GENETIC EFFECTS ASSOCIATED WITH BAND 3C1 OF THE SALIVARY GLAND X CHROMOSOME OF *DROSOPHILA MELANOGASTER*¹

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IN a previous paper (GERSH, 1962) attention was drawn to the uncertain attributes of band 3C1 (BRIDGES 1938) of the salivary gland chromosome of *Drosophila melanogaster*. A study of the pertinent literature shows a state of confusion due to errors which have been made and compounded. I shall attempt to dispose of these errors and to present a revised description of this band's properties.

GREEN (1963) stated that "cytogenetic investigations have established the association of the white mutants with the salivary gland chromosome bands 3C1, 2 and 3. . . ." He did not say so, but it would seem from previous papers that his reason for including 3C1 as a site of part of the white (*w*, eye color) locus is based on salivary analyses made by SLIZYNSKA (1938) and SUTTON (see BRIDGES and BREHME 1944) of three X-ray induced white mutants, *w*^{258.14}, *w*^{258.45} and *w*^{258.48}. All three changes were accompanied by male-lethality and were associated with deficiencies which seemed to include 3C1 but not 3C2. One, *w*^{258.45}, was recorded by SUTTON as a deficiency for one band only, namely, 3C1.

In recent intensive work on the *w* locus, (GREEN 1959; JUDD 1961) this deficiency, *w*^{258.45} has been used as a tester for the presence of band 3C1, while a deficiency for 3C2.3, also male-lethal, has been used as a tester for the presence of the doublet 3C2.3. It has been assumed that male-lethal deficiencies which survive in females whose other X chromosome is deficient for 3C2.3, but not in females with the *w*^{258.45} chromosome, share in common "the loss of band 3C1 and the presence of bands 3C2.3." (GREEN 1959).

LEFEVRE and WILKINS (1966), however, provide evidence that a deficiency for the single band 3C2 is male-viable and that deficiency for 3C3 alone is male-lethal; and further, that the *w*^{258.45} chromosome lacks 3C2 as well as 3C1. This means that a lethal effect covered by the 3C2.3 deficient chromosome, but not by *w*^{258.45}, may lack 3C2 as well as 3C1. It also explains, in a simple way, the fact that the *w*^{258.45} and 3C2.3 deficiencies complement each other's lethal effects, but do not complement for the *w* effect. This failure of complementation led BEERMANN (1962) to suggest that 3C1 was an operator for the structural *w* locus at 3C2, so that the heterozygote for the double deficiency, with the operator and structural locus in the *trans* position, was ineffectual. But if both chromosomes

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lack 3C2, with which the w locus is associated, then the w phenotype would simply be due to a homozygous deficiency for the w locus.

GREEN and JUDD have both described deficient X chromosomes obtained by unequal crossing over in the zeste-white region. They have lethal effects which are covered by the 3C2.3-deficient chromosome, but not by $w^{258.45}$. Although the chromosomes from which they were derived both carried *intermediate* alleles at the w locus, the combination of the new deficiency with the 3C2.3 deficiency was white. Both authors assumed that 3C2 was present in these chromosomes. Again, a simpler explanation is that 3C2, or part of it, was included in the new deficiencies.

Nevertheless, though LEFEVRE and WILKINS have made it clear that 3C2 is the site of w alleles, deficiency for which is nonlethal, it has not yet been proven incontestably that *part* of the w locus is not associated with 3C1. Moreover, although these authors attribute the male-lethality of $w^{258.45}$ to the 3C1 locus, this conclusion is not clear-cut, either. First, RATTY (1954) stated that the $w^{258.45}$ chromosome contained an independent sex-linked lethal factor in addition to the deficiency for 3C1; and second, LEFEVRE and WILKINS found a second small deficiency, to the left of 3C1, in their $w^{258.45}$ stock.

Questions to be answered, therefore, are: (1) Is any part of the w locus associated with 3C1? (2) Is deficiency for 3C1 male-lethal, or is this effect in $w^{258.45}$ due to an independent lethal, possibly a second deficiency? Another pertinent question would be: (3) Can any other genetic effect be attributed to a locus in 3C1?

