INTERACTIONS OF ZESTE MUTATIONS WITH LOCI EXHIBITING TRANSVECTION EFFECTS IN *DROSOPHILA MELANOGASTER*

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ABSTRACT

Zeste (1-1.0; 3A3) mutations have been known to modify the expression of two gene complexes: white (1-1.5; 3C1.5) and bithorax (3-58.8; 89E1-4) in *Drosophila melanogaster*. Certain mutations of these complexes have been shown to behave in a synapsis-dependent fashion. That is, certain bithorax and white genotypes exhibit one level of expression when the two copies of these loci are able to synapse in somatic tissues and another level when heterozygosity for chromosomal rearrangements interferes with their ability to pair. Such phenomena are termed transvection effects by LEWIS (1954). In the case of the white locus, asynapsis leads to a more normal state, whereas at bithorax, asynapsis leads to a more mutant phenotype. Recently, a third case of transvection was described at the decapentaplegic (2-4.0; 22F1-3) gene complex (GELBART 1982); phenomenologically, it is very similar to transvection at bithorax. In this report, we demonstrate that zeste mutations can also interact with those decapentaplegic mutations that exhibit transvection effects. In addition, we present more information on the zeste interactions with white and bithorax. Interactions with zeste may be diagnostic of loci that can exhibit transvection effects. However, different groups of zeste alleles interact with each complex. z^1 interacts with white, z^a alleles interact with bithorax and all tested zeste mutants interact with decapentaplegic. These differential effects of zeste mutations may be a reflection of the neomorphic nature of the z^1 allele.

THE interaction of the X-linked loci zeste and white is one of the most well characterized in *Drosophila melanogaster*. The phenotype of zeste (*z*:1-1.0, polytene location 3A3) mutant individuals is exquisitely sensitive to the number of copies of the white locus (*w*:1-1.5, polytene location 3C1.5) (GANS 1953; SORSA, GREEN and BEERMANN 1973), or more precisely, to the number of functional copies of the proximal portion of the white locus (GREEN 1959). Individuals with two or more functional copies of this region (termed w^{prx} by JACK and JUDD 1979) and homozygous for the z^1 mutation have yellow eyes; with only one copy of w^{prx}, they have wild-type eyes.

The chromosomal locations of the two copies of w^{prx} are crucial in determining the eye color phenotype. For example, unpaired insertions of w^+ into the autosomes result in a wild-type eye color, regardless of the number of copies of z^1 and w^+ on the X chromosome (GELBART 1971). JACK and JUDD (1979) summarized these phenomena and their own extensive studies in their elegant formal model of the zeste-white interaction. Their pivotal observations are that paired white

alleles are "repressed" to engender a zeste level of pigmentation in the presence of z^1, whereas unpaired white alleles are not modified in their expression. Thus, although an unpaired w^+ allele will confer a wild-type eye color, a mutant allele will confer its particular mutant phenotype. They propose that a diffusible product of the zeste locus interacts with a labile RNA product of w^{prx} to produce an active repressor of the white structural gene, located in the distal portion of the white locus (w^{dst}). In strains with the altered diffusible product of the z^1 mutant, closely apposed or paired copies of w^{prx} produce a sufficient concentration of w^{prx}-RNA to increase the local concentration of the active repressor complex. This in turn reduces the amount of product of the adjacent w^{dst} structural gene sufficiently to produce the mutant yellow eye color characteristic of homozygous z^1 w^+ females. Unpaired copies of w^+ accumulate less w^{prx}-RNA in their vicinity, and so less of the active repressor is present, raising the level of w^{dst} function and engendering a wild-type eye color, such as is characteristic of z^1 w^+ males.

Another class of zeste alleles is known to interact with mutations at a second locus exhibiting synapsis-dependent phenotypic interactions—the bithorax gene complex. KAUFMAN, TASAKA and SUZUKI (1973) reported that the z^a class of alleles, which behave as amorphic or hypomorphic mutations, included a mutation identified originally as an enhancer of bithorax [*e(bx)*]. These z^a alleles enhance bithorax (*bx*) alleles shown by LEWIS (1954) to display transvection effects (*i.e.*, synapsis-dependent interallelic complementation) with *Ubx* mutations. The original eye color mutant, z^1, which interacts with white does not act as an enhancer of bithorax.

