Report

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Adaptive Processes in Thalamus and Cortex Revealed by Silencing of Primary Visual Cortex during Contrast Adaptation

Highlights

- Multiple stages along the visual pathway adapt to the contrast of a visual stimulus
- We sought to dissect the processes contributing to adaptation in V1
- Optogenetic cortical silencing causes adaptation in V1 to resemble dLGN adaptation
- V1 adaptation can be modeled by divisive scaling of weakly adapting dLGN input

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In Brief

What is the neural basis of visual adaptation illusions? King et al. silence mouse primary visual cortex (V1) during contrast adaptation to determine the weighting of contributing adaptive processes. V1 adaptation mainly arises locally through activity-dependent divisive scaling, with most of the remainder coming from inherited thalamic adaptation.

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Adaptive Processes in Thalamus and Cortex Revealed by Silencing of Primary Visual Cortex during Contrast Adaptation

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SUMMARY

Visual adaptation illusions indicate that our perception is influenced not only by the current stimulus but also by what we have seen in the recent past. Adaptation to stimulus contrast (the relative luminance created by edges or contours in a scene) induces the perception of the stimulus fading away and increases the contrast detection threshold in psychophysical tests [1, 2]. Neural correlates of contrast adaptation have been described throughout the visual system including the retina [3], dorsal lateral geniculate nucleus (dLGN) [4, 5], primary visual cortex (V1) [6], and parietal cortex [7]. The apparent ubiquity of adaptation at all stages raises the question of how this process cascades across brain regions [8]. Focusing on V1, adaptation could be inherited from pre-cortical stages, arise from synaptic depression at the thalamo-cortical synapse [9], or develop locally, but what is the weighting of these contributions? Because contrast adaptation in mouse V1 is similar to classical animal models [10, 11], we took advantage of the optogenetic tools available in mice to disentangle the processes contributing to adaptation in V1. We disrupted cortical adaptation by optogenetically silencing V1 and found that adaptation measured in V1 now resembled that observed in dLGN. Thus, the majority of adaptation seen in V1 neurons arises through local activity-dependent processes, with smaller contributions from dLGN inheritance and synaptic depression at the thalamo-cortical synapse. Furthermore, modeling indicates that divisive scaling of the weakly adapted dLGN input can predict some of the emerging features of V1 adaptation.

RESULTS AND DISCUSSION

Adaptation in primary visual cortex (V1) depends somewhat on firing rate [12, 13]. Therefore, we reasoned that optogenetically silencing V1 could help disentangle inherited dorsal lateral geniculate nucleus (dLGN) adaptation and thalamo-cortical synaptic depression, both of which should be unaltered by silencing, from the locally produced adaptation that should be minimized by silencing.

Photostimulation Effects

For cortical silencing, we used VGAT-ChR2(H134R)-YFP (yellow fluorescent protein) transgenic mice that express light-sensitive channelrhodopsin-2 (ChR2(H134R)) in all GABAergic interneurons [14]. Therefore, we first sought to confirm that photostimulation could modulate neural activity in a similar way to previous work that induced cortical silencing with ChR2 expressed via adeno-associated viruses [15, 16]. As expected from the diverse and reciprocal connections among GABAergic and excitatory neurons [17], submaximal photostimulation that modulated interneuron activity over a moderate range produced suppression in some cells (Figures 1A and 1B) and activation in others (Figure 1C). Suppressed cells were selected for, and higher-intensity photostimulation used in the adaptation protocol was sufficient to silence neural activity during the adaptation period, or nearly so, for all neurons tested (Figure 1E). Silencing was observed at all recording depths, similar to the effect of strongly driving parvalbumin-expressing (Pvalb+) interneurons alone [15, 16].

Silencing V1 Neurons Alters Contrast Adaptation

We minimized cortical adaptation with a novel photostimulation protocol that optogenetically silenced V1 neurons during the presentation of the adapting stimulus, but not during the test stimulus (Figures 1D and 1E). Contrast adaptation normally caused the adapted contrast response functions of V1 neurons to shift downward and rightward (Figures 2A, 2C, and 2E, white dots); however, photostimulation during the adapting period markedly dampened these effects and actually increased responses to low contrasts (Figures 2A, 2C, and 2E, blue dots). Regular adapted V1 responses to low contrasts are often unchanged (Figures 2A and 2E), or occasionally decreased slightly (Figure 2C), so this facilitation was surprising. The average of the normalized curves (n = 51) illustrates the consistent changes induced both by adaptation and photostimulation (Figure 3A).

Sigmoid fits to each contrast response function are shown as thin curves in Figure 2 (see Supplemental Experimental Procedures), and the R_{max}, c_{50}, and M fit parameters were used to quantitatively analyze changes in our sample's contrast response functions induced by adaptation and photostimulation. Decreases in R_{max} indicate lower adapted responses to maximal contrast (response gain control), and increases in c_{50} indicate a rightward shift in the curve (contrast gain control) [18]. Increases