**Garnett, D.J.** Lovesgrove House, Aberystwyth, Wales, U.K. Detection of colour mutations in Drosophila by biochemical methods.

A sample of male $^{+}/+$ **D.melanogaster** were treated with EMS in the way described by Lewis & Bacher (DIS 43:1 93) for 15 hr and then mated with XXY strain (as Jenkins 1967). The 24hr old $X_1$ were scored for mutations and homogenised in batches of 25, in 1cc of 0.04M HCl. The solutions were filtered and analysed with a Unicam 1800 spectrophotometer at 200nm (5µl per 1.2cc curvette). No attempt was made to fractionate the solution and mutations throughout the body can be picked up; however, due to the fluorescent nature of the pterin eye pigments, mutations occurring elsewhere were less apparent at these higher wavelengths. It was hoped that this kind of analysis could be routinely used in this laboratory, for detecting internal changes, where any organ with distinct pigmentation is involved, e.g., eyes, malpigian tubes, etc.

Wild type - 0.323 ± 0.004

0.125M EMS treated culture - 0.356; 0.025M EMS treated culture - 0.395.

It was found in the course of these experiments that substantial discrepancies occurred when old, killed flies were used as controls for freshly prepared extracts; the solutions, made up as above, keep well at -18°C, less well at room temperature.

I thank the Wellcome Trust and Sigma Chemicals Co.

**References:** Stein, S.P. & E.A. Carlson 1980, DIS 55:139; Jenkins, J.B. 1967, Mut. Res. 4:90-92.

**Garnett, D.J.** Lovesgrove House, Aberystwyth, Wales, U.K. A new method of mutagen application to Drosophila eggs and larvae.

During the course of my work with mosaic eye mutants, I found that chemically induced mutations occurring through development can be shown by treating the embryos and larvae with the compound. Larvae are isolated and the chorion is wetted with the mutagen using an innoculating loop. The larvae are left for 5-10 minutes and are then dried with filter paper (the larvae can be preimmersed in dilute NaOCl to remove the membrane). Embryos were deposited onto microslides coated with a yeast paste hydrated with 0.25M EMS plus 0.1% sucrose and Nipagin. Eggs are counted by transilluminating the slides, so that the percentage survival can be calculated, giving some idea of the toxicity and the mutagenicity of the chemical. The embryos and the larvae are left at 25°C to develop and are scored for mutations 24 hrs after emergence. If the eggs are all deposited in a short time (Gupta 1980, DIS 55:152), the dose (concentration x time) to each will be constant.

It was also shown that insoluble powders could be tested by working into a yeast paste, 1% sucrose, on which the adult males feed for 24 hrs.

**Gazaryan, K.G., S.D. Nabirochkin and E.N. Shibanova.** Institute of Molecular Genetics, USSR Academy of Sciences, Moscow State University, USSR. Induction with high frequency of site-specific visible mutations in the MR-strain of **D.melanogaster** by DNA injected into the polar plasm of early embryos.

In an earlier study we obtained the eye-deformed **(edf)** mutation, an abnormal development of the eye-antennal disk, by introducing the Rous sarcoma virus (RSV) into **D.melanogaster** eggs (Gazaryan et al. 1981, 1982). RSV DNA cloned in pBR322 (pPrC11, see Ambartzumian et al. 1982) rendered similar mutagenic effect (Gazaryan et al. 1984). Neither homologous DNA, nor pBR322 caused visible mutations with such a frequency (1-2 mutants among ∿ 200 flies of $F_2$ progeny of each of 1 to 5 injected embryos ($F_0$). At that time we were dealing with the wild-type Oregon R stock of **D.melanogaster**. In the present study we used **D.melanogaster** (T-007) (MR-strain) for similar experiments. The following DNAs were introduced into the polar zone of eggs at stages 7 to 8 (70-80 min. p.o. at 25°C): pPrC11 plasmid (see above), pBR322, **D.melanogaster** DNA, rat liver DNA. To activate the MR-factor, the males of T-007 strain were crossed with wild-type Oregon R females. The results are listed in the Tables.

The most important thing is that the introduction of pPrC11 containing RSV DNA insert into the polar plasm of the MR-strain embryos causes the mutation in one locus with an extraordinarily high frequency: in up to 50% of the injected embryos. The mutation alters the development of eye facets (about 1/3 of the eye facets are fused) and has been denoted $fe^{m}$ (fused eye, moderate). Apart from this,