MacDonald et al., http://www.jcb.org/cgi/content/full/jcb.201503115/DC1

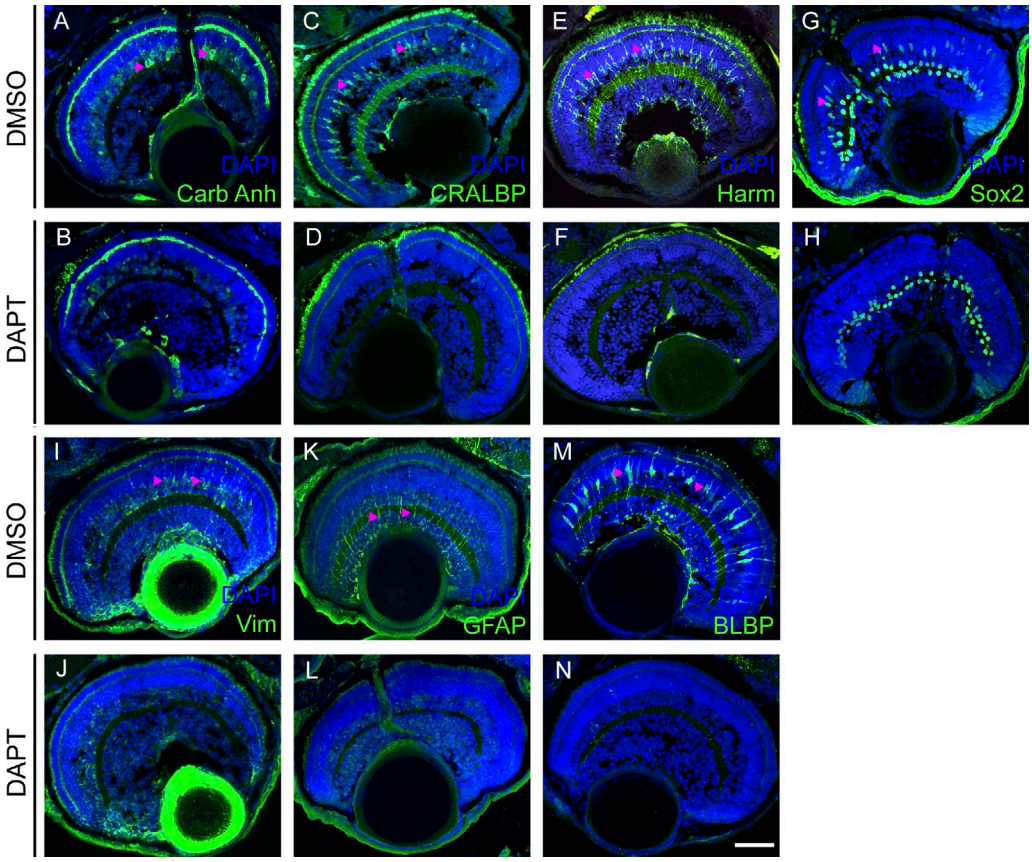


Figure S1. **Blocking Notch with DAPT results in retinas lacking expression of several MG immunohistochemical markers at 96 hpf.** Control retinas are treated with DMSO. DAPT-treated retinas show no expression of MG-specific markers. Shown are carbonic anhydrase (A and B), Cralbp (C and D), Harmonin (E and F), Sox2 (G and H), Vimentin (I and J), GFAP (K and L), and BLBP (M and N). Arrowheads point to MG. Bar, 50 µm.

