A Thymidine-Induced Sex-Linked, Sex-Limited Temperature-Sensitive Lethal and the Significance of Modifier Genes in *Drosophila melanogaster*

In an earlier report, the author[1] investigated a group of eight sex-linked lethals induced in *D. melanogaster* by feeding it on thymidine-containing culture medium[2]. One out of these eight behaved as a temperature-sensitive lethal. This has been maintained in a balanced stock (against *y* Sc^{S1} *In* 49 Sc^{8}) in our laboratory. A pure stock of this lethal (denoted as 1^{ts}) has been established and kept at 26 °C. The fecundity and fertility are relatively good. The homozygous females are viable at 16° and 26 °C, the hemizygous males at 26 °C only. The present note is a complete analysis of this lethal (see also SUZUKI and DUCK[3] and SUZUKI, PITERNICK et al.[4]).

The stock employed for the localisation carried the markers *w sn B* in the *X*-chromosome. By the usual crossing-over technique the lethal factor 1^{ts} was found to be located at $52 \pm 1\%$. An examination of the salivary gland chromosomes stained with acetic-orcein revealed no gross changes and deletion of the size of one band could not be ruled out. It seems very probable that the lethal factor is a point mutation. Further, as the lethal effect of 1^{ts} is found to set in in the late third instar larvae/early pupae stage, this has been termed as a L/Pr-boundary lethal.

To explain the difference in the survival-behaviour of the males and females, as mentioned above, one might presume (see HADORN[5]) that in the females the activity of the ring-gland hormones produced is strong enough to make them cross the L/Pr threshold and let them survive at both temperatures. In males it is insufficient at 16° but strong enough for survival at 26 °C. However, this is just a conjecture.

The rest of the genotype being identical for both of the sexes, the temperature-sensitivity of the present lethal factor may be a result of its interaction with some modifying factor (denoted as '*m*') located on the *X*-chromosome. In males, the modifier is present in a single dose, whereas in females the dose is doubled (2 *X*-chromosomes) and the effect might be potentiated (HERSKOWITZ[6]).

To verify the above hypothesis, the homozygous females 1^{ts} *m*/1^{ts} *m* were mated to normal Oregon males and the emerging females 1^{ts} *m*/+ + in turn mated to *y* Sc^{S1} *In* 49 Sc^{8} males. The resulting recombinant and non-recombinant females were individually mated to *y* Sc^{S1} *In* 49 Sc^{8} males and a large number of cultures started at 26 °C. Out of 154 cultures checked for the presence or abscence of normal males, 13 were without normal males. Assuming the estimated number of the + *m* genotype to be equal to 1^{ts} + genotype, this would give a crossing-over rate of the order of 26/154 or 17%. The 13 cultures, corresponding to the genotype 1^{ts} + (devoid of *m*) behave as lethal both at 26° and 16 °C, being no longer temperature-sensitive. This observation is important from general biological considerations as it points out that the deleterious effects of certain genes might automatically be compensated by others. Further, the terms such as 'sub-vital', 'semi-lethal' and 'penetrance' win new significance in the light of the present observations. More experiments are under progress and will be reported elsewhere[7].

Zusammenfassung. Bei *Drosophila melanogaster* wurde ein temperaturabhängiger Letalfaktor gefunden. Die Temperaturabhängigkeit der Manifestation wird mit dem Vorhandensein von Modifikationsgenen erklärt.

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Modifications with Chelating Agents of the Effects of γ-Ray Fractionated Exposures on Chromosome Aberrations

In previous experiments[1] with *Nigella damascena* seeds, we showed that fractionation of γ-ray doses results in a decrease of chromosome aberration frequencies when intervals of time between the two exposures are longer than 3 min 20 sec and shorter than 5 min 20 sec. In this plant material, the effect of fractionation was concerned with aberrations of the chromosome class only in which exchanges are much more affected than breaks. In this research, it was clear that we were dealing with fast rejoining processes in which ionic bonds are generally imputed[2,3] which excludes the participation of some active metabolism. In view of these experiments, chelating agents were tested to modify the effects of ionizing radiations and found to be active on chromosome breakage as well as cation starvation[4–6]. In this respect, ethylene diamine tetra-acetic acid (EDTA)[7,8], 2, 2′-dipyridyl[9] and sometimes Cupferron[10] were thought to act by a mechanism of chelation of nuclear components.

The present experiments are designed to test whether, in our biological system, chelating agents can influence

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