interiors might meet the objections which Kuiper had made to the classical fission theory of Jeans.

One other point remains to be considered. What happens to the W Ursae Majoris binary if it continues dissipating mass and angular momentum through the medium of the common envelope, which in many respects would play the rôle of v. Weizsaecker's nebulosity? Perhaps what would result is precisely what v. Weizsaecker has predicted: a system of planets and a rapidly dissipating nebulosity which carries off most of the rotational momentum. If this is possible then it is perhaps not too far-fetched to think of the W Ursae Majoris systems as being the parent bodies of what we now observe as single solar-type stars without much angular momentum.

Despite its obviously bizarre appearance, this hypothesis has the attractive feature of giving us some observational evidence of precisely the kind of nebulosity which v. Weizsaecker requires. Otherwise, within the domain of main-sequence stars later than about type A, shell-like formations are not known, at least not among the single stars. Yet one would expect to observe various kinds of dilution effects in a mass of gas having the properties of the v. Weizsaecker nebulosity.

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THE ACTION OF ALLELIC FORMS OF THE GENE A IN MAIZE II. THE RELATION OF CROSSING OVER TO MUTATION OF A^b *

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Inherited changes other than those which may be regarded as true mutations of the gene occur frequently in plant and animal material. Among such changes in *Drosophila* some are associated with crossing over

between intrachromosomal duplications.[1-3] These latter may be distinguished from chromosomal aberrations of a more gross nature since the changes are localized within small regions and the duplicated segments involved carry either the same allele or factors closely related in their effects. It has been suggested that this highly localized duplication of chromatin followed by mutation and selection may constitute an important source of new genes. Further, depending on its frequency of occurrence, this phenomenon has a direct bearing on the interpretations of studies inquiring into the manner of action of genes. These studies assume, as the simplest situation, that each member of an allelic series is a single unit of action. If this assumption is maintained in those cases in which the effects of alleles are non-linear it becomes necessary to consider the action of this single unit as correspondingly more complex from a physiological viewpoint. However, if it can be demonstrated that the alleles in question are represented on the chromatin thread by separable entities it is possible to view their action according to schemes which are not so complex from the standpoint of action of the individual unit.

Evidence is presented here for the compound nature of the A^b gene, a member of the relatively extensive A series of alleles in maize. As noted in the original report,[4] in its determination of brown pericarp this allele is dominant to the wild type A, which is associated with red pigmentation. Since A itself is dominant to recessive a (brown pericarp) the dominant effect of A^b is not expected on the basis of any simple scheme representing the action of these alleles. From a study[5] of the dosage and dominance effects of certain mutants derived from A^b and designated A^d it is known that these alleles retain the distinctly antimorphic effects of the parent allele; their action is unexplained on a simple basis.

A^b mutates spontaneously with high frequency to alleles of lower level. Measured in the female germ line, Stadler[6] found a rate of 0.0005 (19: 36,661) for mutation to the A^d allele. Among 55,765 tested female gamates, no mutations of A^b to a were found. However, mutations to a occur indirectly. A^d, itself a mutant derivative of A^b, has produced 4 mutations to a (4: ca. 50,000).[7] It appears from this that either the mutation of A^b is highly directed or this gene may be compound. In the event of the latter the production of the mutant a by two successive changes, but never in a single step, is explained if it is assumed that two components of A^b are independent in their mutation.

To test the possibility that A^b is represented by closely linked determinants experiments were undertaken to determine whether there is a relation between mutation of this allele and crossing over in or near the A locus. For such an experiment it is desirable to study mutation in $A^b\ A^b$ plants heterozygous for marker genes which are closely linked and located on either side of A^b. If a choice of marker genes can be made such that only

