per cent.   Other experiments have been performed to determine whether
the oxygen effect is exerted by way of the initial breakage mechanism or
on the reunion process.   These consisted of comparative exposures to a
single dose of 300 r in one minute of inflorescences in a vacuum or in oxygen
with the addition or removal·of oxygen either immediately after or during
part of the irradiation period.   In addition, buds were pretreated in the
presence and absence of oxygen before exposure to X rays.   These experi-
ments show that the presence of oxygen during the actual exposure to X
rays rather than during the pre- or postirradiation period is the important
factor, thus indicating that oxygen alone does not influence the recovery
process.   It seems likely that the oxygen effect is an indirect one, resulting
from the production during irradiation in oxygen of some substance such
as hydrogen peroxide.   Although it appears probable that the effect of such
a substance on aberration frequency would result from an increased pro-
duction of chromosome breaks, the alternative possibility, that such a sub-
stance might modify the restitution process, cannot yet be excluded.

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THE ORIGIN AND BEHAVIOR OF MUTABLE LOCI IN MAIZE

BY BARBARA McCLINTOCK

DEPARTMENT OF GENETICS, CARNEGIE INSTITUTION, COLD SPRING HARBOR, NEW YORK

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In the course of an experiment designed to reveal the genic composition
of the short arm of chromosome 9, a phenomenon of rare occurrence (or
recognition) in maize began to appear with remarkably high frequencies
in the cultures.   The terms mutable genes, unstable genes, variegation,

mosaicism, mutable loci or "position-effect" have been applied to this phenomenon. Its occurrence in a wide variety of organisms has been recognized. The most extensive investigations of this phenomenon have been undertaken in *Drosophila melanogaster*.[1] In this organism, the conditions associated with the origin of genic instability have been well defined. The part played by the heterochromatic materials of the chromosomes, in inducing and controlling the type of variegation and its time and frequency of occurrence, has been established. It has not been generally recognized that the instability of genic expression in other organisms may be essentially the same as that occurring in Drosophila.

As stated above, a large number of mutable loci have recently arisen in the maize cultures and are continuing to arise anew. The loci affect variegation for many different kinds of plant characters, each locus being concerned with a particular character or occasionally several characters. Some of these loci are c, yg_2, wx, a_2, y, pyd, which are well-investigated units in maize.[2] Others involve previously unknown genetic units. The same types of genic instability appearing in the maize cultures have been described in many other organisms. The behavior of these new mutable loci in maize cannot be considered peculiar to this organism. The author believes that the mechanism underlying the phenomenon of variegation is basically the same in all organisms. The reasons for this conclusion will be made apparent in the discussion.

The initial appearance of the burst of newly arising mutable loci occurred in the progeny coming from the self-pollination of about 450 plants which had each undergone a series of events in their early development where the short arm of chromosome 9 was subjected to drastic structural modifications. These events took place during the "chromosome type" of breakage-fusion-bridge cycle.[3] The modifications that this mechanism produces are: one or more duplications of segments of the short arm, deficiencies of one or more segments of various lengths, structural modifications of the heterochromatic knob substance, duplications of the knobs with or without structural modifications, and various combinations of these several types of modifications. The chromosome complement of over 150 of these plants were examined at pachytene to determine the nature of the structural modifications that had occurred. In addition to the modifications of the short arm of chromosome 9 listed above, some of the plants had other modifications, many of which are particularly significant because they involve the substances in the chromosome that are believed to be responsible for the origin and behavior of mutable loci—the heterochromatic knobs and centromeres. Altogether, 48 such structural modifications have been analyzed, most particularly in the above-mentioned plants but also in some other plants that had received a chromosome 9 with a newly broken end. Fourteen involved modifications of chromo-