In fact, another effect *has* been attributed to the 3C1 locus. This is suppression of the mutant zeste (z , eye color) first discussed by GANS (1953). GANS showed that two doses of w^+ (or a very closely linked locus) were necessary for the expression of the z phenotype, and that some mutant alleles of the w locus were suppressors of z . She assumed that the w locus was in band 3C1—partly because of my analysis of $w^{258.45}$ as a deficiency for that band alone, partly because she found that w is covered by duplication 20G1L, which she believed to include 3C1 but not 3C2. It has been shown, however, (GERSH, unpublished) that this duplication includes 3C2.3.

The only other stock used by GANS which was supposed to involve a change in dosage for 3C1 but not 3C2 was a duplication, $Dp(1-1)z^4$, "probably involving a tandem reversed repeat of bands 3C1 to 2C1" (p. 23; my translation). The order of bands is given as (0-3B)-(3C1-2C1)-(3C1-centromere), but in the drawing of this duplication (Figure 5b, p. 25) as in Table V, p. 36, GANS suggests that not only 3C1, but also 3C2.3 may be duplicated. Thus, as all her duplications and deficiencies for 3C1 may also have involved 3C2, it is impossible to say that the suppressor-zeste effect was due to loss of the one rather than the other.

GREEN (1959) based conclusions as to 3C1 on work with $Dp(1-1)z^4$, with $w^{258.45}$, and with deficiencies which were also assumed to be normal for 3C2, based on tests with $w^{258.45}$. He followed this up in 1961 by discussing new genetic data which were acquired by using a duplication (referred to as $Dp(1)3C1$, and referred to by GERSH [1962] as $\gamma ac z Dp(1;1)w^{60h21}$) whose "precise limits are

not clear except that 3C1 is duplicated" (GREEN 1961). Although in my 1962 paper I made a case for the extent of the duplication being 3A4-3C1, consideration shows that it could equally well be 3A5-3C2. Again, it is not at all clear that there is a suppressor-zeste effect due to 3C1, distinct from that due to the *w* locus at 3C2. We can follow our third question with a fourth: (4) Specifically, does 3C1 modify the expression of zeste?

MATERIALS AND METHODS

The following symbols for mutants will be used:

<i>ac</i> : achaete (bristles)	<i>rst</i> : roughest (eye surface)
<i>bb</i> : bobbed (bristles)	<i>sc</i> : scute (bristles)
<i>car</i> : carnation (eye color)	<i>spl</i> : split (bristles)
<i>fa</i> : facet (eye surface)	<i>su-w^a</i> : suppressor of white-apricot
<i>l</i> : recessive lethal	<i>w^a</i> : white-apricot (eye color)
<i>N</i> : dominant Notch (wing)	<i>w^m</i> : white-mottled (eye color)
<i>pn</i> : prune (eye color)	<i>y</i> : yellow (body color)

The following are symbols for chromosome rearrangements:

Df(1): deficiency in the X chromosome
Dp(1): duplication of X chromosome bands
In(1): inversion in the X chromosome
Tr, *TR*: triplication

The following special chromosomes were used in experiments:

Dp(1)20*G1L* (GANS 1953), a duplication consisting of the left end of X (bands 1A1 to 3C2) translocated to chromosome 4.
In(1)*w^m* (LEFEVRE and WILKINS 1966), an X chromosome inversion with breaks (1) to the right of *w* and (2) to the left of *bb*, and extending from 3C3 to 19. The inverted 3C3 is attached to chromosome 4 at 102C, the X centromere and tip of 4R are lost.
In(1)*w^m* (BRIDGES and BREHME 1944), an X chromosome inversion with breaks (1) to the left of *w* and (2) to the left of *bb*, extending from 3C2 to about 20B.
In(1)*w^m51b19*, unpublished, an X chromosome inversion, obtained by DR. W. R. BAKER, with breaks (1) to the left of *w* and (2) to the right of *bb*, and extending from 3C2 to about 20D.
 Flies were raised at 25° ± 1°C and when comparisons were to be made the flies were raised simultaneously on the same batch of food.