This report, presents data accumulated over the last 12 years bearing on zeste interactions with loci exhibiting transvection effects. We will further characterize the zeste interactions with white and bithorax. In addition, we will demonstrate that the only other well documented transvection system (decapentaplegic—GELBART 1982) also exhibits interactions with zeste alleles. Interactions with zeste may be diagnostic of loci that can exhibit synapsis-dependent phenotypes. Different spectra of zeste alleles interact with the three synapsis-dependent systems.

MATERIALS AND METHODS

Mutations: With the exception of the decapentaplegic alleles described in the text, all mutations and balancer chromosomes are described in LINDSLEY and GRELL (1968). A summary of the genetic properties of these mutations appears in Table 1.

Culture conditions: Flies were cultured on standard Drosophila cornmeal-yeast extract-sucrose medium in half-pint milk bottles or 25-mm x 95-mm shell vials. Unless otherwise noted, crosses were reared at 25°.

Cytology: Temporary larval salivary gland squashes were made by dissecting the glands directly into 45% acetic acid and staining in 2% orcein in equal parts of 85% lactic acid and glacial acetic acid. Polytene chromosomes were analyzed under phase optics using a Zeiss Universal Research Microscope.

Wing angle measurements: The phenotype being studied affects the orientation of the wings to the body of the fly. To quantitate the phenotypes of various mutant genotypes, the angle of each wing to the long axis of the body was measured on a standard grid dividing a 90° arc into six 15° sectors. In general, an average measurement for a given genotype is based on the scoring of at least 30 individuals (60 wings).

TABLE 1

A summary of the mutations and balancer chromosomes used in this study[a]

Mutant symbol	Mutant phenotype	Genetic position
y, y^2	Yellow body color	X-0.0
sc	Scutellar bristles absent	X-0.0
z^1	Zeste-1 (homozygous yellow eyed)	X-1.0
z^a	Zeste-a (homozygous wild-type)	X-1.0
$z^{a69\text{-}2\,b}$	Zeste-a69-2 (homozygous wild-type)	X-1.0
z^{11G3}	Zeste-11G3 (homozygous wild-type)	X-1.0
w^{11E4}	White eye color	X-1.5
w^a	Apricot eye color	X-1.5
w^{sp}	Spotted-white	X-1.5
ec	Echinus (rough eye)	X-5.5
$dpp^{ho2\,c}$	Decapentaplegic (class I allele)	2-4.0
$dpp^{4\,c}$	Decapentaplegic (class EL allele)	2-4.0
$dpp^{5\,c}$	Decapentaplegic (class II allele)	2-4.0
$dpp^{19\,c}$	Decapentaplegic (class III allele)	2-4.0
Cy	Curly wings	2-6.1
$bx^{34\,e}$	Bithorax-34e	3-58.8
Cbx	Contrabithorax	3-58.8
Ubx	Ultrabithorax	3-58.8
Ubx^{130}	Ultrabithorax-130	3-58.8
Balancers		
In(2LR)Cy0	Second chromosome balancer marked with Cy	
In(3LR)TM2	Third chromosome balancer marked with Ubx^{130}	

[a] Unless otherwise noted, these mutations are listed in LINDSLEY and GRELL (1968).
[b] GELBART (1971).
[c] SPENCER, HOFFMANN and GELBART (1982).

RESULTS

Zeste-white interactions: Early in these investigations (GELBART 1971) it was noted that insertional translocations of w^+ did not behave as normal w^+ alleles *vis-à-vis* the zeste interaction. Thus, $z^1\ w^+$; $Dp(1;2)w^{+51b7}$/– males were wild-type in eye color, even though the fly contained z^1 and two w^+ genes and hence should have been yellow-eyed. This observation was pursued by examining the pairwise interactions of six independently isolated insertional translocations (Table 2) with various zeste bearing X chromosomes. Some of these results are referred to in JACK and JUDD (1979).

