LeFever, H.M. Kansas State Teachers College, Emporia. Analysis of three White mutants resulting in two new recombination sites at the white locus in Drosophila melanogaster.

Three viable white alleles have been analyzed in respect to their location in the locus and their relationship to the mutant zeste. One of the mutants was white-carrot (w<sup>crr</sup>) which arose spontaneously in a wild type X-chromosome (Judd 1964). The two remaining mutants were recovered by this author and designated as w<sup>65a25</sup> and

sp-w<sup>4</sup>. When tested with sp-w<sup>1</sup>, the mutants w<sup>2</sup>Tr and w<sup>6</sup>5a<sup>2</sup>5 gave the uniform brown eye color characteristic of a white allele rather than a white deficiency. The spotted white phenotype is relatively rare among white mutants and three previous mutants of this type were localized at what is now site 7 (Figure 1). Females with the genotype sp-w<sup>1</sup>/sp-w<sup>4</sup> had a phenotype indistinguishable from females with the genotype sp-w<sup>4</sup>/sp-w<sup>4</sup> or sp-w<sup>1</sup>/sp-w<sup>1</sup> indicating that sp-w<sup>4</sup> was possibly a true allele of site 7.

	w <sup>Bwx</sup>	wbf	wcrr	<sub>w</sub> 65a25	wa	w1	sp-w
	1	1				•	1
Judd	0.01+	. 1	0.001	+	' 0.0	1+ ' 0.00	5 <b>+ '</b>
1964	, –		-	-	<b>'</b>	- ,	- '
	1	2	3	4	5	6.	7
LeFever	•	' 0.000	4 <u>+</u> ' 0.0003-	' 0.002 <u>+</u>	1	$0.014 \pm$	
1973	•	:					
	•	'	•	•	'		•

Figure 1. Map illustrating the seven recombination sites of the white locus. For descriptions of the mutants and symbols used, see Lindsley and Grell, 1968. The map distances are corrected for the presence of the autosomal inversions which increase the rate of crossing over by a factor of approximately 3X.

Recombination analysis was used to determine the spatial relationship of the three mutants with reference to the white locus. All constructed parental females carried the autosomal inversions SM1/+ and Ubx $^{130}$ /+ which were employed to increase crossing over in the distal portion of the X-chromosome. Judd (1964) reported that  $w^{crr}$  recombined with  $w^{a}$  and was located to the left of it (Table 1). Females were constructed with the genotype  $w^{crr}$ /y z  $w^{Bwx}$  and mated to y w spl sn $^{3}$  males. Ten exceptional progeny were recovered which place  $w^{crr}$  to the right of  $w^{Bwx}$  (Table 1). Females were constructed with the genotype  $w^{crr}$ /y z  $w^{b}$ f and mated to y  $^{2}$   $^{2}$  w  $^{3}$  spl sn $^{3}$ . Two exceptional progeny were recovered which place  $w^{crr}$  to the right of  $w^{bf}$  (Table 1). Females were constructed with the genotype z  $w^{65a25}$  spl sn $^{3}$ /y  $^{2}$  w  $^{3}$  spl ec and mated to y w spl sn $^{3}$  males. One exceptional male progeny was recovered which places  $w^{65a25}$  to the left of  $w^{a}$  (Table 1). Females were constructed with the genotype z  $w^{65a25}$  spl sn $^{3}$ /w  $^{crr}$  and mated to y w spl sn $^{3}$  males. Two exceptional progeny were recovered which place  $w^{65a25}$  to the right of  $w^{crr}$  (Table 1). The mutant sp- $w^{4}$  was tested in the following manner: females were constructed with the genotype y  $^{2}$  sp- $w^{4}$  spl sn $^{3}$ /y  $^{2}$  w  $^{2}$  spl ec and mated to y w spl sn $^{3}$  males. Nine exceptional progeny were recovered which place the mutant sp- $w^{4}$  to the right of  $w^{2}$  (Table 1).

When the mutants  $w^{crr}$  and  $w^{65a25}$  were placed in the heterozygous state with two doses of zeste (z w<sup>+</sup> dup ec/w<sup>crr</sup> and z/z w<sup>65a25</sup> spl sn<sup>3</sup>) the results were a female with a zeste phenotype. This indicated that  $w^{crr}$  and  $w^{65a25}$  do not affect the expression of zeste. The mutant sp-w<sup>4</sup>, according to its placement by recombination, should be a zeste suppressor (Green 1959c). This was the case as females with the genotype  $y^2$  sp-w<sup>4</sup> spl sn<sup>3</sup>/z w<sup>+dup</sup> ec had a nearly wild type phenotype characteristic of a zeste suppressor effect.

Analysis of the mutant wcrr and w65a25 which places them in separate locations between former recombinational site 2 ( $w^{bf}$ ) and site 3 ( $w^{a}$ ) indicated that the white locus has at least 7 recombination sites of which sites 1 through 5 are non-suppressors of the mutant zeste (Figure 1).

Acknowledgments: I wish to thank Dr. Burke H. Judd for his assistance during the early part of this study, u dertaken while I was a graduate student at the University of Texas, and (Continued at bottom of next page)

Van Valen, L. University of Chicago, Illinois. A method that might estimate age in Drosophila.

In a previous note (DIS 46:125) I reported failure in an attempt to estimate age in Drosophila by use of daily growth layers in the cuticle. Recently Schlein and Gratz (1972) have had success with this method for mosquitoes

and three families of muscoid flies. Their methods differ from ours in perhaps relevant ways. Success with Drosophila could make its ecology anemable to standard ecological procedures. I now lack the relevant equipment but suggest that my previous failure not be taken as definitive.

Reference: Schlein, J. and N.G. Gratz 1972, Bull. World Health Org. 47:71-75.

## (Continued from preceding page)

Table 1. Exceptional recombinant types recovered from heterozygous females.

Heterozygous female	Exceptional recombinant types recovered	Number	Total offspring
$y^2 + w^a \text{ spl ec}$	+ w <sup>crr</sup> w <sup>a</sup> spl ec	4	52713
+ w crr + + +	$y^2 + + +$	1	32713
+++ w <sup>crr</sup>	y z wBwx warr	3	
y z wBwx +	z w <sup>B</sup> wx wcrr	1	103586
,	+ + + +	6	
+ + + + crr y z wbf +	+ + + +	. 2	170725
$\frac{+ \ 2 \ w^{6 \cdot 5 a^{2 \cdot 5}} + \ spl + \ sn^{3}}{y + + \ w^{a} \ spl \ ec +}$	$y^2 + sp1 sn^3$	. 1	14506
$\frac{z + w^{65a25} \text{ spl sn}^3}{+ w^{crr} + + +}$	z + + + +	2	255341
$y^2 + sp-w^4 sp1 + sn^3$	$y^2 + + spl ec$	. 5	21550
$y^2 w^a + spl ec +$	$y^2$ $w^a$ sp- $w^4$ sp1 sn <sup>3</sup>	4	21330

<sup>\*\*</sup> Burke H. Judd personal communication

for his continued support and encouragement on this project.

References: Green, M.M. 1959c, Hered. 13:302-315; Judd, B.H. 1964, Genetics 49:253-265.