ANALYSIS OF PRODUCTS FROM REGULARLY OCCURRING ASYMMETRICAL EXCHANGE IN DROSOPHILA MELANOGASTER[1]

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THE regular occurrence of a deficiency product, apparently resulting from crossing over within an asymmetrically-paired segment of the *Drosophila melanogaster* X chromosome was reported by GREEN (1959). JUDD (1961) recovered the complementary duplication and deficiency strands from an asymmetrical exchange occurring within the white locus. Although the recombinant products analyzed by these two investigators are quite different, they are related since both demonstrate recombination in chromosome segments which are not specifically paired in a point-by-point manner. Prior to these investigations, asymmetrical crossover products were, in general, expected only from chromosomes bearing tandem duplications.

The regular recovery of duplication and deficiency recombinant products from structurally normal chromosomes allows several questions bearing on the mechanism of crossing over to be raised. First, since the exceptional types appear among the offspring from several different crosses, it is of interest to determine whether the non-homologous pairing is accomplished in precisely the same manner in each case, with subsequent crossing over always occurring at the same point. This would lead to a single type of duplication and the complementary deficiency. On the other hand, if pairing may occur in any one of several ways or if crossing over may take place at one of several places within the non-homologously paired region, duplication and deficiency products of several different types should be obtained. A related question is whether the asymmetrical pairing involves truly non-homologous chromosome regions or whether seemingly unrelated parts have retained some specific synaptic affinities during their evolution. In other words, how specific must pairing be in order to allow crossing over? Also, since the exceptional products are recovered from some crosses but not from others, a question is raised as to what genetic elements might be involved in determining this process.

Unlike the results reported by GREEN (1959), three different deficiency products and one duplication type have thus far been recovered from various crosses during the course of a study involving the spatial relationships between some white homoalleles and heteroalleles. A detailed genetic and cytological analysis

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of each exceptional recombinant has been carried out in an effort to determine its nature and mechanism of origin.

MATERIALS AND METHODS

A description of most of the mutants employed in this study may be found in BRIDGES and BREHME (1944). A list, along with symbols and map positions, is given in Table 1. Other special stocks are described in the text.

The flies were reared at 24°C on a standard Brewer's yeast, cornmeal, molasses, agar medium.

For the cytological analysis, larvae were reared at 20°C in uncrowded, yeast supplemented vials. Salivary glands were fixed in 1N HCl for 20 seconds, transferred to distilled water until swelling and clearing occurred and then transferred into a few drops of stain on a silicon-treated slide. The stain was two percent orcein in equal parts of glacial acetic acid and lactic acid (85%). A silicon-treated cover slip was placed over the glands and smearing was done by striking the cover slip sharply with a small rubber hammer. After blotting, the preparation was sealed with a mixture of paraffin and lanolin.

TABLE 1

A list of mutants used in this study. For more detailed descriptions, see BRIDGES *and* BREHME *1944*

Mutant symbol	Phenotype	Map position
y	yellow body and bristle color	X 0.0
y^2	yellow body; black bristles	X 0.0
sc	missing scutellar bristles (scute)	X 0.0+
Hw	Hairy wings	X 0.0+
z	zeste (yellow) eye color	X 1 ±
w	white eye color	X 1.5
w^{11E4}	white eye color	X 1.5
w^a	apricot eye color	X 1.5
w^{bf}	buff eye color	X 1.5
w^{Bwx}	brown eye color	X 1.5
spl	split bristles	X 3.0
N	Notch wings	X 3.0
dm	diminutive bristles	X 4.6
ec	echinus (rough) eyes	X 5.5
sn^3	singed bristles	X 21.0
lz^8	lozenge eyes	X 27.7
m^2	miniature wings	X 36.1
g^4	garnet eye color	X 44.4
f	forked bristles	X 56.7
SM1	Curly wings	II chromosome inversions
Ubx^{130}	Enlarged halteres	III chromosome inversions

RESULTS

Analysis of white-deficiency recombinants: Those crosses which yield white-deficiency recombinants (w^{-r}) are listed in Table 2. The parental female genotypes appear in the left-hand column; these females also carried *SM1*/+ and Ubx^{130}/+ in the second and third chromosomes in order to increase crossing over in the X chromosome. The male parents were *y w spl* sn^3 or *y w ec f* in genotype.