some 9 other than those listed above (telocentric chromosomes, isochromosomes, extra chromosomes 9 with particular modifications, etc.). Four arose from fusion of the centromere of chromosome 9 with the centromere of another chromosome. Four resulted from fusion of the knob substance of the short arm of chromosome 9 with the centromere of chromosome 9. Twenty-four resulted from fusions of the knob substance of the short arm of chromosome 9 with other regions in the chromosome complement: eighteen were with other knobs or with regions very close to these knobs, four were insufficiently analyzed as to the positions of the fusion, and two did not involve a known knob region. In two cases, inversions were present in other chromosomes. The regions involved were the knob and centromere in one of these chromosomes and the nucleolus organizer and the centromere in the other chromosome. There can be no question that these "spontaneous translocations" are nonrandom with respect to the location of the breaks and fusions. The heterochromatic knob and centromere regions are mainly involved.

In the cultures arising from self-pollination of the plants that had undergone the chromosome type of breakage-fusion-bridge cycle in their early development, about 40 different mutable loci were recognized. The majority of such mutable loci could not have been present in the parents of these plants, for the stocks from which they arose had been under investigation for some years without showing evidence of the presence of such a large number of unstable loci. It was concluded, therefore, that either some part of the mechanism concerned with the breakage-fusion-bridge cycle or some of the structural modifications resulting from it were responsible for conditions that produced this burst. That some of the mutable loci were located in or associated with chromosome 9 was realized in the first tests. Other mutable loci, on the other hand, did not show any obvious association with chromosome 9.

The mutable loci fall into two major classes: (1) those that require a separate activator factor for instability to be expressed, and (2) those that are autonomous with respect to the factor that controls the onset of mutability. They also may be subdivided on a quite different basis. This is related to the types of expression of the mutations that occur. The following types are present: (*a*) Changes from the mutant to, or close to, the wild-type expression. After such a mutation, the locus may be permanently stabilized. It may no longer show evidence of the instability phenomenon. (*b*) A second group, similar to (*a*) except that the mutation to wild-type does not produce stability of the locus. The wild-type-producing locus, in turn, may mutate to give the recessive expression. (*c*) A third type where the mutations give rise to a series of alleles of the affected loci. These alleles are distinguished by different degrees of quantitative expression of the normal phenotype. Most of these are

relatively stable; only rarely does instability again appear. (*d*) A fourth type, similar to (*c*). Most of the alleles, however, are not stable for they, in turn, can mutate in the direction of a higher or lower grade of quantitative expression of the phenotype. Mutable loci showing these different types of expression of mutation are found in both the major classes, that is, in the activator-requiring class and in the autonomous class.

The accumulated observations and data from a study of a number of these mutable loci are so extensive that no short account would give sufficient information to prepare the reader for an independent judgment of the nature of the phenomenon. It is realized that this is unfortunate. Manuscripts giving full accounts of some of this phenomenon are in preparation. Since this task will require much time to fulfill, the author has decided to present this short account of the general nature of the study, and the conclusions and interpretations that have been drawn. In this account only short summaries will be given of some of the pertinent information that has led to the conclusions to be presented. These conclusions are concerned with the origin of mutable loci, the events occurring at these loci that result in a change in phenotypic expression, the reasons for changes in the frequency of visible mutations at these loci, the factors controlling the time when mutations will occur, the production of mutations at the a_1 locus in maize without *Dt* being present, and heterochromatin as the probable controlling factor.

A fortunate discovery was made early in the study of the mutable loci which proved to be of singular importance in showing the kinds of events that are associated with their origin and behavior. A locus was found in the short arm of chromosome 9 at which breaks were occurring in somatic cells. The time and frequency of the breakage events occurring at this *Ds* (Dissociation) locus appeared to be the same as the time and frequency of the mutation-producing events occurring at some of the mutable loci.[4] An extensive study of the *Ds* locus has indicated the reason for this relationship and has produced the information required to interpret the events occurring at mutable loci. It has been concluded that the changed phenotypic expressions of such loci are related to changes in a chromatin element other than that composing the genes themselves, and that mutable loci arise when such chromatin is inserted adjacent to the genes that are showing the variegated expression. The events occurring to this inserted chromatin are reflected in a changed expression of the neighboring genes, or sometimes in a loss of these genes. It is the inserted material that is undergoing the "mutational" events. The *Ds* locus is composed of this kind of material.

