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"Moulting" esterases of Drosophila.

During the ontogeny of Drosophila pseudoobscura, a series of 10 β-naphthyl esterase isozymes can be observed by electrophoresis in acrylamide gels. As seen in Figure 1, esterases 1 through 5 appear throughout development, while esterases 6 through 10 are stage specific. Esterases 8, 9 and 10 are present in first instar larvae, but gradually diminish in activity, so that by the third instar they are absent. At the onset of pupation these isozymes reappear, and continually increase in activity through metamorphosis. Esterase 6 and 7 first appear during adult development, increasing in activity until eclosion. At eclosion esterases 6, 7, 8, 9 and 10 simultaneously disappear from the adult. The loss of these isozymic forms is not due to some rapid inactivation, for all five isozymes can be observed in homogenates of the discarded pupal case. Esterases 1 through 5 are absent in these homogenates. Cohomogenization of newly eclosed adults and their empty pupal case restores the 10 banded pre-eclosion pattern.

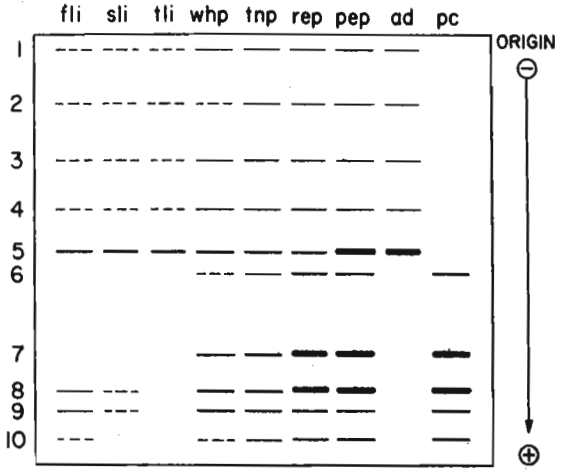

Fig. 1. Ontogeny of esterase isozymes in D. pseudoobscura. Stages are fli - first larval instar; sli - second larval instar; tli - third larval instar; whp - white pupa; tnp - tan pupa, no eye color; rep - pupa, eye colored red, no other pigmentation; pep - pre-emergent pupa, fully pigmented; ad - 9 day old adult; pc - discarded pupal case. D. pseudoobscura strain Est $5^{1.12}$ used in this study was obtained from Dr. R.C. Lewontin, Dept. of Biology, University of Chicago.

While the function of these stage specific esterases is unknown, their developmental timing and post-eclosion localization, suggest a possible role in the moulting process: we therefore designate these enzymes as "moulting esterases". Analagous esterase systems have now been observed in D. melanogaster, D. robusta, D. pattersoni, D. buskii and D. funebris. In D. pseudoobscura we now have allelic variants for several of these systems and are proceeding in genetic analyses.

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(Continued from preceding page)

mental populations is rather surprising. In addition, other tests indicate that the viability and developmental rate of null mutants do not differ from those of wild type flies. The following question, then, might be asked: Is acid phosphatase-1 a relict enzyme? No naturally occurring null alleles have been found in populations of D. melanogaster and, in fact, in this species, the gene-enzyme system is very monomorphic (O'Brien and MacIntyre, 1969; MacIntyre, 1972 and unpublished). The observation that approximately 90% of the soluble acid phosphatase activity detected by our assay methods is due to acid phosphatase-1 also argues against a trivial role for this enzyme in D. melanogaster. (Bell and MacIntyre, 1973.)

The apparent dilemma may be due, in part, to the undoubted real differences between the environments of the laboratory and the fly's natural ecological niche. Certain factors which make this enzyme activity very importnat may be absent under laboratory conditions. We plan to test this possibility as soon as the physiological substrate(s) of the enzyme are determined.

The results described here have another important ramification. If the environment of the laboratory is so different that selection pressure against individuals deficient for a particular enzyme is absent or even reduced, experiments designed to detect selective differences between individuals carrying different allozymic forms of that enzyme, are almost certainly going to give negative results. (For example, see Yamazaki, 1971.) In other words, a demonstration of selective neutrality of certain alleles specifying allozymes is, in fact, a meaningless result if the organism doesn't even need the enzyme under the conditions of the experiment.