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Recessive esterase deficiencies controlled by alleles of Est C and Est 6 in D. melanogaster.

Electrophoretic variation of two esterase zones, Esterase C and Esterase 6, in starch gels is controlled by two independent loci on Chromosome III. A further search for additional inherited esterase pattern differences has revealed one Drosophila strain, sc ec ev et^6 v g^2 f/FM3 y^{31d} sc^8 dm B 1, which exhibits no detectable Esterase C and another, car, which shows no Esterase 6 activity.

Offspring resulting from crosses of individuals lacking either enzyme band to corresponding fast and slow types resemble, with little or no apparent difference in activity, the fast and slow types to which they were mated. Backcross and F_2 progenies include enzyme deficient types in ratios generally not contradictory to single recessive factor inheritance. Control by a recessive suppressor does not seem likely since a heterozygous (FS) pattern did not result from any mating, though, logically a suppressor cannot be completely ruled out. The Aph to Est C and Aph to Est 6 linkage map distances have been reditermined, using the deficiencies rather that electrophoretic variation, and values similar to previous results were obtained.

The segregation pattern, granting allelism and provisionally designating the "silent" allele by an O, in which the deficient types were mated to flies of the most common esterase genotype (Est C^F/Est C^F, Est 6^S/Est 6^S) and then backcrossed and inbred, is summarized in the following table.

Mating		Offspring		Total	χ^2_1
Est C	F/O x O/O	81 F/O	81 O/O	162	0.0
Est 6	S/O x O/O	108 S/O	54 O/O	162	18.0**
Est C	F/O x F/O	64 Fast (F/F & F/O)	30 O/O	94	2.5
Est 6	S/O x S/O	79 Slow (S/S & S/O)	15 O/O	94	4.1*

The tendency for the Esterase C deficient types to be produced in expected ratios and Esterase 6 deficient flies to occur less often than expected has been observed consistently in a number of small scale experiments (designed for other purposes) also. Since there exists a striking quantitative variation between males and females in regard to Esterase 6, and because of the abberant ratios, an important biological function for the enzyme is intuitively suspected, though still not known.

No association between esterase electrophoretic mobility of deficiency and external morphology has yet been detected. It has not been determined whether or not the deficiencies reflect a lack of protein, inactive enzyme, or as suggested by T. R. F. Wright, labile enzyme unable to tolerate the conditions of electrophoresis.

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Chemical selector agents for xanthine dehydrogenase (XDH) mutants of Drosophila melanogaster.

For many years it has been apparent that a chemical selector system for backmutations and wild-type recombinants at the ma-1 and the ry mutants would be very useful. Recently we have discovered that if 0.01 to 0.02% purine is added to our Drosophila media (which is a modification of the media devised by Dr. E. B. Lewis) ma-1 and ry are killed during development, while most lxd and wild-type survive. Presumably this very toxic compound is converted to hypoxanthine by XDH, and only flies having this enzyme survive. Thus, extensive studies on backmutation and genetic fine structure at the ma-1 and ry loci are now possible. When the level of purine is raised to 0.06% even wild-type flies do not survive. After treatment with mutagens it should be possible to produce and select for purine-resistant stocks. Hopefully the mechanism of the resistance will be the presence of high amounts of XDH in these flies. This method supplants the use of 4-hydroxypyrazolo(3,4-d) pyrimidine which inhibits XDH in vivo and which converts wild-type into phenocopies of ma-1 and ry flies as reported by Keller and Glassman (Nature, in press).