A Novel Dominant Mutant Allele at the *white* Locus of *Drosophila melanogaster* Is Mutable

P. M. BINGHAM

Laboratory of Animal Genetics, National Institute of Environmental Health Sciences, Research Triangle Park, North Carolina 27709

Genetic and cytogenetic analyses of a novel mutant allele (w^{DZL}) at the *white* (*w*) locus of *Drosophila melanogaster* and of chromosomal rearrangement mutations produced by the w^{DZL} allele are described here. I suggest on the basis of the observations discussed that the w^{DZL} mutation, which apparently causes the repression of transcription of the *white* locus (Bingham 1980), results from the insertion of a translocatable DNA sequence element into the *white*-region DNA sequences.

The *white* locus is exceptionally accessible to genetic manipulation, and, as a result, the genetic analysis of this locus is now relatively advanced (Judd 1976; Jack and Judd 1979; Bingham 1980). In addition, we have recently cloned the *white* locus DNA sequences (P. M. Bingham et al., unpubl.). In view of these considerations, the *white* locus may represent an opportunity of exceptional value to undertake concerted biochemical and genetic analyses of the regulation of transcription of a gene in a metazoan organism and of the physical nature of mutable alleles in *Drosophila.*

EXPERIMENTAL PROCEDURES

Additional descriptions of the strains and mutant alleles used here may be found in Lindsley and Grell (1968), Judd et al. (1972), Lefevre and Green (1972), Jack and Judd (1979), and Bingham (1980). The methods employed in the cytological analyses are described by Lefevre (1976).

RESULTS

Mutant Phenotype

The w^{DZL} mutation was originally detected in a single heterozygous female in a wild-type (Oregon R) *D. melanogaster* stock and presumably arose spontaneously. The wild-type eye-color in this species is a deep bright red. w^{DZL} produces a yellow eye-color in females homozygous for the allele or heterozygous for the allele and a wild-type allele. w^{DZL} produces a dark red-brown eye-color in males hemizygous for the allele. Thus, w^{DZL} is dominant to the wild-type allele and produces an extreme sexual dimorphism for eye-color phenotype. The mutant displays no maternal effect and is not associated with detectable cytological abnormality (Bingham 1980).

Map Position of the w^{DZL} Mutation

The w^{DZL} mutation has been previously reported to reside approximately equidistant between the *zeste* (*z*) locus and the *Notch* (*N*) locus, at 1.0 and 3.0, respectively, on the standard meiotic map of the X chromosome. On the basis of this positioning and the behavior of the mutant, it was proposed to be an allele at the *white* locus (Bingham 1980).

The map position of the mutation has been further refined as follows: First, 8 y^+ sc^+ w^{DZL} rst^{CT} males were recovered from among approximately 1700 male progeny of females of genotype *y sc* w^+ rst^{CT}/y^+ sc^+w^{DZL} rst^+. The rst^{CT} marker maps between the *white* and *Notch* loci (Lefevre and Green 1972), and these results define the map order of the relevant markers to be w^{DZL}-rst^{CT}-*N*. Second, 2 presumptive y^+ sc^+ $w^a w^{DZL}$ rst^{CT} males were detected among approximately 7000 male progeny of females of genotype *y sc* w^{DZL} rst^{CT}/y^+ sc^+ w^a rst^+. Both of these recombinants produce a very light yellow eye-color in hemizygous males (unlike either w^a alone or w^{DZL} alone), are dominant to w^+ ($w^a w^{DZL}/w^+$ females have yellow eyes), and revert to a form indistinguishable from the parental w^a allele (apricot eye-color phenotype and recessive to w^+). (As will be discussed, the w^{DZL} allele alone frequently reverts to w^+.) These recombinants strongly suggest a map order as follows: w^a-w^{DZL}-rst^{CT}.

In summary, the w^{DZL} mutation resides to the left of the rst^{CT} mutation and apparently to the right of the w^a mutation. As the rst^{CT} mutation resides immediately to the right of the *white* locus and the w^a mutation resides in the central portion of the *white* locus (Judd 1976 and references therein), these observations place the locus of the w^{DZL} mutation in or immediately to the right of the right-hand set of previously defined *white* locus mutant sites.