a small segment of the chromosome lies between them, multiple exchanges in this region may be eliminated or greatly reduced. Under these conditions the frequency of strands which experience multiple exchanges in the marked segment but which appear as non-crossover products is negligible. Where the distance between marker genes is greater the efficiency of the experiment is reduced in proportion to the frequency of double crossover strands which appear as non-crossovers. Unfortunately, until very recently there were no known mutant genes showing close linkage with *A*. At the time the present experiments were begun the best choice of markers was that of lg_2 and *et*, which lie, respectively, 33 units to the left and 13 units to the right of *A* in the long arm of the third chromosome. Since the recombination value between *lg* and *et* is high (ca. 42%), it was considered that even in the event that mutations of A^b were always associated with crossing over many of the mutants would appear as non-crossovers owing to the high frequency of double exchanges between *lg* and *et*. This disadvantage may be lessened if plants of the constitution $A^b\ a$ are employed instead of the homozygotes. This substitution allows the use of *a* as a third marker gene in the experiment; instead of 42 units of recombination the values corresponding to the *lg-a* and *a-et* intervals may now be employed. Since mutants of the A^d type have never been obtained from *a*, it is certain, after suitable testing, that those which occur in these experiments have originated from A^b. Moreover the A^d mutant seeds which are pale in appearance are distinguishable from both the deep purple seeds associated with A^b and the colorless phenotype associated with *a*.

The data reported here are derived mainly from one type of cross: $A^b/lg\ a\ et \times lg\ a\ et$. Plants having these constitutions were grown at Princeton in the summers of 1947 and 1948. In order to avoid the occurrence of pale seeds due to contaminating pollen grains the plants were started and reached the flowering stage earlier than any other families in the field; moreover the mass pollination technique was discarded in favor of individual hand pollinations. The A^d mutants from the 1947 experiments have had two generations of testing since their occurrence. These will be considered separately from those obtained in 1948 since the latter have not been as thoroughly tested.

Approximately half of the seeds produced by this cross have deep purple aleurones ($A^b\ a\ a$) and half are colorless ($a\ a\ a$). Ears of two families which produced a total of 27,936 purple seeds in 1947 were examined for the occurrence of pale seeds. Fifty-one pale seeds (suspected A^d mutants) were obtained for testing. Of these, 36 had etched endosperms (*et et et*) and 15 were normal (*Et et et*). Not all were expected to be true mutants since these included many seeds which deviated only slightly in phenotype from the purple class. Unfortunately only twenty of the 51 cases survived beyond the seedling stage. Thirteen of these were indistinguishable at

maturity from sib $A^b\ a$ plants grown as controls. That these plants carried an unmutated A^b gene is established from their crosses with sib $A^b\ a$ plants. If the plants in question had carried a mutant A^a gene ($A^a\ a$) three types of seeds would have resulted from these crosses: purple, pale and colorless. Only purple and colorless seeds were produced.

The remaining seven plants of the twenty which grew to maturity had a reddish brown plant color in distinct contrast to the purple phenotype of sib $A^b\ a$ plants. That these plants carried a mutated allele of A^b producing an intermediate phenotype is confirmed by their selfed and outcrossed progenies. These data are summarized in table 1.

Six of the seven plants yielded progenies following self pollination. As indicated in the table only pale and colorless seeds were produced on these ears. Crosses of each of the pale plants with $A^b\ a$ individuals gave purple, pale and colorless seeds. From this it is apparent that all seven of the pale plants were heterozygous for a mutant gene associated with intermediate plant and aleurone phenotype and originating in the female germ line of the $A^b\ a$ plants of the original cross.

TABLE 1

SUMMARY OF DATA ON ALEURONE COLOR FROM SELFED AND CROSSED PROGENIES OF THE SEVEN PALE PLANTS ORIGINATING FROM THE CROSS: $A^b/lg\ a\ et \times lg\ a\ et$

	SELFED PROGENIES			CROSSES WITH *a a*		
PLANTS	PURPLE	PALE	COLORLESS	PURPLE	PALE	COLORLESS
1A-1	0	125	48	0	788	866
8A-1	0	94	49	0	469	472
9A-1	0	68	26	0	148	144
14A-1	0	146	46	0	434	465
19A-1	0	40	14	0	693	695
78-1	0	76	18	0	143	159
78H-1	..	...	..	0	360	365