EXPERIMENTAL RESULTS

The lethality of w^{258.45}: In the work reported here, a *w^{258.45}* stock from the University of Chicago was used. It probably came in turn from the Oak Ridge laboratory. The *w^{258.45}* X chromosome from this stock appeared to differ from that of LEFEVRE and WILKINS in being normal in the 3A-B region, and to resemble it in being deficient for 3C1 and 2 (Figure 1). The male-lethal effect of this chromosome is not covered by *Dp*(1;3)264.58 (RATTY's *Dp^m*(*w-N*)1, a duplication for bands 3B3.4 to 3D5.6 inclusive), but is covered by *Dp*(1)20*G1L*. Assuming that there is a lethal to the left of 3C1 (covered by 20*G1L*) and beyond the limit of *Dp*(1;3)264.58, i.e., to the left of 3B3, it should be possible to separate the 3C1, 2 deficiency from this lethal by crossing over, and one might expect that the 3C1, 2 deficiency would then be covered by *Dp*(1;3)264.58.

An attempt was made to obtain a recombinant deficiency 3C1, 2 chromosome of this type from females of constitution *y w^{258.45}/w^a fa spl* mated to *y w* males.

FIGURE 1.—Photomicrograph of X-chromosome tip from salivary gland of $w^{258.45}/+$ larva. $\times 3,100$.

From a total of about 18,000 offspring, 42 daughters inherited a γ^+w recombinant chromosome. When the females were mated with $w/Y; Dp(1;3)264.58/+$ males, this chromosome was never recovered, with or without the duplication, in their sons: the crossovers did not eliminate the lethal effect which the duplication fails to cover.

Two explanations of this result may be offered. One is that there is indeed an independent lethal to the left of 3C1, and the two are so closely linked that the crossover between them was not recovered in the experiment. The frequency of recombination in the hypothetical lethal-white interval would be less than 0.005%. The true map distance would, however, be greater, for crossing over in the $\gamma-w$ region was obviously reduced. A total of 42 recombinant chromosomes among the daughters, with an equal number of the complementing class and a corresponding number among the sons which would be lost owing to lethality, gives a crossover value of about 0.9%, as compared with the standard 1.5%.

If this explanation were correct, the lethal in the Chicago $w^{258.45}$ would appear to be different from that in RATTY's stock, for it is very closely linked to w and is covered by $Dp(1)w^{Vco}$ (2C1-3C4), whereas RATTY's lethal was not covered by his $Dp^+(pn-rst)$ (1E3.4-3C6) and presumably would have crossed over with w more readily. The hypothetical lethal thus acquires a somewhat will-'o-the-wisp quality.

Alternatively, there may not be an independent lethal in this $w^{258.45}$ chromosome. The failure of $Dp(1;3)264.58$ to cover the lethal effect in 3C1 may be due to a weakening of dominance of the normal allele at 3C1 in the duplication. This would not be at all surprising in view of the fact that $Dp(1;3)264.58$ did not cover the deficiency for the corresponding region in the duplication-deficiency stock in which it arose (see $N^{264.58}$, BRIDGES and BREHME 1944). Following William of Occam's principle, this explanation should be preferred.

Visible effect of change at 3C1: The first clue to another effect of the 3C1 locus was obtained from the following considerations. $w^{258.45}/Y; Dp(1)w^{Vco}$ males are viable, and depart from wild-type only in having mottled eyes (a variegated position effect at the w locus, owing to insertion of the duplication into a heterochromatic region). Whereas the 3C1 locus in the duplication obviously functions efficiently in covering the lethality and any visible effects of the 3C1 deficiency, the w locus in the duplication does not cover the 3C2 (w locus) deficiency in all cells, hence the mottling of the eyes. The efficiency of the w^+ allele in the duplication is decreased in the absence of a Y chromosome, with the result that more facets in the eye show a mutant phenotype. The absence of a Y might also decrease the efficiency of the nearby 3C1 locus, possibly resulting in a sublethal effect accompanied by visible abnormalities.