General Features of w^{DZL} Mutability

Spontaneous revertant frequencies for mutant alleles in *Drosophila* are typically low enough (less than 1 in 100,000 gametes) as to preclude detection of reversion (or of secondary mutation) during casual observation and in the absence of mutagenesis. However, a few mutant alleles have been observed to produce mutant and/or revertant frequencies orders of magnitude higher than the figure already mentioned. These frequently mutating alleles are referred to as mutable alleles. Recent reviews of studies of mutable alleles in *Drosophila* can be found in Green (1976, 1980).

The w^{DZL} allele is mutable. In particular, among the progeny of mass matings of homozygous w^{DZL} individuals, new mutants are observed at frequencies of 0.5% to 1.5% (Tables 1 and 2).

Table 1. Spontaneous Mutation in Homozygous w^{DZL} Stocks

	Male progeny	
Pair-mating no.	white eye-color	wild-type eye-color
1–13	0	0
14	0	1
15	0	4
16	0	1
17	0	3
18	0	1
19	1	0
20	4	3
21	0	3
22	4	0
23	0	1
24	1	0
25	2	3
26	3	0
27	2	1
28	0	1

The parents in each pair-mating consisted of a single, virgin, homozygous w^{DZL} female and a single w^{DZL} male. Each pair-mating produced between 150 and 250 progeny. The total number of male progeny examined as determined by weight was 2550. The pair-matings have been renumbered to place those producing no new mutant progeny at the beginning of the table.

The focus of succeeding sections here will be the classes of new mutants produced by the w^{DZL} chromosome that are associated with chromosomal rearrangement mutations. However, the majority of new mutant derivatives of w^{DZL} are not associated with detectable chromosomal rearrangement. This majority class of new mutant alleles may be divided into two categories on the basis of eye-color phenotype. The first category of these new mutant alleles comprises individuals displaying a fully wild-type eye-color and will be referred to as simple revertants. The second category of these new mutant alleles comprises individuals displaying a bleached white eye-color phenotype. Alleles in this second category are recessive for the wild-type allele (unlike the parental w^{DZL} allele) and will be referred to as colorless derivatives of w^{DZL}.

Table 2. Spontaneous Mutation in Homozygous w^{DZL} Stocks

	Female progeny	
Pair-mating no.	notched wings	wild-type eye-color
1–147	0	0
148	0	6
149	0	1
150	0	1
151	0	2
152	0	1
153	0	1
154	1	0
155	1	0
156	1	0
157	2	0
158	1	0
159	1	0
160	1	0
161	7	0
162	1	0
163	3	0
164	1	0
165	6	0
166	1	0
167	1	0

The parents in each pair-mating consisted of a single, virgin, homozygous w^{DZL} female and a single w^{DZL} male. Each pair-mating produced between 150 and 250 progeny. The total number of female progeny examined as determined by weight was 11,970. The pair-matings have been renumbered so that they are grouped according to the kind of new mutant progeny detected.

The salient features of the behavior and origin of these two classes of mutant derivatives are as follows: First, when these derivatives are collected from among the progeny of pair-matings (a single male and a single virgin female is a pair-mating), the distribution of new mutant progeny deviates significantly from that predicted when it is assumed that each new mutant individual arises as the result of an independent event (Table 1). (This clustering of new mutant derivatives is also observed when attention is restricted to chromosomal rearrangement mutations, as may be seen in Table 2.) This phenomenon has been observed previously in the cases of several mutable alleles in *Drosophila* (Green 1976) and has been interpreted to suggest that the mutational events in question often occur gonadally (premeiotically) and thus give rise to clones of mutant individuals. Second, seven independently derived revertants of the w^{DZL} mutation collected from pair-matings of $z^l w^{DZL}$ individuals are indistinguishable from standard w^+ alleles in producing yellow-eyed females when homozygous ($z^l w^+/z^l w^+$), wild-type males when hemizygous ($z^l w^+/Y$), and wild-type females when heterozygous with a $z^+ w^+$ chromosome ($z^l w^+/z^+ w^+$). The reader is referred to Jack and Judd (1979) and references therein for a discussion of the z^l mutation at the *zeste* locus. In addition, each of more than 100 independently derived simple revertants collected from among the progeny of pair-matings of $z^+ w^{DZL}$ individuals displays a fully wild-type eye-color phenotype in homozygous females and in hemizygous males and is fully viable and fertile in both of these cases. Third, 17 of 17 colorless derivatives of w^{DZL} produce a very light yellow eye-color phenotype when heterozygous with the w^{sp} allele at the *white* locus (w^-/w^{sp}) and produce a nearly wild-type eye-color phenotype when heterozygous with the $z^l w^{rdp+}$ chromosome ($z^+ w^-/z^l w^{rdp+}$). These observations may be interpreted on the basis of previously reported observations (Green 1959; Judd 1976; Jack and Judd 1979) to indicate that the colorless derivatives of w^{DZL} are small deletion mutations (small in that all of the 17 chromosomes in question fail to carry a recessive lethal mutation as would be expected if these colorless derivatives of w^{DZL} were associated with deficiencies deleting complementation groups in addition to *white*) and that these small deletions affect at least the rightmost (w^{sp}) portion of the *white* locus. The possibility that the colorless derivatives of w^{DZL} represent small deletions will not be considered further here; however, this possibility is consistent with the hypothesis to be developed on the basis of the analysis of large chromosomal rearrangements generated by the w^{DZL} chromosome. Fourth, none of 7 simple revertants examined and none of 17

