pale green stable to become mutable. (The symbols associated with the particular alleles in the cross are described in Table 1.)

The results show that *pg* mutability is associated with mutability of the colorless allele ($a_1^{m(r)}$—Table 1) and conversely the *pg* allele does not become mutable when the $a_1^{m(r)}$ allele remains immutable (Table 5). Since the factor controlling *pg* mutability is *En*, the factor causing a_1^m mutability must also be *En*. These correlative tests are a confirmation that the new mutable does indeed belong to the *En* system. In retrospect, one can say that a previously dominant allele (A_1) is changed to a mutable form, a_1^m, by the transposition of mutable components from the *Pg* locus to the A_1 locus. These two mutables respond to the same specific elements (*I* and *En*). They have no relation to other systems such as *Ac–Ds* or *a*$_1$*–Dt*. The $a_1^{m(r)}$ allele which becomes mutable in the presence of *En* does not respond to *Dt* nor to *Ac*; in the presence of *Dt* and without *En*, the kernels are stable and colorless. Demonstration of the relationship between a_1^m and pg^m seems proof that the transposition of the elements of the *En* system, *I* and *En*, lead to the origin of a new mutable.

Autonomous mutability: It was originally hypothesized that the dense allele was autonomous. In view of the high frequency of colorless types, consideration should be given to the possibility that this allele is not autonomous but that these colorless types arise from the independent assortment of more than one *En*. It may be recalled that the presence of *En* is necessary for the colorless form to become mutable and, in addition, it is known that more than one *En* may exist in the genome. If, for example, three *En* elements were assorting independently in a testcross ($En_1/+, En_2/+, En_3/+$), one eighth of the resulting progeny would lack *En* and be nonmutable. Such a rate (12 percent) is very similar to the rate

TABLE 5

The test of similarity in response of $a_1^{m(r)}$ *and* pg^s *to* En. *The cross*
$+/pg^sA_1A_1 \times Pg/Pg\ a_1sh_2/a_1sh_2$
Two kinds of F_1 plants.*
One half $Pg/pg^s\ A_1Sh_2/a_1sh_2$ no mutable factor.
One half $Pg/pg^s\ A_1Sh_2/a_1sh_2$ with mutable factor.
F_1 selfed and tested on $a_1^{m(r)}$.

Source	Total no. of ears	Gave a_1 mutable in progeny of ⊗		Did not give a_1 mutable in progeny of ⊗	
		Segregates pg^m and pg^s	Segregates Only pg^s	Segregates pg^m and pg^s	Segregates Only pg^s
1958	.	.	.	.	.
1370	8	4	0	0	4
1371	4	1	0	0	3
1372	3	2	1†	0	0
1373	12	8	0	0	4
1959	. .	.	.	.	.
1040	20	9	0	0	11
		24	1	0	22

* Only the F_1 plants with pg^s are shown, since only these are relevant to the experiment.
† Only exception to simultaneous induction of instability in pg^s and $a_1^{m(r)}$. In all other cases, the occurrence of pg^m is correlated with a_1 mutability.
⊗ Self-pollination.

of stables appearing among the testcross progeny of the autonomous alleles (Tables 2 and 3). Crosses were made to try to resolve this problem.

The autonomous alleles in crosses have always been carried as heterozygotes with a^{dt} and sh_2 on the homologous chromosome in the form $a_1^{m(dense)}Sh_2/a_1^{dt}sh_2$. It was previously noted that a_1 and sh_2 are so closely linked that colorless crossovers (a_1Sh_2) appear at the rate of 1/800 in testcrosses. Under the assumption that the high rate of colorless forms is based on many *En* assorting independently, one could then test for the presence of *En* in the a_1sh_2 segregants. The presence of *En* is detected by testing the a_1sh_2 segregants as well as the colorless exceptional types originating from the cross $a_1^{m(dense)}Sh_2/a_1sh_2 \times a_1sh_2/a_1sh_2$. This was done by crossing these two types, a_1sh_2 segregants and colorless forms, by $a_1^{m(r)}$. If mutability is observed in these tests, *En* must be present. If only colorless forms appear, then *En* is absent. Progenies from the two crosses involving this a_1sh_2 and colorless Sh_2 segregants were examined.