Some of the *lg a et* plants used as a source of pollen in the 1947 crosses carried *Dt*. This gene induces the mutation of *a* to *A* and to other alleles of higher level. Rhoades[8,9] has shown that the frequency of this change is conditioned by the number of doses of both the *a* and *Dt* genes. The *a a a Dt dt dt* endosperms of seeds resulting from crosses with pollen which transmitted *Dt* showed a moderate degree of dotting on a colorless background. The cells of the purple endosperms ($A^b\ A^b\ a\ Dt\ dt\ dt$) carry both *a* and *Dt* and presumably some of these carry an *A* gene resulting from the *Dt*-induced mutation of *a*. However, no dots have been observed on purple endosperms of this constitution and it may be inferred that the mutant cells ($A^b\ A^b\ A$) are indistinguishable from $A^b\ A^b\ a$ cells. This failure of the mutated *A* to modify the phenotype of cells which already carry A^b has an application in establishing with certainty the mutant character of the pale seeds suspected of carrying an A^a allele. If the pale

phenotype of these seeds is owing to the action of modifier genes and not to a change in the A^b gene they as well as any pale seeds in their progeny are expected to be without dots. If, however, a mutation of A^b to A^d is involved, such that the endosperms are of the constitution $A^d\ A^d\ a\ Dt\ dt\ dt$ the cells carrying the *Dt*-induced, mutant *A* gene ($A^d\ A^d\ A$) are expected to appear as purple dots on a pale background. Three of the seven pale seeds whose progeny test are given in table 1 had dotted endosperms. Two other seeds, though they had no dots, carried *Dt*. The absence of dots on these seeds may be attributed to the fact that dots frequently are absent on seeds which carry single doses of *Dt* and *a*. Each of the selfed progenies from these five seeds produced many pale, dotted endosperms. In a later generation all seven mutant cases were tested for the presence of dots on pale background by crossing the $A^d\ a$ plants with $a\ a\ Dt\ Dt$ individuals. In all cases the presence of pale seeds with dots confirms the changed nature of the A^d gene.

In view of the fact that the A^d mutants originated in $A^b\ a$ plants it is necessary to rule out the possibility that they were derived from *a*. In a previous study[5] it was established that a number of mutants of the A^d type which could have come only from A^b share several distinctive types of effect: (1) These mutants, like the parent A^b allele, are dominant to *A* in their brown pericarp effect; (2) though the mutants are associated with a decidedly intermediate plant and aleurone phenotype, there is no cumulative effect with increasing doses; (3) in compounds with an allele of a higher level of action the A^d mutants detract from, rather than add to, the effect of the former. Since mutants having this combination of effects have never been obtained from *a*, a study of the seven A^d mutants under consideration may be expected to establish conclusively their origin. The original seven plants having the constitution $A^d\ a$ were crossed with $A\ A$ individuals. The pericarp color of the F_1 plants indicated, for each of the seven cases, that the A^d gene is dominant to *A* in its effect on this tissue. To test the effects of the A^d mutants in compounds with stronger acting alleles the $A^d\ a$ plants were crossed with $A^{br}\ A^{br}$ individuals. In the F_1 progenies plants having the constitutions $A^{br}\ A^d$ and $A^{br}\ a$ were compared. Five of the seven mutants have been tested in this manner and all show a striking effect in the displacement of the phenotype of $A^{br}\ A^d$ plants in a direction away from wild type as compared with sib $A^{br}\ a$ plants. The tests of the dosage effects of the A^d mutants require an additional generation and these studies are not completed. However, on the ears of the selfed $A^d\ a$ plants pale endosperms of three types occur, having one, two and three doses of the A^d gene. The phenotypes of the pale seeds on these ears are quite uniform suggesting that the A^d mutants show no dosage effect. More critical evidence on this point is being sought at the present time from studies involving comparisons in which a deficiency, instead of *a* is em-

ployed. These tests indicate that the A^d mutants which were obtained in the present experiment from A^b *a* individuals are indistinguishable from those which have a known origin from A^b A^b plants. It may be concluded that the former also are derivatives of A^b.

