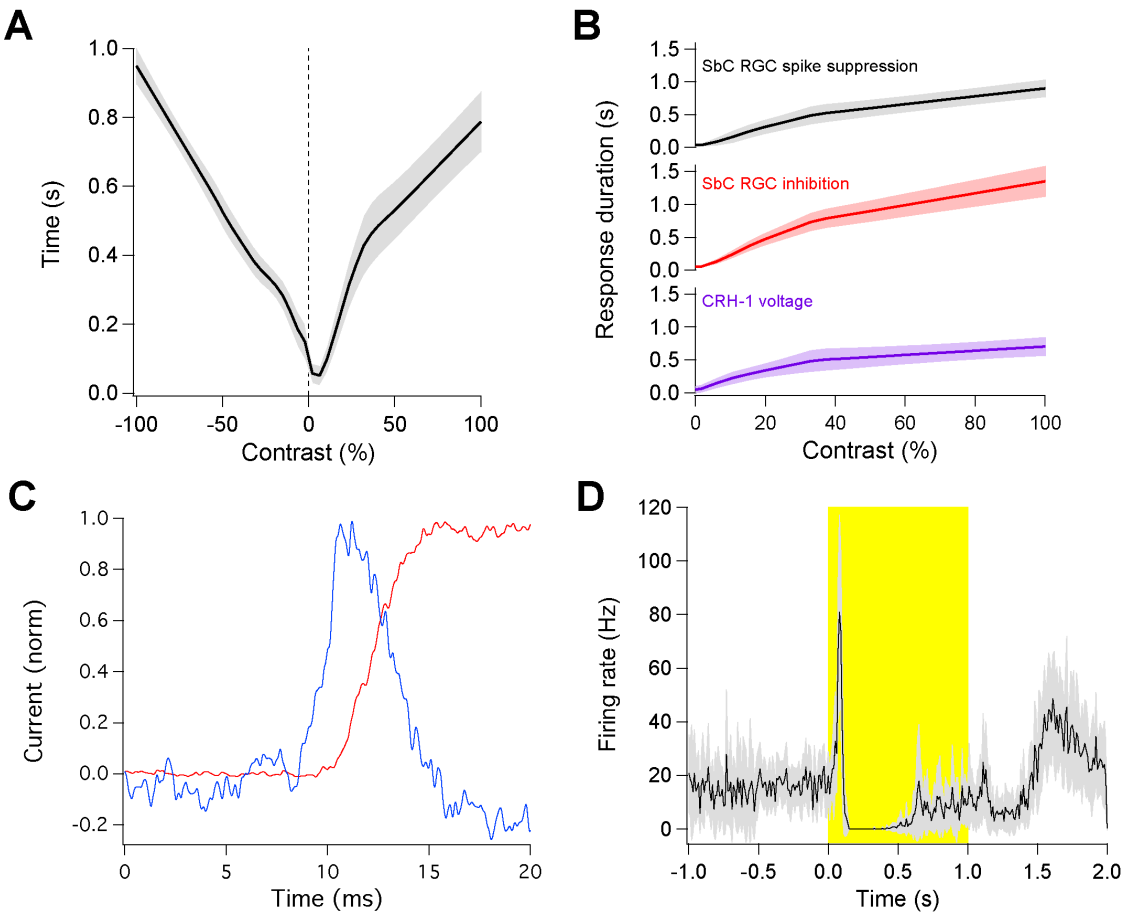

Figure S1. The Suppressed-by-Contrast computation, related to Figure 1. (**A**) The duration for which spike rate is reduced below baseline as a function of contrast for the same population of cells as in Figure 1E. Shaded region is s.e.m., *n* = 14 cells. (**B**) *Top*, spike suppression re-plotted from **A** for only positive contrasts. *Middle*, duration of the inhibitory current in SbC RGCs across contrast, *n* = 4 cells. *Bottom*, duration of the voltage response of CRH-1 amacrine cells to the same contrast stimuli, *n* = 6 cells. Averages shown with shaded regions as s.e.m. across cells. (**C**) Magnification of the initial rise in excitation (blue, inverted) and inhibition (red) to a positive 100% contrast step. Traces normalized to directly compare time course. Data from Figure 1A, *top*. (**D**) Population spike rate for a positive 100% contrast step from the cells in **C.** Shaded region is s.e.m., *n* = 14 cells.

