

in the centre of food by means of an incision practised with a lancet. (2) All larvae seeded at the same time in a side of the medium just beside the vial wall with incision on food. (3) Larvae seeded in 3 groups of 24 larvae each, separated as far as possible with incision on food. (4) All larvae seeded on the centre of food, without incision. In this case larvae were placed on a piece of paper (0.5 x 0.5 cm) which was put on the surface of food.

Monocultures and tricultures (24 larvae for each strain) were carried out. A total of 8 replicates for monocultures and 10 replicates for tricultures were made. Cultures were incubated at $25 \pm 1^\circ\text{C}$ and at $60 \pm 5\%$ relative humidity. Data were analyzed by ANOVA and Student-Newman-Keuls test.

Figure 1a shows viabilities (V) and mean developmental times (MDT) for the three strains in monocultures. Viability shows only significant differences among the different seeding sites in the cardinal strain which has a higher viability when seeding in site 1. As regards MDT, cardinal and sepia strains show differences, cardinal being faster in 1 and 2 situations and sepia in 1 and 4.

Figure 1b shows viabilities (V) and mean developmental times (MDT) in tricultures. Though wild strain shows a slight decrease in viability when seeded in the 4th way, no significant differences among viabilities appear. As regards MDT, statistical tests show that wild strain is faster in situation 1; the MDT of the cardinal strain remain unchanged in all situations, and sepia is faster in situation 1 and slower in the 4th.

In monocultures, wild strain seems to be unaffected by the seeding sites. Cardinal strain, on the other hand, is slower in situations 3 and 4, it showing that, perhaps it is more sensitive than wild strain to gregarism and to help which may represent the incision of the medium. These ideas are supported by the highest viability exhibited by cardinal in situation 1. It seems that sepia has higher sensitivity to gregarism than wild strain though not face to cardinal strain. The incision does not change its response.

In tricultures, viabilities do not show differences among the different seeding sites, while mean developmental times show a phenomenon of facilitation among the strains, mean developmental times being lower in tricultures than in monocultures. The first seeding method gives rise to the fastest developmental rate in the three strains. This result supports some kind of mutual facilitation. This facilitation is present in spite of the existence of the different competitive abilities of genotypes being reflected as differences among the MDT. In this way, the concept of larval facilitation is extended. Moreover, this effect seems to be important for the understanding of genetic polymorphisms. Since in our uncrowded cultures facilitation is put into evidence, it may be thought that under more restrictive conditions its role may be determinant. However, when food and space are limited, facilitation might be hidden under other factors more relevant.

References: Barker, J.S.F. 1971, *Oecologia* 8:139-156; Beardmore, J.A. 1963, *Am. Nat.* 97:69-74; Bos, M. et al. 1977, *Evolution* 31:824-828; Bos, M. 1979, *Evolution* 33(2):768-771; Lewontin, R.C. 1955, *Evolution* 9:27-41; Tomic, M. & F.J. Ayala 1981, *Genet.* 97:679-701.

Chanteux, B., J. Lechien, C. Dernoncourt-Sterpin, M. Libion-Mannaert, S. Wattiaux-De Coninck and A. Elens. F.N.D.P., Namur, Belgium. Ethanol metabolizing enzymes subcellular distribution, in *D.melanogaster* flies homogenates.

A method of homogenization and subcellular fractionation originally described for Rat liver (de Duve et al. 1955) has been slightly modified and used for *Drosophila* flies homogenates (Liétaert et al. 1984). First, a nuclear fraction (N) is separated from a total cytoplasmic extract (E). From the cytoplasmic extract, four fractions are isolated: a heavy mitochondrial fraction (M), a light mitochondrial fraction (L), a microsomal fraction (P), and a final supernatant (S). The same reference enzymes have been used as for Rat liver: cytochrome c oxidase and malate dehydrogenase for mitochondria, acid phosphatase and beta-galactosidase for lysosomes, NADPH cytochrome c reductase for endoplasmic reticulum, and catalase (which plays a part in ethanol metabolism) for peroxisomes.

Five *D.melanogaster* genotypes have been considered: the strain y v f ma¹ b² z lacks aldehyde oxidase (AO) but has a normal alcohol dehydrogenase (ADH) activity; the strain bAdhⁿ⁴ lacks both AO and ADH; the HA and LA lines result from a long term selection for "male sexual activity" combined with brother-sister mating which has given, after 330 generations, a "highly active" line HA and a "lowly active" line LA; the wild e⁺ strain is used as a control. These three last genotypes are endowed with normally high ADH and AO activities. All these genotypes differ in tolerance to ethanol, in oviposition preference for ethanol supplemented mediums, and in larval preference for ethanol supplemented or acetaldehyde supplemented mediums, in relation with their ADH activity level (Deltombe-Liétaert et al. 1979; Hougouto et al. 1982; Depiereux et al. 1985). Adult flies (from 5 to 10 days of age) were used.