Since the heterozygous egg parents of the original cross were A^b/lg *a et* and the pollen parents were *lg a et*, a linkage of *lg* or *et* with A^b in individuals from this cross is attributable to crossing over between A^b and the loci of the marker genes. Table 2 summarizes the information on recombination

TABLE 2

SUMMARY OF DATA ON CROSSING OVER IN THE *lg-a* (REGION I) AND *a-et* (REGION II) SEGMENTS FROM PROGENIES OF BACKCROSSES OF THE TYPE: A^b/lg *a et* × *lg a et**

	CROSSOVERS							
	REGION I		REGION II		REGIONS I AND II			
TOTALS	NUMBER	FREQUENCY	NUMBER	FREQUENCY	NUMBER	FREQUENCY	EXPECTED FREQUENCY	COINCIDENCE
17,298	5714	0.3303	2220	0.1283	405	0.0234	0.0424	0.55
(51 ears)		±0.0036		±0.0025		±0.0004		
22,706†	..		2946	0.1297				
(60 ears)				±0.0022				

* The data reported are from the two families which were involved in the original crosses and which produced the seven individuals carrying the mutant A^d gene.

† These more extensive data on crossing over in Region II include the 51 ears in the first row of the table along with additional progenies from the same families classified for *a* and *et* only.

for the *lg-a* and *a-et* regions obtained from progenies of the original crosses which produced the mutant individuals. Table 3 summarizes information

TABLE 3

SUMMARY OF THE LINKAGE INFORMATION FOR THE MUTANT A^d-BEARING INDIVIDUALS FROM THE CROSS: A^b/lg *a et* × *lg a et*

	CONSTITUTION OF THE A^d-BEARING STRAND			
	NON-CROSSOVER	CROSSOVER TYPES		
MUTANT PLANT	TYPES	REGION I	REGION II	REGIONS I AND II
1A-1	...	.....	*Lg* A^d *et*	.....
8A-1	...	.....	" " "	.....
9A-1	...	.....	" " "	.....
14A-1	...	.....	" " "	.....
19A-1	...	.....	" " "	.....
78-1	...	*lg* A^d *Et*	.....	.....
78H-1	...	.....	.....	*lg* A^d *et*
Totals	0	1	5	1

as determined in progeny tests on the linkage phase of the etched endosperm and liguleless characters for the seven original plants carrying the mutant A^d gene. The interesting feature of these data is that in six of the seven cases the chromosome carrying the A^d gene emerged from the original cross combined with *et*.

Unless the change of A^b to A^d is in some way related to crossing over it would be expected that the frequency of crossing over in the *a-et* interval would be the same in sporocytes which carry A^d as in those which transmit A^b. On this basis, of the original mutant plants approximately 13% (table 2) A^d *et/a et* (crossover) and 87% A^d *Et/a et* (non-crossover) individuals are expected. The actual results were almost exactly the reverse of this; six of the seven individuals were crossover types for the *a-et* region. On the basis of 13% recombination and with no association between crossing over and mutation this number of mutants which are crossover types would be expected to occur among a total including approximately 40 mutants which are non-crossovers for the *a-et* region. There was only one of the latter.

The foregoing data suggest a relation between the mutation of A^b and crossing over in the *a-et* region. This interpretation receives further support from experiments conducted in 1948 on a larger scale. As before, marked heterozygotes carrying A^b were crossed with homozygous pollen parents of the constitution *lg a et*. In this case all of the pollen employed

TABLE 4

SUMMARY OF DATA ON CROSSING OVER IN THE *lg-a* AND *a-et* SEGMENTS FROM BACKCROSSES OF THE MARKED HETEROZYGOTES CARRYING A^b *

	REGION I		REGION II		REGIONS I AND II			
TOTAL	NUMBER	FREQUENCY	NUMBER	FREQUENCY	NUMBER	FREQUENCY	EXPECTED FREQUENCY	COINCIDENCE
5790	2068	0.3572 ±0.0063	804	0.1389 ±0.0045	184	0.0318 ±0.0023	0.0496	0.64