colorless derivatives examined displayed any detectable cytological abnormality. It is not excluded, of course, that these derivatives are associated with DNA sequence rearrangements too small to be detected using conventional cytological techniques.

Chromosomal Rearrangement Mutations Stimulated by w^{DZL}

The first mutable allele in *D. melanogaster* to be described was the w^c allele at the *white* locus (Green 1967). This allele was observed to give rise to new mutant derivatives at an unusually high frequency. Among these new mutants was a class of deletion variants in which deleted segments began at the *white* locus (the locus of the mutable allele) and extended rightward to or beyond the *Notch* locus. On the basis of the instability of w^c and the instability of mutants in maize when apparently associated with the presence of translocatable elements, as studied by McClintock (1965), Green suggested that the w^c mutation may result from the presence of a translocatable element at the *white* locus.

On the basis of what is now quite advanced genetic and biochemical analyses of translocatable elements in bacterial systems (Kleckner 1977; Kleckner et al., this volume), a very specific set of predictions as to the nature of mutations expected to be produced by a translocatable DNA sequence element is possible. In particular, in *Drosophila,* where highly detailed cytological analyses are possible, we expect to be able to recognize several classes of simple chromosomal rearrangement mutations: deletions, duplications, inversions, and reciprocal translocations. These rearrangement mutations should, under the translocatable element hypothesis, have two breakpoints; one precisely at the locus of the translocatable element and a second at an unpredictable position elsewhere in the genome, with the position of this second breakpoint being different for each independently isolated rearrangement mutation.

The w^{DZL} chromosome has been examined for its capacity to produce three of the four classes of rearrangement mutations expected to be detectable under the translocatable element hypothesis. (Duplications are more difficult to detect initially than are mutations of the other three types, and no systematic attempt has been made here to isolate them.)

Deletion mutations beginning at the* white *locus and extending rightward. The occurrence of this class of mutations may be assessed by screening for *Notch* mutations. The *Notch* locus resides nearby (1.5 map units away) and to the right of the *white* locus; its deletion produces a dominant mutation causing notches in the tips of the wings. The results of one such screen for *Notch* mutations is given in Table 2. A total of 11,970 female progeny were examined in this screen, and 28 Notch individuals emerged, corresponding to a frequency of approximately 0.2%. In contrast, examination of approximately 28,000 female progeny from a mass mating of the Oregon-R stock in which the w^{DZL} mutation was originally detected failed to reveal any Notch individuals.

If the mutability associated with the w^{DZL} chromosome results from the presence of a translocatable DNA sequence element near the *white* locus and if this translocatable DNA sequence element is responsible for the generation of the deletion mutations presumed to produce the observed *Notch* alleles, then these deletion mutations should correspond to a deleted segment beginning near or within the *white* locus and extending into or beyond the *Notch* locus.

