Table 1. Enzymes specific activities (percentage of total recovered activity/percentage of total recovered proteins), for the five genotypes. The specific activities are given in units per gram proteins. For acid phosphatase, beta-galactosidase, and aldehyde dehydrogenase, the values have been multiplied by 10^2.

STRAINS:	wild e^+		HA		LA		yvf mal^{bz}		bAdh^{n4}	
ENZYMES	Mean	σ	Mean	σ	Mean	σ	Mean	σ	Mean	σ
Cytochrome c oxidase	0.1636	0.0160	0.1395	0.0368	0.1695	0.0535	0.1454	0.0254	0.1496	0.0391
Malate dehydrogenase	2.1352	0.5756	1.6434	0.3473	1.4681	0.3460	2.0636	0.4652	2.4134	0.3949
Acid phosphatase	2.7093	0.4037	2.4348	0.8748	2.9246	1.7960	3.3772	0.8349	3.0263	0.7651
Beta galactosidase	0.7077	0.1623	0.9545	0.0940	0.8137	0.1656	0.6525	0.864	0.9235	0.1410
NADPH cytochrome c reductase	0.1790	0.0344	0.1314	0.0099	0.1219	0.0173	0.1563	0.0324	0.1206	0.0384
Catalase	0.0529	0.0130	0.0474	0.0241	0.0579	0.0089	0.0674	0.0087	0.0638	0.0240
Alcohol dehydrogenase	0.3032	0.0280	0.1350	0.0345	0.1535	0.0322	0.3262	0.118	-	-
Aldehyde oxidase	5.5915	1.0535	2.5416	1.0281	2.4723	0.5790	-	-	-	-
Aldehyde dehydrogenase	2.8450	2.2997	3.8454	1.6485	4.3905	0.6647	2.1533	0.0115	5.1252	1.1137

of the subcellular fractions obtained by differential centrifugation are shown in Fig. 1. A high proportion of cytochrome c oxidase is present in M and N fractions. Malate dehydrogenase is found in M and N fractions and also in the soluble fractions. The presence of large amounts of the two mitochondrial enzymes in N fractions denotes a high sedimentation coefficient and the presence of large mitochondria. About 40% of the lysosomal enzymes are recovered in M and L fractions. However, the relative specific activities are about the same in L and M fractions for acid phosphatase. Beta galactosidase is most purified in L fraction. A high proportion of the two enzymes is present in the soluble fraction. Catalase is mainly recovered in S fraction and seems not to be associated with a particulate fraction. By electron microscopy, peroxisomes have not been detected in the isolated fractions. NADPH cytochrome c reductase is most purified in the microsomal fraction P. In **D.melanogaster** flies endowed with ADH activity, this enzyme is mainly recovered in the soluble fractions. In wild e^+ strain and in the HA and LA lines, most of AO is unsedimentable, but a low proportion of the enzyme is associated with heavy and light mitochondrial fractions, specially in HA and LA flies. Aldehyde dehydrogenase (ALDH) seems to be associated with mitochondria, but it is also present in the S fraction; perhaps two different ALDH exist in **D.melanogaster** (Garcin et al. 1983) and are localized as in Mammals (Dawson 1983): an essentially mitochondrial ALDH and another soluble one.

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Evidence that the locus of a novel type of suppressor mutation regulates transcription of the white locus.

Members of the w^{sp} class of mutant alleles at the white locus (w^{sp1}, w^{sp2}, w^{2p3}, and w^{sp4}) apparently map outside of and 5' to the white transcription unit and exert tissue-specific effects on white expression (Zachar & Bingham 1982; O'Hare et al. 1983; Davison et al. 1985; Figure 1). The mutations causing these alleles, thus, appear to affect regulatory sequences. The suppressor-of-white-spotted mutation [su(w^{sp})] was isolated as a partial revertant of w^{sp1} and proved to be a suppressor of w^{sp1} mapping distal to white on the X chromosome (W. Gelbart, pers. comm.).

We have extended the genetic analysis of su(w^{sp}) and find it to map approximately at position 0.16 (assuming a map position of 0 for yellow and 1.5 for white) based on the recovery of 2 crossovers on the y-su(w^{sp}) interval and 17 on the su(w^{sp})-white interval.

We further find that su(w^{sp}) strongly suppresses (restores to nearly wild type) the mutant eye color phenotype produced by all four of the w^{sp} mutations, but exerts no detectable effect on the eye color phenotypes produced by any other tested white alleles. The non-w^{sp} alleles tested were w^{bf}, w^{b1}, w^{a1}, w^{a2}, w^{a3}, w^{ch}, w^{sp55} and w^+. su(w^{sp}) effects on expression of any of the non-w^{sp} mutant alleles tested comparable in magnitude to its effects on the w^{sp} alleles would have been readily detected as demonstrated by gene dosage experiments. The non-w^{sp} alleles tested are distinguishable in structure from

