# THE RATE OF INDUCTION OF DOMINANT LETHALS IN *DROSOPHILA MELANOGASTER* SPERM BY X-RAYS

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(With Two Text-figures)

## 1. INTRODUCTION

The induction of a dominant lethal in a sperm results in the death of the individual growing from the zygote produced when a normal egg is fertilized by the affected sperm. The dominant lethal may kill the individual at any stage between the zygote and the adult, but in practice it is found that death usually occurs early in life before the stage of hatching from the egg.

The existence of radiation-induced dominant lethals was first proved by Muller (1927), who showed that their 'number was so great that through egg counts and effects on the sex-ratio evidence could be obtained of them *en masse*'. Since the zygotes never develop to maturity, such lethals cannot be detected individually. Hanson (1928) irradiated *Drosophila* males belonging to the Oregon-R strain and made counts of eggs laid and hatched and of flies that emerged. Thus he determined the mortality rates in the egg stage and the larval or pupal stage. Unfortunately, no measurements of the doses of X-rays given was made in terms of the now established physical unit, the roentgen. However, an estimate of the doses used can be made by a biological method, namely, the rate of induction of sex-linked recessive lethals, since Hanson used the same treatments, T-2 and T-4, for which Muller (1928) has presented recessive mutation data. Other data on dominant lethal induction by X-rays have been given by Timoféeff-Ressovsky (1931), Gowen & Gay (1933), Sonnenblick (1940), Demerec, Kaufmann & Hoover (1938), Fano & Demerec (1941), Demerec & Kaufmann (1941). In several of these papers data is given for treatments at only one dose of X-rays. A comparison of the different sets of data discloses two discrepancies.

In the first place Gowen & Gay's data yield a straight line when plotted semi-logarithmically, suggesting that the rate of induction of dominant lethals is constant per unit X-ray dose at all doses. Fano & Demerec, however, found that the rate of induction per X-ray unit increased with higher doses, so that when the data are plotted semilogarithmically a curve is obtained, the gradient of which increases with increasing dose. Sonnenblick's data agree with this.

Secondly, there is a considerable discrepancy between the rates of induction of dominant lethals found by different workers for a given dose. Thus, compared with the latest estimate, namely, that given by Fano & Demerec, the data of Sonnenblick indicate a higher rate and those of Gowen & Gay a lower rate. Undoubtedly some of this disagreement is due to unsatisfactory dosimetry or perhaps to absorption of some of the incident energy before it reaches the spermatozoa, as is possible in the case of the soft Cu-*K* and Cr-*K* radiations used by Gowen & Gay. However, some of the difference can perhaps be attributed to genetic differences between the stocks of flies used. For Demerec *et al.*

(1938) have shown that Oregon-R shows a higher rate of induction of dominant lethals than Swedish-*b*, whereas these two races differ in the opposite way in respect of the rates of induction of recessive sex-linked lethals in them. These authors suggest two explanations for the difference: (*a*) that the dominant and recessive lethals are basically similar changes differing mainly in their degree of effect; (*b*) that they are unrelated as regards their mechanism of origin. In the first case, Oregon-R would be regarded as physiologically more sensitive than Swedish-*b* to X-ray-induced changes. Hence, because of that greater sensitivity a greater proportion of Oregon-R individuals would die early in ontogeny so that Oregon-R would show more dominant and fewer recessive lethals. On the second hypothesis, the difference could be accounted for by the presence of some biological factor that made one race more sensitive to one type of change and the other race more sensitive to the other type. Possibly the chromosomes are more susceptible to breakages and the genes less susceptible to change in Oregon-R. Or, the different sensitivity may only be an expression of some physiological condition determining the survival rate of the various changes. Then the higher sensitivity of Swedish-*b* would mean that certain changes semi lethal in Oregon-R are completely lethal in Swedish-*b*.

On the other hand, adopting an interpretation of recessive and dominant lethals that we develop in another paper (Lea & Catcheside, 1945) the effect might be accounted for by a change in the relative proportion of breaks that recombine (by restitution or structural change) to those that do not recombine.