In one type of cross, that between the shrunken segregants and their colorless sibs from testcrosses of assorted mutable alleles (Table 2), mutable progeny were not obtained. This indicates that the colorless types were not due to the segregation of *En* and must arise from mutation events. In crosses of these same a_1sh_2 segregants with a known $a_1^{m(r)}$ mutability resulted, but mutability also resulted when the colorless sibs were tested in this same manner; therefore *En* must be present. These colorless forms were then tested by crossing them with a known *En* stock. In these tests, *En* again was not effective since mutability did not result.

It is obvious from these tests that the colorless forms are not mutable in the presence of *En*. Such results indicate that these colorless forms derived as exceptional types from testcrosses of the dense mutable alleles are nonresponsive to *En* and therefore are designated $a_1^{m(nr)}$. This allele would correspond to a^{dl} originating from a_1^{dt} in *Dt* stocks (RHOADES 1941). A similar nonmutable allele was found in the mutable pericarp series. This stable type possesses the mutability controller, Modulator, at the *P* locus (BRINK 1958a). In the *Ac–Ds* system, similar nonresponding *bz* derivatives are obtained (MCCLINTOCK 1956).

It has been shown that the high rate of incidence of colorless forms among the testcrosses of autonomous alleles is due to mutation. Although *En* is carried in multiple in these stocks, the colorless forms do not result from the independent assortment of *En* since they are not mutable when *En* is present.

In addition, this experiment has uncovered a second type of colorless allele, $a_1^{m(nr)}$, which does not respond in the presence of *En*. This colorless allele, $a_1^{m(nr)}$, is differentiated from the other colorless allele, $a_1^{m(r)}$ that does respond in the presence of *En*. (The superscript r = responds and nr = does not respond to *En*). These two alleles are then isoalleles in the original sense since they are phenotypically alike and can be differentiated only by more definitive tests (in this case, response to *En*). These are similar to isoalleles described in Neurospora (HOROWITZ and FLING 1953; WEBBER 1960) and in Drosophila (STERN and SCHAEFFER 1943) which are distinguished by temperature sensitivity of the gene product, histidine biosynthesis and by mutation sequence, respectively. There have been many reports of wild-type isoalleles which are distinguishable mainly on their differential rate of mutability (LEFEVRE 1955; GREEN 1959). A similar difference in mutability of seemingly identical alleles is evident among a number of $a_1^{m(dense)}$ alleles in their rate of change from mutable to colorless forms (Tables 2 and 3). Many of these cases of isoalleles reaffirm the notion that phenotypic expression is not the only indicator of the potential expression of the gene in question. This suggests a wide latitude in genic alterations that are subliminal and do not result in phenotypic differences. These are therefore not readily ascertained.

In a sense the origin of the $a_1^{m(nr)}$ allele reminds one of BRINK's paramutation event in that this colorless form is a permanent change caused by the presence of another element, *En*. It is distinguished from a paramutation event (BRINK 1958b) in that the change is not universal and not necessarily invariable since changes can result in altered pattern expressions, in addition to the nonmutable colorless forms. It might, however, be a form of the same kind of event.

SUMMARY

A new mutable gene at the a_1 locus (a_1^m) originated from an A_1 allele in a pale green mutable stock.

The mutability of the original a_1^m allele is autonomously controlled, i.e., the control of mutability is intimately associated with the a_1 locus. From this type an independent controller located independently of the a_1 locus has been isolated.

In addition it was found that the mutability controller of a_1^m also causes *pg* to be mutable. From these and other results, the a_1^m controller must be identical to *En* (Enhancer) of the *pg* system. This finding is indicative of a transposition event—the transfer of the mutable components of *pg* to the a_1 locus.

A wide range of patterns is derived from the original mutable pattern (dense) and these include various pigmentation types. In addition colorless types arise, some of which respond to *En*. The nonresponding type occurs at a high rate and represents an irreversible loss of color potential at this locus.

The relations between different alleles of a mutable series and isoalleles in general and paramutation events are discussed.

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