A total of 26 independently derived *Notch* mutations have been isolated from w^{DZL} stocks. Whether or not these mutations are associated with deficiencies, and, if so, what are the precise dimensions of the deleted segment, has been determined by genetic complementation. A map of the known complementation groups in the immediate vicinity of the *white* locus is as follows: *zw11-zw9-w-rst-vt-N.* It should be noted that this genetic region is among the most intensively studied in metazoan biology, and all complementation groups in the region whose alleles are either recessive lethal alleles or alleles producing a detectable morphological effect have probably been found (Judd et al. 1972; Lefevre and Green 1972; Judd and Young 1974). As assessed by complementation with the *white* alleles w^{Bwx}, w^a, w^e, w^l, and w^{sp}, 22 of the 26 *Notch* mutations examined are associated with w^+ alleles (all N^-/w heterozygotes display wild-type eye-color), 3 of 26 are associated with *white* alleles quite similar to the parental w^{DZL} allele (N^-/w^e and N^-/w^l females display a wild-type eye-color and all other heterozygotes are yellow-eyed [Bingham 1980]), and 1 of 26 is associated with a w^- allele (N^-/w^l females have bleached white eye-color). All 26 are $zw11^+$, $zw9^+$, rst^- (N^-/rst^{CT} females display a roughest phenotype), vt^- (N^- chromosomes heterozygous with the *wrv-4* deletion [Lefevre and Green 1972] produce extreme roughest and verticals phenotypes), and N^-. These observations demonstrate that all 26 of the *Notch* mutations in question are deletion mutations and place the left-hand (distal) endpoint of the deleted segments at or immediately to the right of the rightmost portion of the *white* locus. (The single *Notch* mutation associated with a w^- allele arose in a vial producing two such mutants as well as three *Notch* mutations associated with w^+ alleles. I suggest that this w^-N^- chromosome is the product of two consecutive mutational events and is thus an anomaly in this context.)

The dimensions of the deleted segments in these deficiency chromosomes have been corroborated and refined by cytological analyses. Nine of the 26 deletion chromosomes characterized by genetic complementation analyses have been examined cytologically (seven associated with w^+ alleles, one associated with a w^{DZL} allele, and one associated with a w^- allele). In all nine cases, the left-hand terminus of the deleted segment is in or very near the interband between polytene chromosome bands 3C1 and 3C2. I estimate the uncertainty in the positioning of these and subsequent breakpoints to be less than one band. In particular, in the cases of these

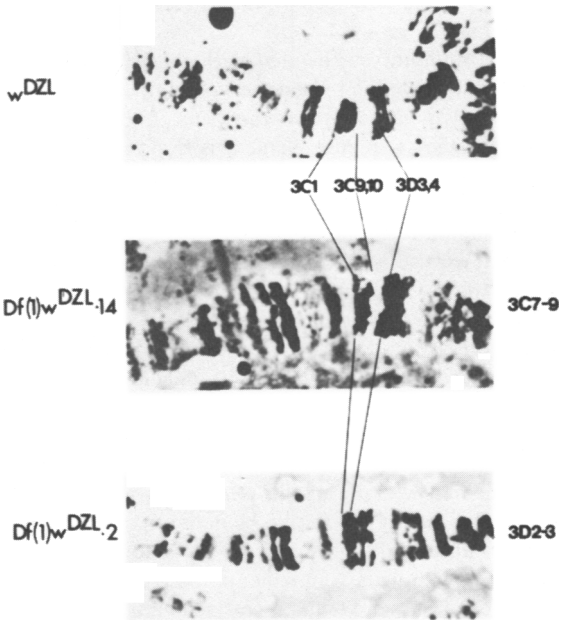

Figure 1. Polytene chromosome squashes of the w^{DZL} chromosome and two deficiency derivatives of w^{DZL} whose deleted segments begin at the *white* locus and extend rightward (toward the centromere). The designations on the left indicate the chromosomes in question and those on the right indicate the polytene chromosome bands near which the non-*white*-locus-associated breakpoint for the deletion has been localized.

derivatives, more than half and probably all of 3C1 remains and more than half and probably all of 3C2 has been deleted. Photographs of polytene squashes of two of these deletion mutations are shown in Figure 1. The most refined analyses reported previously of the cytological position of the *white* locus place it in the 3C1-2 interband (Lefevre and Green 1972; Sorsa et al. 1973; Judd 1974). The right-hand termini of the deletion mutations in question have not been examined as exhaustively as have the left-hand termini; however, at least three distinct right-hand termini (3C7-9, 3C1-1, and 3D2-4) have been distinguished among the nine examined.