In view of these discrepancies and the general paucity of reliable data, we have thought it worth while to describe the results of an experiment we carried out several years ago. The data show the effects of some uncontrolled errors, but it does not appear likely that we shall be able to improve the technique in the near future. The results are in general agreement with those referred to briefly by Fano & Demerec (1941), and in view of the fact that we make use of these data in a theoretical discussion of dominant lethals (Lea & Catcheside, 1945) it is desirable that the observations should be presented in detail.

## 2. Experimental methods

The experiment was planned to measure the rate of induction of dominant lethals by X-rays in *Drosophila* sperm, and to determine whether the criterion used, e.g. failure of the eggs to hatch or failure to reach adulthood, made any serious difference in the estimate obtained.

Males of an Oregon-R strain were irradiated in small gelatin capsules, perforated with small holes, without food, and afterwards mated to females of the Oregon-R strain or of a *w* strain. The males had been kept separate from females for 2 days before the irradiation. All the sets of males, including the control sets, were kept in the gelatin capsules for the same length of time before being given food. The irradiation was carried out at 170 kV. and 5 mA. at 20 cm. focal spot distance, with a filtration of 0·7 mm. of Cu plus 1·2 mm. of Al, at a constant intensity of 66·1 r. per min. The dosages originally planned were 1000, 2000, 4000, 8000 and 12,000 r., but a technical breakdown interrupted the heaviest dose at a little over 10,000 r.

Opportunity occurred subsequently to calibrate the dosemeter used in these experiments against a Victoreen dosemeter recently standardized at the National Physical Laboratory, and the revised doses given in the tables are believed not to depart from the correct doses in international roentgen by as much as 5 %. The output of the X-ray tube

was kept constant during the irradiations and the measurements by having an ionization chamber permanently in the beam and coupled to a device which automatically adjusted the tube milliamperage to compensate any fluctuations in the tube output (Lea, 1939). Errors in the *relative* values of the different doses used should therefore be negligible.

The flies were mated in $2 \times 1$ in. glass vials, closed at the open end by a moderately fine white net. This end was set on the food plate so that the eggs could be laid through the meshes. The food plates were prepared by pouring the usual corn meal-agar-treacle mixture, to which animal charcoal had been added, into 3 in. diameter Petri dishes. The surface of each food plate was then seeded sparingly with yeast before the mating vials were set in position. The dishes were covered by crystallizing dishes, and the females were allowed to lay for 12 hr. at 25° C. The mating vials were removed and the number of eggs laid counted. The Petri dishes were then covered and allowed to incubate for 24 hr., at the end of which time the eggs not hatched were counted, the number hatched being found by difference. Next the food plate in the Petri dish was divided into three portions, each being placed in a separate half-pint milk bottle containing the standard yeasted food-cake, and a piece of towelling paper on which pupation could take place. This subdivision was intended to avoid overcrowding in the cultures with the greater numbers of viable individuals.

The bottles were kept at room temperature as soon as hatching of the adults appeared imminent. The counting of the adults was then continued until no further flies hatched, the criterion adopted being an absence of further flies on three successive days. The numbers of males and females amongst the adults were noted. Finally, each bottle was carefully inspected for unhatched pupae. Thus the number of unhatched pupae added to the number of adult flies gave the number of larvae reaching maturity and pupating. The data obtained are summarized in Table 1.

## 3. ANALYSIS OF THE RESULTS

In each series and at each dosage separate counts are available for usually two or three cultures. $\chi^2$ tests were made to see whether the differences in the percentages of lethals obtained in the two or three different cultures at each dose were greater or not than the variation expected on the basis of the binomial frequency distribution. The tests showed that the differences significantly exceeded the inevitable statistical fluctuation. The excess variation existed in the controls as well as in the irradiated cultures, and was greater in series (*a*), white females, than series (*b*), Oregon-R females, and is presumably to be attributed to some non-homogeneity in the stock of flies or in the cultural conditions. If the variation had followed the binomial frequency distribution, then the standard deviation to be attached to the fraction $q$ of eggs hatching, or to the fraction $p = 1 - q$ of the eggs not hatching, would be $\sqrt{(pq/n)}$, where $n$ is the number of eggs treated. Under the present circumstances, this estimate of the standard deviation needs to be increased by multiplying it by $\sqrt{(\chi^2/\text{D.F.})}$, i.e. the square root of the ratio of the value of $\chi^2$ to the number of degrees of freedom.