* These data from 34 ears in 23 families in 1948.

carried the *Dt* factor. Progeny tests of suspected mutant individuals from these crosses have not been made as yet. In the absence of these, however, all of the pale seeds with dotted endosperms have been selected and classified for normal versus etched phenotype. As indicated above by reason of their dotted phenotypes these seeds carry a mutant derivative of A^b and their selection on this basis is not subject to the error and prejudice which may be involved when classification is made solely on the basis of a character which may be quite variable such as the degree of pigmentation. Of the total of 34 pale, dotted, mutant seeds, 30 are crossover types for the *a-et* interval. Here as in the previous experiment there is an unexpected preponderance of the crossover class. In order to determine the normal rate of crossing over in the stocks employed in this experiment the total progenies from those ears on which mutant seeds occurred were classified. Since the mutant seeds occurred singly on the ears, a total of 34 ears is involved. The value obtained (table 4) for recombination in the *a-et* region (13.9%) agrees reasonably well with that obtained in the 1947 experiment.

The association of crossing over with mutation of A^b may be explained if it is assumed that there is some causal relation between the two. On one hypothesis it may be argued that occasionally a crossover in the *a-et* region results in an unequal exchange of chromatin at the locus of a gene which, like A^b, affects the pigmentation of plant and endosperm tissues. On this basis the pale phenotype of the "mutant" individuals would not be due to a change in the A^b gene but to a modification of chromatin somewhere between the *a* and *et* loci. Two types of evidence are against such an interpretation. (1) In all cases dots, resulting from the *Dt*-induced mutation of *a* to *A*, have been observed on the pale background of the mutant seeds or of pale seeds in their progenies. If the pale background of these seeds is owing to the action of a modifier gene, the phenotype of

TABLE 5

ACTUAL AND THEORETICAL FREQUENCIES OF CROSSOVERS AND NON-CROSSOVERS RELATIVE TO THE *a-et* INTERVAL FOR THE A^d-BEARING STRANDS OF THE FORTY-ONE MUTANT INDIVIDUALS*

BASIS ON WHICH FREQUENCIES ARE COMPUTED	CROSSOVERS	NON-CROSSOVERS	STANDARD ERROR
Observed	0.878	0.122	0.051
No relation between mutation of A^b and crossing over	0.132	0.868	0.053
Mutation of A^b conditioned by crossing over at that locus. No allowance for interference	0.868	0.132	0.053
Same as above but allowing for coincidence of 0.5	0.934	0.066	0.039

* Actual values are based on the total of 41 mutants, 36 of which were crossover types. Theoretical values are based on the average rate of crossing over for this region (13.2%) taken from tables 2 and 4.

cells included in the dots should be modified to pale also. (2) If the pale phenotype of mutant seeds is held due to a modifier it is clear that the latter must be dominant since the cells of these endosperms carry a normal chromosome from the pollen parent. In this case the selfed or suitably crossed progenies of mutant individuals should produce some offspring which are homozygous recessive for the modifier condition and carry A^b; these would appear as recovered cases having purple endosperms and purple plant phenotypes. No such recoveries have been noted in the seven cases for which progenies are presently available.

These two types of evidence not only rule out the possibility of a modifier effect in the occurrence of the mutant seeds but indicate further that the pale effect of the mutation must be due to a change in the A^b gene itself. On any other basis the mutations of *a* (for which these seeds are heterozygous) to *A* through the action of *Dt* would not be recognizable as dots on these seeds; nor would the absence of purple seeds and plants in the

progenies of the mutant individuals be expected. Since it is necessary to relate the high frequency of crossing over to this change the simplest and most direct hypothesis is that these mutations of A^b to A^a are a result of crossing over within the locus of A^b.