In summary, the *white*-locus-associated breakpoints for all of these nine deletion mutations are identical, within the limits of resolution of cytological analysis, and each is, within these same limits, precisely at the locus of the dominant mutant allele w^{DZL}.

Inversion mutations. w^{DZL} often reverts to w^+ in association with the occurrence of the *Notch* deletion mutations discussed in the preceding section. Furthermore, the dominance of w^{DZL} to a w^+ allele is apparently dependent upon somatic synapsis of the alleles. If the w^+ allele is placed elsewhere in the genome other than in the position homologous to that occupied by the w^{DZL} mutation (for example on a w^+-bearing insertional translocation), w^{DZL} is rendered recessive to the w^+ allele (Bingham 1980). This last feature of the behavior of the w^{DZL} allele may be considered an example of a transvection effect (Lewis 1954). These two observations suggest a selection scheme for inversion mutations expected to be generated by the w^{DZL} chromosome on the basis of the translocatable element hypothesis. In particular, if inversion mutation stimulated by the w^{DZL} chromosome is often associated with the reversion of the w^{DZL} mutation to w^+, these inversion chromosomes are expected to produce a wild-type phenotype in individuals heterozygous for w^{DZL} and the inversion (w^+) chromosome, as the inversion (tightly linked to the *white locus) is expected to disrupt somatic synapsis between the* w^+ and w^{DZL} alleles.

Six independently arising w^{DZL} derivatives producing a wild-type eye-color in females heterozygous for the derivative and the parental w^{DZL} allele were collected from the screen whose results are recorded in Table 2. These comprise 12 individuals out of 11,970 females examined, corresponding to a frequency of approximately 0.1% (recall that *Notch* deletions are generated with a frequency of approximately 0.2%). All six of these new derivatives produce fully viable and fertile individuals in the homozygous and hemizygous conditions and produce a fully wild-type eye-color in these same conditions. When examined cytologically, two of the six were clearly found to be associated with inversions of segments of the X chromosome immediately contiguous to the *white* locus. Photographs of squashes of these two chromosomes are shown in Figure 2. *In(1)*w^{DZL}*-1* is associated with the inversion of a segment extending from the 3C1-2 interband through around band 2F6, a segment beginning at the *white* locus and extending leftward (Fig. 2). *In(1)*w^{DZL}*-3* is associated with the inversion of a segment extending from the 3C1-2 interband through approximately band 3E3-7, a segment beginning at the *white* locus and extending rightward (Fig. 2). Two of the remaining four derivatives in question

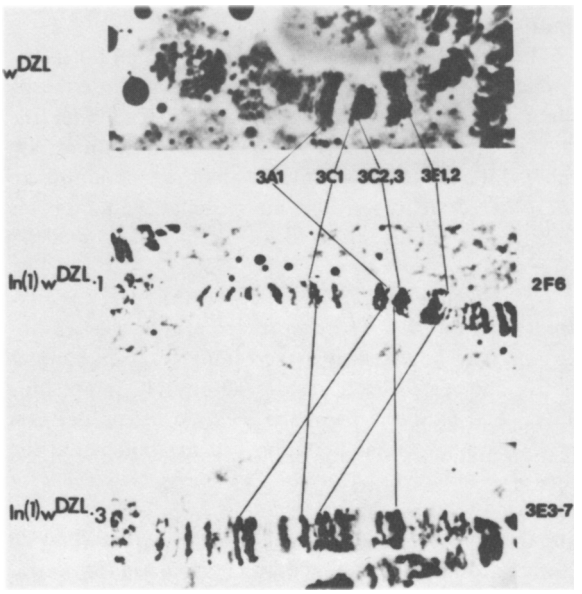

Figure 2. Polytene chromosome squashes of the w^{DZL} chromosome and two inversion derivatives of w^{DZL}. See the legend to Fig. 1 and the text for further details.

showed indications of cytological abnormality suggestive of the presence of inversions smaller than those on the *In(1)w^{DZL}-1* and *In(1)w^{DZL}-3* chromosomes (data not shown), but unambiguous classification was not attempted. The remaining two derivatives did not show cytological abnormality.