The $\chi^2$ tests led to the following estimates of this factor:

| | |
|---|---|
| *w* females, controls | 6·3 |
| *w* females, irradiated (mean) | 2·6 |
| Oregon-R females, controls | 3·4 |
| Oregon-R females, irradiated (mean) | 1·6 |

The errors indicated in Table 1 are standard deviations calculated as $\sqrt{(pq/n)} \times \sqrt{(\chi^2/\text{D.F.})}$.

Inspection of Table 1 shows that the individuals coming from the *w* females × Oregon-R males mating were somewhat less viable than those from the Oregon-R females × Oregon-R

Table 1. *Experimental data*

| Dose r. | Eggs No. | Larvae No. | Larvae % of eggs | Pupae No. | Pupae % of eggs | Flies No. | Flies % of eggs |
|---|---|---|---|---|---|---|---|
| | | Series (*a*). *w* females × Oregon-R irradiated males | | | | | |
| 0 | 679 | 662 | 91·8±5·3 | 607 | 82·9±7·2 | 607 | 82·7±7·2 |
| | 307 | 244 | | 208 | | 208 | |
| | 62 | 56 | | 54 | | 54 | |
| 1,124 | 868 | 725 | 83·8±2·6 | 541 | 66·3±3·2 | 535 | 65·9±3·2 |
| | 295 | 247 | | 221 | | 521 | |
| | 230 | 196 | | 162 | | 162 | |
| 2,240 | 483 | 271 | 56·1±5·8 | 207 | 42·9±5·8 | 203 | 42·0±5·8 |
| 4,490 | 959 | 404 | 40·2±3·2 | 238 | 24·1±2·8 | 231 | 23·7±2·8 |
| | 321 | 136 | | 95 | | 95 | |
| | 288 | 90 | | 45 | | 45 | |
| 9,030 | 632 | 28 | 4·4±1·6 | 28 | 4·3±1·6 | 26 | 4·1±1·6 |
| | 470 | 21 | | 19 | | 19 | |
| 11,420 | 111 | 6 | 1·7±1·7 | 5 | 1·2±1·4 | 5 | 1·2±1·4 |
| | 293 | 1 | | 0 | | 0 | |
| | Series (*b*) | Oregon-R females × Oregon-R irradiated males | | | | | |
| 0 | 262 | 258 | 96·5±1·5 | 244 | 91·6±2·1 | 239 | 88·3±2·6 |
| | 1323 | 1269 | | 1230 | | 1187 | |
| | 246 | 240 | | 204 | | 190 | |
| 1,124 | 317 | 258 | 82·5±2·3 | 241 | 74·1±2·6 | 236 | 67·9±2·8 |
| | 347 | 290 | | 251 | | 215 | |
| 2,240 | 316 | 192 | 61·6±3·4 | 177 | 55·5±3·5 | 165 | 52·6±3·5 |
| | 172 | 111 | | 96 | | 94 | |
| 4,490 | 293 | 122 | 41·6±4·5 | 95 | 32·4±4·2 | 92 | 31·4±4·2 |
| 9,030 | 214 | 14 | 3·7±0·9 | 9 | 3·0±0·8 | 9 | 3·0±0·8 |
| | 906 | 27 | | 25 | | 25 | |
| 11,420 | 479 | 3 | 1·1±0·5 | 3 | 0·9±0·4 | 2 | 0·7±0·4 |
| | 66 | 3 | | 0 | | 0 | |
| | 600 | 7 | | 7 | | 6 | |

Table 2. *Survival to larval, pupal, and adult stages*

(corrected for deaths in the controls)

| Dose r. | Percentage of eggs reaching stage of | | |
|---|---|---|---|
| | Larvae | Pupae | Flies |
| 1,124 | 87±2 | 81±3 | 78±3 |
| 2,240 | 63±3 | 58±3 | 58±4 |
| 4,490 | 44±3 | 32±3 | 32±3 |
| 9,030 | 4·1±0·8 | 3·7±0·8 | 3·7±0·8 |
| 11,420 | 1·3±0·4 | 1·0±0·4 | 0·8±0·4 |

