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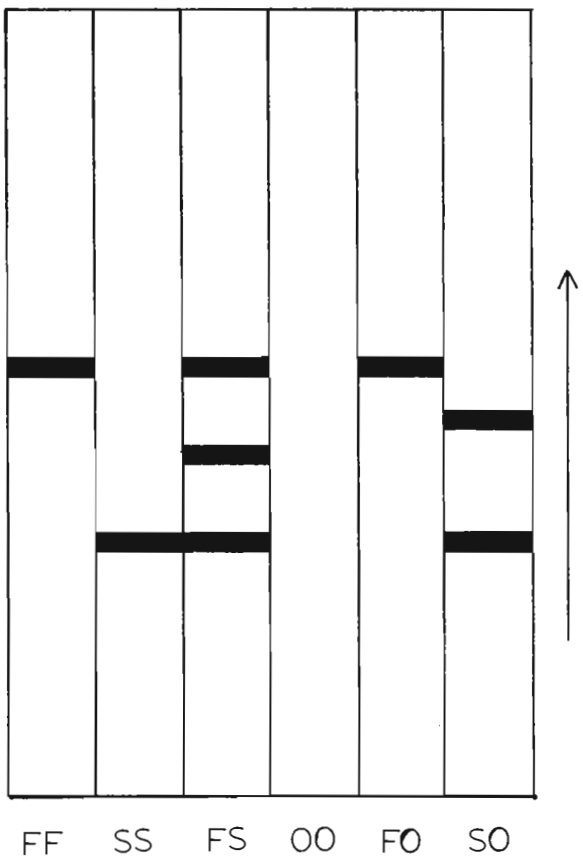
The inheritance of a deficiency of larval alkaline phosphatase in D. melanogaster.

During the latter hours of larval development a densely staining alkaline phosphatase (APH) zone is detectable when single individuals are electrophoresed in starch gels. Previous reports (Nature 20: 321, 1964; Genetics 49: 829, 1964) have described the genetic control of an electrophoretic variation in that zone and noted the appearance of a hybrid enzyme in heterozygotes. The observations are consistent with the hypothesis that the acitve enzyme is a dimer consisting of two identical subunits in homozygotes and that combination of unlike subunits in heterozygotes produces the enzyme of intermediate mobility.

An investigation of the various isozyme patterns of an inbred car strain of Drosophila revealed a complete lack of demonstrable APH activity in the area of the normally dense APH component. After confirming the deficiency in several consecutive generations, adult flies fron the stock were mated with Fast (Aph^F/Aph^F) and Slow (Aph^S/Aph^S) types and the F_1 larval offspring examined. As shown in the accompanying diagram the deficient x Fast hybrid produces only a Fast band, the intensity of which is only slightly if any less dense than that of Aph^F/Aph^F type. Alternately, when deficient x Slow hybrid larvae are examined, in addition to the expected Slow band a band in a position slightly above that of the hybrid zone of Aph^F/Aph^S heterozygotes is also observed.



Schematic comparison of larval APH phenotypes in phosphate buffer, 0.01 M, pH 6.5. Genotypes are indicated below their respective patterns.

Except for the "extra" band in the deficient x Slow heterozygotes the phenotypes are compatible with deficient control by a "silent" Aph^O allele. This is supported by the segregation ratios resulting from backcrosses, outcrosses and F_1 x F_1 mating of deficiency heterozygotes. A summary of the mating experiments is shown in the Table. Preliminary

Summary of crosses demonstrating segregation of Aph^O

Parental Aph Combinations	Offspring FF and/or FO	OO	SO	FS	SS	Total
FF x OO	31					31
SS x OO			29			29
FO x OO	74	52				126
FO x FF	84					84
SO x OO		97	69			166
SO x SS			83		87	170
SO x FF	24			21		45
FO x SS			70	67		137
FO x FO	84	29				113
SO x SO		27	60		20	107
						1008

support for allelism has been obtained by comparing map distances between Aph and a nearby locus using the deficiency as well as the electrophoretic variation.