The mating of $w^{258.45}/Y; Dp(1)w^{Vco}/+$ males with $\gamma^2 su-w^a w^b bb \cdot w/0$ females (with attached-X chromosomes and no Y) yielded sons with pale mottled eyes and occasional reduction or loss of one or more of the following bristles: orbitals (anterior, median or posterior), ocellars, postverticals, verticals (anterior or posterior), dorsocentrals (anterior or posterior), scutellars (anterior or posterior). The average number of affected bristles per fly was more than three. Though these males lacked a Y chromosome as indicated by their pale eyes and sterility, the bristle effect was not due to the absence of a Y chromosome *per se* (other X/0 males such as $\gamma w/0$ and $w^{m4}/0$ were carefully examined and found to have normal bristles). Nor was it due to the presence of $Dp(1)w^{Vco}$ ($w^{258.45}/Y/w^{Vco}$ had normal bristles), nor to the combination of these two ($\gamma w/0/w^{Vco}$ males had normal bristles). These were contributing causes to the manifestation of the effect, but the $w^{258.45}$ chromosome was also necessary for the effect.

If this phenotypic character was due to partial or local uncovering of the 3C1 deficiency, it should also be obtained if another 3C1 deficiency chromosome were used. $Df(1)w^{258.48}$ is such a chromosome, with a deficiency for 3B1 to 3C1 (BRIDGES and BREHME 1944) and probably also, like $w^{258.45}$, for 3C2. Its bristle phenotype is complicated by the fact that the stock is $\gamma sc^5 w^{258.48}$. The sc^5 allele is said to cause "little bristle reduction except of sternites and scutellars" (BRIDGES and BREHME); nevertheless, ocellars, verticals and postverticals (these most

markedly) were found to be affected in males of constitution $w^{258.48} \gamma sc^5/Y/w^{Vco}$. Similar males lacking a Y ($w^{258.48}/0/w^{Vco}$) had many more bristle defects, however, as shown in Table 1. Thus it seems highly probable that the 3C1 locus is responsible for this bristle effect.

3C1 and zeste: Table 2 shows some of the phenotypes recorded by GANS, GREEN and JUDD for genotypes involving zeste with different doses of 3C1.

As noted in the introduction to this paper, GANS' comparison of $w^{258.45}$ with + (lines 1 and 2) is ambiguous, because changes in dosage of both 3C1 and 3C2 are involved.

JUDD's data (lines 4 to 6) provide a stepwise series from w^+ through w^{11E4} (zeste-suppressive w mutant as shown in line 3, 3C1 normal) to $w^{258.45}$ (3C1 and 2 missing). The phenotypes are not distinguishable, although one might expect the two latter, with zeste-suppressors present, to be wild-type, rather than intermediate between wild and zeste.

Although it would seem, from a comparison of the duplications and triplications in lines 7 to 10, that maroon-variegated is converted to zeste by an extra dose of either 3C1 or w^+ , there is a possibility that $zTr(1)z-w$ has both, so that w^+ could be the effective agent in both triplications, and 3C1 might be ineffective.

Taking advantage of GREEN's zTR , which is known to involve only the w locus (3C2 or 3) but not 3C1, and which produces a zeste phenotype in combination with $z^+ w^+$, I have obtained a series somewhat comparable to JUDD's, namely, $z^+ w^+/zTR$, $z^+ Df(w)/zTR$ and $z^+ Df(3C1), Df(w)/zTR$. The w deficiency involved band 3C2 only, and was obtained by combining the left end of $In(1)w^{m51b19}$ (break between 3C1 and 2) with the right end of $In(1)w^{mJ}$ (LEFEVRE and WILKINS, break between 3C2 and 3). This chromosome will be designated $Ins(1) L+R$. The combined 3C1 and w deficiency was $w^{258.45}$.

The results were the following:

$z^+ w^+/z TR$	—zeste
$Ins(1) L+R, z^+ w^-/z TR$	—maroon variegated
$z^+ w^{258.45}/z TR$	—sprinkling of dark flecks on zeste background.

Comparing these results with JUDD's in Table 2, it is clear that adequate dosage of the zeste-enhancing w locus alleles may be critical in permitting differentiation of heterozygous z^+/z phenotypes. The results also suggest that deficiency for

TABLE 1

Comparison of $\gamma sc^5 w^{258.48}$ males with $Dp(1)w^{Vco}$ and with or without a Y chromosome

Genotype	Flies scored	Flies with one or more specified bristles reduced or missing						Average affected bristles per fly
		Ocellars	Post- verticals	Anterior verticals	Posterior verticals	Dorso- verticals	Scutellars	
$\gamma sc^5 w^{258.48}/Y; w^{Vco}/+$	20	1	12	1	5	0	20	4.25
$\gamma sc^5 w^{258.48}/0; w^{Vco}/+$	20	20	12	18	15	11	20	9.65