The effects of the duplications were analyzed in several backgrounds. *C(1)RM*, z^1, an attached X chromosome homozygous for zeste and otherwise wild-type, was used to assay the abilities of autosomal duplications of w^+ to interfere with the yellow eye color engendered by *C(1)RM*, z^1 females (Table 3). These females were XXY; the Y chromosome partially suppresses the variegating position-effect because of the heterochromatic location of two of the duplications: $Dp(1;3)w^{m\,49a7}$ and $Dp(1;3)w^{m\,264\text{-}58}$ (SPOFFORD 1976).

A similar matrix was analyzed for the following phenotypes in males bearing X chromosomes of one of the following constitutions: *sc* z^1 $Dp(1;1)w^{rG2}$ *ec* (Table 4), $z^1\ w^+$ (Table 5), $z^1\ w^a$ *spl ec* (Table 6), $z^1\ w^{11E4}$ (Table 7), $z^1\ w^{65a25}$ (Table 8), and y^2 $su(w^a)$ $z^1\ w^{sp3}$ (Table 9). These crosses were done at a later time than

TABLE 2

The cytogenetics of duplications of the white region[a]

Duplication	X chromosome breakpoints	Insertion site
rG2	3C1; 3C3	Tandem repeat
51b7[b]	3C1-2; 3D6-E1	52E
m64b13	3C1-2; 4E2-3; 5A1-2	26D7
+70h[c]	3A7-8; 3C2-3	31A3
m49a7[b]	3B1-2; 3E2-3	81
m264-58	3B2-3; 3D6-7	80D-F
+67k27[b]	3A4-5; 3E8-F1	86E17

[a] Rearrangement breakpoints are described in terms of their standard polytene chromosome locations. Unless otherwise noted, these duplications are described in LINDSLEY and GRELL (1968).
[b] LEFEVRE (1970).
[c] JUDD *et al.* (1972).

TABLE 3

The eye color phenotypes of C(1)RM, z^1 w^+/Y; Dp(1;A)P/Dp(1;A)Q *females*[a]

	Dp(1;A)Q						
Dp(1;A)P	*m64b13*	*51b7*	*+70h*	*m264-58*	*m49a7*	*+67k27*	None
None	Int2	+	+	+	+	Int1	Z
+67k27	Int2	+	+	+	+	Z	
m49a7	+	+	+	Z[b]	Z[b]		
m264-58	+	+	+	Z[b]			
+70h	+	+	Z				
51b7	+	Z					
m64b13	Z						

[a] Second chromosome duplications were balanced by *In(2LR)CyO* and third chromosome duplications by *In(3LR)TM2*. In Tables 3–9, the following symbols are used: + = wild-type eye color, Int3 = nearly wild-type eyes, but with some lighter red ommatidia, Int2 = brown eyes with a few red ommatidia, Int1 = orange eyes with a few red ommatidia, Z = yellow eyes, W = white eyes, and SP = spotted eyes like w^{sp} males. The abbreviation *Int* indicates that these eye colors are intermediate to Z and wild type. The abbreviation n.t. indicates genotypes that were not tested because their balanced, duplication-bearing chromosomes had accumulated recessive lethals.
[b] These flies had eyes with very few red patches.

TABLE 4

The eye color phenotypes of sc z^1 Dp(1;1w^{rG2}) ec; Dp(1;A)P/ Dp(1;A)Q *males*[a]

	Dp(1;A)Q				
Dp(1;A)P	*51b7*	*+70h*	*m264-58*	*m49a7*	*+67k27*
None	Int3	Int3	Int3	Int3	Int2
+67k27	Int3	Int3	Int2/Int3[b]	Int2/Int3[b]	n.t.
m49a7	Int3	Int3	Z/Int3[b]	n.t.	
m264-58	Int3	Int3	n.t.		
+70h	Int3	Z			
51b7	n.t.				

