Rajaraman, R. and O.P. Kamra. Dalhousie University, Halifax, N.S., Canada. On Ruby laser mutagenicity to Drosophila melanogaster male germ cells.

In the past decade lasers emitting at different wavelengths have been employed to study the interaction of monochromatic radiations on various biological systems. Biological effects of laser radiations depend on their wavelength, energy density as well as the pigmentation and absorbance of the target (Rajaraman and Kamra, 1970). Ruby laser (RL)-induced (6943Å) changes in somatic cells have been reported by several workers. Contradicting observations like growth enhancement by increase in cell division (Jamieson et al., 1969), blockage of mitosis in prophase and retardation of growth by cell division (Gordon et al., 1968) in mammalian cells in culture due to RL radiation have been reported. Chromosomal clumping and aberrations were observed in RL irradiated rabbit endothelial cells (Okigaki and Rounds, 1972). Exposure of D. melanogaster larvae to unfocused RL (0.1J/mm^2) produced significant variations in the life span of different stages that were transmitted as true breeding mutations (Zuzolo, 1966). In view of these observations, we decided to test the mutagenicity of RL radiation in D. melanogaster by screening for sex-linked recessive lethals.

Male Drosophila larvae of X^{c2} y/sc^8 Y stock were collected from the culture tubes when they were climbing up for pupation and were allowed to pupate on moist filter paper. 24 hrs old male pupae were irradiated with 5 or 10 pulses of RL radiation (217 mJ/pulse) to sample the spermatids which are most sensitive to radiation damage. The pupae were arranged during irradiation so that only the posterior ventral part of the pupae were exposed to radiation, to avoid unnecessary damage to the thoracic and head regions. On emergence, the males were individually mated with six 3-day old y sc^{S1} In-49 sc^8;bw;st p^p virgins for two days. The F_1 offspring were screened for lethal mutations. The treatments and the results are shown in the table.

Treatments	No. chrom. tested	No. lethal	% lethal
Control (a)	383	0	0.1
(b)	473	1	
5 x 217 mJ (a)	431	0	0.0
(b)	394	0	

It is apparent that RL radiation did not induce sex-linked recessive lethal mutation even in the spermatid stage, which is most sensitive to radiation damage. The males that were exposed to 10 pulses of 217 mJ showed temporary sterility (for two days) indicating that either the males were not able to mate due to any possible injury by irradiation or the germ cells in spermatid stage were killed, fertility being regained by repopulation due to continuously maturing germ cells. These results indicate that RL radiation is not mutagenic as regards to sex-linked recessive lethals and it may cause cell death or division delay in the spermatogenous cells probably due to secondary heat damage.

References: Gordon, T.E., C.A. Waldron and L.S. Gordon, Cancer 25:851; Jamieson, C.W., M.S. Litwin, S.E. Longo and E.T. Krementz 1969, Life Sci. 8:101; Okigaki, T. and D.E. Rounds 1972, Radiat. Res. 50:85; Rajaraman, R. and O.P. Kamra 1970, Photochem. Photobiol. 11:121; Zuzulo, R.C. 1966, NASA Rep. 1-66-1:1.

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Table 3. χ^2 values for the differences of sex-linked recessive lethals for generations compared

S1 No	Group	BROODS A	B	C	TOTAL
1	Control vs F_1 treated	5.58	1.35	0.403	0.9142
2	Control vs F_2 treated	0..21	0.272	0.610	0.07997
3	Control vs F_3 treated	0.523	0.208	1.437	5.317
4	Control vs F_4 treated	0..21	0.546	3.124	1.7804