In the first two crosses listed, the w^{-r} products appear with regularity, while in the last three the number recovered is not large enough to support such a statement. The last cross, however, also yields a duplication type which appears to be the complementary strand of this w^{-r}; therefore, it is felt that this cross ($y^2 w^{r,dup}$ *ec*/+) along with the first two listed do give rise to asymmetrical exchange products with regularity. The w^a/w^{bf} and w^a/w^{Bwx} crosses are included here because the products recovered did without any doubt arise by crossing over, that is, in association with marker exchange between homologous chromosomes.

The w^{-r} females were originally recognized by their white phenotype and by the sex ratio of 2♀:1♂ among their offspring. Stock cultures were established from each exceptional female by balancing the deficiency against *In(1)*dl-49, bearing the markers *w* lz^s or *y Hw* m^2 g^4.

Each w^{-r} chromosome was subjected to a series of genetic tests as well as a careful cytological examination in order to characterize fully the change brought about by the recombination event. One of the genetic tests involved the use of three different white locus duplications compounded with each w^{-r}. If the duplication covered the deficiency, male w^{-r} offspring were obtained. Since the cytological limits of the duplications are known, coverage or failure to cover gives information concerning the limits of the deficiency in each w^{-r} type.

The duplications, Dp(1;2) w^{51b7} and Dp(1;4) w^{51c20}, described by RATTY (1954), extend over bands 3C2 to 3D2 and 3C2 to 3C6 respectively, using BRIDGES (1938) salivary gland chromosome map as the cytological reference. Dp(1;3) $w^{v,co}$ (BRIDGES and BREHME 1944) covers bands 2C1 to 3C4. The results from these tests are given in Table 3, and serve to separate the w^{-r} chromosomes into two groups. All of the w^{-r} types are covered by Dp $w^{v,co}$, with the exception of the one derived from w^a/w^{Bwx}, which in turn is covered by Dp w^{51b7}; Dp w^{51c20} fails to cover any of the w^{-r} types. This means that w^{-r} from w^a/w^{Bwx} can be de-

TABLE 2

The occurrence of exceptional recombinant females. All parental females were heterozygous for SM1 *and* Ubx^{130}. *Male parents were* y w spl sn^3 *or* y w ec f

Parental female genotype	Exceptional females	Total progeny
y^2 w^a/w^a *spl ec*	4 y^2 w^{-r} *spl ec*	49,884
y^2 w^a *spl ec*/+	6 w^{-r} *spl ec*	65,232
y^2 w^a *spl ec*/w^{bf} f^5	1 y^2 w^{-r} f^5*	63,001
y^2 w^a *spl ec*/*y z* w^{Bwx}	1 y^2 w^{-r} *N*	38,021
y^2 $w^{r,dup}$ *ec*/+	2 w^{-r} *ec*	34,085

* For other recombinant exceptions from this cross, see JUDD 1959.

ficient at a maximum for 3C2 to 3D2. This deficiency must extend through 3C7 to at least 3C10 since it exhibits a Notch phenotype and uncovers the recessive mutant *dm*. Diminutive is placed at 3C9 by SCHULTZ and at 3D1, 2 by DEMEREC (see BRIDGES and BREHME 1944). All the remaining types must be deficient for some part of the region extending from 2C1 to 3C4.

