

at the upper right of this figure. Axon numbering in this pseudocartridge is arbitrary. The perikaryon of a satellite glial cell is positioned to the left of the monopolar's neck. We have on rare occasions observed a pseudocartridge with 8 axons, confirming our contention that a few ommatidium may have 8 photoreceptor cells, including R7. Fig. 5 (sev, bar = 1 μ m) shows a cross sectioned optic cartridge with its centrally localized L1, L2 and L3 interneurons surrounded by R1-6 axon terminals. To the right, and surrounded by an electron dense epithelial glial cell (EG) is the R8 axon without its R7 counterpart which is normally contiguous to R8. The closely paired R7 and R8 axons normally pass through the lamina without synapse on their way to terminations in the second neuropile, the medulla.

In summary, our micrographs show that, for the most part, the compound eye lacks the R7 cell. In addition, we have depicted the occasional fused condition of rhabdomeres in the peripheral retina of the compound eye. The structure of the lamina ganglionaris is unaffected by the loss of R7 except that R7's axon, which normally traverses the lamina, is lacking.

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References: Broda, H. & R. Willmund 1981, *J. Insect Physiol.* 27:789-792; Campos-Ortega, J.A., G. Jurgens & A. Hofbauer 1979, *Wm. Roux Arch.* 186:27-50; Coombe, P.E. 1984, *J. Comp. Physiol.* 155:661-672; Fischbach, K.F. 1979, *J. Comp. Physiol.* 130:161-171; Fischbach, K.F. & H. Reichert 1978, *Biol. Behav.* 3:305-317; Harris, W.A., W.S. Stark & J.A. Walker 1976, *J. Physiol. (Lond.)* 256:415-439; Heisenberg, M. & E. Buchner 1977, *J. Comp. Physiol.* 177:127-162; Hu, K.G., H. Reichert & W.S. Stark 1978, *J. Comp. Physiol.* 1978, 126:15-24; Hu, K.G. & W.S. Stark 1977, *J. Comp. Physiol.* 121:289-305; Hu, K.G. & W.S. Stark 1980, *J. Comp. Physiol.* 135:85-95; Jacob, K.G., R. Willmund, E. Folkers, F.F. Fischbach & H.Ch. Spatz 1977, *J. Comp. Physiol.* 116:209-225; Labhart, T. 1977, *Naturwissen.* 64:S.99; Miller, G.V., K.N. Hansen & W.S. Stark 1981, *J. Insect Physiol.* 27:813-819; Stark, W.S. 1977, *J. Comp. Physiol.* 115:47-59; Stark, W.S., K.L. Frayer & M.A. Johnson 1979, *Biophys. Struct. Mech.* 5:197-209; Stark, W.S., A.M. Ivanyshyn & K.G. Hu 1976, *Naturwissen.* 63:513-518; Stark, W.S. & M.A. Johnson 1980, *J. Comp. Physiol.* 140:275-286; Willmund, R.J. *Comp. Physiol.* 129:35-41.

Stocker, R.F. and M. Schorderet. University of Fribourg, Switzerland. Sensory projections of homoeotically transformed eyes in *D.melanogaster*.

Homoeotic mutants transform particular body parts into others and thereby create displaced sensory neurons, whose axons reach the central nervous system (CNS) at abnormal sites. This allows one to study how the position of sensory neurons affects the specificity of their central connections (Palka & Ghysen 1982; Stocker 1982). The pattern of sensory projections from all types of homoeotic structures tested so far are remarkably constant, i.e., terminals occupy their normal projection area or centers of serially homologous structures. Similar observations have been made in the pattern of afferents from surgically generated ectopic appendages (Stocker & Schmid, in prep.). These data suggest that sensory axons are able to recognize specific structures in the CNS, even if they arrive through an ectopic nerve.