The presence of the "extra" band in Aph^S/Aph^O heterozygotes is possibly the result of combination of an S subunit and the product of the "silent" allele, in which case Aph^O is producing protein which cannot dimerize or does dimerize but is inactive for some other reason (under the test conditions employed). No indication of a double band has yet been found in Aph^F/Aph^O type larvae. All attempts in starch with continuous and discontinuous buffer systems at pH 9.5, 8.5, 7.5, 7.0, 6.5, 6.0 and 3.0, polyacrylamide slabs at pH 8.5 and 7.0, and disc electrophoresis (Ornstein and Davis, 1961) running at pH 9.5 and 6.6 (cf. brochure, Canal Industrial Corporation) show only the single band. This might suggest a structural difference between Fast and Slow APH which prevents the Fast and "silent" protein subunits from combining into active enzyme.

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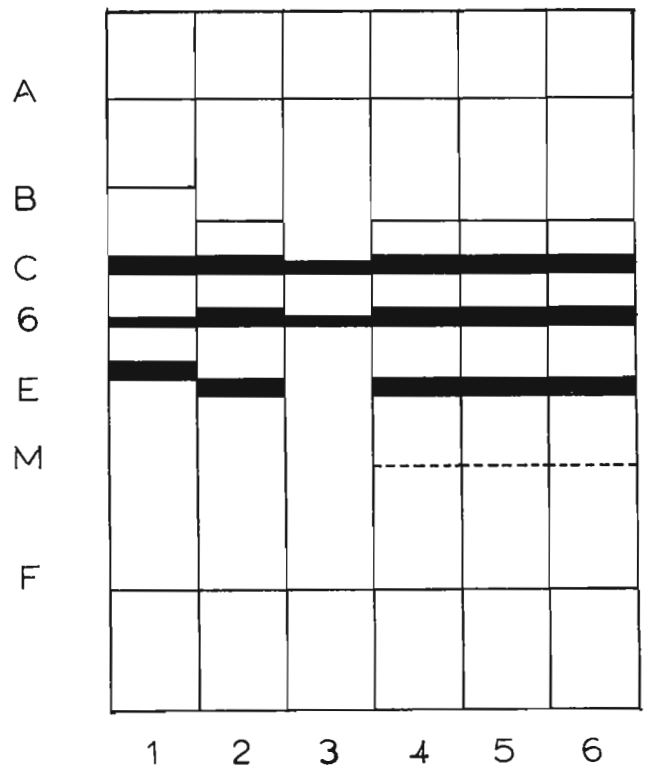
Esterase differences between male and female D. melanogaster.

Esterase zymograms of single adult Drosophila show a quantitative difference in Esterase 6 between males and females - the males having a greater amount of the enzyme or a more active form. A minor esterase component. Esterase M, sometimes not observable, when present is of greater intensity in males than in females or entirely absent in females, depending apparently on the strain under examination.

Multiple homogenates, adjusted to equivalent protein concentrations, show in addition to the quantitative variations, a slightly greater mobiltiy of Esterase B and Esterase E in females. Repeated freezing and thawing aids in making the mobility difference apparent.

Suspecting either a male esterase enhancing substance or a female esterase suppressing substance and assuming a non-random distribution of such a substance within the body, individuals of both sexes were severed in the mesothorax with a lancet and homogenates of the anterior and posterior parts prepared. On examination of the homogenates in starch gels, very little difference could be observed among female anterior, male whole, male anterior and male posterior homogenates. Only female whole homogenates show the increased mobility of Esterase bands B and E while female posterior homogenates have a decreased intensity in nearly all bands. Hence, the complete set of esterase differences can be explained on the basis of a female suppressing substance located in the posterior portion of the fly. The pertinent esterase patterns are diagrammed in the figure.



Females of the constitution XXY show a typical female esterase pattern and XO males show the pattern of normal males; XXY tra/tra females with a phenotypic resemblance to males show a male esterase pattern. The inheritance, therefore, appears to be sex-limited.

A tendency for males to exhibit a female-like esterase pattern when raised on media containing progesterone or stilbestrol has been indicated from preliminary experiments. Unfortunately the purity of the hormones was questionable in these first experiments.

Legend for figure:

1) Female multiple whole fly homogenate
2) Female multiple anterior homogenate
3) Female multiple posterior homogenate
4) Male multiple whole fly homogenate
5) Male multiple anterior homogenate
6) Male multiple posterior homogenate