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Table 1. Y-maze orientation.

Group	Mutant	Choice Coeff.	
Wild type	WT	0.75	
Receptor	so	0.55	
	w	0.49	
	ey ²	0.70	
	sevLY3	0.74	
	ora ^{JK84}	0.50	
	rdgB ^{KS222}	0.58	
ERG	X-37*	0.65	
	X-61*	0.66	
	X-72*	0.53	
	nonA ^{H2}	0.54	
	nonA ^{H17}	0.47	
	nofCB1	0.48	
	nofCB2	0.48	
	nofCB5	0.53	
	nofCB8	0.49	
	nofCB9	0.52	
	nofCB10	0.51	
	t	0.45	
	no object fixation	nofAS100	0.81
		nofDB11	0.59
nofEB12		0.42	
nofFS71		0.48	
nofGS131		0.73	
apoS129	0.51		
Optomotor-blind	nbAH18	0.65	
	nbAH47	0.53	
	elfH37	0.75	
	ombH31	0.26	
	opm53*	0.54	
e	0.64		
Motor	Hk ¹	0.63	
Brain	sol ^{KS58}	0.41	

* laboratory name

The orientation of flies towards a 5x5° dark spot in a multiple Y-maze was tested in mutants known for partial or complete blindness or other defects in the processing of visual information. The maze resembles "Galton's Board" and consists of 14 consecutive decision points at which the running fly has to decide to go either to the left or to the right (Bülthoff, Götz & Herre 1982). A probability of choice $0.5 < p < 1$ indicates preferred orientation towards the dark spot whereas a probability $0 < p < 0.5$ is associated with orientation away from the pattern. A probability around 0.5 suggests inability to orientate visually towards the pattern. The choice coefficients in Table 1 are calculated from the end distributions of Y-maze runs with approximately 1300 flies for most mutant strains, and for none with less than 500 flies.

A few of the mutant strains show normal orientation effects as compared to the behavior of the wild type. These strains include the eyeless mutant (ey²) with rudimentary compound eyes and the mutant sevenless (sevLY3) in which the inner rhabdomere 7 is missing, the mutant with an extra lamina fiber (elf^{H37}) (Heisenberg 1979) and, surprisingly, also two of the S-mutants (nofAS100, nofGS131) which have been selected because of a defect in their attitude towards visual objects (Heisenberg & Wolf 1979). The orientation effect is almost absent in the blind mutant sine oculis (so), in the mutant white (w) lacking screening pigment, in the receptor mutants ora^{JK84} and rdgB^{KS222}, and mutants disturbed in the primary process of phototransduction (X), no on-response (non), night blind (nb^{H47}) and tan (t) (Pak 1975). The same holds for the ERG mutants of the nofC group (Bülthoff 1980), for the no-object-fixation mutants (nofFS71, apoS129, nofDB11, nofEB12 (Heisenberg 1979; Bülthoff 1980) and for the hyperkinetic (Hk¹) mutant (Kaplan & Trout 1969) and the recently described small optic lobe (sol^{KS58}) mutant (Fischbach & Heisenberg 1981). Most surprising are the scores obtained for the optomotor blind mutant ombH31 (Heisenberg, Wonneberger & Wolf 1978), a mutant with severely diminished movement

induced course control response which is structurally impaired in the lobula plate giant neurones of the visual system. The flies of this strain avoid the visual object in the Y-maze. The avoidance reaction, or antifixation is remarkably strong ($p = 0.26$) and seems to be in contradiction to the almost normal fixation response obtained in other experimental paradigms (Bülthoff, Götz & Herre 1982).

References: Bülthoff, H. 1980, Dissertation Tübingen; _____, K.G. Götz and M. Herre 1982, in prep.; Fischbach, K.F. and M. Heisenberg 1981, Proc. Natl. Acad. Sci. USA 78:1105-1109; Heisenberg, M. 1979, in: Handbook of Sensory Physiology, ed. H. Autrum, 7:6A:665-679; _____ and R. Wolf 1979, J. comp. Physiol. 130:113-130; _____, R. Wonneberger and R. Wolf 1978, J. comp. Physiol. 124:287-296; Kaplan, W.D. and W.E. Trout 1969, Genetics 61:399-409; Pak, W.L. 1975, in: Handbook of Genetics, ed. R.C. King, 3:703-733.