Additional information bearing on the limits of the w^{-r} types has been obtained by compounding them *inter se*, as well as with a series of known deficiencies. The assumption made in this test is that lethality will result if both chromosomes lack a common region. If the two deficiencies do not overlap, females will survive despite the fact that both of their X chromosomes would be lethal in the homozygous or hemizygous state. The deficiencies used as tester chromosomes were: w^{258-11}, deficient for bands 3A3 to 3C3 inclusive; a chromosome derived by crossing over between *In(1)rst*^{3} and *In(1)w*^{m4} which is deficient for bands 3C2, 3; w^{258-45}, deficient for band 3C1.

The results from these combinations are given in Table 4, and again serve to divide the w^{-r} chromosomes into the same two groups distinguished by the previ-

TABLE 3

Tests for coverage of w^{-r} *chromosomes by three white locus duplications. The cytological limits of each duplication are placed in parentheses. + = coverage by duplication; 0 = failure of coverage*

w^{-r} chromosomes	Dp(1;2)w^{51b7} (3C2–3D2)	Dp(1;4)w^{51c20} (3C2–3C6)	Dp(1;3)$w^{V,co}$ (2C1–3C4)
y^2 w^{-r} *spl ec* (from w^a/w^a)	0	0	+
w^{-r} *spl ec* (from $w^a/+$)	0	0	+
y^2 w^{-r} f^5 (from w^a/w^{bf})	0	0	+
y^2 w^{-r} *N* (from w^a/w^{Bwx})	+	0	0
w^{-r} *ec* (from $w^{r,\,dup}/+$)	0	0	+

TABLE 4

Tests of w^{-r} *chromosomes* inter se, *and with three white region deficiencies of known size. L = lethal combination; w = viable white-eyed females*

	w^{258-45} (3C1)	w^{258-11} (2C1–3C4)	$w^{-}rst^{3*}$ (3C2, 3)	y^2 w^{-r} *spl ec* (w^a/w^a)	w^{-r} *spl ec* ($w^a/+$)	y^2 w^{-r} f^5 (w^a/w^{bf})	y^2 w^{-r} *N* (w^a/w^{Bwx})	w^{-r} *ec* ($w^{r,\,dup}/+$)
w^{258-45}	L	L	w	L	L	L	w	L
w^{258-11}	..	L	..	L	L	L	..	L
$w^{-}rst^{3*}$	..	..	L	w	w	w	L	w
y^2 w^{-r} *spl ec* (w^a/w^a)	..	..	..	L	L	L	w	L
w^{-r} *spl ec* ($w^a/+$)	..	..	..	..	L	L	w	L
y^2 w^{-r} f^5 (w^a/w^{bf})	..	..	..	..	..	L	w	L
y^2 w^{-r} *N* (w^a/w^{Bwx})	..	..	..	..	..	..	L	w
w^{-r} *ec* ($w^{r,\,dup}/+$)	..	..	..	..	..	..	..	L

* Derived by crossing over between *In(1)w*^{m4} and *In(1)rst*^{3}.

ously described use of the white duplications. Stricter limits may now be placed on the break points of some of the w^{-r} types, however. All of the w^{-r} losses with the exception of the type obtained from w^a/w^{Bwx} have one feature in common, that is the loss of band 3C1. The w^{-r} from w^a/w^{Bwx} on the other hand lives when compounded with a known 3C1 deficiency or with any of the other w^{-r} types. Interestingly enough, these combinations produce viable females which are white in phenotype.

The information gathered thus far allows the following conclusions to be drawn: (1) The w^{-r} from w^a/w^a, $w^a/+$, w^a/w^{bf} and $w^{r,dup}/+$ all have their rightmost break between bands 3C1 and 3C2, and extend left from that point, but not beyond 2C1. (2) The deficiency derived from w^a/w^{Bwx} has its leftmost break between 3C1 and 3C2 and extends to the right to some point between 3C9 and 3D2.