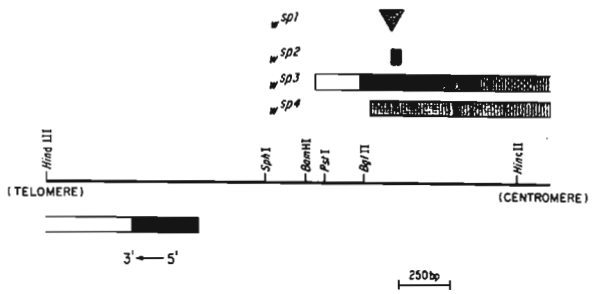

Figure 1. Physical map of relationship of w^{sp} mutations to the 5' end of the major white transcription unit. The central line with appended restriction sites represents white locus DNA sequences extending from coordinates +3.1 (HindIII) to +5.5 (HincII) on the conventional white locus map (see Zachar & Bingham 1982). The lower bar indicates the position of the 5'-most known exon (solid portion) and intron (open portion) of the major white transcript (Pirrotta & Brockl 1984; Davison et al. 1985). The stippled triangle represents the position of the B104 insertion responsible for the w^{sp1} mutation and the three stippled bars represent the extents of deletion lesions presumably responsible for the w^{sp2}, w^{sp3}, and w^{sp4} alleles (Zachar & Bingham 1982; Davison et al. 1985).

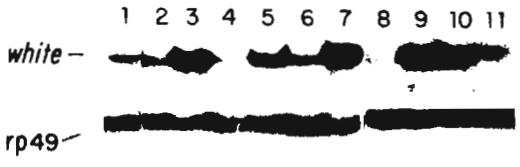

Figure 2. Effects of the su(w^{sp}) mutation on accumulation of white locus transcripts in adult head tissues. Shown are the results of hybridization to polyadenylated RNAs (10 micrograms per channel) isolated from adult heads with white locus or rp49 DNA sequence probes. The genotypes analyzed were as follows by lane: [1] w^{+}; [2] w^{sp1}; [3] su(w^{sp})w^{sp1}; [4] w^{sp2}; [5] su(w^{sp})w^{sp2}; [6 and 8] w^{sp3}; [7 and 9] su(w^{sp})w^{sp3}; [10] su(w^{sp})w^{a2}; [11] w^{a2}. The RNA blot was cut in half; the top portion was hybridized with the white probe and the bottom portion (as a control for the amount of polyadeylated RNA) was probed with the rp49 probe. The white probe has approximately 900 bases of homology to the major white transcript and is a combination of fragments from -1.2 to -0.4 kb and from +3.1 to +6.6 kb on the conventional white locus map (see Figure 1). The rp49 probe is the 0.6 kb EcoRI to HindII fragment containing most of the transcription unit (O'Connell & Rosbash 1984). The su(w^{sp}) mutations produce elevation in white transcript levels in adult body tissues in these genotypes quite similar to those shown here for head tissues (results not shown). The procedures for RNA blot analysis and tissue and RNA isolations are as described in Bingham & Zachar (1985) except that culture temperature was carefully controlled between 21° and 22°C here.

the w^{sp} alleles by having an intact w^{sp} region (Zachar & Bingham 1982; Figure 1). Numerous allele-specific suppressors are known in Drosophila (Lindsley & Grell 1968). In contrast to su(w^{sp}), the specificity of previously characterized suppressors is based on interaction with specific transposons (Bender et al. 1983; Modollel et al. 1983; L. Searles & R.A. Volker, pers. comm.). Thus, suppressor-of-white-spotted represents the first identified member of a novel class of suppressor mutation whose allele specificity is detemined by the portion of the target locus mutationally altered.

We have investigated the effects of su(w^{sp}) on accumulation of white transcripts in mature adult tissues. We find that w^{sp1}, w^{sp2}, w^{sp3}, w^{a2} and w^{+} alleles produce indistinguishable levels of a 2.6 kb transcript in adult head tissues and adult body tissues (Figure 2 and results not shown). This transcript is indistinguishable in size and transcriptional polarity from the major white transcript previously observed in unfractionated w^{+} tissues (O'Hare et al. 1983; Pirrotta & Brockl 1984). The su(w^{sp}) mutation produces a substantial elevation in white transcript levels in mature adult head and body tissues (Figure 2 and results not shown) in the cases of all tested genotypes (Figure 2).

In summary, the suppressor-of-white-spotted mutation is partially recessive and results in elevation of adult white transcript levels. The w^{sp} mutations cause apparently tissue-specific effects on white expression in immature adult eye tissues and the suppressor-of-white-spotted mutation largely relieves this effect without having a measurable effect on eye pigment deposition engendered by white alleles having an intact w^{sp} region.

On the basis of these results, we propose the following model for the function of the su(w^{sp}) locus product and the w^{sp} genetic element in regulating white expression in adult tissues: the su(w^{sp}) locus causes the production of a repressor active in several (and possibly all) adult tissues, mature and immature. The repressor is partially responsible for defining the levels of white transcripts in most of these tissues. However, in immature eye pigment cells (where eye pigment deposition occurs) the effect of this repressor on white transcription is completely antagonized by the action of a positive effector requiring or acting at the regulatory genetic element inactivated by w^{sp} mutations. This positive effector is present in immature adult eye pigment cells but exerts little or no effect on white transcript levels in mature adult tissues (much later).

