Table 2. Total sterility load (T), partial sterility load (P) and complete sterility load (C) in the irradiated and control populations.

Population	T	P	C	P:C
Males:				
A: Control	0.0755±0.0288	0.0550±0.0250	0.0205±0.0295	2.7
B: 25KR-77G	0.0703±0.0320	0.0438±0.0253	0.0266±0.0352	1.6
C: 50KR-75G	0.0954±0.0465	0.0139±0.0361	0.0815±0.0487	0.2
D: 75KR-75G	0.1636±0.0493	0.0288±0.0282	0.1348±0.0520	0.2
Females:				
A: Control	0.0640±0.0108	0.0454±0.0177	0.0186±0.0168	2.4
B: 25KR-77G	0.1066±0.0332	0.0661±0.0256	0.0504±0.0405	1.6
C: 50KR-75G	0.1112±0.0326	0.0489±0.0205	0.0623±0.0349	0.8
D: 75KR-75G	0.1623±0.0355	0.0700±0.0234	0.0923±0.0437	0.8

the populations the fertility rates are higher for heterozygotes than for homozygotes. Furthermore, in populations C and D, which had been exposed to 50,000r and 75,000r of X-rays respectively, the fertility of both the homozygotes and the heterozygotes is lower than that in the control population. The loads have been computed from the mean fertility ratios by the same method as Temin's (1966). As seen in Table 2, the total load for males and females markedly increases as an accumulated dose of X-rays increases. In populations A and B most of the total load for males and females is due to mutant genes leading to partial sterility. On the other hand, in populations C and D the total load for males is mainly due to mutant genes causing complete sterility and for females the ratio of P:C is approximately 1:1.

The results described above indicate that some of the radiation-induced mutant genes with detrimental effects on viability or fertility are maintained for a number of generations in the populations with radiation histories, although these detrimental genes may partly be eliminated by natural selection in early generations after the irradiation is suspended.

References: Temin, R.G. 1966, Genetics 53:27-46; Tobari, I. and M. Murata 1970, Genetics 65:107-119; Murata, M. and I. Tobari 1973, Jap. S. Genet. 48:349-359.

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An approximate χ^2 test as applied to mutation experiments with D. melanogaster.

The rapidly increasing interest in the development of methods for the detection of environmental mutagens has been accompanied by an interest in the statistical procedures to be employed by mutation researchers (see e.g., Armitage 1971; Berchtold 1975; Kastenbaum and Bowman 1970; Katz 1978, 1979; Traut, in press; Würgler et al. 1975). One of those procedures is the chi-square (χ^2) test. The following approximation to that test facilitates the computation of χ^2 considerably and yields nevertheless P values almost identical with those calculated in the usual way. (Note that by the help of Patau's (1942) graphs the P values belonging to the calculated χ^2 values can be obtained.) Although this approximation has already been described (Armitage 1971), it is, as far as I know, not employed to test the significance of the difference between mutation frequencies. The approximate procedure should be applied to low relative frequencies only, amounting to at most a few percent. This requirement, however, is generally complied with in mutation experiments carried out with D. melanogaster (main types of mutations studied: recessive sex-linked lethals, autosomal translocations and X-chromosomal aneuploidy). It is true that there are cases allowing the applicaiton of the following approximate formula also to high mutation frequencies (Traut, unpublished). However, it would be difficult to consider this possibility in practice and it seems, therefore, wise to use the approximate formula only when the mutation frequencies are small. As well formulae (1) to (4) presented below as the two examples illustrating the performance of the approximate test consider Yates' correction for continuity.

The following χ^2 formula is usually applied to test the significance between two mutation frequencies x_1/n_1 and x_2/n_2 (x = number of mutated units, e.g., cells, chromosomes, loci; and n = number of units analyzed), where $x_1(x_2)$ = number of mutated units and $y_1(y_2)$ = number of non-mutated units of experiment 1 (experiment 2), and $N = x_1 + y_1 + x_2 + y_2$. For low mutation frequencies (see above), formula (1)

$$(1)\quad \chi^2 = \frac{([x_1y_2 - x_2y_1] - 0.5\ N)^2\ N}{(x_1 + x_2)(y_1 + y_2)(x_1 + y_1)(x_2 + y_2)}$$

(2) $\chi^2 \approx \frac{([x_1 - x_1'] - 0.5)^2}{x_1'} + \frac{([x_2 - x_2'] - 0.5)^2}{x_2'}$ can be replaced by the simpler approximate formula (2), where $x_1' = n_1([x_1+x_2]/N)$ and $x_2' = n_2([x_1+x_2]/N) = x_1 + x_2 - x_1'$ are the expected absolute frequencies belonging to x_1 and x_2, respectively. This approximation can be derived as follows. When the formula for χ^2 is expressed in its extended version instead of by formula (1), we obtain:

(3) $\chi^2 = \frac{([x_1 - x_1'] - 0.5)^2}{x_1'} + \frac{([x_2 - x_2'] - 0.5)^2}{x_2'} + \frac{([y_1 - y_1'] - 0.5)^2}{y_1'} + \frac{([y_2 - y_2'] - 0.5)^2}{y_2'}$

where y_1' and y_2' are the expected absolute frequencies belonging to y_1 and y_2, respectively. For low mutation frequencies the contribution to χ^2 from the non-mutated units, y_1 and y_2, is so small when compared with the contribution from the mutated units, x_1 and x_2, that it can be neglected. It is this omission which transforms formula (3) to formula (2). In addition, the numerical computation carried out with formula (2) is facilitated by the fact that in formula (2), $([x_1 - x_1'] - 0.5)^2 = ([x_2 - x_2'] - 0.5)^2$, as can be shown by a simple consideration. Furthermore, when formula (2) but not when formula (1) is used, one automatically learns whether there is an expected absolute frequency (x') smaller than 5, and, therefore, whether the application of the χ^2 test is legitimate. Formula (2) could be expressed also in other ways; however, the numerical computations are carried out best when this formula is used as it stands.

Example illustrating the application of formula (2):

$x_1/n_1 = 100/1000 = 10.0\%$ (experiment 1)
$x_2/n_2 = 40/50 = 8.0\%$ (experiment 2)
(a) $x_1' = 1000([100 + 40]/1500) = 93.3$
(b) $x_2' = 100 + 40 - 93.3 = 46.7$
(c) $([x_1-x_1']-0.5)^2 = ([x_2-x_2']-0.5)^2 = 6.2 = 38.4$
(d) $\chi^2 \approx 1.24$ [The exact value, computed with formula (1) or (3), amounts to $\chi^2 = 1.36$.]

When the two frequencies to be compared with each other (e.g., one belonging to the treated, the other belonging to the control sample) are based on equal (or almost equal) sample sizes ($n_1 = n_2$), formula (2) can be simplified, because in this formula both x_1' and x_2' can now be substituted for $\frac{1}{2}(x_1 + x_2)$. We then obtain formula

(4) $\chi^2 \approx \frac{([x_1 - x_2] - 1)^2}{x_1 + x_2}$

Example illustrating the application of formula (4):

$x_1/n_1 = 30/5000 = 0.600\%$ (treated sample)
$x_2/n_2 = 5/5000 = 0.100\%$ (control sample)
$\chi^2 \approx ([30 - 5] - 1)^2/(30 + 5) = 16.46$ (The exact value, computed with formula (1) or (3), amounts to $\chi^2 = 16.52$.

References: Armitage, P. 1971, Statistical Methods in Medical Research, Blackwell Scientific Publ., Oxford, London, Edinburgh, Melbourne, pp. 138-140; Berchtold, W. 1975, Arch. f. Genetik 48:151-157; Kastenbaum, M.A. and K.O. Bowman 1970, Mutation Res. 9:527-549; Katz, A.J. 1978, Mutation Res. 50:301-307; _____ 1979, Mutation Res. 64:61-77; Pätau, K. 1942, Zeitschr. Abst. Vererb. lehre 80:558-564; Traut, H. (in press), Biometrical Journ.; Würgler, F.E., U. Graf and W. Berchtold 1975, Arch. f. Genetik 48:158-178.

Triantaphyllidis, C.D. Aristotelian University of Thessaloniki, Greece. The esterase-A of D. auraria.

In three laboratory strains of D. auraria, an enzyme polymorphism of esterase A (Est-A) could be detected by means of starch gel electrophoresis. There exist two variants of Est-A with different electrophoretic mobility, which were called Fast and Slow (Fig. 1). To analyze the genetic basis of these electrophoretic variants, homozygous stocks for each of them were constructed. Then single-pair matings in many combinations were performed. The hybrids resulting from these crosses as well as the progenies resulting from the backcrosses and from F_1 x F_1 crosses were analyzed electrophoretically. The results showed that the two esterase A variants were controlled by codominant alleles at an autosomal gene. Heterozygous individuals show two electrophoretic zones, and there is no indication for the formation of a hybrid enzyme. As far as substrate specificity is concerned the two Est-A variants showed an α-naphthyl acetate specificity in an α-β mixture. Furthermore the Est-A zones show increased activity in the presence of 10 ml n-propanol in 100 ml