TABLE 2
Variation in expression of the zeste phenotype as a function of dosage at the w and/or 3C1 loci

Genotype	Bands extra or missing	Bands questionable	z alleles		w alleles		Phenotype	Reference
			z ⁺	z	3C1	not su-z		
1. z ⁺ w ⁺ /z Dp(1-1)z ⁺	+ 2C1 — 3C1	+ 3C2, 3 ?	1	2	3	2 (73) 0	wild type	GANS 1953
2. z ⁺ w ^{258.45} /z Dp(1-1)z ⁺	— 3C1, 2 + 2C1 — 3C1	+ 3C2, 3 ?	1	2	2	1 (22) 0	nearly zeste	GANS 1953
3. z w ^{11E4} /z w ⁺	none	none	0	2	2	1 1	wild type	GANS 1953
4. z ⁺ w ⁺ /z w ^r , dup ⁺	+ 3C2	none	1	1	2	3 0	mottled reddish-brown	JUDD 1961
5. z ⁺ w ^{11E4} /z w ^r , dup ⁺	+ 3C2	none	1	1	2	2 1	mottled reddish-brown	JUDD 1961
6. z ⁺ w ^{258.45} /z w ^r , dup ⁺	— 3C1, 2 + 3C2	none	1	1	1	2	mottled reddish-brown	JUDD 1961
7. z ⁺ w ⁺ /z Dp(1)z-w	+ 3A4 — 3C1	+ 3C2, not 3A4 ?	1	1	3	2 (73) 0	maroon variegated	GREEN 1963
8. z ⁺ w ⁺ /z DP	+ 3C3	+ 3C2, not 3C3 ?	1	1	2	3 0	maroon variegated	GREEN 1963
9. z ⁺ w ⁺ /z Tr(1)z-w	Tr 3A4 — 3C1	Tr 3C2, not 3A4 ?	1	1	4	2 (24) 0	zeste	GREEN 1963
10. z ⁺ w ⁺ /z TR	Tr 3C3	Tr 3C2, not 3C3 ?	1	1	2	4 0	zeste	GREEN 1963

3C1 does not suppress the zeste effect: on the contrary, it seems to counteract the suppressive effect of deficiency for 3C2.

One reservation must be made about all these results: the stocks used were not coisogenic. But if deficiency for 3C1 suppresses *z*, there must be a powerful accumulation of modifiers in the $w^{258.45}/z$ *TR* flies to counteract almost completely the suppressive effect of deficiency for both 3C1 and the *w* locus.

In order to throw further light on this question, an attempt was made to obtain a number of new deficiencies for 3C1 and to see whether they had a consistent effect on the zeste phenotype.

The method used was to irradiate *Ins(1) L+R*, z^+w^- males with 2500r of X rays and mate them to *zTR* females. Any females among the progeny that approximated to zeste in eye color were saved and mated to γ *w* males. If the irradiated X chromosome proved to be male-lethal, a stock was established for further testing, as described below.

There were several reasons for using this particular procedure: (1) It would have been desirable to obtain a deficiency for 3C1 without affecting 3C2. This could be done by irradiating an X chromosome such as that of *In(1)w^m*, in which these two bands are widely separated by a previous rearrangement. This is why the inversion chromosome was selected. (2) *In(1)w^m* is accompanied, however, by a V-type position effect of the *w* locus. As GANS has shown, this would act as a suppressor of zeste whose effect could not be overcome by additional doses of w^+ . Ultimately, this would affect the possibility of testing interaction of 3C1 and *z*. On the other hand, the *w* deficiency in the combination *Ins(1) L+R*, $z^+ w^-/z$ *TR*, as already shown, allows partial expression of zeste, and modifications of the phenotype in either the plus or minus direction should be detectable. The inversion would still serve the purpose of limiting the changes obtained at 3C1 so that they might involve bands of 3B and regions further to the left, but would not involve bands to the right of 3C2. (3) In selecting modifications towards zeste rather than towards wild type, the assumption was made that the zeste-like phenotype of $z^+ w^{258.45}/z$ *TR* reflected an enhancing effect of deficiency for 3C1. If this should prove to be untrue after all, the method still held a good chance of screening out some 3C1 deficiencies, specifically those that extended to the left at least as far as 3A3, so as to remove the z^+ allele from the *Ins(1) L+R*, $z^+ w^-$ chromosome. Flies of the constitution *Ins(1) L+R*, $z^- w^-/z$ *TR* would probably be zeste-like in appearance, even though they would be deficient for 3C1, 3C2 and other bands.

