of the Figure presented here, the inversion extends from the tip of the arm to the end of the 75A section in the reference map constructed by Hinton & Downs (1975).

The complete pairing of both ends to the last visible bands in the heterozygote and the differences in banding patterns of tips in standard and inversion homozygotes suggest that the inversion is completely terminal.

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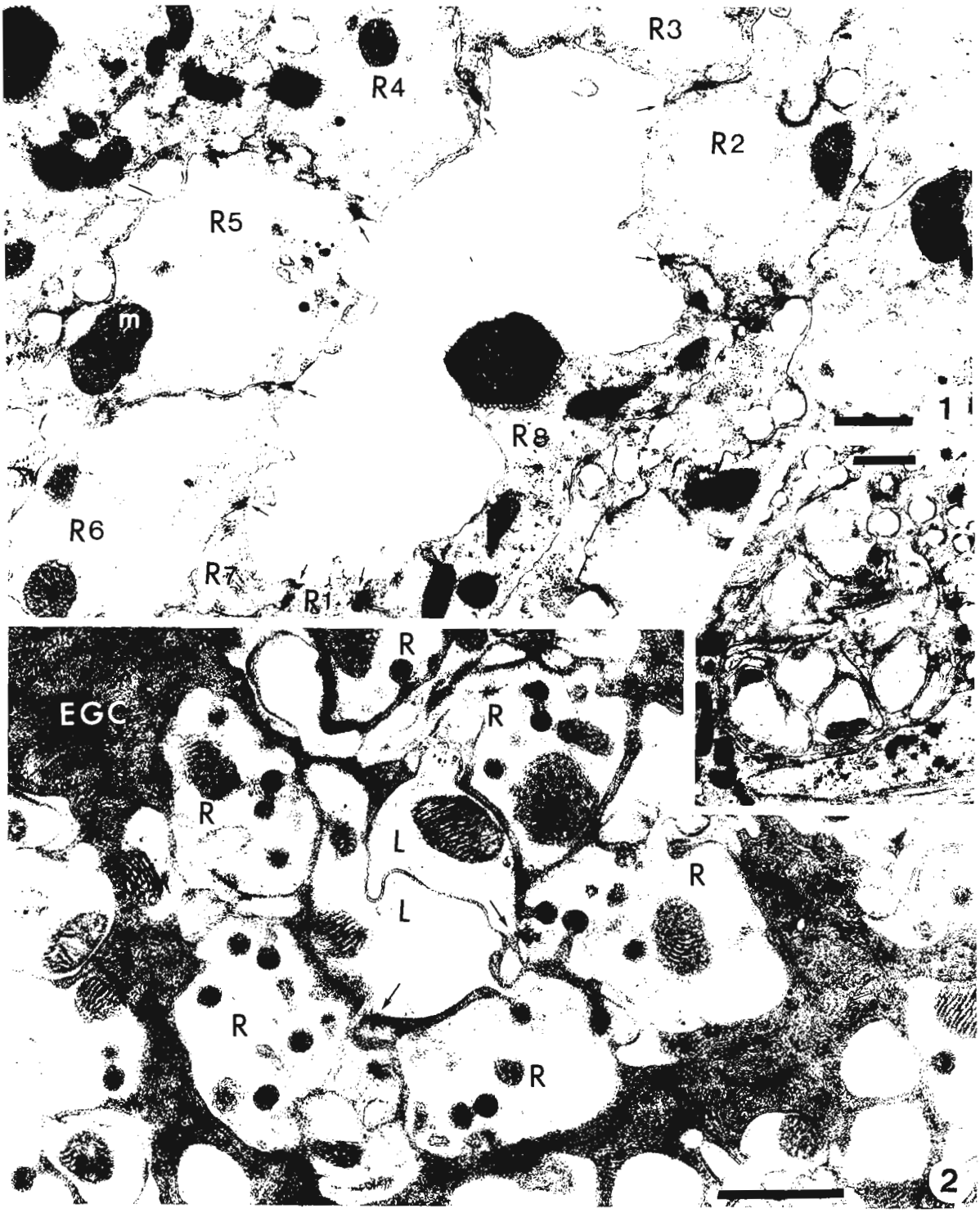
Stark, W.S. and S.D. Carlson.* University of Missouri, Columbia, USNA. *University of Wisconsin, Madison, USNA. Retinal degeneration in rdgB^{KS222} is blocked by ora^{JK84} which lacks photoreceptor organelles.