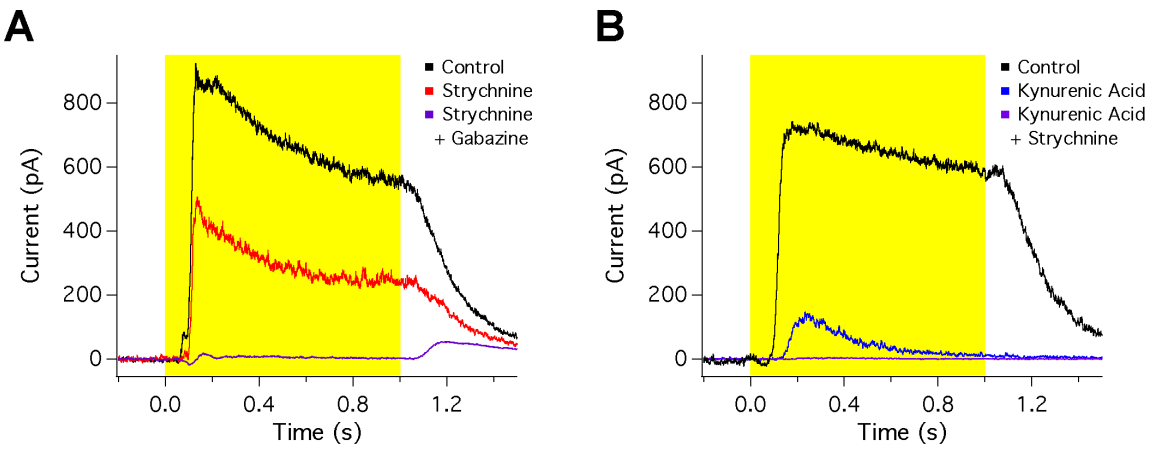

Figure S2. Pharmacology of inhibitory current in the SbC RGC, related to Figure 1. (**A**) Inhibitory currents to a step of light from darkness to 200 R*/rod/s measured in control conditions (black), in the presence of strychnine (1 µM, red), or strychnine and gabazine (10 µM, purple). (**B**) Inhibitory currents in a different SbC RGC responding to the same visual stimulus in control conditions (black), in the presence of kynurenic acid (1 mM, blue), or kynurenic acid and strychnine (1 µM, purple). The ionotropic glutamate receptor (iGluR) antagonist kynurenic acid (Stone and Burton, 1988) was used to isolate a circuit in which AII amacrine cells are driven through gap junctions with ON cone bipolar cells (Ke et al., 2014; Manookin et al., 2008; Murphy and Rieke, 2008). In kynurenic acid, we still observed an inhibitory current in the SbC RGC (173 ± 32 pA vs. 805 ± 78 pA in control, n = 5). Consistent with its origin in the AII amacrine cell, the glycine receptor antagonist strychnine (1 µM) completely eliminated the remaining current (8.0 ± 8.2 pA; not significantly above noise level, p > 0.8). It is notable that the outer stratum of the SbC RGC's dendritic field is exclusively for inhibitory input (at least partially from AII amacrine cells), as we measured no OFF excitation (Figure 1D). Traces are averages of 10-25 trials.

