Sobels, F.H. University of Leiden, The Netherlands. A study on the possible effect of the gene segregation distorter, SD 72, on the radio-sensitivity of sperm and spermatids in Drosophila melanogaster.

Late Drosophila spermatids are characterized by considerably lower radiosensitivity, with regard to the induction of recessive lethals or translocations, than mature spermatozoa. Earlier studies with X-irradiation in air, O_2 or N_2 (Sobels, Mutation Res. 8:111, 1969) showed that the lower radiosensitivity of late spermatids in comparison to that of spermatozoa originates from a lower degree of oxygenation, and this conclusion was further strengthened by results obtained with fast neutron irradiation (Sobels and Broerse, Mutation Res. 9:395, 1970). It was considered of interest to determine whether genotypic differences in intracellular oxygen tension would be reflected in changes of radiosensitivity. A strain carrying the gene segregation distorter was chosen to study this problem. Recent observations with the electron microscope by both Takahashi and Peacock (unpubl.) and Nicoletti (Atti. Ass. Genet. Ital. 13:1, 1968) have shown that in strains carrying the gene segregation distorter, SD, about half the cells (i.e. those carrying the SD^+ allele) contained within a cyst of late spermatids become pycnotic and decay away. It did not seem altogether improbable that the processes involved in the breakdown of the SD^+ cells require oxygen, and that consequently the intracellular oxygen tension within the remaining spermatids carrying the SD allele is possibly reduced with a concommitant effect on their radiosensitivity.

The SD 72 strain was selected for this study because of its high K value. As control, a Tokyo wild type strain was used. Every generation, both the SD and Tokyo strains were outbred to the same cn bw strain. Because this procedure had been continued for a great number of generations the two strains were, except for the SD gene, otherwise considered to be isogenic. One-day-old males of both strains were exposed to 2000 R X-rays. Mature spermatozoa were sampled by leaving the irradiated males individually with 1 female during one night, following the day of irradiation (brood A). To sample spermatids, the males were mated to 3 females per male for 1 (brood B), 2 (brood C) and 2 (brood D) days, respectively. Since the morphological manifestation of the SD gene is restricted to a late stage of spermatid development, it was thought that this sampling procedure would bring out the effect, if any.

Table 1. The frequencies of sex-linked recessive lethals, as induced by an exposure of 2000 R X-rays in sperm and spermatids of a segregation distorter (SD 72) and an isogenic wild (Tokyo) strain; pooled data of 3 replica experiments.

	Broods* - Days after treatment							
	A 0 - 1		B 1 - 2		C 2 - 4		D 4 - 6	
	N chr.	% l	N chr.	% l	N chr.	% l	N chr.	% l
SD 72/cn bw	1691	6.03	2236	4.96	1998	5.06	1418	8.25
Tokyo/cn bw	1622	6.04	2231	4.84	1818	4.40	967	7.65

* For brood A the males were mated individually to one female during one night only. For brood B, C and D the males were mated to 3 females per male, during 1, 2 and 2 days, respectively.

In two experiments tests for recessive sex-linked lethals were carried out by means of the Basc method. No differences in mutation frequencies in the successive broods were observed between the two strains. A third experiment was then carried out in which the irradiated males were mated to Inscy;bw;st p^p females; F_1 females heterozygous for the SD, or Tokyo chromosome could then be recognized and used for further testing. The induced mutation frequencies in the two different strains were not, however, different. Since there was not significant heterogeneity between experiments, the data were pooled, as they are shown in Table 1. It can be seen that the effect of the SD gene on spermatids carrying the homologous chromosome is not parallelled by a change in sensitivity to the mutagenic action of X-irradiation.

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