[a] Second chromosome duplications were balanced by *In(2LR)CyO* and third chromosome duplications by *In(3LR)TM2*. For an explanation of the symbols used in this table, see Table 3.
[b] This genotype had eyes with large patches of two different eye colors. Fine grained variegation occurred within both types of patch.

TABLE 5

The eye color phenotypes of z^1 w^+; Dp(1;A)P/ Dp(1;A)Q *males*[a]

	Dp(1;A)Q				
Dp(1;A)P	*51b7*	*+70h*	*m264-58*	*m49a7*	*+67k27*
None	Int3	Int3	Int3	Int3	Int2
+67k27	Int3	+	Int2	Int2/Int3[b]	n.t.
m49a7	Int3	+	Int3	Int3	
m264-58	Int3	+	Int3		
+70h	+	Int3			
51b7	n.t.				

[a] Second chromosome duplications were balanced by *In(2LR)CyO* and third chromosome duplications by *In(3LR)TM2*. For an explanation of the symbols used in this table, see Table 3.

[b] This genotype had eyes with large patches of brown and red pigmentation. Fine grained variegation occurred within both types of patch.

TABLE 6

The eye color phenotypes of z^1 w^a spl ec; Dp(1;A)P/ Dp(1;A)Q *males*[a]

	Dp(1;A)Q				
Dp(1;A)P	*51b7*	*+70h*	*m264-58*	*m49a7*	*+67k27*
None	Int3	Int3	Z/Int3[b,c]	Z/Int3[b,c]	Int2
+67k27	Int3	Int3	Int2	Int2	n.t.
m49a7	Int3	Int3	Z/Int3[b,d]	Z/Int3[b,d]	
m264-58	Int3	Int3	Z[d]		
+70h	Int3	Z[d]			
51b7	n.t.				

[a] Second chromosome duplications were balanced by *In(2LR)CyO* and third chromosome duplications by *In(3LR)TM2*. For an explanation of the symbols used in this table, see Table 3.

[b] This genotype had eyes with large patches of yellow and red pigmentation. Fine grained variegation occurred within both types of patch (see text).

[c] This Z (yellow) eye color presumably represents patches in which *Dp(1;3)*w^{m-} is inactivated (because of its insertion into heterochromatin) allowing the w^a eye color to be expressed.

[d] Some of the Z (yellow) tissue may represent w^a (due to inactivation of both duplications) whereas some may represent zeste expression, because of activation of both duplications.

TABLE 7

The eye color phenotypes of z^1 w^{11E4}; Dp(1;A)P/Dp(1;A)Q *males*[a]

	Dp(1;A)Q				
Dp(1;A)P	*51b7*	*+70h*	*m264-58*	*m49a7*	*+67k27*
None	+	+	W/Int3[b]	W/Int3[b]	+
+67k27	Int3	Int3	Int3	Int3	n.t.
m49a7	Int3	+	Z/Int3[b]	Z/Int3[b]	
m264-58	+	+	Z		
+70h	+	Z			
51b7	n.t.				

[a] Second chromosome duplications were balanced by *In(2LR)CyO* and third chromosome duplications by *In(3LR)TM2*. For an explanation of the symbols used in this table, see Table 3.

[b] This genotype had eyes with large patches of two different colors. Fine grained variegation occurred within both types of patch.

TABLE 8

The eye color phenotypes of z^1 w^{65a25}; Dp(1;A)P/ Dp(1;A)Q *males*[a]

	Dp(1;A)Q				
Dp(1;A)P	*51b7*	*+70h*	*m264-58*	*m49a7*	*+67k27*
None	Int3	Int3	W/Int3[b]	W/Int3[b]	Int2
+67k27	Int3	Int3	Int2/Int3[b]	Int2/Int3[b]	n.t.
m49a7	Int3	Int3	Z/Int3[b]	Z/W/Int3[b]	
m264-58	Int3	Int3	n.t.		
+70h	Int3	Z			
+51b7	n.t.				