Table 3. *Percentage of larvae reaching adult stage*

| Dose r. | No. of Larvae | No. of Adults | % | Corrected % | Post-egg lethals % |
|---|---|---|---|---|---|
| 0 | 2729 | 2483 | 91·0 | 100·0 | 0·0 |
| 1,124 | 1716 | 1369 | 79·8 | 87·7 | 12·3 |
| 2,240 | 574 | 462 | 80·5 | 88·5 | 11·5 |
| 4,490 | 752 | 463 | 61·6 | 67·7 | 32·3 |
| 9,030 | 90 | 79 | 87·8 | 96·5 | 3·5 |
| 11,420 | 20 | 13 | 65·0 | 71·5 | 28·5 |

males matings, this being true of the controls as well as of the irradiated series. When the surviving percentages were corrected by multiplying by the appropriate factors to bring the survival of the controls to 100 %, it was found that there was no longer any significant

difference between the series (*a*) and series (*b*) surviving percentages. Weighted means were therefore taken which are given in Table 2.

In Fig. 1 we plot semi-logarithmically the percentages of eggs reaching the larval and adult stages respectively as a function of the dose. At small doses the curves are linear,

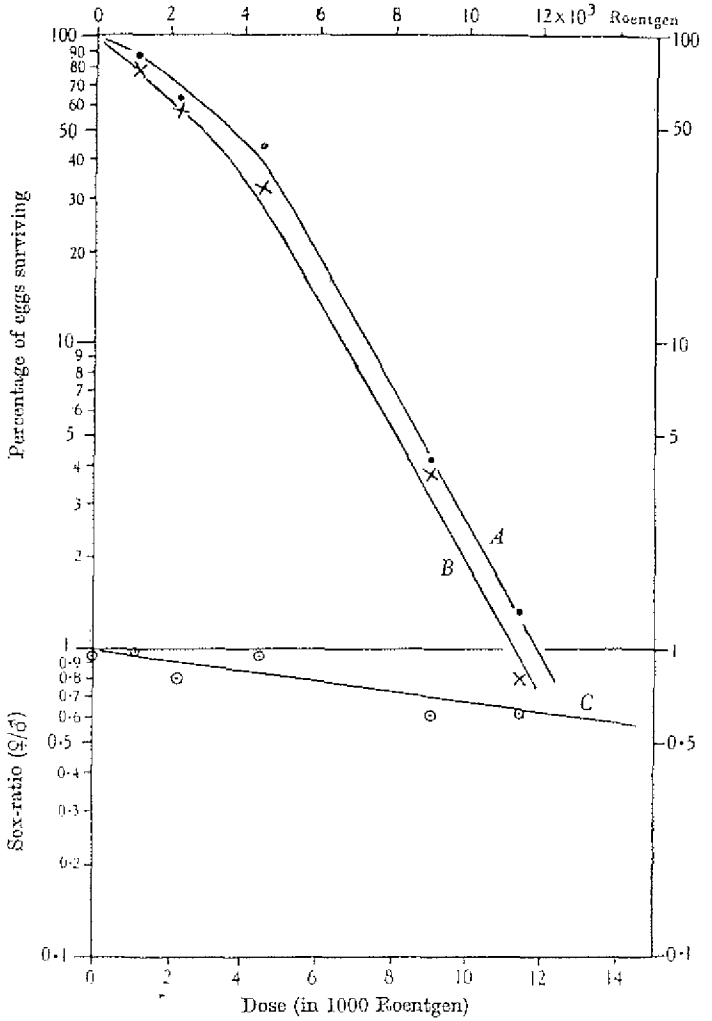


Fig. 1. Rates of X-ray induction of dominant lethals in *Drosophila melanogaster* Oregon-R race sperm. *A* shows the percentage of eggs hatching, the dots being experimental values; *B* the percentage of eggs surviving to adult stage, the crosses being experimental values; *C* the sex ratio (females/males), the circles being experimental values.

but the gradient increases at large doses, suggesting that a mixture of 'single-hit' and 'multiple-hit' effects contribute to the total yield of dominant lethals.

