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California. Negative geotaxis and mating  
behavior in control and gamma-irradiated  
*Drosophila*.

Oregon R wild type male *Drosophila melanogaster* were used in this study. They were housed at 21°C and 45% relative humidity in bottles containing standard corn meal-molasses medium enriched with brewer's yeast. Once each week the flies were shaken into bottles containing fresh food.

Negative geotaxis was measured every week using a glass-stoppered volumetric cylinder of 250 ml fitted with a layer of soft plastic on its bottom. When shaken to the bottom by tapping the cylinder against a rubber mat, the flies quickly ran or flew to the upper region. The technique was standardized by using 50 flies for each measurement, tapping 10 consecutive times, and counting the number of flies that crossed the 250 ml mark in 20 sec.

For investigation of mating behavior, 24 control and 24 irradiated flies were used. The mating behavior of the irradiated flies was observed every week after shaking them into a bottle containing 72 virgin females that were 8 days old. The same procedure was used to investigate the mating behavior of the controls. At the end of 10 min., the number of matings was recorded and, after spontaneous separation of the males and females, the females were discarded and the males were saved for future matings.

#### EFFECTS OF 50 KR 60 CO $\gamma$ -RADIATION ON NEGATIVE GEOTAXIS OF DROSOPHILA

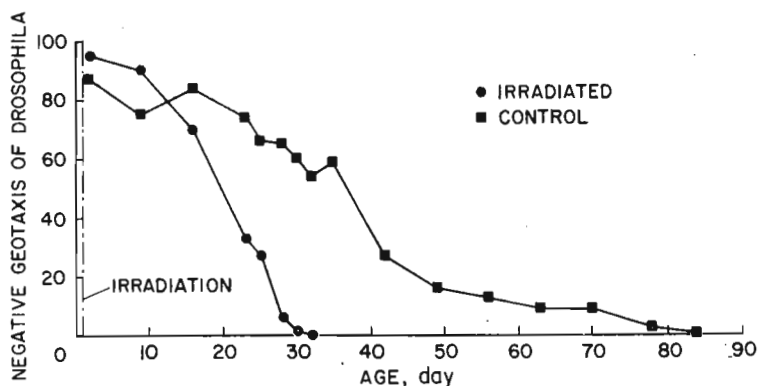


Fig. 1. Effects of 50 kR  $^{60}\text{Co}$   $\gamma$ -radiation on negative geotaxis of *Drosophila*. The points represent the percentage of flies that reached the 250 ml line on a volumetric cylinder 20 sec. after shaking them to the bottom. Each point is the mean of 10 consecutive readings.

Figure 1 shows that negative geotaxis of the irradiated *Drosophila* is in striking contrast with that of the controls. Whereas unirradiated *Drosophila* show a plateau of high activity until approximately 20 days, the negative geotaxis of irradiated flies starts declining sharply 8 to 16 days after irradiation. Zero values are attained at about 30 days of age versus 80 to 85 days for control flies. The shape of the curve for exposed *Drosophila* is

#### MATING OF CONTROL AND $\gamma$ -IRRADIATED (50 KR) DROSOPHILA

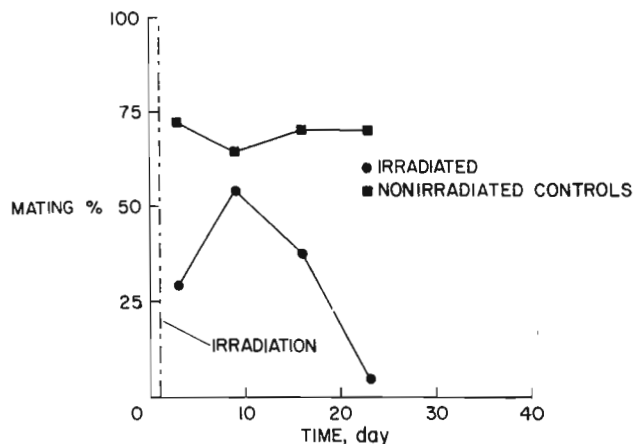


Fig. 2. Mating of control and  $\gamma$ -irradiated *Drosophila*. The points indicate the percentage of males that were able to start copulation in 10 min. Twenty-four control and 24 irradiated males were used in this study. Each group was left in a container with 72 virgin females that were 8 days old.

consistent with a radiation syndrome that rapidly progresses until death of the fly.

