adaptation (see Hu and Stark 1977 for arguments), resulting in behavior dominated by R7/8. At lower intensities, selected to be near R1-6 electrophysiological and behavioral thresholds, flies are photoneutral in our straight or Y arena experiments. In these arenas, reasonably light-adapted flies are shaken and given 30 s for a choice. In experiments with less agitated, dark-adapted flies orienting to extremely dim lights at their leisure, flies show strong photo-positive phototaxis probably mediated by the sensitive R1-6 photoreceptor system (Schümperli 1973; Jacob et al. 1977). These differing conditions operationally define additional phototaxis variables, namely fast vs. slow phototaxis (see Heisenberg and Götz 1975). In straight, T or Y arenas, R1-6 may mediate positive slow phototaxis near R1-6 threshold while R7/8 mediates fast phototaxis at R7/8 threshold. Phototaxis in a Hirsch-Hadler maze is obviously different. Clearly, numerous variables affect phototaxis under the differing conditions of straight, T or Y arenas vs. Hirsch-Hadler mazes; we have shown that fly strain or illumination condition cannot completely account for these discrepancies.


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Stark, W.S., R.B. Srygley and R.M. Greenberg. The Johns Hopkins University, Baltimore, Maryland. Analysis of a compound eye mosaic of outer rhabdomeres absent marked with cardinal.

Drosophila with mutant compound eye receptors have been investigated by developmental biologists and vision researchers. Harris, Stark and Walker (1976) introduced characterizations of 3 such mutants, frequently studied since. Two of these mutants, rdgB and sev (causing degeneration of retinula cells R1-6 and non-formation of R7 respectively) were shown to be cell autonomous by mosaic studies. The third mutant, ora'84, was not studied by mosaic means at that time because its third chromosome location (65.3) made mosaic induction and combination with autonomous markers more difficult. Outer rhabdomeres absent, ora, discovered and mapped by Koenig and Merriam (1977) causes non-formation of R1-6 rhabdomeres, i.e., the microvillar photopigment-containing organelles. Here we present a mosaic study of ora.

An ora stock with eye color markers, bw; ora cd, was constructed with the aid of microscopic optical techniques and histology (see Harris, Stark and Walker 1976). Brown (bw) blocks and red drosoppterin synthesis while cardinal (cd) is an eye-autonomous mutant lowering brown ommochromes to about 15%; cd (3-75.7) is near ora on the right arm of chromosome 3 (see Lindsley and Grell 1968). The bw and bw; ora cd stocks were crossed to produce heterozygotes which were irradiated at 24 to 75 hours after egg laying (rearing at 24°C) with 1200 r of gamma rays (from 137Cs source, Gammarator) to induce somatic crossing over. Several eyes mosaic for eye color were found. Heads were fixed shortly after eclosion with a hypertonic aldehyde fixative followed by osmium tetroxide (see Stark and Clark 1973) and embedded in Spur, a low viscosity epoxy. One large right eye mosaic was serial sectioned at 1 micron and examined (without staining to enhance eye color pigment contrast) for reconstruction.

The accompanying figure shows reconstruction of much of this large mosaic. The trapezoidally arranged R1-6 rhabdomeres were scored for their presence or absence. The central R7/8 rhabdomeres, not affected by ora, were always present and are thus always drawn in. Secondary pigment cells (SPC's), 6 of which surround an ommatidium and are shared between ommatidia, were scored for presence (dark) or absence (clear) of brown pigment granules. Primarily pigment cells (PPC's), 2 of which surround the distal light-focusing pseudocone in each ommatidium, were scored for the presence (dark) or absence (clear) of conspicuous large brown pigment granules. The mosaic patch is located at the eye's equator (shown by a line and arrows).
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 ommochrome 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).


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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 oocytes cannot be ini-