samples. M69 and M70 were supplied to us by Dr. P.T. Ives, and were trapped from his Amherst population in 1969 and 1970. The M69 sample comprised 98 lines and the M70, 102 lines. The S sample consisted of 265 lines derived from an artificial population descended from several hundred flies collected at Rochester, N.Y., and maintained for several years by Dr. S. Saul. Electrophoresis on starch and acrylamide gels was used to score each line for its genotype with respect to the five loci. Lethal-bearing chromosomes were scored as well as quasi-normals.

Of the thirty tests for linkage disequilibrium between pairs of loci, four significant results were obtained, as shown in the table below.

Population	Loci	Significance level	Map distance
M69	2 and 4	.05	8.6
M70	1 and 4	.05	15.2
S	2 and 3	.01	2.9
S	3 and 5	.01	10.3

Every locus is involved in at least one significant disequilibrium. There is no sign that disequilibrium is confined to the closest pairs of loci: 2.9 is the smallest map distance in our set of loci, 8.6 is the 5th smallest (out of 10), 10.3 is the 7th and 15.2 is the 9th. There is no significant decrease of the correlation values as map distance increases.

For the pairs of loci that had a significant correlation in one population, the absolute values of the correlation coefficients for the other populations are also strikingly high. We tested these values against those for pairs of loci that had no significant correlations in any population, using the Mann-Whitney U-test, and the difference is significant at the .01 level. This is not due to high correlation between the M69 and the M70 data, since a significant result is still found when these are pooled, and the test repeated. In other words, a high correlation coefficient in the S data tends to be associated with high values in the M69 and M70 data, and vice-versa, despite the fact that these are independent populations. Furthermore, we have evidence that the M69 and M70 populations can be regarded as essentially independent, since they differ significantly both in gene frequencies at loci 2 and 5 and in the linkage disequilibrium between loci 2 and 4. This strengthens the evidence that high correlations between certain pairs of loci tend to occur in all three separate populations, although the direction of the association may vary, and suggests that these correlations may be caused by specific selective interactions between the alleles at these loci rather than by chance events occuring in the history of the populations.

Using the gene frequency estimates that we obtained, we have also calculated the expected numbers of chromosomes of each genotype, and compared them with the observed numbers. The X^2 was not significant for the M69 and M70 data, but was significant at the .05 level for S. The discrepancy in the S data could all be ascribed to the significant pairwise associations. These results, therefore, show no evidence for correlations of higher order. There is no indication of the extreme non-random association between linked genes proposed by I. Franklin and R.C. Lewontin (Genetics 65:707-734).

Inversions are not segregating at appreciable frequencies in these populations, so that the significant correlations reported cannot be ascribed to associations with inversions.

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The alteration of Di-ethyl-sulphate induced genetic damage by penicillin in D. malenogaster.

The genetic damage induced by X-rays could be increased or decreased by pre- or post-treatment with chemicals, various gases and antibiotics in Drosophila (Sobels, 1961, 1963, 1964, 1965, Burdette 1961, Clark 1963 and M.S. Rao 1965). Very few experiments were conducted to assess the possibility of altering the chemically-induced genetic damage. (Sobels 1956; Sobels and Simons 1956; Brink 1963). With a view to find out the feasibility of reducing the genetic damage induced by chemicals experiments were undertaken. Di-ethyl-sulphate known for its high mutagenic activity has been used to induce genetic damage and penicillin is tested so as to screen any alteration.

Oregon-K males of D. melanogaster were injected with 8 international units of penicillin. The treated flies were allowed to feed on a medium containing 0.4% of Di-ethyl-sulphate for 24 hours; the feeding technique was adopted from Pelecanos and Alderson (1964).

The mutagenicity was screened by the incidence of sex-linked recessive lethals. Since

Di-ethyl-sulphate cannot induce chromosomal breaks (Pelecanos and Alderson 1964) no attempt was made to score translocations. A brood pattern of 3 days interval was used (Auerbach and Sonbati 1960) and 3 broods were studied. Treated Oregon-K males were mated individually with 3 virgin females of Y sc^{Sl} In-49 sc^{8};bw:st. The virgin F_1 females were mated individually with Y sc^{Sl} In-49 sc^{8} males. In F_2, the absence of a wild type body colour male is an indication that a sex linked recessive lethal has been induced.

A series of experiments were conducted as follows: (1) control, (2) penicillin treated, (3) Di-ethyl-sulphate treated, (4) penicillin injection + Di-ethyl-sulphate, and (5) penicillin injection + 24 hours rest + Di-ethyl-sulphate.

The chi-square test has been done to compare the following groups: (1) control versus penicillin treatment, (2) Di-ethyl-sulphate versus penicillin + Di-ethyl-sulphate, (3) Di-ethyl-sulphate versus penicillin + 24 hours rest + Di-ethyl-sulphate, (4) penicillin + DES versus penicillin + 24 hours rest + DES.

If the calculated values exceed the chi-square values at 5% level for one degree of freedom, the groups compared are taken to be significantly different from each other. The results are presented in Table 1.

Table 1

Treatment	Brood A			Brood B			Brood C			Total		
	N	l	%	N	l	%	N	l	%	N	l	%
1. Control	1366	3	0.22	1716	8	0.46	1894	5	0.26	4986	16	0.32
2. Penicillin by injection	541	-	-	373	4	1.07	316	8	2.53	1230	12	0.97
3. DES feeding for 24 hr	300	39	13.00	673	84	12.84	279	25	8.96	1252	148	11.83
4. Penicillin + DES feeding for 24 hr	591	26	4.39	669	27	4.03	738	33	4.47	1998	86	4.30
5. Penicillin + 24 hr rest + DES feeding for 24 hr	338	35	10.35	346	27	7.77	510	22	4.31	1194	84	7.03

N = Total number of X-chromosomes scored l = Lethals recorded

Table 2. Chi-square values for the difference in sex linked recessive lethal frequency for the groups compared.

Sl No	Group	Brood A	Brood B	Brood C	Total
1.	Control vs penicillin	-	1.89	20.09	10.74
2.	DES vs penicillin + DES feeding for 24 hr	21.79	31.55	7.57	65.12
3.	DES vs penicillin + 24 hr rest + DES feeding for 24 hr	1.07	5.15	6.95	16.32
4.	penicillin + DES feeding vs penicillin + 24 hr rest + DES feeding	11.4	6.4	0.017	11.4

The results of statistical analysis are presented in Table 2. Analysis of the data in Table 2 clearly indicates that penicillin reduces the genetic damage induced by chemicals similar to the reduction observed with X-rays. The rest of 24 hours between penicillin and DES treatments also showed a significant reduction in the spermatid and spermatocyte stages of the male germ plasm, while the data on the penicillin + DES and penicillin + 24 hours showed a significant deviation in spermatozoa and spermatid stages. This is probably due to the elimination of penicillin from the system.