Various types of alterations are observed as the consequence of events occurring at the *Ds* locus. Some of these alterations resemble the effects produced by x-rays, ultra-violet light, chemicals, etc. They involve

chromosome breakage and fusion. The breaks are related, however, to events occurring at this one specific locus in the chromosome—the *Ds* locus. The *Ds* designation was given to this locus because the dissociation, now known to be related to dicentric and associated acentric chromatid formation, was recognized before the other events occurring at *Ds* had been disclosed. Some of the events occurring at *Ds*, when considered without reference to all the known events, would not by themselves suggest that changed conditions at this locus are associated with a breakage-inducing phenomenon. All of them can be explained, however, by the assumption that one kind of alteration of the inserted chromatin (the chromatin of the *Ds* locus) takes place, and that the various kinds of changes observed represent consequences of this one altered condition. This condition is assumed to be a stickiness of the materials composing the *Ds* locus, which arises only at precise times in the development of a tissue. The control of the timing of this changed condition will be considered shortly. The reasons for assuming the change to be a stickiness will be obvious from the following list of known events that involve the *Ds* locus. These are: (1) Dicentric chromatid formation with fusion of sister chromatids at the location of *Ds*. This is accompanied by formation of an acentric fragment composed of the two sister segments of this arm, from *Ds* to the end of the arm. (2) Loss of detectable *Ds* activity without visible alteration of the chromosome. In some cases, the loss of *Ds* activity is presumably due to loss of the locus itself. (3) Deletions of chromatin segments of various lengths adjacent to *Ds*, usually with concomitant loss of *Ds* activity but occasionally without loss of this activity. (4) Reciprocal translocation involving chromosome 9 in which one breakage point is at *Ds*. (5) Duplications of segments of chromosome 9, inversion or ring chromosome formations involving chromosome 9 with one break at the *Ds* locus. (6) Transposition of *Ds* activity from one position to another in the chromosomal complement with or without an associated gross chromosomal rearrangement. (7) Changes at the *Ds* locus itself which result in precise changes in the relative frequency of occurrence of the above types of events in future cell and plant generations. This last event, which is of considerable importance, has been termed "change in state" of the *Ds* locus. From a study of the progression of changes in state of *Ds* through cell and plant generations, it appears that the various states may reflect the quantity of the inserted chromatin, the *Ds* loci with larger quantities of this material showing a high frequency of consequences (1), (3), (4), (5) and (6) above, and those with less of this material showing high frequencies of consequence (2) above.

It is from the transpositions of *Ds* that some of the new mutable loci may arise. The mechanism of transposition has received considerable study. Some cases of transposition of *Ds* are associated with a gross

chromosomal rearrangement. In these cases, two chromosome breaks occur to give rise to the rearrangement; one break marks the known position of *Ds* in the chromosome, before the rearrangement occurred, and the second break marks the new position of *Ds* activity. Sister chromatids are affected at each of these two positions of breakage. It has been determined for several of these cases that the appearance of *Ds* activity at the new position most probably arose at the time of origin of the gross chromosomal rearrangement. One case of transposition of *Ds* has been of particular importance because it illustrates how new mutable loci, associated with changes in genic expression, can arise. This transposed *Ds* locus appeared in a single gamete of a plant carrying chromosomes 9 with the dominant *C* allele. This gamete carried a *Ds* locus that had been transposed from a known position in the chromosome 9 to a new position in the same chromosome. The chromosome having *Ds* at this new position was morphologically normal in appearance. This new position of *Ds* corresponded to the known location of *C* (*C*, colored aleurone, dominant to *c*, colorless aleurone). All of the above-enumerated events were now occurring at this new position. Significantly, the appearance of *Ds* activity at this new location was correlated with the disappearance of the normal action of the *C* locus. The resulting phenotype was the same as that produced by the known recessive, *c*. It has been determined from previous studies that a deficiency of the *C* locus will give rise to a *c* phenotype. That the *c* phenotype in this case was associated with the appearance of *Ds* at the *C* locus, and was not due to a deficiency, was made evident because mutations at this locus from a *c* to a full *C* phenotypic expression occurred. It could be shown that when *C* action reappeared, the *Ds* action concomitantly disappeared from this locus. The restored action of *C* was permanent; no further *Ds*-type events occurred at this *C* locus. In most cases, the event giving a restored *C* action did not result in an altered morphology of chromosome 9. Loss of *Ds* activity without concomitant structural alterations of the chromosome result from event (2) above.

