

Heisenberg, M. Max-Planck-Institut für biologische Kybernetik, Tübingen, Germany. Isolation of mutants lacking the optomotor response.

A multiple-Y-maze was used to select D.m. mutants lacking the optomotor response. The procedure for mutagenesis which yields sex-linked recessive mutations was the same as used by S. Benzer (Proc. Nat. Acad. Sci., 1967, 58: 1112), for the isolation of non-phototactic

mutants. From 60,000 flies tested, 17 mutants were obtained which belong to 5 complementation groups.

| <u>Group</u> | <u>Mutants</u>    | <u>Location on X-chromosome</u> |
|--------------|-------------------|---------------------------------|
| I            | opm 3,4,5,9,10,14 | 7 ± 1                           |
| II           | opm 8 (tan)       | 27.5 (tan)                      |
| III          | opm 1,7,13,15,16  | 29 ± 1                          |
| IV           | opm 6,11,12       | v < X < f                       |
| V            | opm 2,17          | 56 ± 5                          |

All mutants have abnormal electroretinograms (ERG's) and belong most likely to the same complementation groups as the ERG mutants isolated by S. Benzer and co-workers (in preparation).

In Group I mutants de- and/or re-polarisation of the retinula cells seem to be disturbed. No ERG has been found in mutants opm 4 and 9; mutants opm 3, 10 and 14 show a comparatively small and extremely slow de- and re-polarisation. In mutant opm 5 depolarisation is fast but repolarisation is very slow. The ERG of this mutant consists of a large lamina potential in addition. In all mutants of this group the optical axes of the retinula cells as judged by the deep pseudopupil are properly oriented but in contrast to wild type no green reflecting screening pigment is observed at the rhabdomers 1-6 under strong illumination. The mutants opm 3 and 5 are not completely blind behaviorally.

The ERG's of Group II and V mutants show an altered lamina potential. The receptor potential, the orientation of the rhabdomers and the pigment migration mechanisms are about normal. Light sensitivity determined by slow phototaxis is diminished by a factor of 50. The optomotor response is not completely lost. (A detailed description is in preparation.)

Group III and IV: The ERG's range from less than 1/50 to about 1/5 the normal amplitude with the lamina potential missing. Mutants without a detectable ERG do not show any slow phototaxis nor an optomotor response. In most cases the deep pseudopupil is greenish-grey and less clearly visible than in wild type. No pigment migration is observed.

Young flies of mutant opm 12 have a normal pseudopupil which, however, does not show pigment migration. The optomotor response is normal at high light intensity but absent in dim light. The ERG has about 1/5 the normal amplitude and consists of the receptor potential only. More than 10 day old flies have no pseudopupil, no ERG and no optomotor reaction. The retinula cells of this mutant seem to degenerate.

Gvozdev, V.A., V.J. Birstein, L.G. Polukarova and V.T. Kakpakov. Kurchatov's Institute of Atomic Energy, Moscow, U.S.S.R. Expression of the sex-linked genes in the established aneuploid sublines of *Drosophila melanogaster*.

A number of aneuploid sublines of embryonic cells of *Drosophila melanogaster* were obtained by cloning of the tetraploid subline. 70 - 90% cells in these sublines were characterized by their specific karyotypes remaining unchanged during at least 60 - 80 cell generations. The karyotypes of these aneuploid sublines may be represented as 4X+6A and 5X+5A as compared to

diploid karyotype 2X+4A (X-sex chromosomes, A-large autosomes without taking into account the microchromosomes of the fourth pair). The cell size and protein content per cell is equal for both aneuploid and diploid cells.

The increase in the number of X-chromosomes in both aneuploid sublines is not accompanied by the raising of the specific activities of the X-linked 6-phosphogluconate and glucose-6-phosphate dehydrogenases. However in flies with different doses of corresponding structural genes, due to the duplications or deletions of a part but not whole X-chromosome the specific activities of these enzymes have increased almost proportionally to gene dose (Seecoff et al., 1969; Gvozdev et al., 1969).

The absence of proportionality of specific activities of 6-phosphogluconate and glucose-6-phosphate dehydrogenases to the number of X-chromosomes carrying their structural genes may be therefore attributed to a phenomenon of dosage compensation for sex-linked genes which may take place not only in whole flies but also at the cell level in culture.

