Orevi, N. The Hebrew University, Jerusalem, Israel. Prune temperature-sensitive mutations in D. melanogaster.

A search for prune temperature sensitive (pn^{ts}) mutations was attempted in order to gain more information on the interaction between prune (pn) and killer of prune (Kpn).

Wild-type (Q.A.) males were treated with EMS and mated en-masse to sc^8 pn^1/FM6 females. pn^1 is a deletion in the prune region. Cultures were maintained at 29°C. F_1 females which were suspected to carry a pn-mutation were mated individually to FM6 males. Two cultures were established from each to these females, one was kept at 22°C, the other at 29°C. Sons were checked for the pn phenotype.

50 mutations that proved to be allelic to classic pn mutations were recovered. Of these, 10 were temperature-sensitive, namely at 29°C the eye color was brownish while at 22°C it resembled the wild-type. The pteridine pattern of the ts mutants could be distinguished also chromatographically from that of the wild-type. Nine of the ts mutations are insensitive to Kpn at both temperatures. Females heterozygous for the pn^{ts} alleles and classic pn alleles were uneffected by Kpn at both 22°C and 29°C, while their eye color was intermediate at 22°C and prune at 29°C.

One ts allele displayed temperature-sensitivity also in relation to the pn-Kpn interaction: at 29°C pn; Kpn flies died, while at 18°C at least some survived.

Woodruff, R.C. and J. Bortolozzi. University of Texas, Austin. Induction of mutations in D. melanogaster by 2-nitrosofluorene (a frameshift mutagen in prokaryotes).

Ames et al. (1972) have observed that 2-nitrosofluorene (2NF) is a potent frameshift mutagen in Salmonella typhimunium. A preliminary experiment (Woodruff and Johnson, 1973) indicated that by larvae feeding 2NF was not mutagenic in Drosophila melanogaster. Yet, it was conjectured that this lack of detected mutagenic activity may have been due to the method of administration. This report shows that this conjecture was correct, 2NF when administered to adults by injection increases the frequency of recessive sex-linked lethal mutations.

Oregon-R-C males 2-3 days old were injected with 2NF that was dissolved in dimethylsulfoxide (DMSO) and added to 0.7% saline so that the final concentration of 2NF was 0.06% and DMSO was 5%. The control solution contained 5% DMSO in 0.7% saline. A standard recessive sex-linked lethal mutation experiment was performed using FM7, y^{31d} w^a 1z v B/sc^{10-1} females. The P1 flies were pair mated and all crosses were coded to facilitate the detection of lethal clusters. To determine if visible light has an influence on the mutagenicity of 2NF, treated and control flies were divided into two groups. One group (light) received light for about nine hours per twenty-four, and the other group (dark) was kept in the dark for the entire experiment. The results of this experiment are shown in the accompanying table.

Frequency of recessive sex-linked lethal mutations

2-nitrosofluorene treatment		control	
Light	Dark	Light	Dark
6/911 (0.66%)	11/795 (1.38%)	1/956 (0.10%)	5/925 (0.54%)

The frequency of lethal mutations in the treated group (17/1,1706 = 1.0%) is significantly higher at the 1% level (Stevens, 1942) than the control group (6/1,881 = 0.32%). Among the 23 total lethal mutations recovered in this experiment one 2NF induced lethal mutation which was recovered in the light was temperature sensitive, i.e., it was lethal at 24°C but partially viable at 29°C.

There was an unexpected result in this experiment which should be mentioned. The frequency of lethal mutations produced in the dark (16/1,720) is significantly higher at the 1% level than the frequency of lethal mutations produced in the light (7/1,867). The implications of this preliminary observation are unknown. One possibility is that the absence of light increases the frequency of mutations. We have further tested this possibility and the results are reported elsewhere in this volume (Bortolozzi, Woodruff and Johnson).

References: Ames, B.N., E.G. Gurney, J.A. Miller and H. Bartsch 1972, PNAS 69:3128; Woodruff, R.C. and T.K. Johnson 1973, Genetics 74:s299; Stevens, W.L. 1942, J. Genetics 43: 301.

The junior author was supported by Fundação Amparo À Pesquisa do Estado São Paulo, Brazil.