It is seen from Table 2 and Fig. 1 that the dominant lethal effect is expressed mainly in the egg stage. It is of interest, however, to consider how the percentage of post-egg lethals varies with dose, since this might give some information on the nature of the damage to the sperm which expresses itself in this way as a semi-lethal effect. In Table 3 the relevant data are set out. They are very erratic, but suggest that the percentage of

post-egg lethals increases less rapidly, rather than more rapidly, than the first power of the dose, in contrast to the egg-lethals. Comparison of curves *A* and *B* in Fig. 1 suggests that the proportion of larvae and pupae dying tends with increasing dose to a limiting value of 25–30 %, which is attained at about 4000 r.

The number of females in the offspring of an irradiated male parent is lower than the number of males, owing as is well known to the extra probability of a dominant lethal being induced in an *X*-bearing sperm as compared with a *Y*-bearing sperm receiving the same dose. The ratio of females to males is plotted against the dose on a semi-logarithmic scale as curve *C* of Fig. 1. The data are not adequate to determine the shape of the curve; it can be satisfactorily fitted by a straight line ($\chi^2=4{\cdot}1$, $n=4$, $P=0{\cdot}4$) of gradient $3{\cdot}8\pm1{\cdot}4$ % change of sex ratio per 1000 r. The line *C* of Fig. 1 was drawn with this gradient (which was obtained from the data by assigning weights proportional to the numbers of flies counted at each dose and using the method of least squares). The sex ratios for the different doses given by line *C* are listed in the last column of Table 4, assuming that the sex ratio in the controls should be unity.

## 4. Discussion

In Table 5 and Fig. 2 we have collected together all the dominant lethal data available in the literature. The two curves shown in Fig. 2 we have drawn through the two most recent and extensive sets of data available, namely, Sonnenblick's (curve *A*) and our own (curve *B*). It is seen that these agree well in general shape. The principal characteristic is that at small doses the curve is linear, but at higher doses the gradient increases. This same shape has been found also by Fano & Demerec (1941) in extensive experiments not yet fully published. The general shape of the curve can therefore be considered known with certainty, although in early experiments Gowen & Gay (1933) obtained points which do not depart significantly from a straight line. In plotting the data of Gowen & Gay in Fig. 2 we have made the assumption, proposed by Schultz (1936), that Gowen & Gay's doses, which were stated in electrostatic units per c.c. and which should be practically identical with roentgen, actually need to be divided by the factor 2·44 to convert them to roentgen. This factor was chosen by Schultz to bring Gowen & Gay's yield of sex-linked recessive lethals into agreement with those of other authors; we see from Fig. 2 that the same factor brings their yield of dominant lethals within the range of values obtained by other authors.

It is rather unsatisfactory to find so large a quantitative difference as exists between curves *A* and *B* of Fig. 2. The discrepancy between the different experiments is even greater than appears in the figure, since curve *A* refers to survival to the larval stage, and curve *B* to survival to the adult stage. It is difficult to believe that errors in dosimetry in modern experiments are great enough to account for the discrepancies. It is possible that the yield of dominant lethals is different in different stocks of flies. Demerec *et al.* (1938) found a difference between the yields when Oregon-R and Swedish-*b* males were irradiated under the same conditions. However, curves *A* and *B* of Fig. 2 both refer to results obtained with Oregon-R males, but in different laboratories.

In the experiments of Hanson (1938) and Muller (1928), included in Table 5 and Fig. 2, doses were not measured in roentgen, and the values given are those inferred from the yields of sex-linked recessive lethals obtained with these same doses (described as 'T-2' and 'T-4') in Muller's (1928) experiments.

