

Whole wild-type (Oregon-R) were used as source of enzyme extracts for larvae, pupae and adults of various ages. The figure shows that there is a slight dip in enzyme activity or concentration (measured on specific activity basis) at approximately 60 hours after hatching and again just prior to emergence from the puparium at 180 hours. These dips are reproducible. There is a slight break in continuity of the curve at emergence but only minor. The activity of the adult increases to a maximum at approximately 4-5 days of age. The slight drop in activity at about day 7 of the adult may not be real, although it occurs in every assay.

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Mutation rates at the loci controlling esterase activity of *D. virilis*.

Recent studies on electrophoresis of single flies have made it possible to know the genetic variabilities of enzymes and their selection mechanisms in populations. Such studies were performed with some *Drosophila* species and indicated that there were considerable amounts of

enzyme variations in most natural populations of *Drosophila*. No one knows, however, at the present time the exact nature of the mechanisms through which the genetic variations have been maintained in the populations. One of the possible mechanisms, suggested by Lewontin and Hubby (1966), is that selection tends to eliminate alternative alleles but mutation restores them. In order to accept this hypothesis it is necessary to assume the extraordinary high mutation rates or very, very weak selection on the average. The purpose of the present study is to estimate the X-ray induced mutation rates for esterase alleles of *D. virilis* and to see whether or not the mutation rates are much higher for the esterase alleles than for visible or dysgenic alleles, such as recessive lethals.

The present study consists of two experiments; in Experiment-I the mutation rate from "inactive" to "active" is estimated, while in Experiment-II the reverse is done.

In Experiment-I, male flies of *D. virilis* taken from "null" strain, which was homozygous for the silent allele at all loci concerned, and therefore had no esterase band, were irradiated with 2,000r of X-rays and thereafter mated with the homozygous females from the same "null" strain. The progenies emerging in the next generation were examined by thin layer agar electrophoresis.

In this experiment the total number of flies examined was 9,372; no mutation was observed at all esterase loci except for Est-2 locus. At this locus we detected 2 mutations from Est-2<sup>0</sup> to Est-2<sup>B</sup>, where Est-2<sup>0</sup> was a silent gene producing no esterase band. This mutation rate was estimated to be  $1.05 \times 10^{-7}/r$ .

In Experiment-II, males homozygous for both the Est-2<sup>B</sup> and Est-9 were exposed to 2,000r of X-rays. Immediately after irradiation they were crossed to the females taken from the "null" strain used in Experiment-I. In the next generation F<sub>1</sub> flies heterozygous for "null" and Est-2<sup>B</sup> were examined by the thin layer agar electrophoresis.

A total of 14,020 flies were examined in this experiment. At the Est-2 locus 2 mutations from Est-2<sup>B</sup> to Est-2<sup>0</sup>, and one from Est-2<sup>B</sup> to Est-2<sup>D</sup> were detected. At Est-9 locus, 7 mutations to "null" were found. The mutation rates were  $0.72 \times 10^{-7}/r$ ,  $0.36 \times 10^{-7}/r$  and  $2.50 \times 10^{-7}/r$ , respectively, for Est-2<sup>B</sup>→Est-2<sup>0</sup>, Est-2<sup>B</sup>→Est-2<sup>D</sup> and Est-9→"null". No mutation from the Est-9 band to another esterase band was found. Furthermore, we found 27 cases showing that both of Est-2<sup>B</sup> and Est-9 genes mutated together to "null" genes. In this case, it is not obvious that this event is responsible for either point mutation or chromosomal aberration.

Demerec (1934) has reported the mutation rate to be  $5.2 \times 10^{-8}/r$  on the average at 9 loci on the autosome of *D. melanogaster*. The same order of the mutation rate has been presented by Alexander (1954), i.e.,  $1.5 \times 10^{-8}/r$ . At white locus which is located on the X-chromosome of *D. melanogaster*, Bonnier and Luning (1949) has estimated the mutation rate to be  $0.8-1.2 \times 10^{-7}/r$ . Girvin (1949) has estimated it to be  $7.6 \times 10^{-8}/r$  on the average at 7 visible loci on the sex-chromosome of *D. virilis*.

Comparing the results obtained in this study with those mentioned above, it seems very unlikely that the X-ray induced mutation rate at isozyme loci is considerably higher than those at visible loci. However, it cannot be determined from the present results that the genes controlling isozyme activity have either an extremely high mutation rate or a very low selective value because of the small number of chromosomes examined. Further studies should be done to accumulate data on this problem.

(The nomenclature of the esterase loci used in this study has been made by Ohba (1968), see Proc. XII Inter. Congr. Genet., Vol. II: 156.)