In summary, at least two inversion mutations stimulated by the *w^{DZL}* chromosome have been found. These mutations arose in association with reversion of the *w^{DZL}* mutation. The *white*-locus-associated breakpoints for these inversion mutations are, within the limits of resolution of cytological analysis, identical and, in addition, are identical to the *white*-locus-associated breakpoints of the nine *Notch* deletions described in the preceding section.

Deletion mutations beginning at* white *locus and extending leftward. Two deletion mutations arising in homozygous *w^{DZL}* females and deleting segments beginning at the *white* locus and extending leftward (distally) were isolated as follows: Exceptional wild-type eye-color females emerging from the cross y^+ *w^{DZL}*/y^+ *w^{DZL}* × y^1 w^+/*Y* were examined for the presence of a recessive lethal mutation. Two independently derived chromosomes of this type corresponding to 3 individuals were found among approximately 3700 female progeny produced by 47 pair-matings of the above type. This corresponds to a frequency of the order of 0.1%.

The two derivatives in question were subjected to genetic complementation analysis using the same set of mutant alleles employed in the analysis of the *Notch* deletion mutations discussed above. Although the majority of classes of the *Notch* deletion chromosomes proved to be *zw11*$^+$, *zw9*$^+$, w^+, *rst*$^-$, *vt*$^-$, and *N*$^-$, the two derivatives in question here displayed a precisely reciprocal pattern of complementation: *zw11*$^-$, *zw9*$^-$, w^-, *rst*$^+$, *vt*$^+$, and *N*$^+$. These results demonstrate that these derivatives are deletion mutations in which the deleted segment begins within or immediately to the right of the *white* locus and extends leftward into or beyond the *zw11* locus.

The cytogenetic analysis of these two variants shows them to be cytologically visible deletion mutations in which the deleted segment begins within or very near the 3C1-2 interband and extends leftward, in one case to approximately 3B4-5 and in the other case to approximately 3A2-4 (Fig. 3).

In summary, the genetic and cytogenetic analyses of these two derivatives shows them to be deletion mutations in which *white*-locus-associated breakpoints apparently reside at the identical position, and that position is, in turn, apparently identical to that of the *white*-locus-associated breakpoints for the rearrangement mutations discussed in the preceding two sections.

***A reciprocal translocation derivative of* w^{DZL}.** A single wild-type female arising in a homozygous *w^{DZL}* stock and detected during routine stock transfers produced wild-type female progeny and yellow-eyed female progeny in a one-to-one ratio when crossed to her *w^{DZL}*/*Y* brothers. When these wild-type daughters were

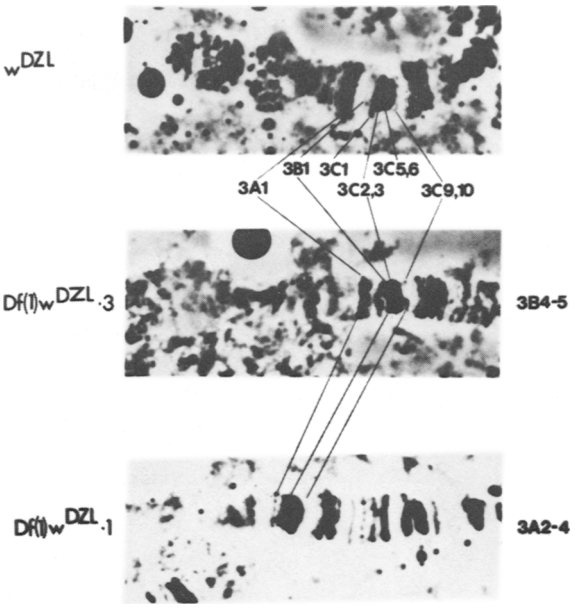

Figure 3. Polytene chromosome squashes of the *w^{DZL}* chromosome and two deficiency derivatives of *w^{DZL}* whose deleted segments begin at the *white* locus and extend leftward (toward the telomere). See the legend to Fig. 1 and the text for further details.

again crossed to *w^{DZL}*/*Y* males, approximately half of the female progeny (7 of 16 female third instar larvae) were found, upon cytological analysis, to carry a reciprocal translocation of the tips of the X chromosome and the left arm of the third chromosome. This derivative arose in association with the reversion of the *w^{DZL}* allele (as evidenced by the wild-type eye-color of females heterozygous for the reciprocal translocation and the parental *w^{DZL}* allele) and is therefore presumed to be a *w^{DZL}*-generated rearrangement.