[a] Second chromosome duplications were balanced by *In(2LR)CyO* and third chromosome duplications by *In(3LR)TM2*. For an explanation of the symbols used in this table, see Table 3.

[b] This genotype had eyes with large patches of at least two eye colors. Fine grained variegation occurred within each patch.

TABLE 9

The eye color phenotypes of y^2 su(w^a) z^1 w^{sp3}; Dp(1;A)P/ Dp(1;A)Q *males*[a]

	Dp(1;A)Q				
Dp(1;A)P	*51b7*	*+70h*	*m264-58*	*m49a7*	*+67k27*
None	+	+	SP/Int3[b]	SP/Int3[b]	+
+67k27	Int3	+	Int3	Int3	n.t.
m49a7	+	+	n.t.	SP/Int2	
m264-58	Int3	+	SP		
+70h	+	SP			
51b7	n.t.				

[a] Second chromosome duplications were balanced by *In(2LR)CyO* and third chromosome duplications by *In(3LR)TM2*. For an explanation of the symbols used in this table, see Table 3.

[b] This genotype had eyes with large patches of two different eye colors. Fine grained variegation occurred within both types of patch.

those utilizing *C(1)RM,* z^1 and, in the intervening period, lethals had accumulated on some of the duplication-bearing autosomes. Thus, some homozygous duplications were not analyzed. Frequently, the eyes of *Dp(1;3)*w^{m49a7} and *Dp(1;3)*$w^{m264\text{-}58}$ flies exhibit large patch color variegation. Each eye color in such variegated flies is presented in the tables.

The results of these crosses form a consistent pattern completely compatible with the model of JACK and JUDD (1979). In genotypes in which the X chromosome constitution should generate a zeste eye color when the autosomes are normal [*C(1)RM,* z^1 and *sc* z^1 *Dp(1;1)*w^{rG2} *ec*], the addition of one copy of *Dp(1;A)*w^+ shifts the eye color back to virtually wild type, except in the cases of *Dp(1;3)*w^{+67k27} and *Dp(1;2)*w^{m64b13}, which only partially suppress the zeste phenotype. Homozygotes for any *Dp(1;A)*w^+ have the yellow eye color characteristic of z^1 w^+ females. This is also true of *Dp(1;3)*w^{m49a7}/*Dp(1;3)*$w^{m264\text{-}58}$. All other doubly heterozygous *Dp(1;A)*w^+ duplications produce virtually wild-type eyes. (See below for an explanation of variegating phenotypes.)

When the X chromosome bears z^1 w^+ (Table 5), most duplications are unable to alter the wild-type eye color significantly. The exception to this is *Dp(1;3)*w^{+67k27}. As in many other genotypes, this duplication seems partially

capable of producing a more mutant eye color. The other four X chromosomes engender mutant eye colors in males because of their white mutations. The addition of a single *Dp(1;A)w*$^{+}$ to each of their genomes covers the white mutation, leading to an almost wild-type eye color. This is true of all *Dp(1;A)w*$^{+}$ duplications except for *Dp(1;3)w*$^{+67k27}$, which yields a brownish eye color in combination with z^{1} w^{+}, z^{1} w^{a} *spl ec* and z^{1} w^{65a25}. w^{a} and w^{65a25} are alleles that do not interfere with zeste expression (GREEN 1959; JUDD 1976).

Dp(1;3)w^{67k27} is partially able to promote the zeste eye color. *Dp(1;A)w*$^{+}$ homozygotes exhibit (a) the eye color of the unpaired white allele if it is more pigmented than zeste (w^{sp3}, or w^{+}) or (b) zeste if the unpaired white allele is less pigmented than zeste (w^{11E4} or w^{65a25}). The w^{a} eye color is sufficiently close to zeste to make them difficult to distinguish by phenotypic criteria. These generalizations also hold for *Dp(1;3)w*^{m49a7}/*Dp(1;3)w*$^{m264\text{-}58}$. All other doubly heterozygous duplications generate intermediate or wild-type eye color. In the variegating genotypes, the patches representing inactivation of *Dp(1;3)w*$^{m264\text{-}58}$ or *Dp(1;3)w*^{m49a7} can be inferred from a comparison of *Dp(1;3)w*^{m}/*Dp(1;A)w*^{x} with *Dp(1;A)w*^{x}/−. For example, in Table 7, z^{1} w^{11E4}; *Dp(1;3)w*^{m49a7}/*Dp(1;3)w*$^{m264\text{-}58}$ males have variegated eyes with yellow and near wild-type patches. The near wild-type patches are characteristic of cells in which each *Dp(1;A)w*^{m}/− is active (see z^{1} w^{11E4}; *Dp(1;3)w*^{m49a7}/− males). Hence, the yellow patches must result from cells in which both duplicated genes are active.

