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A multiple Y-maze paradigm was used to select *D. melanogaster* mutants disturbed in visual orientation. Sex-linked recessive mutations were obtained by EMS mutagenesis (Lewis & Bacher 1968). Screening of a total of 15,000 flies resulted in 11 mutants which belong to six complementation

groups. The probability of visual attraction towards the pattern is shown in the last column of the following table. A probability around 0.5 suggests absence of preferred orientation towards the pattern, which is 0.75 in the wild type. The probability of 0.26 in the optomotor blind mutant *omb<sup>H31</sup>* (Heisenberg, Wonneberger & Wolf 1978) indicates an equally strong avoidance response in this particular strain.

Group	Mutants	ERG	Pig.	DPP	Sens.	Y-Maze
ERG						
1	<i>nofCB</i> (1,2,5,8,9,10)	-	+	+	0.001	0.48-0.53
OPTIC						
2	<i>nofIB3</i>	+	-	-	1	0.55
3	<i>nofKB6</i>	+	-	-	1	0.56
4	<i>nofLB7</i>	+	-	-	1	0.56
BEHAVIOR						
5	<i>nofDB11</i>	+	+	+	1	0.59
6	<i>nofEB12</i>	+	+	+	1	0.42

abbreviations: ERG - Electretinogram; DPP - Deep Pseudopupil  
Pig. - Screening Pigment; Sens. - Sensitivity

receptor potential. In some individuals of the second group the return to baseline after light-off is delayed by 2-5 s. In other flies the receptor potential is not sustained during light-on. The decay to base line during the light stimulus is similar to that of the transient receptor potential group (*trp*). The absolute sensitivity (Sens.) as shown by the ERG is reduced by a factor > 1000 for all mutants of Group I tested so far. The ERG defects in the mutants of the present complementation group partially overlap with known ERG defects in mutants of different genotype. The deep pseudopupil (DPP) and the screening pigment (Pig.) was normal in all mutants of Group I.

The three optic mutants (different complementation groups), however, show normal ERG responses, but abnormal eye color and DPP's. The eye color in these three mutants is darker and more brownish than in wild type. This is probably due to a disrupted screening pigment which can be observed in semi-thin sections. These mutants also show irregularities in the pattern of the rhabdomere endings, which can be investigated by the optical neutralization technique (Franceschini & Kirschfeld 1971). In some flies the endings are packed more closely than in the wild type and are arranged in a more circular pattern which deviates from the normal trapezoidal arrangement. This, however, was not due to a missing receptor as in the mutant *sevenless* (*sev<sup>LY3</sup>*). The number of receptors in a rhabdome was normal in all of the mutants. In none of the mutants, however, the DPP's were visible except for a few individuals in which the DPP was blurred. The defect is most probably due to a displacement of the distal rhabdomere endings from the focal plane of the dipotric apparatus of the ommatidia. This is seen by comparison of real and virtual images of the antidromically illuminated receptor endings in individual intact ommatidia through adjustment of the focal plane of the microscope. For the mutants *nofIB3*, *nofKB6* and *nofLB7* the receptor endings are located between the lenslet and its focal plane. This would suggest that the flies are unable to respond in the Y-maze, because the tiny test pattern is blurred so much that it falls below the detection threshold.

Of the 11 selected mutants only *nofDB11* and *nofEB12* do not seem to be disturbed in early visual processing. While optics and ERG appear to be normal, these mutants differ significantly from wild type in object-induced orientation. The disturbed orientation behavior of *nofEB12* can be seen already in the culture vial or in free flight: Mutant flies seem to turn more often than wild type. It seems that *nofEB12* is not able to maintain a straight course towards an object over extended periods of time.

The physiological and structural integrity of the peripheral stages of information processing in the visual system of the mutants were checked by measuring the electretinogram (ERG) and observing the deep pseudopupil (DPP). The mutants in the first group belong to one complementation group with abnormal transient responses (off-transient of lamina potential is absent) but can be separated further into a group of normal (*nofCB1*, *nofCB2*, *nofCB5*) and abnormal (*nofCB8*, *nofCB9*, *nofCB10*) time course of the