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Changes of DNA replication pattern of the polytene chromosomes of **Drosophila melanogaster** resulting from chromosomal rearrangements.

Bender et al. (1971) showed that replication of a duplicated region of polytene chromosome is changed as a result of a rearrangement. A similar situation has also been noted by Kalisch & Haegele (1973) who also observed that, at comparable replication phase, the labelling intensity of duplicated subdivision is different from those of homologous subdivisions in +/+ chromosomes even when the different DNA amount of both genotype was taken into consideration. Ananiev & Gvozdev (1974), on the basis of their work on DNA replication in a Eu-heterochromatin rearrangement of Drosophila, also indicated that the replicative behaviour of a replicating unit of the transposed region is altered from the parental type. The following sets of experiments were therefore carried out to characterize the factor or factors presumably responsible for determining the replication pattern in greater detail by genetically dissecting the units. For this pur-

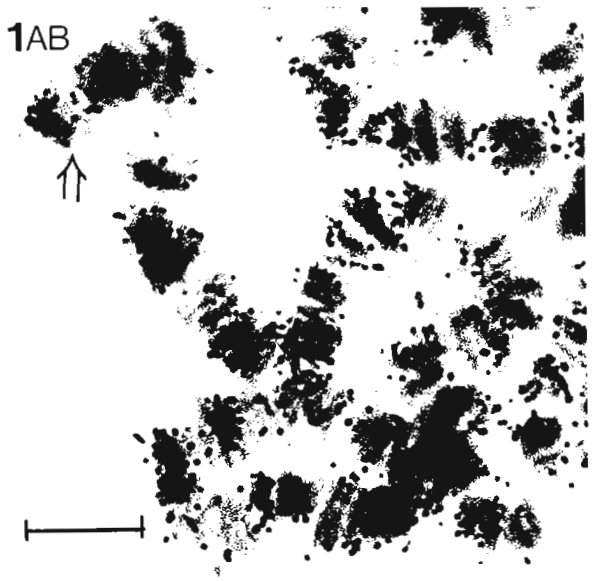

Figure 1. ^{3}H-TdR labelled autoradiogram of inversion heterozygote In(1)d149, 1(1)J1/+, showing the 3D pattern in 1(1)J1/+. Arrow indicates asymmetric distribution of label at 1AB site. Scale indicates 10 µm.

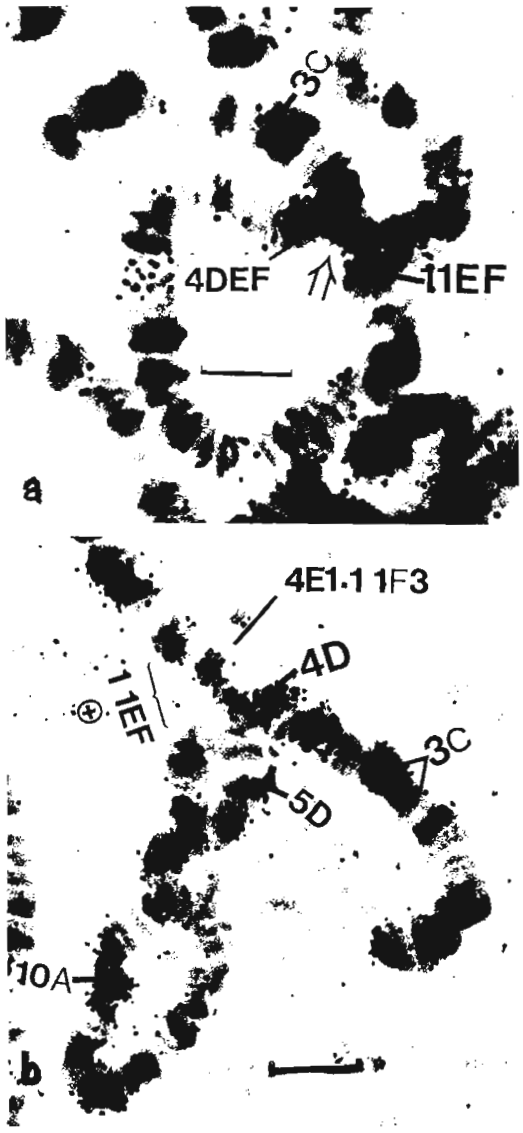

Figure 2. ^{3}H-TdR labelled autoradiograms of the X chromosomes of the inversion heterozygote In(1)d149, 1(1)J1/+. (a) An early pattern in In(1)d149/+. Arrow indicates absence of label at the point of break. (b) A terminal pattern in In(1)d149/+. Arrow points to the asynapsed homologues segments, one of which is unlabelled and the other labelled. Scale indicates 10 µm.