To delimit the extent of the deficiencies more closely, one other test was employed. Among the group with the missing region extending left from 3C1, it is of interest to determine whether the locus of zeste, located in band 3A3 (GANS 1953), is or is not included in the deficiency. To test for the presence of the zeste locus is not a simple operation since its mutant expression depends on the presence of two normal white regions. It was discovered, however, that an intralocus duplication for part of the white locus derived by crossing over between $w^{r,dup}$ (JUDD 1961) and a wild-type X chromosome, would serve to differentiate between those chromosomes having z^+ and those lacking z^+. The $w^{r,dup}$ chromosome was originally recovered from *y sc z* w^a *ec*/y^2 w^{bf} *spl* attached-X chromosomes and represents an intralocus duplication for white. The mutant w^{bf} is present in $w^{r,dup}$ and accounts in part for the "light buff" phenotype. The replacement of w^{bf} with a wild-type allele by crossing over results in a chromosome which still carries the intralocus duplication but which phenotypically is wild type. This crossover product is designated as $w^{r,dup+}$. Cytologically both $w^{r,dup}$ and $w^{r,dup+}$ appear normal; both, however, interact with the mutant *z*. It is this interaction with the zeste locus that allows the test for presence or absence of z^+.

To demonstrate the validity of the test, two of the white deficiencies employed earlier were used. w^{258-11}, which is deficient for 3A3 to 3C3 inclusive, lacks the *z* locus, while w^{258-45} is deficient for 3C1 only and thus has z^+ present. When these two deficiencies were compounded with *z* $w^{r,dup+}$, the class of genotype w^{258-11}/ *z* $w^{r,dup+}$ was zeste in phenotype while w^{258-45}/*z* $w^{r,dup+}$ females were a dark reddish-brown. As a further check on this test, flies of genotypes *z* w^+/*z* $w^{r,dup+}$ and *z* w^{11E4}/*z* $w^{r,dup+}$ were obtained and both proved to be zeste in phenotype, while z^+ w^+/*z* $w^{r,dup+}$ and z^+ *w*/*z* $w^{r,dup+}$ were a dark reddish-brown mottle in eye color. Both *w* and w^{11E4} are suppressors of *z*, just as is a deficiency for the white locus (GANS 1953). It is reasonable to assume then that this test does distinguish between the presence or absence of z^+ regardless of whether the deficiency extends through the white locus.

This same test was performed with each of the w^{-r} chromosomes and served to further subdivide the classes. The w^{-r} chromosomes obtained from w^a/w^a lack

the zeste locus while those from $w^a/+$, w^a/w^{bf}, $w^{r,dup}/+$ all have it present, as of course does w^{-r} derived from w^a/w^{Bwx}.

Taking into consideration all of the genetic tests, three categories may be established among the w^{-r} chromosomes. (1) The w^{-r} from w^a/w^a is deficient for 3A3 to 3C1 inclusive. The left break is not established with certainty by these tests but must lie between 3A3 and 2C1. (2) w^{-r} chromosomes derived from $w^a/+$, w^a/w^{bf} and $w^{r,dup}/+$ have the deficiency extending from 3A4 to 3C1 as a maximum. Again, the left break is not definitely established by the genetic tests, but it is clearly different from that of w^{-r} recovered from w^a/w^a. (3) The w^{-r} from w^a/w^{Bwx} is deficient for 3C2 to 3D2, with the right break not established with certainty.

It should be noted that all three types of recombinant chromosomes have one break in common, that is, between 3C1 and 3C2; also, that any given cross yields but a single type of deficiency. With these results in mind, it was decided to do a detailed cytological study of each of the exceptional chromosomes in order to more accurately define the break points.

Cytological analysis of white-deficiency recombinants: The cytological studies served to confirm the categories established through the genetic tests and allowed a more definite establishment of break points. Despite a very careful analysis, no differences were found among the deficiencies recovered from any given cross. Utilizing the data collected from both the genetic and cytological studies, the limits of the three different w^{-r} types may now be stated as follows:

w^{-r} from w^a/w^a extends from 3A3 to 3C1 inclusive;

w^{-r} from $w^a/+$, w^a/w^{bf} and $w^{r,dup}/+$ extends from 3A8 to 3C1, with the left break somewhat uncertain due to the very faint bands present in this area;

w^{-r} from w^a/w^{Bwx} extends from 3C2 to 3C12, with 3D1,2 uncertain.