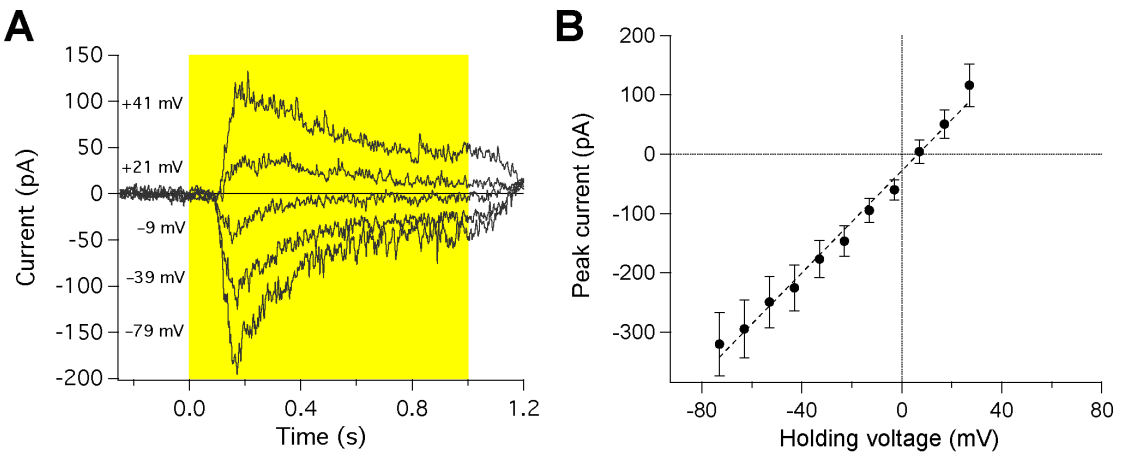

Figure S3. Current-voltage relationship of the CRH-1 amacrine cell, related to Figure 2. (**A**) Currents measured in a CRH-1 amacrine cell held at different membrane potentials as indicated. Stimulus is a step of light from darkness to 200 R*/rod/s. Traces are averages of 4 trials. (**B**) Average current-voltage relationship across CRH-1 amacrine cells. Dashed line is a linear fit. Error bars are s.e.m., *n* = 7 cells.

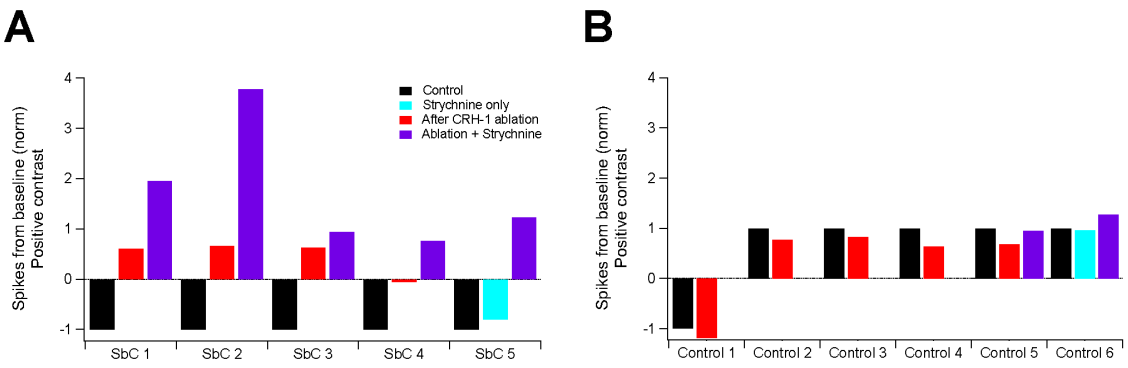

Figure S4. SbC RGC and control RGC recordings before and after ablation, related to Figure 4. Cell-attached recordings of the total number of spikes from baseline to positive contrast from SbC RGCs (**A**) and control RGCs (**B**) before ablation (black), after ablation (red), or after ablation in the presence of strychnine (purple). Experiments where strychnine was added prior to ablation are colored in cyan. Each cell recorded was normalized to pre-ablation conditions.