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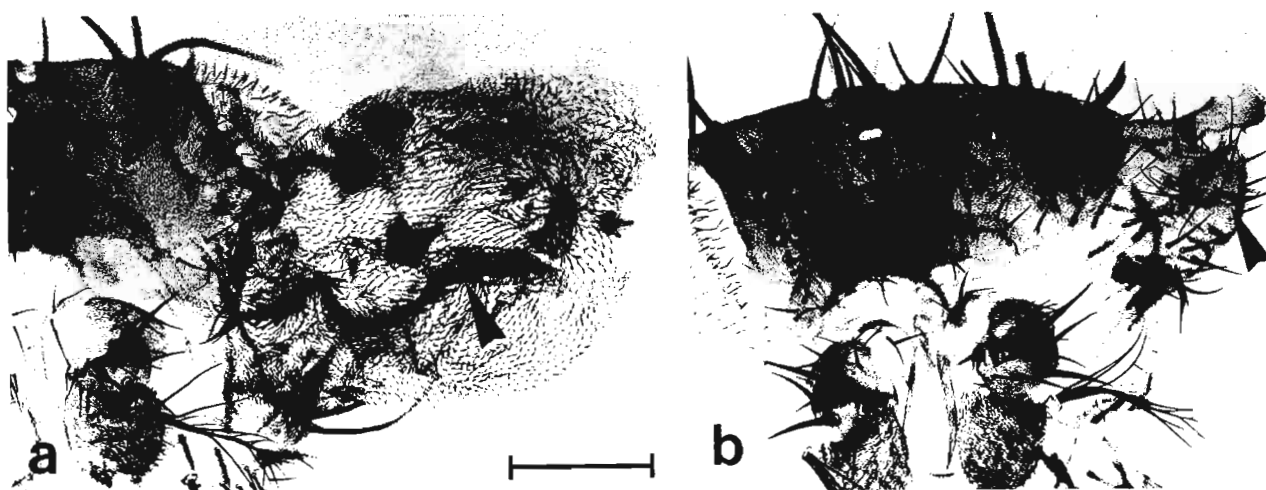


Figure 1. Homoeotically transformed eyes in *ey^{opt}* (a) and *tuh* (b). The wing tissue present in *ey^{opt}* is characterized by sensilla of the triple row (arrowhead). In *tuh* transformation leads to sensory bristles typical of abdominal tergites (arrowhead). Bar 200 μ m.