The X-chromosome-associated breakpoint is in or very near the 3C1-2 interband (Fig. 4). Thus, the X chromosome breakpoint for this reciprocal translocation is, within the limits of resolution of cytological analysis, identical to the *white*-locus-associated breakpoints of all of the rearrangement mutations discussed previously.

DISCUSSION

The *w^{DZL}* allele at the *white* locus of *D. melanogaster* has several important properties as a mutation apparently causing the repression of transcription of the locus. These properties, suggested by previously reported genetic studies (Bingham 1980), are as follows: (1) *w^{DZL}* causes the repression of expression of the *white* allele linked to it (in *cis*) and of standard w^+ alleles (in *trans*). (2) This repression of *white* locus expression in *trans* is apparently dependent upon somatic synapsis of the *w^{DZL}* and w^+ alleles (the dominance of *w^{DZL}* to w^+ can be said to be sensitive to a synapsis or transvection effect). (3) The z^1 mutation at the *zeste* locus causes the selective repression of somatically synapsed *white* locus (w^+) pairs but not of an isolated *white* locus copy even if

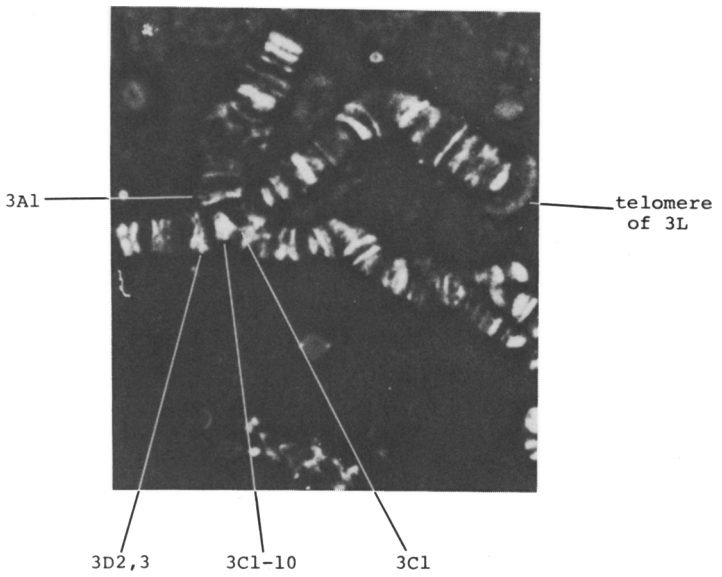

Figure 4. A polytene chromosome squash from a female individual heterozygous for the two products of an X-3L reciprocal translocation and a normal X and third chromosome. This reciprocal translocation apparently was stimulated by the w^{DZL} allele.

this isolated copy resides in the same nucleus with a synapsed *white* locus pair (Jack and Judd 1979). Several features of the behavior of the w^{DZL} allele (at the *white* locus) suggest that this mutation effects the same repression process as that effected by the z^{1} mutation (at the *zeste* locus).

Results of meiotic recombination analyses described here strongly suggest that the locus of the dominant mutant w^{DZL} allele is in or immediately to the right of the rightmost elements of the array of previously defined *white* locus mutant sites.

In addition to its properties as a dominant mutant allele, w^{DZL} is associated with an unstable or mutable allele as described here. This mutable allele generates chromosomal rearrangement mutations (deletions, inversions, and a reciprocal translocation) all sharing a common breakpoint, as determined by cytological analysis. These data support the hypothesis that the mutability associated with the w^{DZL} allele results from the presence of a translocatable DNA sequence element with properties similar to those of the well-characterized elements in bacterial systems. As assessed by genetic complementation analysis of the deletion variants produced by this mutable allele, the common breakpoint for these rearrangement mutations is in or immediately to the right of the rightmost elements of the *white* locus array. (Recall that the right-oriented deletions described are almost always associated with retention of *white* locus function, whereas the left-oriented deletions described are associated with the loss of *white* locus function.) According to the translocatable element hypothesis, the position of these breakpoints is the position of the translocatable element.