References: Gvozdev, V.A., V.J. Birstein and L.Z. Faizullin In: Structure and genetical functions of Biopolymers, Moscow, 1969, Vol. I: 137-165 (in Russ.); Seecoff, R.L., W.D. Kaplan and D.G. Futch, 1969 Proc. Natl. Acad. Sci. 62, 2: 528-36.

Alexandrov, I.D. Research Institute of Medical Radiology, Academy of Medical Sciences of U.S.S.R., Obninsk, U.S.S.R. Mutation isoalleles or modification of frequencies of radiation-induced viable point mutations by attendant chromosome rearrangements in *D. melanogaster*?

First Timofeeff-Ressovsky (Biol. Zbl. 52: 468-476, 1932) for  $w^+$  locus and then Lefevre (Genetics 40: 374-387, 1955) for  $y^+$  locus reported the existence of alleles with significantly different rates of mutations following X-irradiation of adult males. Such alleles were termed "mutation isoalleles". In both cases the detected frequencies of viable point mutations served essentially as an estimate of

mutability of the wild-type homoalleles.

In a previous note (DIS 44: 78) the preliminary data on comparative mutability of five wild-type genes in two stocks of *D. melanogaster* (D-32 and D-18) induced gamma-irradiation (4000 r) of adult males were presented. The frequencies of point mutations permitted to suggest the existence of the mutation isoalleles for  $y^+$ ,  $w^+$  and  $cn^+$  loci. Genetics and cytogenetics analyses allowed further to determine more precisely the frequencies of these mutations. They are listed in the Table below. It may be seen that the detected frequencies of viable point mutations for each locus are nearly three times as high in one stock (in D-18 for the  $y$  and  $w$  but in D-32 for the  $cn$ ) as in the other. The inter-stock difference for  $w$  is statistically significant. The  $w^+$  locus is known to be one of the highly mutable loci. Therefore, lack of the significant difference for  $y$  and  $cn$  point mutations may well depend on the insufficient samples of examined flies. All the same, this finding, in principle, is parallel to those reported by Timofeeff-Ressovsky for  $w^+$  alleles and Lefevre for  $y^+$  alleles.

However, if  $y$ ,  $w$  and  $cn$  mutations being taken into consideration with attendant physiological effects (sterile  $F_1$  mutations, mutations with recessive lethality) and viable chromosomal rearrangements, the mutation rates for each of the loci are practically the same in our two stocks. It seems reasonable to suppose that many of these physiological effects are associated with chromosomal rearrangements of all kinds (for example, large deletions or translocations for the sterile mutations according to Lefevre, Genetics 55: 263-276, 1967; *ibid.*, 63: 589-600, 1969; Lindsley, Proc. XII Intern. Congr. Genet. I: 144, 1968).

Thus the same gene mutation may be accompanied by rearrangements of some kinds occurring with definite but different probability in each of the two stocks. Therefore, the rearrangements occurring with different frequencies in different genotypes may differently modify the frequency of true point mutations. If this is the case, the detected frequencies of viable point mutations in different stocks may hardly provide evidence of different mutational potentialities of homoalleles themselves. Do the "mutation isoalleles" exist in this case? At least, all cases of the "mutation isoalleles" in *D. melanogaster* may be as well explained now by this hypothesis of modificatory effect of independently and simultaneously occurring chromosomal rearrangements as an attendant factor with regard to intragenic mutation.

One more aspect of the finding requires explanation: What does determine in the two stocks the different probability of complication of point mutations by rearrangements? So long as our data were obtained under the same experimental conditions and the pattern of difference in the frequencies of  $w$  and  $cn$  point mutations in D-32 is opposite to that in D-18 the stochastic of complication of these point mutations by rearrangements seems to depend upon some peculiarities of chromosomal environment neighbouring to homoalleles rather than the peculiarity of genotype as a whole. The nature of these chromosomal differences is still obscure although it can point to a possible role of amount of intercalary heterochromatin adjacent to a particular locus. The high radiosensitivity of this heterochromatin in respect to breakage is known, and the quantity of that in different stocks may be quite different (Lefevre, 1955, *loc. cit.*).

(Table on next page:)