reported in *Ustilago*[23], *Aspergillus*[24] and *Saccharomyces*[10,25], which enhance mitotic inter- and intragenic recombination in diploid strains, but the possibility that some of the mitotic segregants may have been due to chromosomal abnormalities has not been ruled out.

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Differential lethality in developmental stages of *Drosophila* following X-irradiation

Daniela Guerra, Mirella Mochi, Sandra Grazia and D.L. Palenzona

Institute of Genetics, University of Bologna, Via F. Selmi 1, I-40126 Bologna (Italy), 1 December 1978

Summary. A *Drosophila melanogaster* line has been treated with ionizing radiations. The dose-response relationship has been studied upon separate treatment of male and female gametes. The results show that while the total survival is similar, at different developmental stages differences can be observed between progenies from treated male and female gametes. It is suggested that developmental patterns may affect the expression of induced mutations.

Induced mutation frequencies result from a number of factors both internal and external to the cell[1,2]. Treatments with mutagens have shown different responses due to age, developmental stage of treated individuals, stage of cellular division, environmental conditions, sex and so on.

The different sensitivity to X-rays treatment of male and female germ cells, already reported[3] as a greater induced mutation frequency in the males, is thought to be a process of cell selection, which could be more effective in virgin females, considering the different stage of germ cells maturation in the adults of the 2 sexes[4]. On the other hand, the mechanisms effective at the molecular level in modifying the response to mutagenic treatments which may be visualized by the differential damage produced, involve enzymatic activities which are crucial for cellular metabolism[5].

Gatti et al.[6] also explain the induced mutation frequency on the 2 sexes by differences in the efficiency of the induced damage recovery mechanisms, linked to particular enzymatic complexes, which are more or less active in the 2 sexes. These enzymatic activities could be the same involved in the recombination process. On the other hand, genetic mechanisms are needed satisfactorily to explain the genetic control of the mutagenic action, since differences in mutagenic responses are often found to be under genetic control. In a previous experiment[7], different mutagenic responses were obtained by irradiation of different selection lines genetically related; on the basis of the experimental design used, it was argued that the different mutation frequencies observed could result from some kind of control of gene activity.

Since differential gene activity could be expressed at various developmental stages, the work reported in the present paper was aimed at investigating the mutagenic effect of ionizing radiation in successive developmental stages of genetically related progenies.

Material and methods. Flies from a line *(K)* of *Drosophila melanogaster* were treated with several doses of X-rays (1.5, 3.0, 4.5, 6.0 Kr; filter 4 mm. Al.; 250 kV; 8 mA).

The *K* line has the following characteristics: 1. it was obtained by selecting for short wing heterozygous flies vg^+/vg (vg = vestigial on the 2nd chromosome); 2. it is still maintained under selection; matings are allowed only be-

't'-values for the comparison between the regression coefficients of A and B crosses within each line

	K b±SE	t	*Canton* b±SE	t
L/E				
Cross A	−5.56±0.359	4.51**	−6.03±0.804	1.98
Cross B	−9.36±0.762		−8.33±0.838	
1-(L-F)/E				
Cross A	−2.17±0.447	6.43**	−1.98±1.699	1.99
Cross B	1.86±0.379		1.54±0.518	
F/E				
Cross A	−7.27±0.564	0.91	−8.86±1.952	0.56
Cross B	−8.07±0.657		−7.62±1.078	

* = $p < 0.05$; ** = $p < 0.01$.

tween males vg^+/vg and vg/vg females. Flies within the line are therefore phenotypically wild type and vestigial in a 1/1 ratio; 3. heterozygous flies carry lethal gene/s linked to vg^+. No lethals linked to the *vg* allele are apparently present.