In summary, the cytogenetic and complementation studies of the rearrangement variants produced by the mutable allele linked to w^{DZL} support the translocatable element hypothesis as a rationale for the mutability of the w^{DZL} chromosome. On this basis, the locus of the translocatable element is placed in or immediately to the right of the rightmost elements of the *white* locus array. Meiotic recombination results described here strongly suggest that the mutation responsible for the behavior of w^{DZL} as a dominant mutant allele also resides in or immediately to the right of the rightmost elements of the *white* locus array.

On the basis of these observations, I propose that the mutational event giving rise to the dominant mutant w^{DZL} allele and the mutational event giving rise to the mutable allele associated with the w^{DZL} allele are, in fact, the same mutational event. I further propose that this mutational event is the insertion of a translocatable DNA sequence element into the DNA sequences corresponding to the rightmost portion of the *white* locus.

REFERENCES

BINGHAM, P. M. 1980. The regulation of *white* locus expression: A dominant mutant allele at the *white* locus of *Drosophila melanogaster*. *Genetics* **95:** 341.

GREEN, M. M. 1959. Putative non-reciprocal crossing over in *Drosophila melanogaster*. *Z. Verebungs.* **90:** 375.

———. (1967). The genetics of a mutable gene at the *white* locus of *Drosophila melanogaster*. *Genetics* **56:** 467.

———. (1976). Mutable and mutator loci. In *The genetics and biology of* Drosophila (ed. M. Ashburner and E. Novitski), vol. 1b, p. 929. Academic Press, New York.

———. 1980. Transposable elements in *Drosophila* and other diptera. *Annu. Rev. Genet.* (in press).

JACK, J. W. and B. H. JUDD. 1979. Allelic pairing and gene regulation: A model for the *zeste-white* interaction in *Drosophila melanogaster*. *Proc. Natl. Acad. Sci.* **76:** 1368.

JUDD, B. H. 1974. Genes and chromosomes of *Drosophila*. In *The eukaryotic chromosome* (ed. W. J. Peacock and R. D. Brock), p. 169. Australian National University Press, Canberra.

———. 1976. Genetic units for *Drosophila*—Complex loci. In *The genetics and biology of* Drosophila (ed. M. Ashburner and E. Novitski), vol. 1b. Academic Press, New York.

JUDD, B. H. and M. W. YOUNG. 1974. An examination of the one cistron-one chromomere concept. *Cold Spring Harbor Symp. Quant. Biol.* **38:** 573.

JUDD, B. H., M. W. SHEN, and T. C. KAUFMAN. 1972. The anatomy and function of a segment of the X chromosome of *Drosophila melanogaster*. *Genetics* **71:** 139.

KLECKNER, N. 1977. Translocatable elements in prokaryotes. *Cell* **11:** 11.

LEFEVRE, G., JR. 1976. A photographic representation of the polytene chromosomes of *Drosophila melanogaster* salivary glands. In *The genetics and biology of* Drosophila (ed. M. Ashburner and E. Novitski), vol. 1a, p. 31. Academic Press, New York.

LEFEVRE, G., JR. and M. M. GREEN. 1972. Genetic duplication in the *white-split* interval of the X chromosome in *Drosophila melanogaster*. *Chromosoma* **36:** 391.

LEWIS, E. B. 1954. The theory and application of a new method of detecting chromosomal rearrangements in *Drosophila melanogaster*. *Am. Nat.* **88:** 225.

LINDSLEY, D. L. and E. H. GRELL. 1968. *Genetic variations of* Drosophila melanogaster. Carnegie Institute of Washington Publication Number 627, Washington, D.C.

MCCLINTOCK, B. 1965. The control of gene action in maize. *Brookhaven Symp. Biol.* **18:** 162.

SORSA, V., M. M. GREEN, and W. BEERMAN. 1973. Cytogenetic fine structure and chromosomal localization of the *white* gene in *Drosophila melanogaster*. *Nat. New Biol.* **245:** 34.

Cold Spring Harbor Symposia on Quantitative Biology

A Novel Dominant Mutant Allele at the *white* Locus of *Drosophila melanogaster* Is Mutable

P. M. Bingham

Cold Spring Harb Symp Quant Biol 1981 45: 519-525
Access the most recent version at doi:10.1101/SQB.1981.045.01.068