Photomicrographs of representatives of each of the three w^{-r} types are presented in Plate 1. Although exact break points cannot be determined from the photographs, they are sufficient to illustrate that the limits of each deficient type are unquestionably different.

Analysis of recombinant duplication: In only one cross, other than the one devised by JUDD (1961) which allows the recovery of the small intralocus white duplication, has it been possible to recover a recombinant chromosome which represents the duplication resulting from asymmetrical exchange. Females of genotype $y^2\ w^{r,dup}\ ec/+$; $SM1/+$; $Ubx^{130}/+$ when mated to $z\ w^{11E4}$ males yield a product, carrying the marker y^2, which is recognized in the female offspring of this cross by its very dark brown, slightly mottled phenotype. Five of these exceptions were recovered among 74,003 offspring. Progeny testing of these exceptional females showed that the recombinant chromosome (designated here as $w^{r,Dp}$) exhibited a wild-type eye color and is male viable. The white duplication, however, interacts with the mutant z to account for the dark brown mottled phenotype of the F_1 female exceptions. It should be noted that the $y^2\ w^{r,Dp}$ chromosome appears to be the reciprocal product of the $w^{-r}\ ec$ derived from the same type of parental females, the analysis of which is reported in the preceding section. Cytological analysis of the five $w^{r,Dp}$ chromosomes shows a duplication

