Counts of adults emerging from water treated bottles (about 10% of the total) were used to estimate the total population of zygotes yielding ry^+ recombinants. X-chromosome non-disjunction rates were also obtained from these counts, and were close to previously established values for wild type and mei-9^a females.

The data in the table indicate that intragenic recombination occurs in mei-9^a females at or above wild type rates. From analysis of flanking marker combinations in the ry^+ recombinant chromosomes it may be inferred (for rationale see Chovnick et al., 1971) that in mei-9^a, most of the recombination events are gene conversions. Indeed, only one crossover was recovered among the 20 ry^+ recombinants tested. Thus, the mei-9^a defect results in a reduction of intragenic crossing over, but not of gene conversion. Several of the ry^+ recombinant progeny of mei-9^a females (4 of 8 from ry^5/ry^{41} crosses, 1 of 12 from ry^{502}/ry^{41} crosses) transmitted ry to their offspring. This is inferred to be the result of post-meiotic segregation of ry and ry^+ in the first mitotic division of the embryo since 2 of the 5 recombinants that transmitted ry to offspring transmitted ry and ry^+ maternally derived chromosomes. Since many mal^+-mal XDH^+-XDH^- mosaic flies do not survive purine treatment although heterozygotes do (see accompanying note), the data presented here are consistent with the hypothesis that gene conversion is actually increased above wild type levels in mei-9^a females.

Current molecular models of recombination (Meselson and Radding, 1975) suggest that gene conversion and crossing over are alternative fates of a heteroduplex DNA intermediate. The reduction in crossing over and concomitant increase in gene conversion observed in mei-9^a females are not inconsistent with these models. Since a high level of post-meiotic segregation is not a feature of recombination at the ry locus or any other locus that has been tested in mei-9^+ flies, these results also suggest that the mei-9^+ excision repair function may be an important agent of excising base pair mismatch from heteroduplex DNA formed during gene conversion in Drosophila.

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Romans, P. Univ. of California, San Diego, La Jolla, California. Effects of purine selection on survival of Drosophila mosaic for Xanthine Dehydrogenase (XDH) activity.

In certain crosses rosy mosaic flies (post meiotic segregants) appear to be produced by recombination (see previous report). Since the recombinants recovered were selected for under conditions of purine treatment which result in death of XDH^- (ry) individuals, and survival of XDH^+/XDH^- (ry^+/ry) heterozygotes, it is of interest to know how well mosaics which have some tissues heterozygous XDH^+/XDH^- and the rest XDH^- survive the treatment to be scored as recombinants. The following experiment was performed to answer this question.

y f^{36a} mal females were crossed to R(1)2 w^{vc}/y^+.Y males to generate mosaic zygotes of the required kind: maroonlike flies, like rosy flies, lack detectable XDH activity (for review, see Dickinson and Sullivan, 1975). The following breeding protocol was followed in order that numbers of zygotes of the various genotypic classes in purine and water treated (control) cultures should be nearly identical. In order to insure healthy culture conditions and minimize culture dependent effects of purine, large numbers of rosy flies were allowed to lay eggs in bottles used for the crosses for about six hours prior to introduction of the experimental parents. Then a three-day brood was collected from each set of parents and treated with either 0.8 ml deionized distilled water or 0.8 ml of 0.165% (w/v) aqueous purine at the time the parents were removed from the bottles. Three additional broods were obtained in exactly the same way. Half the cultures begun on a particular day received the purine treatments in broods 1 and 3, water in 2 and 4, and the other half, the reverse.

Table 1. Results of crosses of y f^{36a} mal ♀♀ X R(1)2 w^{vc}/y^{+}.Y ♂♂

Treatment	y f^{36a} mal/ y . Y ♂♂	y f^{36a} mal/ R(1)2 w^{vc} ♀♀	Genotype gynandromorphs	y f^{36a} mal/ 0 ♂♂	other	total
Water	3660	660	198	419	1*	4938
Purine	6	668	117	0	0	791

* y f^{36a} mal/ y f^{36a} mal/ y^{+}. Y ♀

The progeny recovered from the crosses are summarized in Table 1. Equal numbers of y f^{36a} mal/R(1)2 w^{vc} non-mosaic females were recovered following both treatments: because of the protocol employed, it is assumed that close to 100% of the XDH^{+}/XDH^{-} heterozygotes survived at the dose of purine used. However, many fewer (58.3% of control) XDH^{+}-XDH^{-} mosaic gynandromorphs were recovered from the purine treated cultures. This suggests that purine treatment caused pre-adult death of a large proportion of mosaic zygotes. This might be because either a large fraction of their tissue was XDH^{-} or each of the lethal mosaics was XDH^{-} in some critical tissue or tissues.

In order to examine these possibilities, the distribution of y f^{36a} mal tissue in the recovered mosaic gynandromorphs was determined by scoring y and f^{36a} in their cuticle. Forty-six cuticular structures were scored per side. All of the mosaics recovered from water treatments and 115 of the 117 recovered from purine treatments were scored. In these mosaics the average proportion of y f^{36a} mal tissue was 60.8% for water treated zygotes and 45.6% for purine treated ones. The 60.8% f^{36a} mal tissue found in the control is higher than values usually reported for loss of R(1)2 w^{vc} (values are in the range 49-51% for maternally inherited R(1)2 w^{vc}, Hall et al., 1976). Since the two groups of mosaics were identified and scored concurrently, it is unlikely that mosaics with a low percentage of y f^{36a} cuticle were missed in one case but identified in the other. A more reasonable interpretation is that the paternally contributed ring X was very unstable and could be lost at more than one cell division.