The specific activities of the reference enzyme and of the main ethanol metabolism enzymes are shown in Table 1, for the five genotypes. The distribution pattern of the same enzymes for each

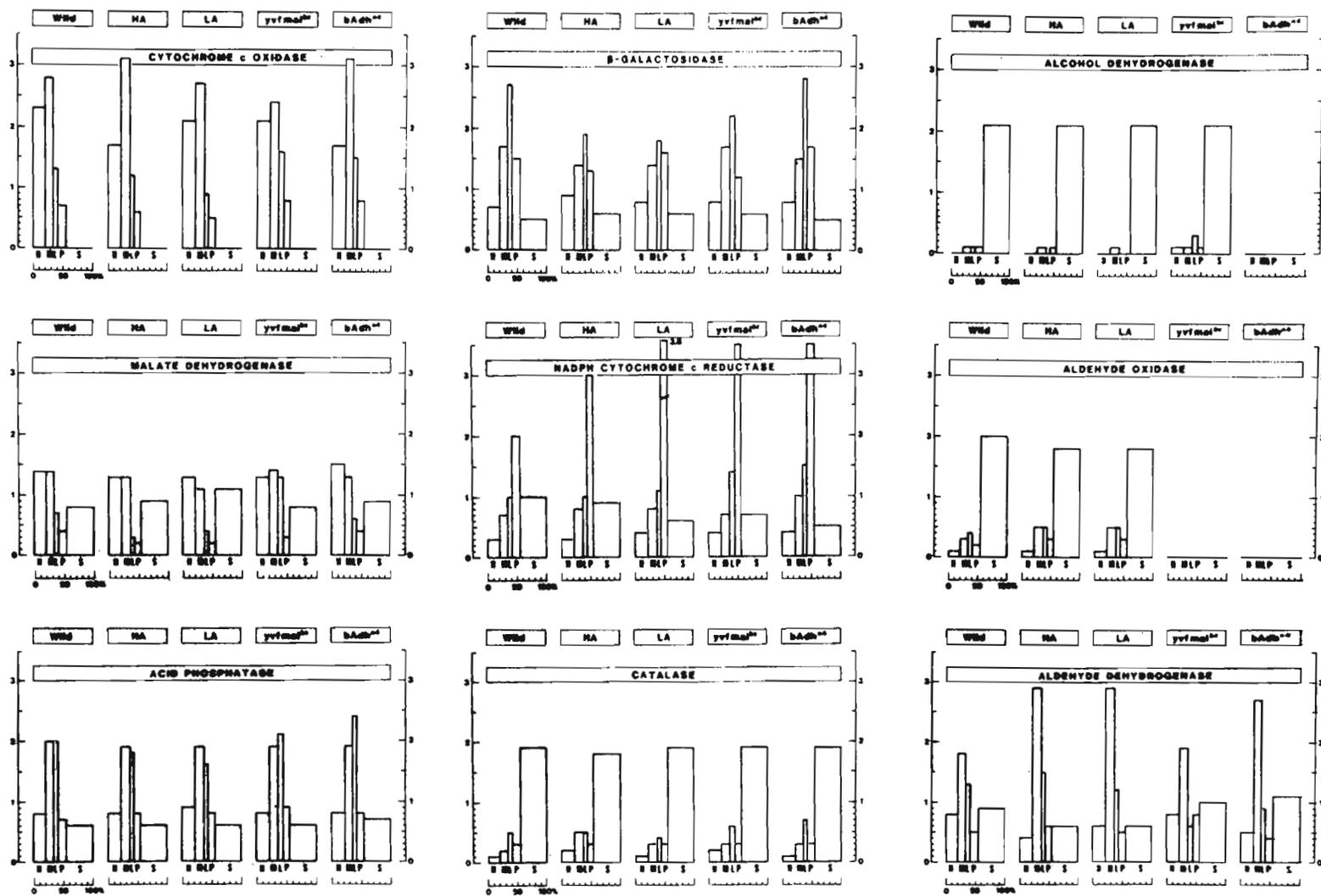


Figure 1. Distribution pattern of enzymes. Ordinate: relative specific activity of fractions (percentage of total recovered activity/percentage of total recovered proteins). Abscissa: relative protein content of fractions (cumulatively from left to right). N: nuclear fraction, M: heavy mitochondrial fraction, L: light mitochondrial fraction, P: microsomal fraction, S: supernatant.

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 .

ENZYMES	STRAINS: wild e ⁺		HA		LA		yvf mal ^{bz}		bAdh ⁿ⁴	
	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.

References: Dawson, A.G. 1983, T.I.B.S. 8:195; de Duve, C., B.C. Pressman, R. Gianetto, R. Wattiaux & F. Appelmans 1955, Biochem. J. 60:604; Deltombe-Liétaert, M.C., J. Delcour, N. Lenelle-Monfort & A. Elens 1979, Experientia 35:579; Depiereux, E., N. Hougouto, J. Lechien, M. Libion-Mannaert, M.C. Liétaert, E. Feytmans & A. Elens, Behav. Genet. (in press); Hougouto, N., M.C. Liétaert, M. Libion-Mannaert, E. Feytmans & A. Elens 1982, Genetica 58:121; Garcin, G., J. Coté, R. Radouco-Thomas, S. Chawla & C. Radouco-Thomas 1983, Experientia 39:1122; Kaidanov, L.Z. 1980, Genetica 52/53:165; Liétaert, M.C., M. Libion-Mannaert, S. Wattiaux-De Coninck & A. Elens 1985, Experientia 41:57.

Chapman, C.H. and P.M. Bingham. State University of New York, Stony Brook, USNA. 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