When a stock was established from a zeste-like deviant whose irradiated X proved to be male-lethal, it was tested by mating females of constitution *Ins(1) L+R*, l/z *TR* with $w^{258.45}/Y$; $w^{vco}/+$ males. The appearance of white-eyed daughters indicated that the new lethal was not allelic to that of $w^{258.45}$; the stock was then discarded. Failure to obtain white-eyed daughters indicated that the lethals were allelic and that the 3C1 locus was affected. The stock was then retained and examined cytologically.

The eye color of daughters of the irradiated males varied considerably, from almost wild-type to zeste. More than 100 relatively light-eyed *Ins(1) L+R/z* *TR*

females were tested and the great majority of them produced viable, white-eyed and otherwise normal sons. Nine of the females produced only z sons, however, and one produced one white-eyed son, which was so abnormal (reduced bristles, notched wings, sterile) that the stock was maintained.

Of the nine females mentioned, seven proved on testing with $w^{258.45}$ to carry lethals which were not allelic to the 3C1 locus, while two had X chromosomes which were lethal in combination with $w^{258.45}$. These two, and the tenth female, will now be described.

Female No. 1, $l(1)3C1^{65g}$, whose eyes were considerably mottled and lighter than those of its sibs, gave rise in later crosses to females whose eyes were indistinguishable from those of $Ins(1) L+R, z^+ w^-/z TR$. Cytological examination revealed an inversion extending approximately from 12D to 19A, but 3C1 appeared to be unaltered. Males with this X chromosome and $Dp(1)w^{vco}$, but no Y, showed bristle abnormalities similar to those described above for $w^{258.45}/0$; $w^{vco}/+$, with an average of 5.1 affected bristles per fly. Females $l(1)3C1^{65g}/w^{258.45}$; $w^{vco}/+$ also showed occasional missing bristles (average 1.3 per fly).

Female No. 2, $l(1)3C1^{65l}$, had eyes with minute dark flecks on a zeste background. In later crosses the eyes remained flecked but the background tended to be darker, especially after aging. Cytological study indicated a deficiency for 3C1, but it must be emphasized that analysis was difficult and doubtful because of the inversion and proximity of the nucleolus-forming region. No other heavy bands were missing. Presence or absence of 3B3.4 was not determined, nor was it possible to tell whether there was any loss of heterochromatic material from the inverted region adjacent to 3C1. Males with this X chromosome and $Dp(1)w^{vco}$, but no Y, have normal bristles. Whether this is really a deficiency for 3C1 is, therefore, very much in question.

Female No. 3, $T(X;3)3C1$ had eyes resembling those of female No. 2, but in later crosses the eye color of females heterozygous with $z TR$ was very variable, the background being sometimes close to zeste, sometimes darker, and the mottling sometimes speckled and sometimes blotchy. Cytological analysis showed that the left end of the X chromosome had been translocated to the proximal heterochromatic region of 3R (81) and had been replaced by the whole of 3R from 82 to 100. Females heterozygous for this rearrangement and $w^{258.45}$ are viable and normal in appearance except for their white eyes. Not many of the translocation males survive. Those that do are sterile, often have deformed wings, and sometimes show missing or reduced (Minute-like) bristles. This sometimes shows clearly as a variegated effect on the thorax. Some of the males have no visible abnormalities (except white eyes), but are sterile. Males with $Dp(1)w^{vco}$ and no Y usually have some missing bristles (average 3.6 per fly). Males lacking both a Y and the duplication are inviable.