In the cases of 5 of the total of 41 mutants from both experiments the A^a-bearing strand delivered to the egg was seemingly a non-crossover type for the *a-et* interval. In view of the relatively great length of this segment (13.2 units as an average in these experiments) it is possible to explain these apparent non-crossover types as due to double exchanges, one occurring at the locus of A^b, giving rise to the mutation, the other occurring somewhere between this locus and that of *et*, thus reconstituting the parental combination. The data of table 5 are pertinent to this argument. Here are presented the actual frequencies of crossover and apparent non-crossover strands carrying the mutant A^a gene alone with frequencies expected on three theoretical bases. The greatest discrepancy is between observed values and those expected on the basis of no relation between mutation and crossing over (second row). In the third row of the table are given the frequencies expected on the hypothesis that the mutation of A^b is caused by a crossover at that locus. On this basis the non-crossover class results from double exchanges. Here there is good agreement between the theoretical and experimental frequencies. The value 0.122 for apparent non-crossovers among the 41 mutant cases is close to the average value of 0.132 for crossing over in the *a-et* region (from tables 2 and 4). However, in the computation of the theoretical frequencies in the third row of table 5 no allowance has been made for interference. The frequency of 0.122 for non-crossovers (reasoned as cases of double exchange) among the 41 mutant cases corresponds to a coincidence of 0.9. This exceeds the coincidence value of 0.6 corresponding to the *lg-a* and *a-et* regions (tables 2 and 4). In view of the small number of mutant cases it is not clear whether the excess of apparent non-crossover types among them is due to errors in sampling or to the occasional occurrence of mutations of A^b which are not associated with crossing over. If mutations of the latter type occur a large proportion of these would fall in the non-crossover class thus tending to increase unduly the coincidence value. There is at present no basis for estimating the effect of a crossover at the A^b locus on the frequency of coincidental exchange in the *a-et* region but it is reasonable to expect that the value for coincidence in this case would fall below 0.6. In the last row of table 5 are given the theoretical frequencies of crossovers and apparent non-crossovers associated with an arbitrarily chosen coincidence of 0.5; from the corresponding standard error for $N = 41$ it is clear that the actual frequencies could well have been chosen from such a population.

Confirmatory evidence of the association of mutation of A^b and crossing

over is being sought from independent experiments using plants in which the A^b segment of the third chromosome either has no homologous region with which to synapse (in hemizygous plants) or, in cases where A^b is carried in a duplication, competes at a disadvantage for synapsis. Since these experiments are designed to eliminate or suppress those A^b mutations due to crossing over they may be expected to establish also whether mutations of A^b to A^d which are not associated with crossing over can occur.

In the foregoing presentation occasional reference has been made to the "A^b locus." There is strong argument from the present evidence for viewing the "A^b locus" as composed of at least two, physically distinct units which function coöperatively in the determination of the A^b effect. If these are designated alpha and beta, the latter being more distal, the A^d mutants may be described as having alpha and lacking beta since the strands carrying A^d in these experiments are predominantly of the non-parental class for *et*, the more distal marker gene. This consideration poses several interesting problems whose analysis may be viewed as critical tests of the hypothesis. (1) Since on this basis A^b is composed in part of the A^d or alpha component the action of A^b may be interpretable, at least in part, in terms of effects which, among those of the A alleles, are distinctive for A^d. As reported elsewhere[5] these include the determination of brown pericarp which is dominant to the red effect of A, the absence of a cumulative effect with increasing doses, and a striking tendency to reduce rather than enhance the type effect in combinations with various A alleles. Of the alleles associated with purple aleurone and plant phenotypes, only A^b has a brown pericarp effect which is dominant; it is reasonable to infer that this action may be assigned to A^d which has been argued as a component of A^b. It is not known whether A^b shares the dosage and competitive effects of A^d but this information is technically more difficult to obtain for A^b than for A^d because of the closer approach of the former to wild type. (2) If, as on the present scheme, the A^d mutants represent the alpha component which has been separated from the beta constituent by crossing over between the two, it is expected that another class of mutants corresponding to the beta component carried on the reciprocal strands involved in such exchanges should occur with equal frequency. While these have not been isolated this is no argument against their occurrence. A^b is associated with purple aleurone and plant while what has been argued as its alpha component, A^d, is distinctly intermediate in effect. From this it seems likely that the beta component produces a purple phenotype and if so mutants of this type would be indistinguishable from seeds carrying the unchanged A^b. However, on the expectation that the beta component produces a pericarp color distinguishable from A^b, experiments are being conducted to determine whether A^b gives rise to mutants of this type. These would have escaped detection in the experiments reported here