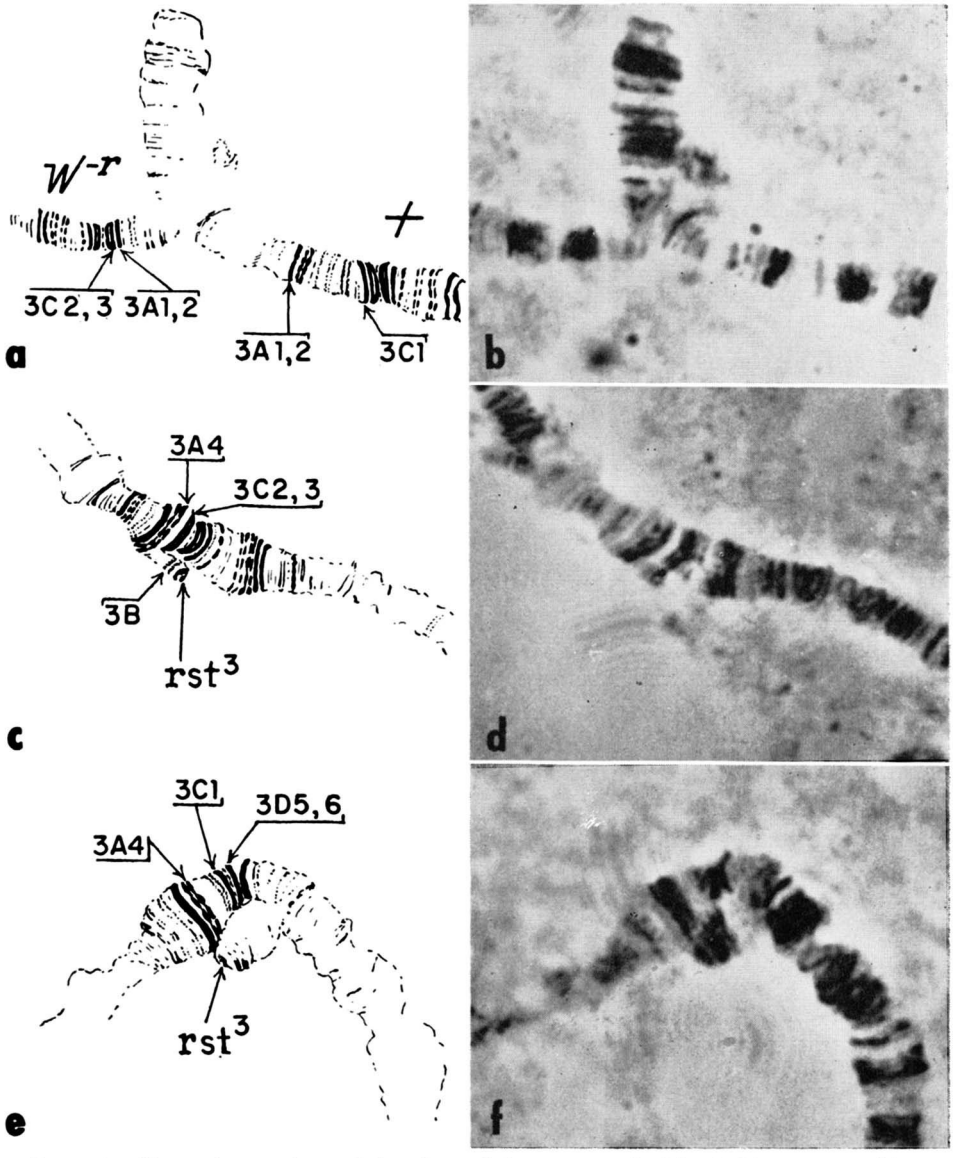

PLATE 1.—Photomicrographs and drawings of three types of white-deficiency recombinants. Sections a and b show w^{-r} from w^a/w^a heterozygous for a structurally normal X chromosome. The two strands are separated from one another with the + strand extending to the right and w^{-r} to the left. Note the absence of bands 3A3 through 3C1 in w^{-r}. Sections c and d show w^{-r} from $w^a/+$ paired with a rst^3 chromosome. Note the absence of region 3A8 through 3C1. Sections e and f show the $w^{-r}N$ chromosome from w^a/w^{Bwx} paired with rst^3. Bands 3C2 through 3C12 are missing in the w^{-r} strand.

which includes 3B1 through 3C1 as a minimum. This corresponds rather closely to the section which appears to be missing in the presumed reciprocal w^{-r} chromosome. Plate 2 is a photomicrograph of one of the $w^{r,Dp}$ chromosomes.

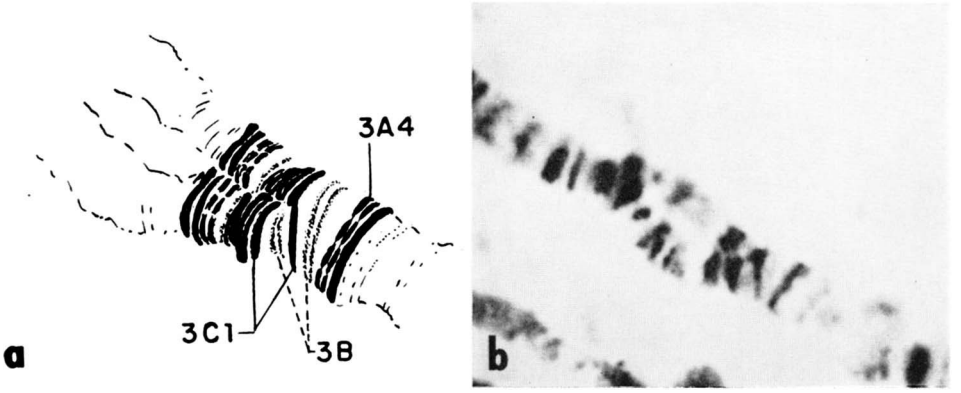

PLATE 2.—Photomicrograph and drawing of $w^{r,Dp}$ from $w^{r,dup}/+$ paired with a structurally normal chromosome. Note the duplication which extends from region 3B through 3C1 as a minimum.