Harris et al. (1976) characterized a number of mutants with defects in the visual receptors which have since been used for studies of photoreceptor development, function and input. In the rdgB^{KS222} mutant, isolated by Hotta & Benzer (1970), the R1-6 receptors of the compound eye degenerate after exposure to light while the other types, R7 and R8, are spared (Harris et al. 1976; Harris & Stark 1977). This mutant has been utilized in over a score of studies (see Stark & Carlson 1982; Stark et al. 1983; Chen & Stark 1983 for references). Another mutant, ora^{JK84}, isolated by Koenig & Merriam (1977), eliminates the most of the photopigment containing organelle (rhabdomere) in each R1-6 cell (Harris et al. 1976). The latter strain has proven equally useful in vision research (see Stark & Carlson 1983 for references).

In a study of the mechanism of light induced degeneration in rdgB flies, rdgB was combined with ora (Harris & Stark 1977). The experimental strategy, after creating this double mutant, was to deprive rdgB flies of photic stimulation by ridding them of their photoreceptive organelle (and thus their capacity to effectively absorb photons). Earlier, Harris & Stark (1977) presented electron micrographs suggesting that ora protected against degeneration in rdgB but not completely. However, those micrographs were technically less than what is possible nearly a decade later. Furthermore, the literature now contains a wealth of electron micrographs which suggest, among other things, that fixation of the fly compound eye is not always achieved successfully. The purpose of this study is to reexamine the protection of rdgB offered by ora now that better EM technique is available. Our work was prompted in part by our extensive and recent ultrastructural examinations of rdgB (Stark & Carlson 1982) and of ora (Stark & Carlson 1983). In addition to the receptor somata, we examined the receptor axon terminals in the optic cartridges of the lamina ganglionaris, the first optic neuropile. This synaptic region is an important area to study because the R1-6 terminals in rdgB are especially sensitive to the deliterious effects of light (Stark & Carlson 1982; Carlson et al. 1984). An abstract of our findings is available (Carlson & Stark 1985).

We isolated rdgB;ora flies upon eclosion and then fixed them either promptly or after 1, 2 or 3 weeks of aging at 23°C on a 12 hr on, 12 hr off cycle of fluorescent laboratory illumination. Our micrograph are from the High Voltage Electron Microscope Laboratory, an NIH Biotechnology Resource, at the University of Wisconsin, Madison. Details of our techniques are given elsewhere (e.g., Stark & Carlson 1983).

Our results are presented in the accompanying plate. All scale bars = 1 µm. Figure 1 is a cross section through one ommatidium in the proximal part of the peripheral retina of an rdgB;ora fly aged 2 weeks. At this level R8 is the only cell possessing a rhabdomere, while the R1-6 receptors (except at the most distal level) lack this organelle. R7's short rhabdomere is distal and in tandem to that of R8. Thus one sees only the R7 axon in this plane. All R cells are joined to each other by belt desmosomes (arrows). The R1-6 cells are ultrastructually normal, i.e., they look precisely like R1-6 cells from flies with only the ora mutation, without signs of degeneration. That diagnosis is backed by checking the mitochondria (m) in this field; they are normal appearing,numerous and particularly conspicuous. Attenuated processes of pigmented glial cells (secondary pigment cells) spatially isolate the ommatidia. "Holes" in these cells represent sites where pigment granules were apparently extracted during the process of fixation. The insert shows the fasicle of 8 axons from one ommatidium just beneath the basement membrane which separates the retina from the lamina. This bundle (pseudocartridge) looks completely normal, similar or identical to that in wild type and ora flies. Figure 2 shows one cross sectioned optic cartridge in the lamina. Six R1-6 terminals make synapses mainly onto the processes of several laminar monopolar neurons. The most conspicuous of these are the pair of electron lucent L1 and L2 cells located in the core region surrounded by the six photoreceptor (R) terminals. The electron dense surround is made up of processes from 3 so called "epithelial" glial cells (EGC). These glial cells insert capitate projections (seen as small, dark spheres or mushroom-like structures) into the R1-6 terminals. Occasionally, T-bar synapses are seen and noted (arrows). The axonal projection to and termination of R1-6 axons in this area look completely normal and identical to that found



Stark & Carlson, "Retinal degeneration ...."

Figure 1. Cross-section through one ommatidium in the proximal part of the peripheral retina of an rdgB;ora fly aged 2 weeks.

