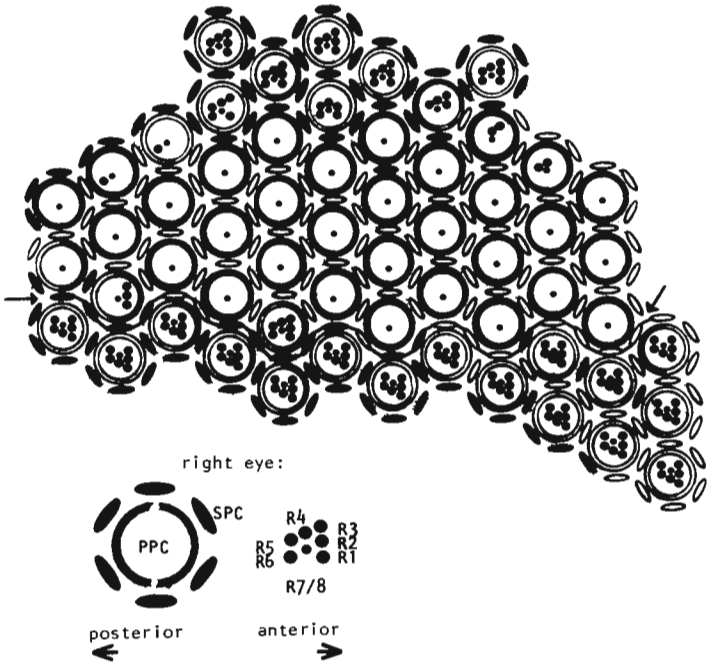

Basically, the ommatidia lacking R1-6 have unpigmented secondary pigment cells and primary pigment cells with large brown granules while ommatidia with normal receptors have pigmented secondary pigment cells and pale primary pigment cells. Most of the rest of the eye's ommatidia not drawn in this reconstruction show this same pattern of normal receptor cells. The apparent reversal from the expected primary pigment cell phenotype is caused by a previously undescribed property of cd; cd, which does not completely eliminate ommochromes, actually increases the size and visibility of primary pigment cell granules. It causes much greater ommachrome loss in secondary pigment cells. Thus, the primary pigment cells scored dark are actually cd phenotype (bw; ora cd genotype) and the paler ones (which do, in fact, have smaller brown granules) are actually phenotypically cd$^+$ (bw; ora$^+$ cd$^+$). The large mosaic studied is thus a bw, ora cd patch in a phenotypically bw (otherwise wild-type) background. Such a mosaic should have a homozygous ora$^+$ cd$^+$ twin patch (undetected in the same phenotype heterozygous background) and would be expected from an early somatic crossover event between the centromere and the closely linked ora cd vs ora$^+$ cd$^+$ in the heterozygotes.

Near the borderline, ommatidia with mixed rhabdomere and pigment cell phenotype were found. The presence or absence of R1-6 rhabdomeres was not consistently correlated with whether nearly neighboring pigment cells were bw; ora cd or bw; ora$^+$ cd$^+$ phenotype. This mosaic thus suggests that ora and cd are cell autonomous, i.e., that the mutant phenotypes are determined by the cells themselves, not by any possible interaction between receptor and eye color pigment cells or circulating factors. The pattern of receptor cell autonomy is consistent with other receptor cell mutants (e.g., see Campos-Ortega and Hofbauer 1977).

References: Campos-Ortega, J.A. and A. Hofbauer 1977, Wilhelm Roux's Arch. 181:227-245; Harris, W.A., W.S. Stark and J.A. Walker 1976, J. Physiol. 256:415-439; Koenig, J. and J.R. Merriam 1977, DIS 52:50-51; Lindsley, D.L. and E.H. Grell 1968, Genetic Variations of Drosophila melanogaster, Carn. Inst. Wash. Publ. 627; Stark, W.S. and A.W. Clark 1973, DIS 50:105-106.

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Steiner, Th. and F.E. Würgler. Institute of Toxicology, Swiss Federal Institute of Technology & University of Zürich, Schwerzenbach, Switzerland. Oocyte stages in newly hatched females of some mus and mei mutants.