since the P factor which has a complementary effect in the pigmentation of this tissue was absent. (3) Since the A^d mutants are argued to represent a more proximal component of A^b considerable interest attaches to whether these mutants are similar in their action. If the alpha component is itself compound in nature it might be predicted that the A^d mutants in turn are not a homogeneous lot. Comparisons in constant genetic backgrounds of the mutant alleles of the present experiments are not available now; from observations of related but non-sib mutant plants the impression is given that these alleles will prove to be quite similar in effect. Nevertheless, the possibility of the component nature of alpha remains a particularly inviting prospect. Studies[5] on the action of A^b and several mutants of the A^d type indicate that their action cannot be explained on the basis of a simple relation between gene and reaction. Provisional schemes were offered to account for the exceptional behavior of these alleles. It was suggested (*a*) that the determinants corresponding to these genes function as units but that the enzyme corresponding in each case might participate in more than a single reaction, or alternatively (*b*) that a simple relation biochemically between gene and enzyme might be defended on the assumption that each of these alleles is compound in the sense of being constituted by more than one physically distinct determinant. The evidence reported here suggests that the antimorphic action of A^b resides in such a component nature. It is possible that the A^d mutants likewise owe their unusual behavior to a similar cause but proof of this requires direct evidence of the compound nature of what has been labeled here the alpha component. (4) Is the crossover which gives rise to the mutants preceded by unequal synapsis? Since the mutations occurred in plants heterozygous for a it follows that any statement about such an arrangement must be based on some understanding of the physical constitution of this allele and on this point there is no information. There is hence no basis from these experiments to argue that the change is related to unequal synapsis or even that there is any final inequality of exchange. However the mutants which Stadler[6] obtained and which are similar in effect to the A^d mutants of the present experiments occurred in homozygous A^b individuals. It is not known whether these mutations were associated with crossing over, but if so, there is a basis for the argument that unequal synapsis is involved. Making use of more efficient markers which have become available recently, the relation between crossing over and mutation in A^bA^b plants is being investigated.

It is premature to discuss the relation of the present case to others which have been more extensively studied.[1-3] Nevertheless it is a rather inviting prospect, in analogy with the cases of Bar, lozenge, Star-asteroid and others[10] in *Drosophila* and with that in *Gossypium*,[11, 12] that A^b represents repeated loci and by further analogy with several of these cases has com-

ponents which are derived by duplication from a common ancestral locus and which have since become modified. No less attractive is the possibility that the divergent action of certain of the *A* alleles may find an explanation in terms of their component nature.

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*ABERRANT HETEROZYGOTES IN ESCHERICHIA COLI**

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A mechanism of genetic recombination has been indicated in experiments on *Escherichia coli*, strain K-12.[1] A synthetic agar medium was used as a selective sieve to isolate occasional prototroph recombinants which appear in mixed cultures of complementary biochemical mutants. Later, additional genetic factors were introduced, including fermentation and virus-resistance mutations, and these factors were found to segregate in characteristic ratios, suggesting linkage. All the cells in a given prototroph colony showed the same combination of characters and were stable on further cultivation. Therefore, it was inferred that segregation had occurred before the initiation of the colony, and that the postulated zygote had a very short life, probably a single cell generation. The life cycle would resemble the ascomycete's, in which haploid nuclei fuse to form a transient diploid zygote which undergoes meiosis without any intervening mitoses.

Exceptions to this rule have now been found in the form of unstable prototroph cultures which continually segregate out various recombination types so as to suggest that they are heterozygous and diploid. But