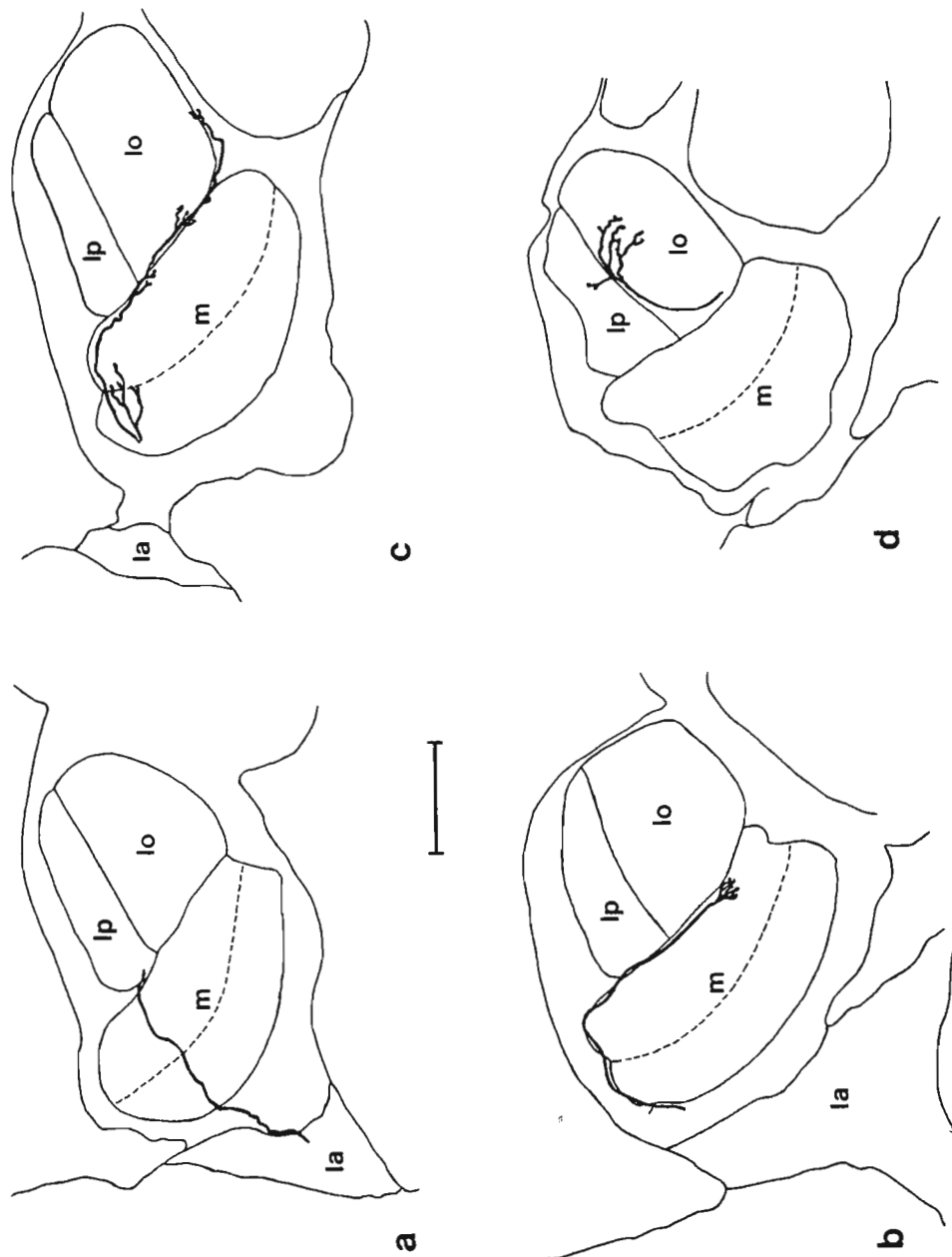


Figure 2. Camera lucida drawings of sensory projections from transformed eyes in the visual ganglia. **a:** A single axon in tuh extends through the medulla (m) into the lobula/lobula plate region (lo/lp). **b, c:** Other tuh fibers by-pass the medulla neuropil at its posterior border and project along the line which divides the medulla and the lobula. **d:** A single axon in ey^{opt} terminates in both lobula and lobula plate. la: lamina, posterior is on top. Bar 50 μ m.

Most of the transformed appendages can be homologized with the original appendage on morphological criteria, such as wings and halteres, or legs and antennae. Moreover, the sensory projection centers involved in these systems reflect the metameric organization (though in the head ganglia individual neuromeres are less obvious than in the thoracic or abdominal CNS). One might therefore argue that the observed terminal patterns are due to structural homologies between the peripheral tissues on the one hand and their projection centers on the other. Here we are studying a system in which particular epidermal structures are replaced by completely different structures, and we allow the sensory axons to grow into a CNS region of distinct architecture. This is the case in the mutants *eyeless-ophthalmoptera* (ey^{opt}) and *tumorous-head* (tuh), which transform the eye partially into wing or abdominal tergite structures, respectively (Fig. 1; Postlethwait et al. 1972; Postlethwait 1974). Will the sensory fibers from these transformed tissues, which are now confronted with the highly ordered lattice of the visual ganglia, still project in a specific manner?

Females of the genotypes *ey^{opt}* (4-2.0) and *tuh-1;tuh-3* (1-64.5;3-58.5) were used. For light microscopic examination of the transformed structures, heads were treated with 5% KOH and mounted in Faure's solution. The projection patterns were observed by filling the sensory axons with horseradish peroxidase (HRP, Sigma type VI, 10% w/v, or type II, 20%) after amputation of the transformed tissues. The HRP marker was visualized according to the standard DAB technique (Cogshall 1978). Heads were embedded in soft Epon and serially sectioned at 20 μ m.

The phenotype of the transformations varies in both mutants. We followed projections only from appendages with well discernible sensory structures, i.e., double or triple row bristles in *ey^{opt}* and tergite bristles in *tuh* (Fig. 1). Surprisingly, only 11% of the HRP-fillings in *ey^{opt}* and 7% in *tuh* yielded labelled sensory axons in the brain. This low success rate is unlikely to be due to incomplete diffusion of the tracer molecule, since filling from another transformed region in *tuh*, the leg-like antenna, resulted in 68% of labelled preparations. We rather suspect that in the majority of cases afferent connections are not formed at all, probably because structures essential for guiding sensory axons to the optic ganglia are missing. The presence of such cues could depend on the precise localization of the transformed tissue in the eye, although there is apparently no simple correlation between the two phenomena. A complete lack of afferent projections is reported of "extra" eyes in the mutant two-faced (Kankel 1984).