The shape of curve, which, as has been remarked, seems to be well established, suggests that the dominant lethals do not form a homogeneous class, but are composed of a mixture of single-hit aberrations, the yield of which is proportional to dose and which

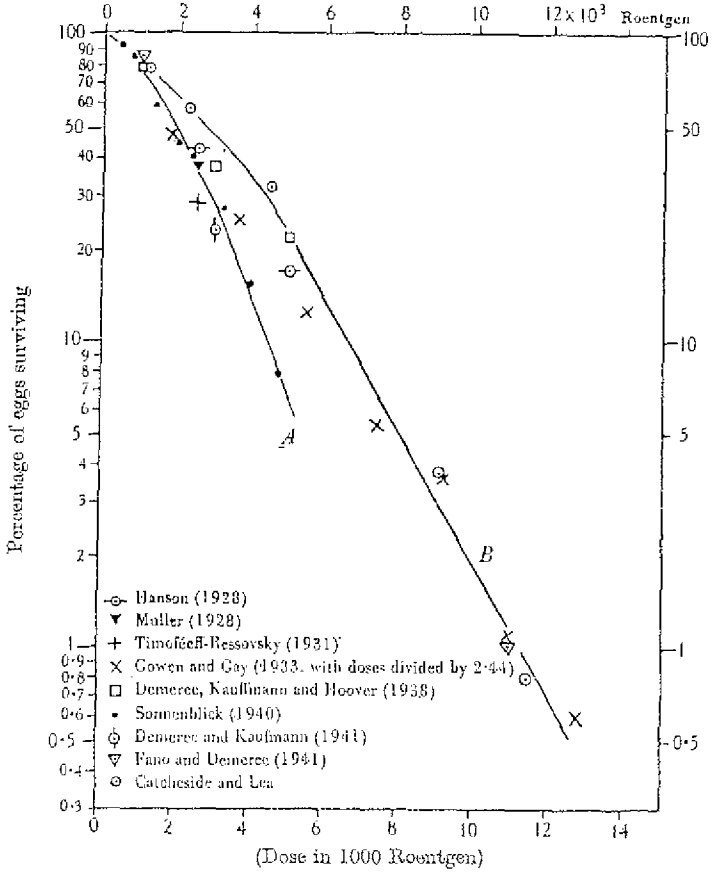


Fig. 2. Rates of X-ray induction of dominant lethals in *Drosophila melanogaster* sperm, recorded in the literature. The curve *A* is drawn through Sonnenblick's experimental points, which record egg lethals. The remaining data show total lethals resulting in death any time before the adult stage; the curve *B* is a reproduction of *B* in Fig. 1. The doses for Hanson's (1928) and Muller's (1928) experimental points are estimated from Muller's (1928) recessive sex-linked data. The doses used in plotting Gowen & Gay's (1933) data are 1/2·44 the values given by those authors (see text).

Table 4. *Variation of sex ratio with X-ray dose*

| | *w* mothers | | Oregon-R mothers | | Combined | | Ratio females to males | |
|---|---|---|---|---|---|---|---|---|
| Dose | Females | Males | Females | Males | Females | Males | Observed | Calculated |
| 0 | 457 | 410 | 753 | 863 | 1210 | 1273 | 0·95±0·038 | 1·000 |
| 1,124 | 453 | 465 | 225 | 226 | 678 | 691 | 0·98±0·053 | 0·958 |
| 2,240 | 99 | 104 | 106 | 153 | 205 | 257 | 0·80±0·074 | 0·918 |
| 4,490 | 182 | 189 | 42 | 50 | 224 | 239 | 0·94±0·087 | 0·843 |
| 9,030 | 13 | 32 | 17 | 17 | 30 | 49 | 0·61±0·14 | 0·710 |
| 11,420 | 1 | 4 | 4 | 4 | 5 | 8 | 0·62±0·34 | 0·652 |

predominate at low doses, and aberrations involving more than one hit which increase more rapidly than the first power of the dose and which become important at higher doses. The quantitative explanation of the dominant lethal curve along these lines is given in a separate paper (Lea & Catcheside, 1945).

Data on the distortion of the sex ratio in the progeny of irradiated males is rather scanty for rod-*X* stocks, though Bauer (1939) has studied this phenomenon in detail in a ring-*X* stock, where the depression of the sex ratio is much more marked. Our data (Fig. 1 *C*) indicate a depression of 3·8 ± 1·4 % per 1000 r.