It seems highly relevant that all three of the flies described above were selected on the basis of their light eye color, without benefit of a deficiency for zeste. (One of the nine females with male-lethal changes in the X chromosome did turn out to have a deficiency for zeste, but it did not extend through 3C1, for it was not lethal in combination with $w^{258.45}$). At the time they were picked up, the chromo-

some of these flies were more properly comparable to those of their darker-eyed *Ins(1) L+R/z TR* sisters than at later stages after the establishment of the stocks. The apparent tendency to darkening of the eye may have been brought about by selection of modifiers in the intervening time.

On the basis of these results it certainly seems safe to conclude that deficiency for 3C1 does not suppress *zeste*: it seems, on the contrary, to enhance the *z* effect, while non-deficient mutants at the 3C1 locus, *l(1)3C1^{65g}* and *T(X;3)3C1*, have a less marked effect in the same direction.

Relation of 3C1 to the white locus: It was now possible to do an experiment which should determine whether 3C1 is functionally related to the *w* locus. In other words, are defects in 3C1 accompanied by impairment of the pigment-producing system of the eye?

In *T(X;3)3C1*, the 3C1 locus is affected but not always to a lethal degree. The effect of the *w* locus deficiency at 3C2 can be covered by means of a duplication that includes 3C2 but not 3C1. In males of the constitution *T(X;3)3C1/Y/Dp*, any effect of 3C1 on eye pigmentation should be expressed in a variable form, corresponding to the degree of manifestation of the bristle character.

Dp(1;2)w^{51b7} is a duplication of bands 3C2 to 3D5.6, inserted into the second chromosome (LEFEVRE 1952). We can be sure that it does not include band 3C1, because it resulted from irradiation of *In(1)w^{m4}*, in which 3C1 is widely separated by inversion from 3C2 and the bands following it. It was described by LEFEVRE (1951) as a nonmottled duplication, and indeed, in the combination *Df(1)N⁵⁴¹⁹, γ w^a N/Y; Dp(1;2)w^{51b7}/+* the eye color is completely wild-type. In some respects, however, the *w⁺* allele still behaves, according to its origin from *In(1)w^{m4}*, as a V-type position effect. For instance in *z TR* males it suppresses the *zeste* effect, producing a maroon mottled eye color. In *Ins(1) L+R, z⁺ w⁻/Y; Dp(1;2)w^{51b7}/+* males the eyes are very slightly mottled with darker flecks. *T(X;3)3C1/Y; Dp(1;2)w^{51b7}/+* males show the same type of mottling, with no perceptible difference from fly to fly. There is thus no reason to believe that the 3C1 locus has any effect on pigmentation of the eye such as one would expect if it were the site of part of the *w* locus.

Conclusions: The foregoing results indicate that 3C1 is the site of a distinct locus, loss or change of which has the following results: (1) The deficiency acts as a recessive lethal. (2) Those flies which, under certain conditions such as coverage by a duplication, survive with an altered 3C1 locus, tend to have some bristles missing or reduced. (3) Changes at the locus counteract the suppression of *zeste* caused by loss of a *w⁺* allele, at least in *z⁺/z* heterozygotes where the expression of *zeste* is made possible by the presence of a minimum of three doses of *w⁺*.

The conclusion, previously arrived at only tentatively, that deficiency for 3C1 in male-lethal, is reinforced by the X ray induced changes described here, all of which are allelic to the *w^{258.45}* lethal and especially *T(X;3)3C1*, which has a break immediately adjacent to 3C1. A suitable name for the locus would seem to be "X chromosome lethal at 3C1," symbolized, as in this paper, by *l(1)3C1*.

The idea that 3C1 is the site of part of the *w* locus must be discarded.

POULSON (1940) described the developmental effects of a group of recessive lethal deficiencies, all lacking 3C1 and all associated with a white phenotype. One of them was $w^{258.45}$, in which, as has been shown, the lethal effect is due to loss of 3C1. As all of this group of lethals showed similar embryological abnormalities, it is highly probable that these are characteristic of $l(1)3C1$.

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SUMMARY

$l(1)3C1$ is a locus associated with band 3C1 of the salivary gland X chromosome. Deficiencies for the locus are recessive lethals. In conditions which permit survival of flies with deficiency, mutation or position effect at this locus, bristles of the head and thorax may be reduced or absent. Deficiency for the locus does not suppress zeste: on the contrary, under certain conditions it enhances the zeste effect. The locus is not functionally related to the w locus.

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