Retinula cells R1-6 of normal eye ommatidia send their axons into the first order visual neuropil, the lamina; fibers from R7 and R8 pass through the lamina and terminate at a particular depth in the second neuropil, the medulla (Fischbach 1983). Both types of axons are characterized by peculiar terminal swellings. The fibers observed in fillings from transformed eyes (8 fillings in *ey^{opt}* and 5 in *tuh*) are of different shape. In both mutants they appear to pass through the lamina and then to choose one of two pathways: some of them project into the medulla and from there further into the third visual neuropil, the lobula/lobula plate complex (Fig. 2a). Others by-pass the medulla neuropil at its ventral border to reach the lobula/lobula plate complex from posterior (Fig. 2b,c). We never saw axon branching in the lamina, and only rarely in the medulla. In contrast, in the lobula and lobula plate the fibers arborize extensively, but the patterns produced are quite variable. In *ey^{opt}* mutants fibers extend mostly along the bundle which divides lobula and lobula plate and send off terminals into both of these neuropils (Fig. 2d). Axons passing along the medulla/lobula border have been found in *tuh*; they branch at the anterior end of this region (Fig. 2b,c). Other fibers in *tuh* project into the lobula and lobula plate like those in *ey^{opt}*. In addition to these patterns, two completely different types of projections were observed. In *ey^{opt}* a single axon was seen to enter the brain via the antennal nerve and to terminate without branching in the mechanosensory antennal center. In the periphery this axon followed the "nervus tegumentalis" (Hertweck 1931) which innervates the dorsal wall of the head close to the eye margin. In another case in *tuh*, axons originating in the eye region reached the suboesophageal ganglion via a side branch of the labial nerve and arborized immediately after arriving in the CNS, below the center of normal proboscis fibers present in the same nerve. Thus, the terminals appear to avoid the center of a heterologous structure.

In fills from transformed eye tissue, labelled axons similar in shape and distribution to those of retinula cells R7-8 were occasionally present. These fibers might originate in normal ommatidia from the vicinity of the homoeotic structures and have been accidentally filled because of damage during the amputation of the transformed tissue. Alternatively, they might stem from the homoeotic structures, but have retained their original identity (cf. Palka & Ghysen 1982).

Our data show that sensory axons from eyes transformed into wing or tergite structures arrive at different sites in the CNS of the head. Moreover, their terminal pattern is most variable. We conclude that sensory axons may choose any available peripheral nerve as guidance cue to reach the CNS (cf. Ghysen & Deak 1978). The fact that terminals of transformed eye structures are not restricted to the normal eye projection regions lamina and medulla, and the absence of a reproducible pattern suggest that the fibers are unable to recognize specific structures in the CNS. These data seem to contradict the pattern specificity observed in other homoeotic systems or in surgical transplants. The specificity of the projections has been explained by the existence of a surface marker common to all projection centers of homologous appendages (Stocker 1982; Stocker & Schmid, in prep.). However, it is conceivable that the visual ganglia possess the same general marker as wing or tergite projection centers, but that retinula fibers relate to additional cues specific for lamina and medulla which homoeotically transformed axons are unable to read. In a comparable situation, sensory terminals from leg-like antennae in aristapedia mutants distribute randomly in the antennal lobe; this is quite in contrast to the well-patterned wildtype antennal terminals (Stocker & Lawrence 1981). We conclude that sensory axons depend on different mechanisms for tracing particular projection centers and for establishing their terminal arborization pattern.

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References: Cogshall, J.C. 1978, *J. Comp. Neurol.* 177:707; Fischbach, K.F. 1983, *Habil. Thesis*, Wurzburg, F.R.G.; Ghysen, A. & I.I. Deak 1978, *Roux' Arch.* 184:273; Hertweck, H. 1931, *Z. wiss. Zool.* 139:559; Kankel, D.R. 1984, *Proc. XV Int. Congr. Genet.* 3:205; Palka, J. & A. Ghysen 1982, *TINS* 5:382; Postlethwait, J.H. 1974, *Devl. Biol.* 36:212; Postlethwait, J.H. et al. 1972, *Devl. Biol.* 29:337; Stocker, R.F. 1982, *Devl. Biol.* 94:31; Stocker, R.F. & P.A. Lawrence 1981, *Devl. Biol.* 82:224.