The data on mating frequency show that young control flies have an approximately constant high activity (Fig. 2). Mating of irradiated *Drosophila*, however, is depressed at 2 days after exposure, recovers at 8 days, and declines sharply thereafter. These observations suggest that 50 kR produce in *Drosophila* both an acute reversible injury and a chronic syndrome that eventually results in death.

Most male flies can copulate even after being completely sterilized by a dose of 50 kR. This finding agrees with our unpublished observations that control senescent male flies can also mate up to 91 days of age, several weeks after they have become sterile. This suggests that the status of the sex organs in *Drosophila* is not as important in mating behavior as the condition of the nervous system and the muscles. The degradation in performance for both negative geotaxis and mating of 9- to 16-day-old irradiated flies correlates well with the pathological changes of the brain observed starting at 14 days after exposure.

Vítek, J. J.E. Purkyně University, Brno, Czechoslovakia. The selection coefficients of heterozygotes for the recessive lethal mutations of *D. melanogaster*.

In many papers, selection coefficients of heterozygotes for recessive lethals were estimated by means of different tests (e.g. Cy/Pm). The adaptive values for lethal mutations in heterozygous condition were studied either in natural populations (Dobzhansky and Wright, 1941; Cor-

deiro, 1952; Dobzhansky and Spassky, 1968; and others), or in population cages (da Cunha, 1963; Sankaranarayanan, 1966; and others).

The majority of these authors found either the increase or the decrease of adaptive values of heterozygotes for lethals from 2% to 4%.

We have studied the selection coefficients of heterozygotes for three lethals of the chromosome 2:  $l(2)ax$  (chromosome  $al\ dp\ b\ bw\ l(2)ax$ ),  $Bl$ ,  $L^2$ ; in the population cages and in five different populations, on the genetic background of Oregon-K. The initial genotypes of these populations were:  $l(2)ax/+$ ;  $L^2/+$ ;  $Bl\ L^2/+$ ;  $L^2\ +/+$ ;  $l(2)ax$ ;  $Bl\ L^2\ +/+$ ;  $l(2)ax$ . The selection coefficient of each studied allele was estimated by comparing the theoretical relation between the frequency of normal allele and of the mutant allele with the empirical relation. The results are presented in the table.

The selection coefficients of heterozygotes for recessive lethal mutations  $Bl$ ,  $L^2$ ,  $l(2)ax$ , regarding the standard alleles, in the different experimental populations

generation	population	$l(2)ax/+$	$L^2/+$	$Bl\ L^2/+$	$L^2\ +/+$	$l(2)ax$	$Bl\ L^2\ +/+$	$l(2)ax$
	mutation	selection coefficients						
1	$Bl$			0.02			0.10	
	$L^2$		0.47	0.05	0.58		0.07	
	$l(2)ax$	0.20			0.09		-0.01	
2	$Bl$			0.36			0.22	
	$L^2$		0.74	0.35	0.63		0.09	
	$l(2)ax$	0.02			0.62		0.08	

Some of the selection coefficients are ten or more times higher than the values estimated by other authors. This increasing of selection coefficients may be caused by the specific environment of populations, further by the specific genetic background (compare the population  $Bl\ L^2\ +/+$  to others), and the specific properties of studied lethal mutations. The selection coefficients of the second generation are higher than those of the first one. This increase is caused by the increasing size of populations.

References: Cordeiro, A.R. 1952 Proc. Natl. Acad. Sci. USA 38:471-478; da Cunha, A.B. et al 1963 Proc. XI Intern. Congr. Genet., The Hague 1, 158; Dobzhansky, Th. and S. Wright 1961 Genetics 26:23; Dobzhansky, Th. and B. Spassky 1968 Genetics 59:411-425; Sankaranarayanan, K. 1967 Genetics 57:653-664.