Table 2. Distribution of amounts of y f^{36a} (mal) cuticle in recovered mosaics

Treatment	Number of flies with indicated % structures y f^{36a} per mosaic					
	1-20	21-40	41-60	61-80	81-100	Total
Water	33	19	30	48	68	198
Purine	36	17	14	21	27	115

The distribution of the amounts of y f^{36a} cuticular tissue found in the mosaics of the two series is summarized in Table 2. A contingency χ^2 test shows that the two distributions are different ($\chi^2_4 = 13.22$, $P = 0.01$). More than half the total χ^2 value (7.00) is contributed by the 1-20% y f^{36a} mal tissue classes. The number of mosaics in this class recovered after purine treatment (35) is about the number to be expected if all mosaic zygotes produced had survived purine treatment, rather than the observed 58%. Thus it appears that all or most mosaics with a low percentage of y f^{36a} mal tissue survived, and progressively fewer survived in the higher percentage groups.

Table 3. Distribution of y f^{36a} cuticular structures in recovered mosaics

Region (number of structures scored)	Average fraction of cuticle y f^{36a} per half mosaic		
	Water	Purine	Ratio Purine/water
Head (11)	.593	.548	.93
Thorax (21)	.623	.503	.79
Abdomen (14)	.537	.326	.61
Tergites 1-4, sternites 2-4	.579	.390	.67
Tergites 5-7	.511	.315	.62
Sternites 5-6	.519	.279	.52
Genitalia and analia	.447	.167	.38

To inquire whether any particular region of the fly must be XDH^{+} in order for it to survive purine treatment, I examined the fraction of cases in which any particular structure was y f^{36a} among mosaics recovered from the same treatment. An obvious feature of this analysis (Table 3) is that abdominal structures, in particular the posterior ventral ones, sternites 5 and 6 and the genital structures and analia, exhibited very low frequencies of y f^{36a} cuticle

in purine treated as compared to control mosaics. This implicates tissues in the posterior ventral parts of the larva as those responsible for lethality of XDH- (mal) and XDH^{+}-XDH- (mal) flies following purine treatment.

The failure of a large fraction of XDH^{+}-XDH- (mal) mosaic zygotes to survive purine selection suggests that recombination values may be underestimated in rosy experiments such as those referred to above in which mosaic recombinants are produced.

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Mutagenesis of the vestigial region of D. melanogaster.

Spontaneous mutations of vestigial are well known and include minor (nicked or notched wings), moderate (excised or antlered wings) and intense (strapped and vestigial) reductions of the wings. The vestigial region is located at 67.0 on the second chromosome. The normal allele is highly mutable with both x-rays and chemical mutagens, but there are many differences in its response to these mutagens than is the case for dumpy, a mutable multiple allelic system with numerous gradations of allelic expression.

Table 1. EMS induced vestigial phenotypes using +♂ x "12 pl"♀ (19°C)

Phenotype	Total	Complete	Mosaic
nick	= 91	6	85
notch	= 102	27	75
excised	= 22	8	14
antler	= 61	5	56
strap	= 11	1	10
vestigial	= 2	1	1

The 289 allelic vestigial phenotypes were obtained among 5380 total progeny (5.37% frequency). Only 33 transmitted (0.61%), mostly from the excised, antler and strap phenotypes.

Table 2. EMS induced vestigial phenotypes using +♂ x cm vg sf ♀ or +♂ x cn vg^{E7} sf ♀ (25°C)

Series	(vg)	Total	%	Transmitted	%
cn vg sf	18	8,521	0.211	4	0.047
cn vg^{E7} sf	17	13,570	0.125	3	0.022

Most of the 35 allelic phenotypes were excised, antlered, or strapped. The higher temperature eliminating the nicked and notched expressions of the F_1 heterozygotes.

In the first series (Table 1), wild type males fed EMS (0.0125M in 2% sucrose for 24 hours) were mated to "12 pl"/Cy females. The features of this series included an abundance of nick and notch F_1 phenotypes which are probably heterozygous penetrance effects (more abundant at 19°C than 25°C). Nevertheless, many transmitted mutants demonstrated the high frequency of mutation of vg^{+} with both EMS and x-rays.

In the second series (Table 2) the EMS fed vg^{+} males were mated to cn vg sf or cn vg^{E7} sf females, the cn (57.5) and sf (71.5) markers reducing the amount of modifiers compared to the "12 pl" chromosome. The vg^{E7} allele, which is homozygous normal, shows an antlered phenotype in the heteroallelic vg^{E7}/vg compound. The mutagen tests were also carried out at 25°C to reduce the penetrance of vg in the F_1 heterozygotes. The EMS induced mutants in this second series consisted of 35 fertile exceptions, 6 phenotypically complete (3 of which were gonadally normal and 3 were gonadal mosaics) and 29 phenotypically mosaic (25 of which did not transmit and 4 of which were gonadal mosaics). The phenotypes of the transmitted alleles included 5 strap alleles, one classical vestigial, and one allele similar to vg^{NO2} (with charring of the vestigial wings).

Vestigial resembles dumpy in (1) being highly mutable, (2) consisting of a range of allelic types, (3) arising mostly as mosaic phenotypes, and (4) transmitting only about 20% of its F_1 phenotypes. It differs from dumpy (1) in giving rise to more mild than extreme induced alleles, (2) in having the opposite respnose to temperature (high temperature enhances mutant dumpy expression and diminishes vestigial expression; low temperature enhances vestigial expression and diminishes dumpy expression), and (3) in being more sensitive to modifiers. Successful mutagenesis studies with vestigial demand the use of warmer temperatures (25°C to 28°C) or milder alleles to act as sifters (such as the vg^{E7} allele).

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