A number of D. melanogaster stocks are known in which larvae exhibit increased sensitivity to chemical mutagens. Several X-chromosomal loci were identified which lead to mutagen sensitivity (mus). In addition to mutagen sensitivity some loci show strong meiotic effects (mei). It is a task for the near future to study the mutagen sensitivity of the germ cells of such stocks. In order to get comparable results with the different mutants it must be possible to treat and test comparable germ cell stages. Studies on ooctyes cannot be ini-

Table 1. Strains from which females were analyzed.

Abbreviation	Formula	Reference
mus101	w mus(1)101^{D1}	Boyd et al. 1976
mus104	w mus(1)104^{D1}	Boyd et al. 1976
mei-41	w mei-41^{D5}	Boyd et al. 1976
mei-9	mei-9^{L1}	Graf et al. 1979
mei-9/M5	mei-9^{L1}/Basc	Graf et al. 1979
w	w	Boyd et al. 1976
B.w.	Berlin wild	Steiner & Würgler 1979
$\bar{X}\bar{X}$	C(1)RM, y^{2} su-w^{a} wa bb/ y In(1)d1-49 v · YL/sc^{V1}.YS	Steiner & Würgler 1979

tiated without some basic information concerning the kinetics of oogenesis in the various mutants. To this aim we analyzed the ovarioles of freshly hatched females of a few mus and mei mutants and some control strains. Table 1 gives the genetic constitution of the strains used, the abbreviated name, and references which give further details concerning the particular mutants. The mutagen-sensitive mutants were chosen because they have known DNA repair defects: mei-9 is excision repair deficient (Nguyen and Boyd 1977), whereas mei-41, mus101, and mus104 are postreplication repair deficient (Boyd et al. 1976). The flies were cultured on our standard Drosophila medium (Würgler, Sobels and Vogel 1977) at 25°C and 60% rh under uncrowded conditions. Females 2.5 ± 1.5 h old were dissected and the ovaries analyzed as described by Bürki and Würgler (1972). Oocyte stages were classified according to King, Rubinson and Smith (1956). The results of our study are compiled in Table 2. The most advanced stages in all types of female are stage 8 oocytes (S8). Only in a few exceptions were stage 9 or even stage 10 oocytes found. Of the younger oocytes stage 7, stage 5/6 and stage 3/4 are found in slightly increasing frequencies. This unexpectedly good agreement of oocyte stages between females of such divergent genotypes indicates that the mus and mei mutants studied do not alter the kinetics of oogenesis. In addition, because the white stock is the ancestor of the mus101, mus104 and mei-41 females (Boyd et al. 1976), our results also indicate that the mutagenic treatment of germ cells of the white stock did not induce other mutations on the X-chromosome which modify the kinetics of oogenesis in these related mutagen-sensitive stocks. It is important to stress that comparable kinetics of oogenesis does not mean comparable "quality" of the oocytes studied. This is easily seen if we look at the last line of Table 2 in which we report the egg-to-adult survival observed with the ooctyes obtained from the different types of females. In contrast to the kinetics of oogenesis these data on spontaneous lethality indicate profound strain differences which seem to be due to the mus and mei mutations.

Table 2. Analysis of ovarioles and oocyte stages in 2.5 ± 1.5 h old females of different D. melanogaster strains.

	Genotypes of females							
	mus101	mus104	mei-41	mei-9	mei-9/M5	w	B.w.	$\bar{X}\bar{X}$
No. flies analyzed	20	20	20	20	20	20	18	20
No. ovarioles analyzed	824	784	817	768	786	800	783	748
Mean numbers per female:								
ovarioles	41.2	39.2	40.8	38.4	39.3	40.0	43.5	37.4
S10	--	--	--	0.05	--	--	--	--
S9	--	--	0.2	0.6	--	0.4	0.2	0.2
S8	3.4	1.8	3.8	5.8	3.8	3.2	2.9	3.0
S7	19.6	17.5	20.2	16.4	17.6	18.6	20.5	11.0
S5-6	20.0	21.6	25.1	19.7	17.8	22.6	17.5	14.4
S3-4	27.1	26.6	28.7	24.7	23.0	27.5	n.d.	n.d.
Class B oocytes	43.0	40.9	49.2	42.6	39.3	44.8	41.1	28.5
Egg to adult survival (%)	88.8	n.d.	34.9	23.8*	61.5*	88.1	92.1	33.6