Gowen & Gay (1933) obtained sex-ratio curves with two wave-lengths of X-rays which were approximately linear when plotted semi-logarithmically, the mean gradient for

Table 5. *Dominant lethal data*

| Author | Stock | Egg lethal or total lethal | Dose in r. | % of lethals |
|---|---|---|---|---|
| Hanson (1928) | — | Egg | 2,500 | 48 |
| | | | 5,000 | 72 |
| | | Total | 2,500 | 57 |
| | | | 5,000 | 83 |
| Muller (1928) | — | Total | 2,500 | 62 |
| Timoféeff-Ressovsky (1931) | — | Total | 2,500 | 72 |
| Gowen & Gay (1933) | — | Total | 1,830 | 52·9 |
| | | | 3,610 | 74·8 |
| | | | 5,490 | 87·4 |
| | | | 7,310 | 94·6 |
| | | | 9,140 | 96·4 |
| | | | 10,970 | 98·9 |
| | | | 12,800 | 99·4 |
| | | | 14,630 | 99·6 |
| Demerec *et al.* (1938) | Oregon-R | Total | 1,000 | 22 |
| | | | 3,000 | 63 |
| | | | 5,000 | 78 |
| Sonnenblick (1940) | Oregon-R | Egg | 488 | 7·8 |
| | | | 780 | 15·5 |
| | | | 1,365 | 41·0 |
| | | | 1,950 | 56·0 |
| | | | 2,340 | 59·7 |
| | | | 3,120 | 72·8 |
| | | | 3,900 | 85·5 |
| | | | 4,680 | 92·8 |
| Demerec & Kaufmann (1941) | Swedish-*b* | Total | 3,000 | 77 |
| Fano & Demerec (1941) | — | Total | 1,000 | 15 |
| | | | 11,000 | 99 |

Table 6. *Sex-ratio depression per* 1000 *r.*

| | |
|---|---|
| Hanson (1928) | 1·8 ± 1·1 |
| Muller (1928) | 4·2 ± 1·3 |
| Gower & Gay (1933) | 2·2 ± |
| Bauer (1939) | 2·0 ± 0·7 |
| Catcheside & Lea | 3·8 ± 1·4 |
| Weighted mean | 2·5 ± 0·5 |

the two wave-lengths being 0·92 % per 1000 e.s.u. per c.c. Again, adopting Schultz's factor of 2·44 to convert Gowen & Gay's doses to roentgen, we infer a gradient of 2·2 % per 1000 r.

Hanson's (1928) data yield a similar value if it is assumed, as previously, that his T-2 dose was about 2500 r., and his T-4 dose about 5000 r. The sex ratios for the two doses, after correction for the control, are respectively 0·959 and 0·911, indicating a depression of sex ratio of 1·8 % per 1000.

Muller (1928) observed the sex ratio in the $F_1$ generation of a cross between irradiated males and *ClB* females. The doses were the same as those used by Hanson, and the depression of the sex ratio, compared to the ratio in the controls, was 4·2 % per 1000 r.

Bauer (1939) obtained a sex ratio, after correction for the control sex ratio, of 0·922 at 4000 r., corresponding to a gradient of 2·03 % per 1000 r.

In Table 6 we have collected the available values of the sex-ratio depression per 1000 r. Where the authors have stated the number of flies counted, we have calculated the standard deviations assuming the numbers of males and females to be distributed in a binomial distribution. The weighted mean value is $2{\cdot}5 \pm 0{\cdot}5$ % per 1000 r. At 4000 r. the ratio of females to males will be $e^{-4\times{\cdot}025} = 0{\cdot}905$.


## Summary

The rate of X-ray induction of dominant lethals in the sperm of the Oregon-R stock of *Drosophila melanogaster* is, at low doses, 12 % per 1000 r. for death in the embryo stage, and 20 % per 1000 r. for death at any stage between the zygote and the adult. At higher doses of X-rays both rates increase. Over the whole dose range, therefore, the dose-action curve does not fit a single-hit type of action, though this is approximated at low doses.

The rate of depression of the sex ratio of females to males, founded on our own and other data, is about 2·5 % per 1000 r.


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## *Note added in proof*

Demerec and Fano (*Genetics*, **29**, 348–60, 1944) have described extensive experiments on the induction of dominant lethals. Their Fig. 1*b* shows the proportion of eggs that develop to the stage of the adult fly, after various doses of X-rays. When their experimental values are corrected for lethals in the controls by dividing by 0·81, the points are found to lie fairly well on curve *B* of our Fig. 2.