The cytological limits of $w^{r,Dp}$ are similar to those reported by LEWIS (1957) for Dp(1;1) w^{55j}, which appeared as a *y ac* (w^{ch}) (*sp-w*) son of a *y ac* w^{ch} *fa*/y^2 *sp-w* female. The origin of Dp(1;1) w^{55j} could be through asymmetrical exchange similar to the cases described here. The genetic analysis by LEWIS indicated, however, that the locus of roughest is also included in the duplicated segment, therefore, it is probably not identical with $w^{r,Dp}$.

A complete analysis of the $w^{r,dup}$ chromosome from which these more extensive duplication and deficiency recombinant products were derived is presently underway and will be reported at a later time. As has already been reported (JUDD 1961), this product, $w^{r,dup}$, appears to be structurally normal even though a portion of the white locus is duplicated. At this point in the investigation, it appears that this intralocus duplication is having little if any effect on the type of asymmetrical pairing reported here and by GREEN (1959). It is significant that the cross involving the intralocus duplication was the only one which allowed the recognition of the larger $w^{r,Dp}$ exception. This is attributable to the use of the mutant *z* in the male parent of this cross, and one would predict that the reciprocal duplications might be obtained from all of the crosses reported here if the mutant *z* is used as a detection device. It is possible of course that not all of the duplication types will give this interaction with zeste and some other indicator system must be used to recover them.

DISCUSSION

Several interesting facts emerge from this study of recombination products. It is considered significant that only one type of asymmetrical exchange is detected from any given cross, despite the demonstration that exchanges at other points in this region can be recognized. This observation can best be explained by assuming that the non-homologous pairing always occurs in the same way in any given genotype; and furthermore, that exchange between homologs paired

asymmetrically may occur only within some very restricted area. The points of pairing and exchange appear to be different for different genotypes.

GREEN (1959) postulates a pairing scheme which when accompanied by crossing over would yield the w^{-r} type which he describes. Such a scheme involves the pairing of band 3A3 (the *z* locus) with band 3C1 of the homologous chromosome. A similar but not identical pairing arrangement can be used to explain the w^{-r} obtained from w^a/w^a. In this case band 3A3 would pair with band 3C2 of the homolog in the following way:

. . . 3A1,2,3,4 . . . 10 3B1,2 3C1,2,3,4 . . .
3A1,2 . . . 3B1,2 . . 3C1,2,3 . . . . . 12 3D1,2,3,4 . . .

A crossover occurring between 3A2,3 in the upper sequence and between 3C1,2 in the lower would give a chromosome deficient for 3A3 to 3C1 inclusive and the other strand would contain a tandem duplication for this region. All of the known white locus mutations appear to be located in the 3C2,3 doublet which under this scheme is pairing with the closely linked zeste locus (also an eye-color determining locus) at 3A3. This might be taken as evidence that the white and zeste loci evolved from a common ancestral unit and that some pairing homology still remains. Additional evidence for this view comes from the functional relationships which exist between these two loci (GANS 1953).

The pairing scheme above brings the 3C2 band in the upper sequence adjacent to 3D3 in the lower sequence if a band for band pairing along the length of this chromosome segment is assumed. A crossover between 3C1,2 and 3D2,3 would give a chromosome deficient for 3C2 to 3D2 inclusive. These limits correspond rather closely to those of the w^{-r} derived from w^a/w^{Bwx}.

In order to explain the exceptional recombinants from $w^a/+$, w^a/w^{bf} and $w^{r,dup}/+$, a pairing arrangement quite different from the one just described must be assumed. In this case, the deficiency extends from about 3A8 to 3C1. The pairing which would most simply give this type of crossover product is as follows:

. . . 3A1,2 . . . 6,7,8,9,10 3B1,2 . . 3C1,2 . . .
3A1,2,3 . . 3B1 . . 3C1,2,3 . . . .