n.d. = not determined *from Graf and Würgler 1978

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References: Boyd, J.B. et al. 1976, Genetics 84:485-506; Bürki, K. and F.E. Würgler 1972, DIS 46:49; Graf, U. and F.E. Würgler 1978, Mutation Res. 52:381-394; Graf, U. et al. 1979, Mutation Res. 59:129-133; King, R.C., A.C. Rubinson and R.F. Smith 1956, Growth 20: 121-157; Nguyen, T.D. and J.B. Boyd 1977, Molec. Gen. Genet. 158:141-147; Steiner, Th. and F.E. Würgler 1979, Int. J. Radiat. Biol. (in press); Würgler, F.E., F.H. Sobels and E. Vogel 1977, in: Kilby, B. et al., Handbook of Mutagenicity Test Procedures, pp. 335-373.

Stevens, P.G. and E.A. Carlson. State University of New York, Stony Brook.

Chromosome mosaics induced in ring-X by ethyl methane sulfonate and by X-rays in D. melanogaster.

Chromosomal mosaics were produced by inducing breakage of a ring-X chromosome. When this resulted in the loss of the ring chromosome during one of the early cleavage stages of the zygote, an XX/XO gynandromorph was formed. In some cases the chromosome was repaired or altered without breakage, resulting in a point mutation rather than chromosome loss. The markers w, m, f, and B were used so that the extent of mosaicism could be observed to distinguish point mutations from gynandromorphs.

Table 1. EMS-induced mosaics and their transmissibility.

Transmissibility	Gynandromorphs	Point mutations $w^+ \rightarrow w$	$B \rightarrow B^+$	$m^+ \rightarrow m$
died	1	1	1	0
nontransmissible	0	4	1	1
sterile	5	0	0	0
transmissible	0	0	1	0
lethal	0	0	2	0
total	6	5	5	1

In the first series X^{c2} y B males were fed ethyl methane sulfonate (0.0125M EMS in 2% sucrose) for 24 hours. They were then mated to w m f virgin females and progeny were examined for mosaics (Table 1). The mosaics obtained from among 4787 total progeny consisted of 6 gynandromorphs (0.1%) and 11 point mutations (0.2%). The data suggest that EMS produces more chemical alterations or repaired breaks on the ring-X chromosome, resulting in point mutations, than unrestituted breaks or aneucentric rings leading to loss and gynandromorphism.

In the second series X^{c2} y B males were exposed to X-rays (2500R) and then mated to w m f virgin females. As in the previous series, the progeny were examined for mosaics (see Table 2). The mosaics obtained from among 920 total progeny consisted of 5 gynandromorphs (0.5%) and 1 point mutation (0.1%). These X-ray results are consistent with the expectation that breakage of the ring-X chromosome is more likely to occur, producing gynandromorphs, than the induction of point mutations.

Table 2. X-ray induced mosaics and their transmissibility.

Transmissibility	Gynandromorphs	Point mutations $w^+ \rightarrow w$
nontransmissible	3	0
sterile	2	1
total	5	1

The distribution of the 11 gynandromorphs obtained is shown in Table 3. Note that in none of these 11 cases was there mosaicism for all five of the phenotypic characteristics used. Most of the gynandromorphs were genital male or female in phenotype and their sterility is probably due to incompatible head tissue of the opposite sex. The fertility of three gynandromorphs, one with an apparently male head and female genitalia, suggests that her head ganglial tissue was female or that males were successful in overcoming her behavioral barriers to reproduction. In two of the three fer-