Zeste-bithorax interactions: In addition to z^{1}, other mutant zeste alleles have been recovered. The commonly obtained allele from EMS treatment is the z^{a}-type of allele (GELBART 1971). These mutations are homozygous and hemizygous wild type, but have yellow-orange eye color as z^{1}/z^{a} heterozygotes. KAUFMAN, TASAKA and SUZUKI (1973) found that *e(1)bx* is a z^{a} allele. That is, mutations specifically selected as enhancers of certain bithorax (*bx*) alleles behave as z^{a} alleles whereas z^{a} alleles also enhance certain *bx* mutations. Interestingly, these *bx* alleles also participate in transvection effects at bithorax.

Synapsis-dependent phenotypes are also elicited by *Cbx Ubx*/+ flies (LEWIS 1955). *Cbx Ubx*/+ flies have wings that arch out laterally because of a partial transformation of posterior mesothorax to posterior metathorax. The addition of a rearrangement that disrupts synapsis between the bithorax homologues (89E1-4) causes the reversion of the Cbx Ubx/+ phenotype to wild type. Four zeste alleles in a total of six different genotypes were examined for their abilities to interfere with the *Cbx Ubx*/+ transvection effect. $z^{a69\text{-}2}$ is a z^{a}-like allele (GELBART 1971). z^{11G3} is an X-ray-induced revertant of z^{1} (GANS 1953; KAUFMAN, TASAKA and SUZUKI 1973). *Cbx Ubx*/+ males bearing z^{a}, $z^{a69\text{-}2}$ or z^{11G3} were wild type whereas males bearing z^{1} had arched wings indistinguishable from those of z^{+}; *Cbx Ubx*/+ flies. Therefore, z^{a} alleles appear to disrupt both the *Cbx Ubx*/+ and *bx*/*Ubx* transvection effects.

Zeste interactions with decapentaplegic: Recently, transvection has been demonstrated at the decapentaplegic gene complex (*dpp*, 2-4.0, 22F1-3) (GELBART 1982). Considering that zeste interacted with the two other well defined transvection systems, we deemed it valuable to examine the interaction of zeste with decapentaplegic.

Several pairs of *dpp* alleles exhibit partial or full synapsis-dependent complementation. The phenotypes of these various genotypes with or without mutant

TABLE 10

The effects of X-chromosome constitution on various synapsis-dependent decapentaplegic genotypes[a]

X genotype	Autosomal genotype					
	4/ho2	*4/H7, ho*[b]	*4/DTD35,*[c] *ho2*	*4/H30,*[c] *ho2*	*4/5*	*4/19*
z^+	A	A	B	A	"+"	"+"
z^1	B	F	F	B/C	enh.	enh.
$z^1\ w^{11E4}$	A	D	Not done	A	enh.	enh.
z^{11G3}	A	Not done	F	A	enh.	enh.
$y\ z^a$	B	B	F	A	enh.	enh.
$z^{a69\text{-}2}\ w^e$	A	Not done	E	A	enh.	enh.

[a] The wing angle symbols A–F represent average wing angles of 0°–15° (A) through 75°–90° (F). "+" indicates the standard phenotype of dpp^4/dpp^5 and dpp^4/dpp^{19} heterozygotes. *enh.* indicates that these phenotypes are enhanced (*i.e.*, more abnormal).