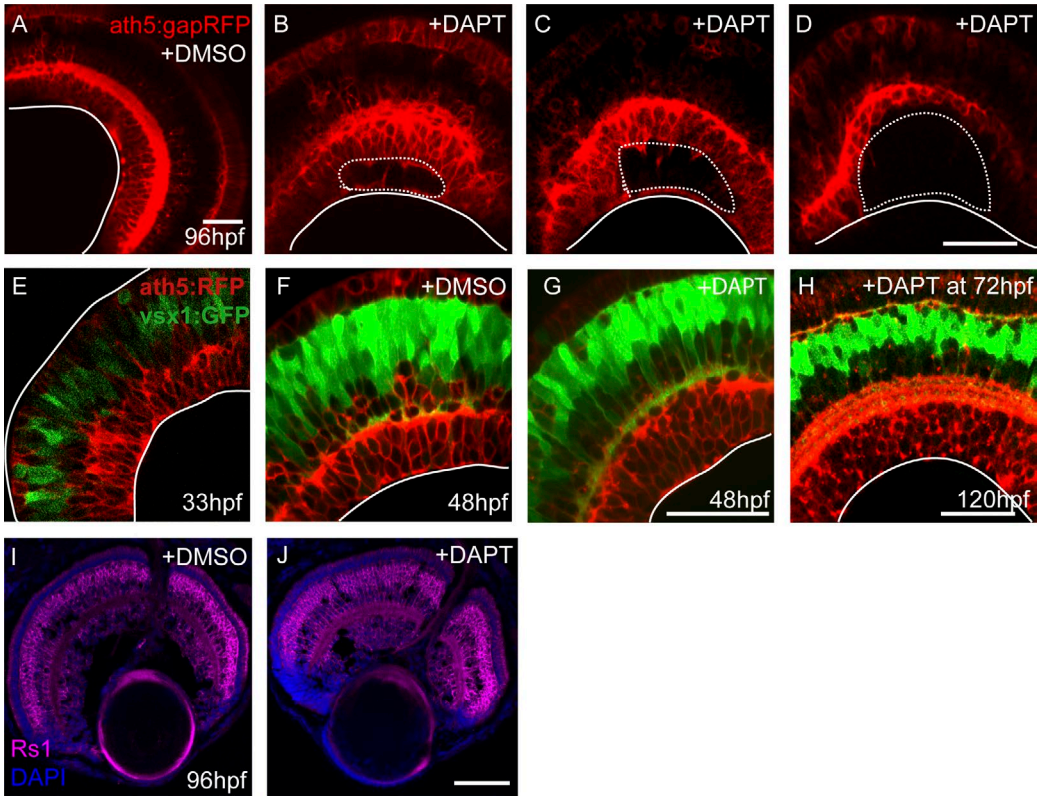


Figure S2. **The extent of ripping in the GCL varies in the absence of MG.** Single z-sections of unfixed transgenic retinas are shown. (A–D) *ath5:gapRFP* strongly labels the GCL. The retinoschisis phenotype varies in severity from retina to retina. The basal surface of the retina is marked with a solid line. The rip is marked with a broken line. (E–H) In the *vsx1:GFP;ath5:gapRFP* transgenic embryos, most retinal neurons are labeled. There is no evidence of ripping within the nascent retina at 33 hpf (E) and 48 hpf (F), when radial progenitors remain and MG would normally not be present. (G) Just after DAPT is added to the media, there are no rips in the retina. (H) Treatment of the embryo with DAPT after 72 hpf, allowing MG to be specified in the central retina, does not result in rips 48 h later. (I) her4:GFP is expressed specifically in the bipolar cells (arrow) and MG (arrowhead) in the retina. (J) Knockdown of her4 expression reduces the number of MG and results in the retinoschisis phenotype in the GCL. (K and L) Cryosection of the retina showing that Retinoschisin1 expression is not altered with DAPT treatment at 96 hpf. Bars: (A) 30 µm; (B–D) 25 µm; (E–H) 30 µm; (J) 50 µm.

