

Wall et al., <http://www.jcb.org/cgi/content/full/jcb.200805155/DC1>

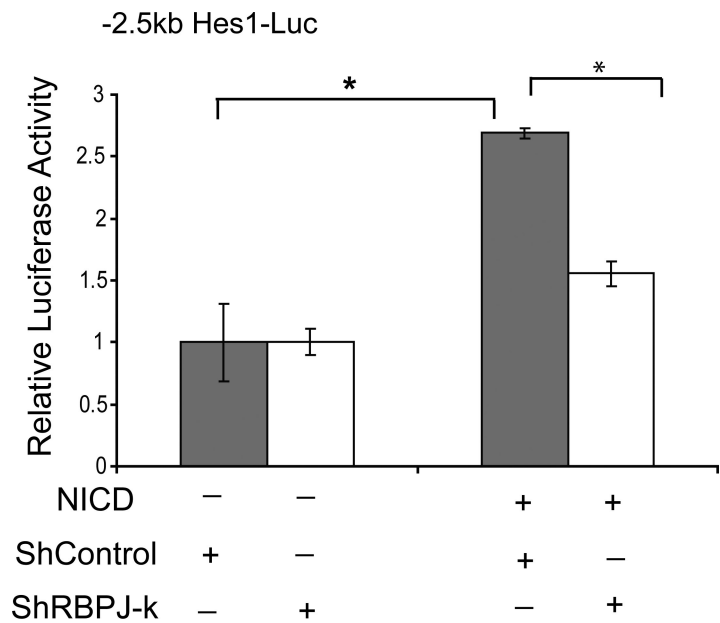


Figure S1. **RNAi for RBPJ- $\kappa$  inhibits Notch-driven activation of a Hes1 luciferase reporter.** Retinal explants were coelectroporated with NICD, -2.5-kb Hes1-Luc, and either an shcontrol or shRBPJ- $\kappa$  construct, and luciferase activity was measured after 48 h in culture. Error bars indicate standard deviation. \*,  $P < 0.05$ .

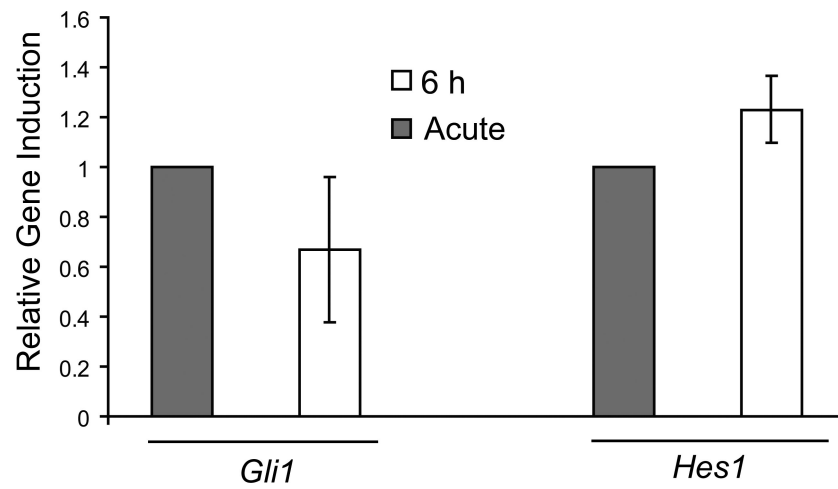


Figure S2. **RT-qPCR was performed on RNA isolated from acutely dissected retinas ( $n = 3$ ) or retinal explants cultured for 6 h in the absence of a Smo agonist ( $n = 4$ ).** There is no significant decay in the levels *Hes1* or *Gli1* transcript in retinal explants 6 h after culture compared with acutely dissected retinas. Error bars represent SEM.

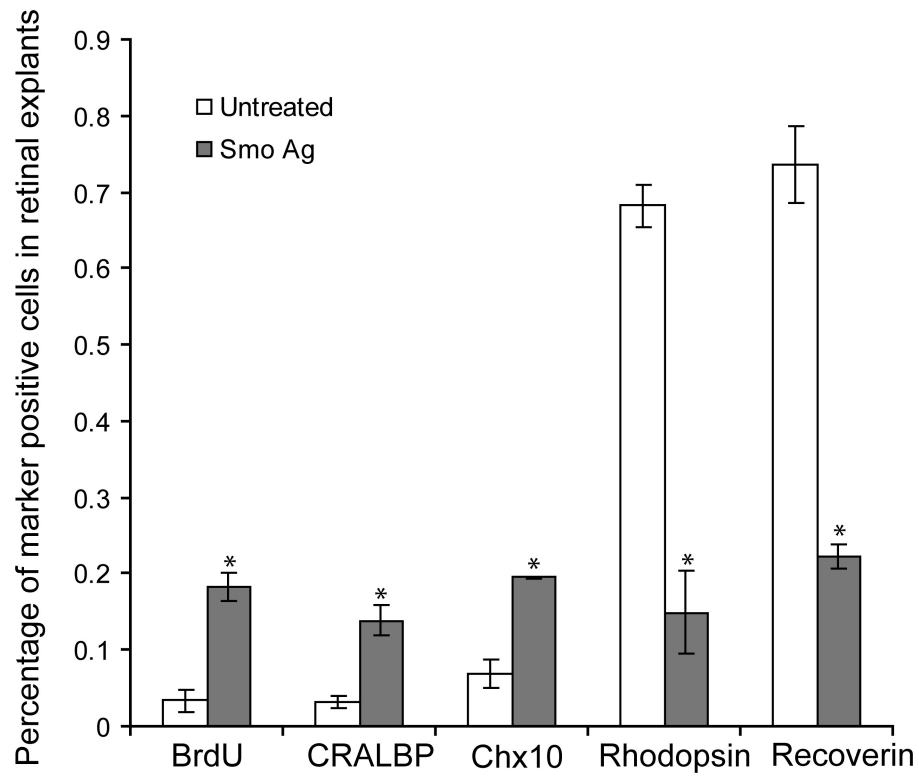


Figure S3. **Activation of the Shh pathway promotes proliferation (BrdU), Muller (CRALBP), and bipolar (Chx10) cell development while suppressing the rod photoreceptor fate (rhodopsin and recoverin).** Retinal explants from P0 mice were electroporated with GFP and cultured with or without a Smo agonist for 3 d to access proliferation, and 7 d to access cell type composition. The explants were dissociated and scored for proliferation and cell type markers among the GF-positive population using IHC. Error bars represent standard deviation. \*,  $P < 0.001$ .