

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-nitrosfluorene (a frameshift mutagen in prokaryotes).

Ames et al. (1972) have observed that 2-nitrosfluorene (2NF) is a potent frameshift mutagen in *Salmonella typhimurium*. 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,  $y^{31d} wa^{1z} v B/sc^{10-1}$  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-nitrosfluorene 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, Genetic 74:s299; Stevens, W.L. 1942, J. Genetics 43: 301.

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