[b] *H7 = T(2;3)H7 [T(2;3)23D1-2; 80F].*

[c] *DTD35 = T(2;3)DTD35 [T(2;3)28E-F; 81F].*

[d] *H30 = T(2;3)H30 [T(2;3)21C1-2; 67F].*

substitution at the zeste locus have been examined (Table 10). No enhancement of the dpp^4/dpp^{ho2} phenotype was elicited by zeste mutations; however, the addition of some rearrangements of *2L* that do not themselves disrupt complementation (*e.g.*, *T(2;3)H7*) to this genotype seems to have sufficiently sensitized it so that enhancement by zeste alleles is noted. The most dramatic enhancement is by z^1; the effects of z^a and z^{11G3} are variable.

The dpp^4/dpp^5 and dpp^4/dpp^{19} phenotypes (SPENCER, HOFFMANN and GELBART 1982) reflect partial complementation between the *dpp* alleles (GELBART and WU, unpublished results). These phenotypes are enhanced by all available zeste mutant alleles. We have analyzed the wing phenotype in detail, although enhancement may extend to the eye, genitalia, halteres and legs, depending on the genotype in question. z^+; dpp^4/dpp^5 flies are often missing the anterior cross-veins of their wings, which are held obliquely to the longitudinal axis of their bodies. In the presence of zeste mutations, there is additional disturbance of the venation and the wings are held perpendicularly to the body. z^+; dpp^4/dpp^{19} flies have wild-type or nearly wild-type-sized wings bearing abnormal venation. Zeste mutations further disrupt venation and reduce wing size to two-thirds to one-half wild type in the case of z^a and one-quarter wild type to vestigial in the case of z^1.

To be sure that enhancement resulted from effects of the zeste locus, mapping experiments were undertaken. In preliminary experiments, the dpp^4/dpp^5 enhancement was found to segregate with the *y-w* interval. The position of the enhancer mutation within this interval was determined by analyzing 27 individuals bearing crossovers in the *y-*w^a region, which were derived from $y^2\ sc\ w^a\ ec/y\ z^a$ mothers. These individuals were progeny tested for their enhancement phenotype as well as for their zeste genotype. Crosses were reared at 23.5°, as the most consistent difference between the parental chromosomes were noted at this temperature. Enhancement of both the dpp^4/dpp^5 and dpp^4/dpp^{19} phenotypes co-segregates with z^a. Similarly, by analyzing 38 recombinants in the *sc-*w^{11E4} interval from $z^1\ w^{11E4}/sc\ ec$ females, we have found that the enhancement of the dpp^4/dpp^{19} phenotype co-segregates with z^1.

DISCUSSION

Zeste mutations clearly interact with all three transvection-sensitive loci: bithorax, decapentaplegic and white. Although casual observations suggest that zeste is not a generalized modifier locus (as many other mutations are not modified by mutant zeste alleles), it is possible that these mutations have not been examined under the proper conditions to elicit an interaction. We speculate that an interaction with zeste is diagnostic of a locus that can exhibit synapsis-dependent phenotypes. A similar suggestion has been made by BABU and BHAT (1981) on the basis of their studies of the interactions of z^a with white and bithorax mutations.

The z^a allelic class is the common type of zeste mutant recovered after EMS mutagenesis (GELBART 1971). One other zeste allele—peppered-69a—(z^{p69a}) which produced homozygous females with brown mottled eyes and males with wild-type eyes also arose in this mutagenesis. Tests with deletions of zeste have led to the suggestion that z^a alleles represent the null or hypomorphic state of the z^+ function (GELBART 1971; KAUFMAN, TASAKA and SUZUKI 1973).