The following experimental design was realized:

	♀		♂
Population genotypes	vg/vg^+ vg/vg		vg/vg^+ vg/vg
cross A	X-rayed vg/vg♀	×	vg/vg^+♂
cross B	vg/vg♀	×	X-rayed vg/vg^+♂
Progeny genotypes			
cross A	vg^*/vg vg^*/vg^+		vg^*/vg vg^*/vg^+
cross B	vg^*/vg vg^{*+}/vg		vg^*/vg vg^{*+}/vg

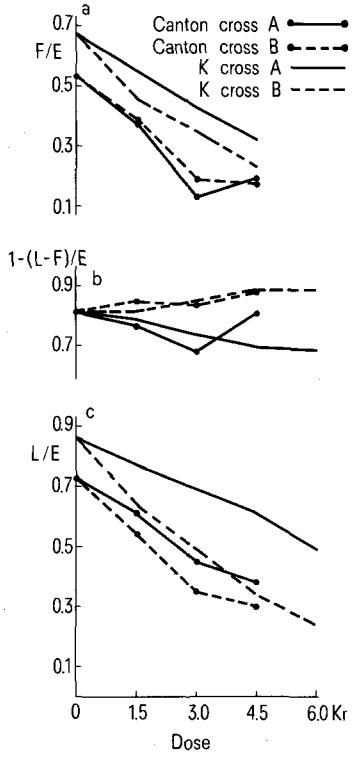
* X-rayed chromosome.

It may be noticed that all progenies carry only one of two 2nd chromosomes X-rayed; in cross A only the *vg*-marked chromosone has been treated while in cross B either vg^+ or *vg* chromosome has been X-rayed. It must be remembered that, except for the 2nd chromosome, the genotype is expected to be similar in vestigial and wild type flies due to the mating scheme followed in several hundreds generations.

The X-ray induced lethal mutants are located in trans in cross A and in cis in cross B progenies in respect to the lethals already present on the vg^+ 2nd chromosome.

To obtain F_1 progenies, only the eggs (3000–4000) layed in the first 3 days of adult life were taken. 3 independent replicates were reared at a constant temperature of 25 °C.

The induced lethality was assessed by scoring the number of eggs (E), the number of larvae (L) and the number of emerged flies (F). The L/E ratio is taken as an estimate of embryonic survival; the F/E ratio is an estimate of total survival; the post-embryonic survival is expressed as [1-(L-F)/E].



Dose-response curves of progenies from treated female (cross A) and male (cross B) gametes of 2 lines (*K* and *Canton*): *a* Total survival; *b* post-embryonic survival; *c* embryonic survival.

In order to estimate the relative importance of the genetic background, the results obtained on the *K* line are compared with those resulting from similar experimental procedures applied to a *Canton* laboratory stock genetically related to *K* line.

Results and discussion. The survival values observed are shown in the figure. In both lines *(Canton* and *K),* the total survival (number of emerged flies on the total number of eggs considered: F/E; figure a) shows a dose-response relationship quite similar when male and female gametes are treated. On the other hand, at various developmental stages, survival values vary according to whether male or female gametes are treated (figures b and c).

This is especially evident in *K* line: in the embryonic stage, survival (L/E ratio) of progenies from treated female gametes is larger, while in the post-embryonic stage [1-(L-F)/E] it is smaller than that of the corresponding progenies from irradiated male gametes.

A similar trend was observed as far as line *Canton* is concerned, but here the statistical test shows no significant difference between progenies from male and female X-rayed gametes (table).

Lethal effects induced by ionizing radiations seem therefore to differ in relation to the developmental stage at which they are detected rather than for their total value; furthermore these differences seem to be present in both *K* and *Canton* flies, being more significant in the *K* line. Differences between cross A and cross B progenies could possibly be traced to differential gametic selection[4] or to differential efficiency of repair mechanisms[6].

The intriguing feature of the results reported in the present paper is the relationship between developmental stage and response to radiations: this suggests the existence of control mechanisms which are either gene-specific or dependent on specific gene activity. In fact lethal effects detectable at different developmental stages may be reasonably thought to depend upon radiation damage involving different genes or biochemical lesions.

It is however difficult to visualize how recovery mechanisms or gametic selection could be related to developmental stages, unless the biochemical mechanisms involved were also involved in molding the developmental patterns.

In *K* line genetic differences are present between vestigial and wild type 2nd chromosome: these differences involve lethal genes which were expected to increase the total lethality of cross A more than that of cross B progenies. This expectation was not verified; moreover any initial genetic difference could not possibly explain changes relative to developmental stages unless some relationship with developmental patterns was assumed.

In conclusion it seems safe to assume that the observed different responses in cross A and cross B progenies (treatment applied on female and male gametes respectively) involve an interaction between developmental patterns and radiation damage. This in turn may have interesting implications for the expression and control of induced mutations and may be a useful tool for the study of developmental genetics.

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