me that swb^{62b} had been tested for allelism with Fahmy's original swb allele.

The swb^{62b} phenotype is not expressed when made heterozygous with a deficiency, Df(1) w^{67k30}, that lacks bands 3C2 through 3C6. However, when made heterozygous with either cytologically normal or deficient Notch mutants or with facet-glossy (fa^g), the swb phenotype is exhibited. In fact, the eyes of swb^{62b} cannot be distinguished from those of fa^g. In heterozygous combination with fa, swb^{62b} produces a fa phenotype; with spl, the eyes are completely +. A new X-ray induced allele, swb^{71b}, behaves exactly like swb^{62b}, although its eyes are rougher and appear mottled, but with a more nearly normal color.

It is clear the "swb" was the incorrect symbol. The mutant should have been symbolized fa^{swb} as a male-viable member of the N locus complex of mutants located at 1-3.0. As a symbol fa^{swb} perhaps deserves priority over fa^g, the symbol given to the allele found in 1962. In appearance, the eyes of both swb^{62b} and fa^g are much more aptly described by the term "strawberry" than by "glossy".

Mittler, S. Northern Illinois University, DeKalb, Ill. Failure of Dimethyl sulfoxide to protect against radiation-induced sex-linked lethals.

Dimethyl sulfoxide (DMSO) has been found to protect mice[1], tissue culture[2], pseudomonas[3], and catalase[4] from radiation. In an attempt to protect cells in spermatogenesis from radiation-induced recessive sex-linked lethals, 0.1 µl of 3.5% DMSO was injected into one-day-old adult Oregon R males. These males were irradiated with 2000 R γ rays from Cs^{137} Gammator 50 at 500 R/min. and mated to M-5 females at a ratio of one male to two females. The males were presented with new harems every two days until six days after irradiation. The control males were injected with saline solution and irradiated at the same time and were also transferred to new females every two days.

Brood	Injection	Lethals	Total Chromosomes Tested	% Lethals
0-2 day	Control	26	517	5.03
0-2	DMSO	24	414	5.8
2-4	Control	26	591	4.4
2-4	DMSO	23	469	4.9
4-6	Control	21	328	6.4
4-6	DMSO	19	290	6.55

DMSO did not protect post-meiotic cells in spermatogenesis from radiation-induced recessive sex-linked lethals.

References: 1. Ashwood-Smith, M.J. 1961, Int. J. Radiat. Biol. 3:41; 2. Vos, O. and M.C.A. Kallen 1966, Int. J. Radiat. Biol. 5:609; 3. Bridges, B.A. 1962, Int. J. Radiat. Biol. 5:101; 4. Lohmann, W., A.J. Moss, Dr. and W.H. Perkins 1965, J. Nuclear Med. 6:519.

Charlesworth, B. and D. Charlesworth. University of Liverpool, England. Linkage disequilibrium in populations of Drosophila melanogaster.

We have carried out an experiment to detect possible linkage disequilibrium between five polymorphic loci located in the middle of chromosome 3 of D. melanogaster. The loci studied, their map positions, and the alleles present in sufficiently high frequency to be useful in this study, are shown in the first table.

Locus	Map Position	Alleles (Relative electrophoretic mobilities)
1. Esterase-6	36.8	1.00, 1.10
2. Phosphoglucomutase	43.4	1.00, 1.20, 1.30*
3. Larval alkaline phosphatase	46.3	1.00, 1.30
4. Xanthine dehydrogenase	52.0	1.00, 1.04
5. Aldehyde oxidase	56.6	1.00, 1.04

* This allele was present only in population S.

For references, see O'Brien, S.J. and R.J. MacIntyre 1971 DIS 46:89-93.

Sets of chromosomes were extracted from male flies by a balancer technique, and maintained either homozygous or as balanced stocks. We studied flies from three population

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