The z^1 allele is definitely not the null state, as z^1/z^a is yellow-orange eyed whereas *Df(1)z*/z^a is wild type. z^1 is not a hypermorph, as z^1/z^+ is wild type whereas z^1/*Df(1)z* is yellow-eyed. The original z^1 allele, which dramatically affects w^+ function, was spontaneous in origin. X-ray mutagenesis gives rise chiefly to rearrangement-associated zeste alleles that behave like z^a; EMS mutagenesis has not given rise to any z^1-like mutants (GANS 1953; GELBART 1971). As a spontaneous mutation that cannot be readily induced with standard mutagens, z^1 is a prime candidate to be a mutation caused by the insertion of a mobile element (W. BENDER, personal communication). Based on studies of mutant derivatives of z^1, E. LIFSCHYTZ has made similar suggestions (personal communication).

Most zeste alleles have been recovered on the basis of eye color phenotypes (that is, by virtue of their interactions with the white gene). The spectrum of zeste mutations that affect the three loci are distinct. Only z^1 causes a pigmentation change associated with the state of the white locus; z^a/z^a or z^a/*Df(1)z* flies are wild type. Only z^a alleles and z^{11G3} enhance the bithorax transvection effects. KAUFMAN, TASAKA and SUZUKI (1973) found that all z^a alleles and z^{11G3} behaved in this manner with respect to the *bx*/*Ubx* transvection effect. We have found that z^a alleles as well as z^{11G3} enhance the *Cbx Ubx*/+ transvection effect. All tested alleles (z^1, z^a, $z^{a69\text{-}2}$ and z^{11G3}) enhance the three decapentaplegic synapsis-dependent genotypes [dpp^4/dpp^5, dpp^4/dpp^{19} and $dpp^4/R(dpp^{ho2})$]. Thus, each locus exhibiting transvection effects responds to a characteristic and different set of zeste alleles.

How these differences relate to the functions of z^+ and its mutant alleles is unclear. Perhaps z^1 alters the temporal or spatial expression of the z^+ product. Conceivably, regulatory sequences brought in by the putative z^1 insertion element could activate the zeste locus at unusual times or in unusual tissues. Such an insertion, which can exhibit developmentally regulated transcription (FLAVELL *et al.* 1980), may put the locus into which it has inserted under completely a different pattern of developmental control, both in terms of spatial and temporal gene expression. Thus, if there is overlap in the tissue distribution of product of the wild-type and insertion mutant alleles, we might classify such

a mutation as a null by one criterion and as normal or even an overproducer by another.

Another curious observation is that enhancement of some bithorax alleles by z^a is dependent upon mutant substitution at white (KAUFMAN, TASAKA and SUZUKI 1973). It is as if white and bithorax (and perhaps a very few other loci) compete for a limiting amount of zeste product. This explanation presupposes that these loci would compete for this product even in tissues where they were presumably not active. Although the analysis is far from exhaustive, we have not observed any competitive interactions with *dpp* and either bithorax or white mutations.

Finally, let us turn to a consideration of our results in terms of the JACK and JUDD (1979) model of the zeste-white interaction. Virtually all of our observations on insertional duplications of w^+ are clearly consistent with this elegant model (see Introduction). A couple of interactions at first glance might appear exceptional, but can be rationalized in terms of the JACK and JUDD scheme. The $Dp(1;3)w^{m49a7}/Dp(1;3)w^{m264\text{-}58}$ double heterozygote behaves in an equivalent manner to the homozygous *Dp(1;A)* duplications (although its analysis is complicated by its variegating phenotype). We interpret this to indicate that these two duplications reside in proximity to one another even though they are inserted into different regions of proximal chromosome 3. Even more striking is the behavior of the largest insertional duplication—$Dp(1;3)w^{+67k27}$. As inferred from the intermediate eye color phenotype of, for example, $z\ w^+$; $Dp(1;3)w^{+67k27}$, a single dose of this duplication is capable of partially repressing w^{dst} activity. We suggest that this partial repression reflects the ability of this large duplication to pair frequently with its normally situated w^+ homologue. Hence, both apparent exceptional observations probably reflect peculiarities of the systems controlling somatic chromosome alignment.

The phenomenology of the zeste interactions with transvection-sensitive loci has become most involved. The understanding of these interactions will await the molecular characterizations of the relevant loci, which are underway in several laboratories including our own.

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