The other enumerated events associated with *Ds* activity were also occurring at this mutable *c* locus. The dicentric chromatid formations were not associated with the appearance of a *C* phenotype, suggesting that the inserted inhibiting material composing *Ds* may be situated proximal to the *C* locus. Several cases of transposition of *Ds* from this location to still another location in the short arm of chromosome 9 were recognized. In each case, a restored *C* action was associated with a disappearance of *Ds* activity at the *C* locus and the appearance of *Ds* activity at the new position. The changes in state of *Ds* at this mutable *c* locus (event (7) above) are particularly significant since it has been determined that a specific change in state of *Ds* is often accompanied by a specific change in the frequency of *c* to *C* mutations.

The origin and behavior of this mutable *c* locus has been interpreted as follows: Insertion of the chromatin composing *Ds* adjacent to the *C* locus is responsible for complete inhibition of the action of *C*. Removal of this foreign chromatin can occur. In many cases, the mechanism associated with this removal results in restoration of the former genic organization and action. The *Ds* material and its behavior are responsible for the origin and the expression of instability of the mutable *c* locus. The mutation-producing mechanisms involve only *Ds*. No gene mutations occur at the *C* locus; the restoration of its action is due to the removal of the inhibiting *Ds* chromatin. The possible nature of the inserted material will be considered later.

In the cultures having *Ds*, other mutable loci continue to arise. They show types of behavior similar to that described for the mutable *c* locus. This mutable *c* locus (called *c-m1* because it was the first of the mutable *c* loci isolated in these cultures) belongs to the (*a*) group of mutable loci. In some of the progeny of the original self-pollinated cultures, other mutable *c* loci have arisen from previously normal *C* loci. One of these, *c-m2*, shows the type (*c*) expression of variegation, which differs markedly from that shown by *c-m1*. A wide range of quantitative expression, for at least two different reactions associated with aleurone pigment formation, appears as the consequence of various mutations at this locus. The intermediate alleles, full wild-type alleles and some alleles showing even stronger phenotypic expressions than the wild-type from which it arose, are produced by mutations at *c-m2*. · The mutations are often expressed as twin sectors, the depth of color in one sector being greater than that in the sister sector. These twin sectors may reflect a single mutation-producing event at the *c-m2* locus that involved both sister chromatids. It has also been determined that chromosome breakage may occur at this locus.

The phenotypic expressions resulting from mutations of *c-m2* and *c-m1* are clearly quite different. That this difference may be related to differences in the inserted chromatin is suggested by the appearance of a mutable *wx* locus arising from a *Wx* locus in a gamete of a plant carrying *c-m2* (*Wx*, starch of endosperm stains blue with iodine; *wx*, recessive allele, starch stains red with iodine; located in short arm of chromosome 9, proximal to *C*). The type of variegation expressed by this mutable *wx* locus (*wx-m1*) is strikingly similar in all respects to that occurring at *c-m2*. It could not be determined in this case that transposition to the *Wx* locus of the same inhibiting substance that induced *c-m2* had occurred. Such an event is suspected from the known transposition capacities of this material.

