and Race B is heterozygous for IIID.

Thus strong non-sexual reproductive isolation between a strain of D. rubida from Honiara, Solomon Islands, and the three established races of D. rubida together with a unique inversion pattern justifies the designation of a fourth race of D. rubida.

Literature Cited: Mather, W.B. 1961. Chromosomal polymorphism in D. rubida Mather. Genetics Princeton 46: 799-810. Mather, W.B. 1964. Speciation in D. rubida. Evolution Lancaster Pa. 18: 10-11. Mather, W.B. 1968. A third race of D. rubida. Pap. Dep. Zool. Univ. Qd. 3: 75-77. Strickberger, M.W. 1962. Experiments in Genetics with Drosophila. Wiley, London.

Lifschytz, E.* and Falk, R. Hebrew University, Jerusalem, Israel. Some further studies of reversion at the K-pn locus.

A.1. An attempt was made to obtain a dose curve for induced reversions of K-pn (RK's) using X-ray in mature sperm. Preliminary results are given in Table I. Details of the experimental procedures are given in Lifschytz and Falk, Genetics, 1969. The number of females/culture indicates larval density. Each female represents ca. 200 tested zygotes or 400 hatched larvae. At the bottom of the table the averaged result of E.M.S. treatment is given.

Table I

Dose	Female Culture	Replicates	Total Females	No. Revertants	Revertants/Recovered Females
500	4	2	1,020	3	1/340
1,000	4.1	3	924	5	1/185
2,000	3.7	4	1,647	13	1/196
3,000	2.0	2	852	15	1/57
4,000	1.7	3	488	10	1/49
Control					1/3000
			E.M.S.		
0.2%	1.54	2	593	15	1/40

2. The conclusions one can draw are:

a. The induction of RK's mutant (recessive lethals, presumably small deficiencies) follows one hit kinetics.

b. The efficiency of E.M.S. in inducing RK mutation, as compared to the efficiency of X-ray, is 20%. This conclusion is based on the fact that with the same E.M.S. treatment and with the same flies, 48% recessive lethals are induced on the X-chromosome. By extrapolation from the known dose-effect relations for X-ray induced sex-linked-recessive lethals, it is possible to estimate that a dose of X-rays that would produce 48% lethals would produce one RK mutant per 10 females. Moreover, this is an underestimate since with 48% lethals at least one-third of the chromosome carries two lethals.

Assuming that RK's are deficiencies, and X-ray induced lethals are mostly deficiencies, one can hopefully use this system for estimation of the point mutation/deficiencies ratio following different mutagenic treatments.

B. Apart from being all recessive lethals and allelic to each other, about 30 RK mutants both from X-ray and E.M.S. were tested for 2;3 translocation or gross inversions. Surprisingly enough none of them was associated with a translocation or an inversion. The implication of this finding will be discussed elsewhere.

In agreement with previous findings, 15 pairwise combinations of different RK's (hence recessive lethals) that were tested for complementation of the K-pn effect turned out to be noncomplementing.

This has been done using free duplication (Falk and Shamai) for the K-pn gene, thus enabling us to test whether the genotype

$$pn/Y;\frac{RK^1}{RK^2},\ Dp(3;f)ca^+bv^+K\text{-}pn^+$$

is lethal. Up to now none of the $RK^1/RK^2/K\text{-}pn^+$ combinations regain the K-pn/K-pn$^+$ interaction with pn.

*Present address: Department of Biology, University of California at San Diego, La Jolla, California.