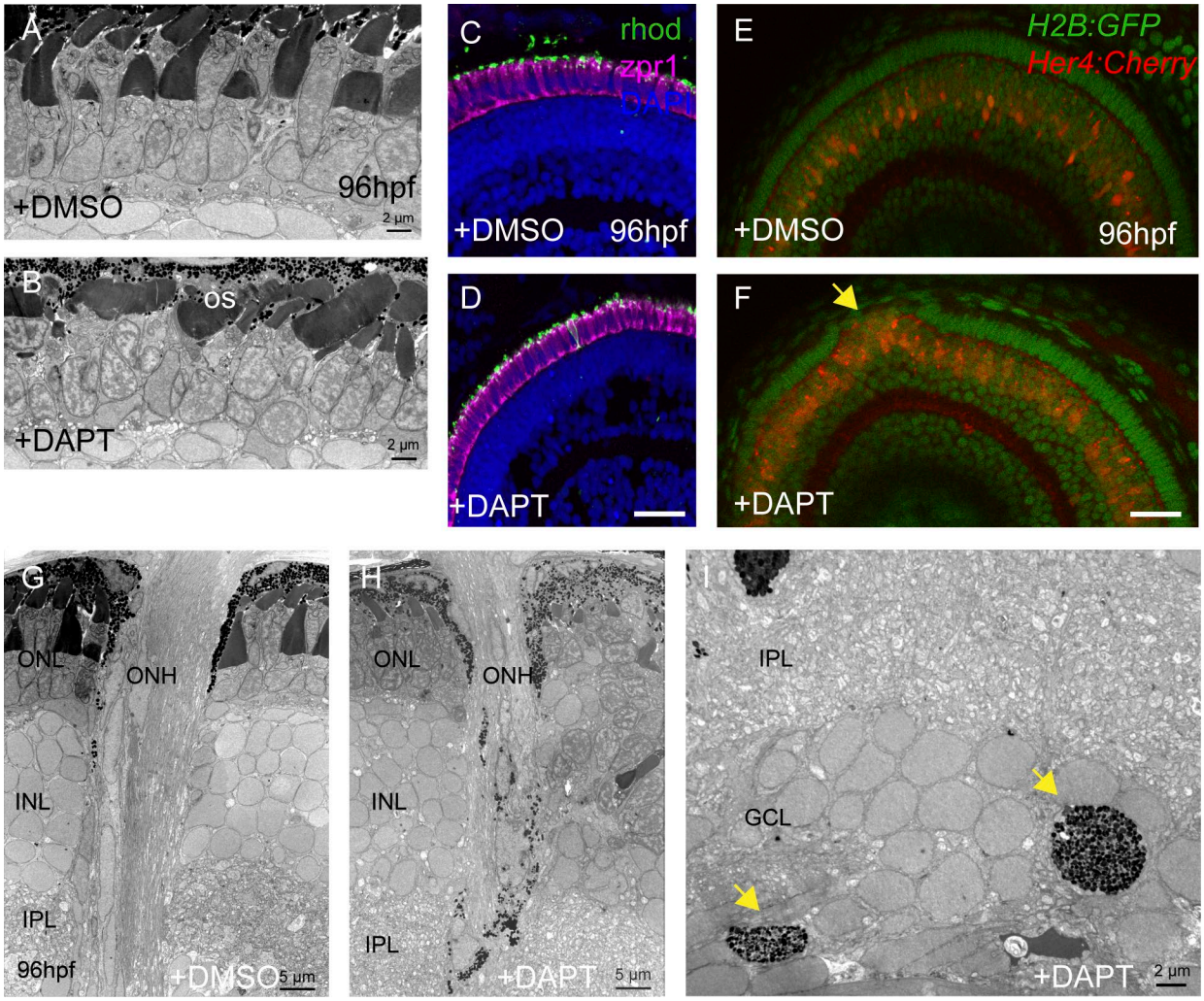
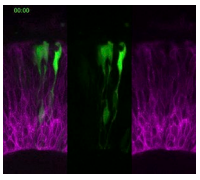
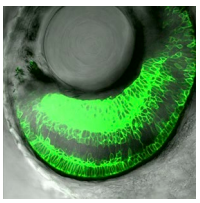


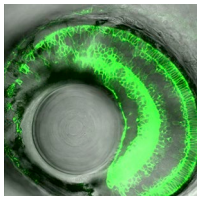
Figure S3. **Defects in the retina lacking MG (related to Fig. 2).** Electron micrographs of the retina lacking MG. (A and B) Photoreceptor cell bodies and their outer segments appear disorganized in DAPT-treated retinas. (C and D) Despite a disorganized outer nuclear layer (ONL), both rods and cones are present in the DAPT-treated retinas, as labeled by rhodopsin and zpr1, respectively. (E and F) However, the photoreceptor layer is occasionally disrupted (n = 1/20 fish; arrow), as shown by the *H2B:GFP* transgene, where all nuclei are labeled (green). (G–I) Electron micrographs show pigmentation extending along the optic nerve head into the retina with DAPT treatment. Pigment granules can also be found in the GCL upon DAPT treatment (I, arrows). ONH, optic nerve head; IPL, inner plexiform layer. Bars in C–F, 30 µm.



Video 1. **In vivo time lapse of Notch activity within clones in the embryonic retina.** Time-lapse confocal imaging of mosaic retinas with *TP1:Venus* (green)-labeled clones within host retinas where many retinal neurons are labeled by *ath5:gapRFP* (magenta). Images are of maximum-intensity projections of five confocal slices from time-lapse confocal microscopy using a laser-scanning confocal microscope (FV1000; Olympus). Time is shown in hours:minutes. Imaging begins at ~38 hpf and frames were taken every 15 min for ~24 h. n = 6 clones observed.



Video 2. **There is no retinoschisis in the retina under control conditions.** Single z stack captured in vivo from a DMSO control *ath5:GAP-RFP* (green) zebrafish retina at 96 hpf using a laser-scanning confocal microscope (FV1000; Olympus). The step size is 2 µm and the video is displayed at 10 frames per second.



Video 3. **There is a significant retinoschisis phenotype in the retina in the absence of MG.** Single z stack captured in vivo from a DAPT control *ath5:GAP-RFP* (green) zebrafish retina at 96 hpf using a laser-scanning confocal microscope (FV1000; Olympus). The step size is 2 µm and the video is displayed at 10 frames per second.