In this report, *Ds*, *c-m1*, *c-m2* and *wx-m1* have been used as illustrations of newly arising mutable loci because all of them require an activator and

all respond to the same activator.   This activator has been designated
Ac.   Extensive studies of *Ac* have shown that it is inherited as a single
unit.   It shows, however, a very important characteristic not exhibited
in studies of the inheritance of the usual genetic factors.   This charac-
teristic is the same as that shown by *Ds*.   Transposition of *Ac* takes place
from one position in the chromosomal complement to another—very often
from one chromosome to another.   Again, as in *Ds*, changes in state may
occur at the *Ac* locus   These changes in state are of two main types:
either changes that resemble the known effects produced by different
doses of the *Ac* locus from which it was derived, or changes that result in
a decidedly altered time of action and dosage response of *Ac*.   *Ac* may be
detected and its action studied by observing the mutations occurring at
the mutable loci requiring its presence for mutability to be expressed.
It should be emphasized that when no *Ac* is present in a nucleus, no muta-
tion-producing events occur at *c-m1*, *c-m2* or *wx-m1;* nor are any chromo-
some breakage events detected at *Ds*, for no such events occur.   As an
example of this interaction it may be stated that *c-m1* has been maintained
in cultures having no *Ac* locus for several generations, and has given com-
pletely colorless aleurone with no evidence of *c* to *C* mutations.   Similarly,
the various quantitative alleles arising from mutations of *c-m2* or *wx-m1*
may be maintained without giving mutations, if *Ac* is removed from the
nucleus by appropriate crosses.   Thus a series of stable recessive mutations
or stable alleles of a mutable locus may be isolated and maintained (if the
chromosome complement is normal, see below).   When *Ac* is returned to
the nucleus, however, instability may again appear.

   The dosage action of *Ac* may be studied in the diploid plant or in the
triploid endosperm tissue of the kernels.   When marked dosage effects
are produced by a particular state of *Ac*, they are registered alike in both
the plant and the endosperm tissues; the higher the dose of *Ac*, the more
delayed is the time of occurrence of mutations at the *Ac*-controlled mutable
loci.   *Ac* determines, therefore, not only the mutation process at these
mutable loci but also the time at which the mutations occur, the different
states of *Ac* giving different times of occurrence in 1, 2 or 3 doses.   The
action of *Ac* on the mutable loci it controls has been described.   It is
believed that this action produces a stickiness of the inhibiting materials
adjacent to the affected loci.   With reference to *Ds*, the observed conse-
quences of this stickiness have been enumerated.   This physical change
probably takes place in the inserted inhibiting materials at all the *Ac*-
controlled mutable loci at the same time in the same cell.   This latter
conclusion rests on the observation that mutations occur concomitantly
at two or more *Ac*-controlled mutable loci when these are present in the
same nucleus.   The similarity in the type of inheritance and the behavior
of *Ds* and *Ac* has been indicated above.   Another similarity is that changes

in state, loss or transposition of Ac occur at the same time that changes take place at the Ac-controlled mutable loci. It would appear that the changes in the physical properties of the specific inhibiting chromatin at the mutable loci and at Ac itself are of the same nature, and that all are expressions of the primary genetic action of the material composing Ac. It is suspected that Ds and Ac are composed of the same or similar types of material. The possible composition of this material will be considered shortly.

The study of Ac and the Ac-controlled mutable loci has made it possible to interpret the many patterns of variegation exhibited by mutable loci. The variegated pattern is an expression of the time and frequency of occurrence of visible changes in the phenotype. The frequency of appearance of a visible mutation need not reflect the frequency of the events that occur at a mutable locus, as the study of c-$m1$ has clearly revealed. The visible mutations reflect only the frequency of one or several particular consequences of one primary type of event occurring to the inhibiting material adjacent to the affected gene. The changes in state of this inhibiting material that arise as one of the consequences of the primary event, lead to changes in the relative frequency of the consequences of this event when it again occurs in future cell and plant generations. Such changes in state are reflected either in increases or decreases in the relative frequency of appearance of visible mutations. The study of Ac has indicated the nature of the control of the time when the mutations will occur at these mutable loci. The different doses of Ac together with the changed states of Ac control the time of occurrence of these mutations. The changes in time of occurrence of visible mutations are thus reflections of changes in dosage or changes in state of Ac.