A crossover between 3A7,8 in the upper sequence and between 3C1,2 in the lower would give duplication and efficiency products with break points at 3A8 and 3C1. It is of course quite likely that the salivary gland chromosome bands do not accurately portray the chromosome region as it is at the time crossing over occurs. Nonetheless, it is difficult to reconcile the origin of all of the w^{-r} chromosomes with a single asymmetrical pairing scheme, unless one assumes that this chromosomal region is lengthened at prophase by a genetically inert substance such as intercalary heterochromatin. PROKOFYEVA-BELGOVSKAYA (1939) notes the possibility of heterochromatin in the 3C region, based on the cytological observation that inert regions tend to pair with one another. Under these conditions, exchanges which occur within some relatively extensive region would all appear to have taken place at the same point genetically and on the salivary gland chromosome. Such a scheme would tend to group the exchanges

at a few sites. This still would not explain why all of the exceptions from any single cross appear to be identical however.

GREEN (1959) attempted to evaluate the genetic factors involved in the determination of the occurrence of w^{-r} chromosomes and came to the conclusion that the mutant apricot or a related mutant was necessary for the origin of the w^{-r} losses. It should be pointed out that all but one of the crosses used in this present study involved the mutant apricot. More than 20 other crosses, some also using apricot, failed to give w^{-r} types.

GREEN also states that the genetic state of the *z* locus, at least in the w^{a2} chromosome he used, plays an important role in the mechanism of origin of w^{-r} chromosomes. This point also can be supported by the present study by citing the following example. Females of genotype y^2 w^a *spl ec*/w^{Bwx} yield the reciprocal crossover types expected from heteroalleles, i.e., y^2w^+ and w^{Bwx} w^a *spl ec*, and in addition a y^2 $w^{r,def}$ class (JUDD 1959) which represents an intralocus deficiency similar to that reported by JUDD (1961). Presumably the reciprocal intralocus duplication class also occurs but was not recognized. If, however, the mutant *z* is placed in the w^{Bwx} chromosome and females of genotype y^2 w^a *spl ec*/*y z* w^{Bwx} are constructed, the intralocus deficiency (y^2 $w^{r,def}$) class is not recovered; instead as reported here, a y^2 w^{-r} *N* type deficient for 3C2 to 3C12 is found. Presumably the duplication product reciprocal to the w^{-r} also occurs but goes undetected because of the way the cross was performed. The substitution of the mutant *z* into the w^{Bwx} chromosome influences the type of asymmetrical pairing in a very dramatic way. This points once more to genetic control of pairing and crossing over, and implies that even asymmetrical pairing is still quite specific.

It is not possible at this point to determine which if any of the w^{-r} types described here corresponds to the one reported by GREEN (1959). His analysis showed that the z^+ locus was present in the w^{-r} chromosome; therefore, it is similar to the type which is deficient for 3A8 to 3C1, obtained from $w^a/+$, w^a/w^{bf} and $w^{r,dup}/+$. Since GREEN did not carry out a cytological examination, it remains to be determined whether these two types are identical.

The regular and relatively frequent occurrence of rather extensive duplication and deficiency products by crossing over provide changes in chromosome structure which are of considerable significance to the evolution of this organism. The deficiencies obviously will be selected against and rather rapidly eliminated. The duplications on the other hand, if we may judge by the type which has been recognized, are completely viable and fertile and for the most part not recognizably different from their normal sibs. The presence of a duplication, however, allows sites for the occurrence of new mutations without doing away with gene functions which might be indispensable. Such duplications could add immeasurably to the variability within a population without seriously affecting that part of the genome which is already selectively adapted. Evolutionary changes in such a population could be more rapid, and the types of mutations tested by natural selection could be larger in number and more varied in type.

It is considered quite plausible that the recombination events described here

will be found in a variety of organisms and will prove to be an important factor in evolution.

SUMMARY

Three types of white deficiencies and one type of white locus duplication which apparently arise by crossing over within an asymmetrically paired chromosome region are described by genetic tests and from cytological observations. Only a single type of asymmetric exchange occurs in any given cross.

Pairing schemes are presented which will allow the formation of the exceptional recombinants. It is concluded that the asymmetric pairing is genotypically controlled and is quite specific.

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