Figure 2. One cross-sectioned optic cartridge in the lamina.

in wild type, a situation which is also the case for non-rdgB ora flies. Thus the R1-6 terminals, which are an especially sensitive indicator of degeneration in rdgB flies, survive well in rdgB; ora flies. We observed no differences in this regard with aging under the cyclic illumination, i.e., among flies fixed when newly emerged or after being aged 1, 2 or 3 weeks. To ascertain whether the stock we studied retained rdgB in combination with ora, we did a genetic cross to separate rdgB from ora. Optical examinations of the progeny verified that degeneration still occurred meaning rdgB, still present, had been uncovered from ora's protection. In summary, we conclude that ora affords fairly complete protection against degeneration in rdgB flies, even more protection than suggested by Harris & Stark (1977). In functional terms, genetic elimination of the photopigment and its organelle prevents light-induced degeneration by depriving rdgB flies of photic stimulation.

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Stark, W.S. and S.D. Carlson.* University of Missouri, Columbia, USNA. *University of Wisconsin, Madison, USNA. Ultrastructure of the compound eye and first order neuropile in the sevenless (sev) mutant of **D.melanogaster.**

The sevenless (sev) mutant has been extremely useful in the analysis of function and development of photoreceptors in the Drosophila visual system (Harris et al. 1976; Stark et al. 1976, 1979; Heisenberg & Buchner 1977; Hu & Stark 1977, 1980; Jacob et al. 1977; Stark 1977; Labhart 1977; Fischbach & Reichart 1978; Hu et al. 1978; Campos-Ortega et al. 1979; Fischbach 1979; Willmund 1979; Stark & Johnson 1980; Miller et al. 1981; Broda & Willmund 1981; Coombe 1984). In the compound eye of a nonmutant fly, each ommatidium contains 8 receptor cells of 3 types: R1-6 has photoreceptive rhabdomeres peripherally oriented around the central axis of the ommatidium while R7 and R8 have distal and proximal central rhabdomeres, respectively. The rhabdomere of each photoreceptor cell is normally never fused to that of its neighboring cell in the open rhabdomere configuration of the fly retina. In the sev mutant the R7 cell does not form (Campos-Ortega et al. 1979) and thus it lacks R7 function (e.g., Harris et al. 1976). Recently, we initiated a program of ultrastructural research on Drosophila visual mutants, sponsored in part by the High Voltage Electron Microscope (HVEM) Laboratory, an NIH Biotechnology Resource, at The University of Wisconsin, Madison. We reexamined sev and a white-eyed strain (w sev) because there was so little ultrastructural data extant on that mutant. Our preliminary observations are now presented which include the premier micrographs from its lamina ganglionaris (first optic neuropile and thus the first synaptic relay station of the retinal projection).

In general, distal sections through the peripheral retina showed 6 rhabdomeres (R1-6) while proximal sections showed 7 (R1-6 and R8) as expected. A rare exception to this generalization is shown in Fig. 1 (bar = 1 μm). Here a distal ommatidium is cross sectioned to reveal the trapezoidal arrangement of photoreception cells, and we suggest that the central cell is R7 as labeled. The neighboring ommatidium is at a more proximal level, and from its shape and orientation it is proposed that the central cell is R8. It is possible that the designated R7 cell is really an R8 cell in the R7 position (see Campos-Ortega et al. 1979) but our observations suggest that a few isolated ommatidia near the equator may actually have R7. The electron dense small spheres in close apposition to the rhabdomeric microvilli are the ommochrome pigments of the retinula cells which migrate during light and dark adaptation in the red eyed fly. Very deep in the retina, there are quite few ommatidia which apparently lack some of the 7 expected rhabdomeres. On closer examination (Fig. 2, sev, bar = 1 μm), it is shown that rhabdomeres of adjacent R1-6 cells occasionally fuse (arrow). It should also be noted that rhabdomeric fusions are found in which the two sets of conjoined microvilli are at very different angles. At this proximal level, intraretinular pigment granules, which are typically concentrated distally, are not seen, as expected, even though this section (Fig. 2) is from a red eyed fly.

Beneath the basement membrane, the axons of each ommatidium are bundled into pseudocartridges (Fig. 3, w sev, bar = 1 μm). As expected, most of these fascicles have 7 axons. Fig. 4 (w sev, bar = 1 μm) shows a longitudinal section through the distal level of the first optic neuropile. A type 1 monopolar interneuron (L1 or L2) is revealed for a considerable length: through the perikaryon (MP), nucleus (Nu), neck (N) and up to a point where dendrites (D) project laterally to retinular cell terminals (R). The L cell soma shows numerous mitochondria as well as a Golgi body (G). The R cell terminal is identified by capitate projections. Another pseudocartridge, obliquely sectioned, lies just outside the L cell's perikaryon