The mutable loci that require no activator show the same kinds of expression of variegation as do the activator-requiring mutable loci. It has been shown that the changes occurring at Ac are much the same as those occurring at Ds. Thus, Ac or Ac-like loci, could be responsible for the origin of new mutable loci when transposed to a position adjacent to a gene whose inhibited action could be detected by a visible change in phenotype. Dosage action could be exhibited by such autonomous mutable loci, as well as various "changes in state," reflected by changes in the phenotypic expression and the time and frequency of occurrence of visible mutations of the affected genes. The study of the behavior of Ds in its several states makes it possible to reinterpret the variegation patterns in Drosophila, which in some cases appear to be associated with loss of segments of chromosomes and in other cases appear to be associated with changes in the degree of action of the genes involved. It also makes it possible to interpret the reported "position-effect" in Oenothera, because the events responsible for the changes in phenotype and the appearance of

duplications and deficiencies in this organism appear to be the same or similar to those described for Ds in maize.[5]

The possible composition of Ac may now be considered. Until recently, the investigation was not focused on this problem. It is believed, however, that this material is probably heterochromatin. This statement is based, in part, on the evident homologies in the expression of variegation in maize and Drosophila, but is more convincingly suggested by the results of a preliminary experiment focused on the induction of mutations at the a_1 locus in maize when the known Dt (Dotted) locus is absent. The action of Dt in chromosome 9 on the a_1 locus in chromosome 3 is very much the same as the action of Ac on the mutable loci it controls.[6] The similarities are too great to be dismissed as being due to causally unrelated phenomena. The Dt locus activates the a_1 locus; mutations to higher A_1 alleles occur (A_1, colored aleurone; a_1, colorless aleurone, recessive to A_1). Without Dt in the nucleus, a_1 has been shown to be completely stable. Dt is located in the *heterochromatic* knob terminating the short arm of chromosome 9. The suspicion is immediately aroused: Is Dt action caused by some modification of the heterochromatic knob in chromosome 9? If so, could this modification be produced anew by subjecting a chromosome 9 to the breakage-fusion-bridge cycle? Would the effective alterations of the knob arise directly because of the induced changes, or would they be produced secondarily by some other induced structural alteration, either within the short arm of chromosome 9 or elsewhere, that would upset, in some way, the normal functioning of the knob substance and thus bring about an alteration in its action? This last question is pertinent because some of the structural alterations in Drosophila appear to affect the functioning of the centrically placed heterochromatin. For example, some of the Minutes bring about chromosome elimination and "somatic-crossingover," both of which may well be related to adhesions of specific heterochromatin that occur at certain times in development.[7] To answer the above questions, plants homozygous for a_1 and having no Dt locus (designated dt by Rhoades) were crossed by plants similarly constituted with reference to a_1 and dt but carrying a rearrangement of the short arm of chromosome 9 that would introduce a chromosome 9 with a newly broken end into many of the primary endosperm nuclei in the given cross.[8] Breakage-fusion-bridge cycles involving such a chromosome 9 with a newly broken end would occur during the development of the kernels. Some of these broken chromosomes 9 would carry a knob, and this knob could then be subjected to modifications as a consequence of the breakage events. If some of these modifications gave rise to the same conditions that were present at Dt, mutations from a_1 to A_1^* could appear in some of the kernels resulting from the cross. A large number of crosses of this type were made. The results were positive with respect to inducing mutations of a_1 to A_1. A small

number of the kernels resulting from these crosses showed mutations of a_1 to A_1. Often, only a single small A_1 spot was present on the kernel. Several of the kernels, however, had a pattern of mutations of a_1 to A_1 that was indistinguishable from that produced by Dt. These kernels could not have arisen by contamination, for stocks with the known Dt locus had never been obtained and thus no plants with this locus could have been present in the field. Furthermore, the stock having a_1 and dt, originally obtained from Rhoades, had been grown for several years. A number of sib crosses were made each year and no mutations of a_1 to A_1 were observed in the kernels on these ears.

The facts (1) that Dt is located in the heterochromatic knob of chromosome 9, (2) that the effect it produces can be recreated by subjecting chromosome 9 to the breakage-fusion-bridge cycle, (3) that Ac appeared in stocks that had undergone this cycle, and (4) that Ac and Dt are alike in their respective actions, all point to heterochromatin as the material composing Ac. The burst of new mutable loci which appeared in the self-pollinated progeny of plants that had been subjected to the chromosome type of breakage-fusion-bridge cycle becomes comprehensible if it is considered that the alterations in the quantity or structure of heterochromatic elements during this cycle were primarily responsible for the initial appearance of these mutable loci. This report has shown that, once such loci arise, other mutable loci arise through transposition of the inhibiting chromatin substances to other loci which in turn become mutable.

Why should altered heterochromatin be responsible for initiating such a chain of events? To answer this question, attention must be centered on the action of heterochromatin in the normal nucleus. That it is associated with the exchange of materials between nucleus and cytoplasm has been indicated.[9] Changes in quantity, quality or structural organization of heterochromatic elements may well alter the kind and/or degree of particular exchanges that occur, and in this way control the chromosome organization and the kind and the relative effectiveness of genic action. There can be little question that transpositions of both Ds and Ac occur and that the time of their occurrence in the development of a tissue is under precise control. This control is determined by the number of Ac loci present and their organization and possibly their position in the chromosome complement. Is this transposition of heterochromatin? Is it a reflection of a process that normally occurs in nuclei? Is it responsible for controlling the rates and types of exchange that occur between nucleus and cytoplasm? Is it usually an orderly mechanism, which is related to the control of the processes of differentiation? If so, induced disturbances in quantity and organization of the heterochromatic elements of the chromosome could give rise to a series of alterations reflected both in chromosome structure and behavior and in genic reactions that could

markedly alter phenotypic expressions.[10]   It is well known that the various knobs and centromeres may coalesce in the resting nuclei.   This coalescence is also frequently observed both in the somatic and the meiotic prophases. Are the transpositions and the changes in state of *Ac* products of this coalescence?   This is suspected because of the frequent transpositions of *Ac* from one chromosome to another.

It may be considered that these speculations with regard to heterochromatin behavior and function have been carried further than the evidence warrants.   This may be true; but it cannot be denied that one basic kind of phenomenon appears to underlie the expression of variegation in maize.   In many cases, there can be little question about the similarities in expression of variegation in Drosophila and maize.   A heterochromatic element has repeatedly been found to be basically associated with the origin and expression of variegation in Drosophila.   That a heterochromatic element likewise is responsible for the origin and behavior of variegation in maize has not been proved, although it is indicated, as the analysis of *Dt* has shown.

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[2] The symbols refer to genes affecting the parts of the plant as follows: *c*, aleurone pigment; *yg₂*, chlorophyll; *wx*, composition of starch in pollen and endosperm; *a₂*, aleurone pigment; *y*, starch composition of endosperm; *pyd*, chlorophyll.

[3] McClintock, B., Proc. Natl. Acad. Sci., **28**, 458–463 (1942).

[4] The annual reports of the author, appearing in the Yearbooks of the Carnegie Institution of Washington, **41–48**, (1942–1949), contain more detailed summaries of some of the observations that are described in this paper.

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[7] Stern, C., *Genetics*, **21**, 625–730 (1936).

[8] McClintock, B., *Ibid.*, **26**, 234–282 (1941).

[9] Vanderlyn, L., *Bot. Rev.*, **15**, 507–582 (1949).

[10] This report deals only with the origin and behavior of mutable loci arising in these cultures.   A number of other heritable changes are also arising.   Many are asscoiated with marked alterations in morphological characters.