How the mechanisms of long-term synaptic potentiation and depression serve experience-dependent plasticity in primary visual cortex

Sam F. Cooke and Mark F. Bear

In figure 1 of this article, two lines were erroneously drawn in part (a) and the axis label of the scale bar at the top of panel (h) was incorrectly shown as 100 mV; it should have been 100 µV. Also, in figure 4, the axis labels of the two scale bars at the top of panels (e) and (f) were incorrectly shown as 100 mV; they should have been 100 µV. The corrected figures are shown below.

Figure 1. Ocular dominance (OD) plasticity resulting from visual deprivation. (a) Head-fixed mice view phase-reversing sinusoidal grating stimuli while visual-evoked potentials (VEPs) and unit activity are recorded. An occluder is used to restrict visual input to one eye or the other. (b) Recordings are made from electrodes implanted in layer 4 of the binocular zone of V1 (green), receiving independent input from the contralateral (blue) and ipsilateral (yellow) eyes. (c) In binocular V1 of the mouse, thalamo-recipient principal cells in layers 2/3 and 4 receive independent inputs from contralateral and ipsilateral eyes. Pronounced feed-forward connections from layer 4 to layer 2/3 and horizontal connections within layers 2/3 also exist. Inhibitory cells receive thalamic and intra-cortical input and inhibit principal cells throughout cortex. (d) OD plasticity is assayed by suturing the contralateral eye for 3 days to deprive this eye of visual input. Monocular deprivation (MD) results in a significant reduction in the amplitude of VEPs driven by visual input through the contralateral eye. Example waveforms are displayed at the top of the figure. (e) Binocularly responsive units can be scored for ocular dominance by assessing the bias towards response to the contralateral or ipsilateral eye prior to and after deprivation. OD plasticity is assayed by suturing the contralateral eye for 3 days to deprive this eye of visual input. Monocular deprivation (MD) results in a significant reduction in the amplitude of VEPs driven by visual input through the contralateral eye. Example waveforms are displayed at the top of the figure. (f) Binocularly responsive units can be scored for ocular dominance by assessing the bias towards response to the contralateral or ipsilateral eye prior to and after deprivation. OD plasticity is assayed by suturing the contralateral eye for 3 days to deprive this eye of visual input. Monocular deprivation (MD) results in a significant reduction in the amplitude of VEPs driven by visual input through the contralateral eye. Example waveforms are displayed at the top of the figure. (g) Binocularly responsive units can be scored for ocular dominance by assessing the bias towards response to the contralateral or ipsilateral eye prior to and after deprivation. OD plasticity is assayed by suturing the contralateral eye for 3 days to deprive this eye of visual input. Monocular deprivation (MD) results in a significant reduction in the amplitude of VEPs driven by visual input through the contralateral eye. Example waveforms are displayed at the top of the figure.

Correction

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Figure 4. SRP and LTP mutually occlude one another: (a) Geniculo-cortical LTP can be induced in vivo by acutely placing a stimulating electrode into the dorsal lateral geniculate nucleus (dLGN) of isoflurane anaesthetised mice and recording electrically-evoked potentials in layer 4 through chronically implanted recording electrodes that can also record VEPs in the same animals when awake. (b) LTP induced with theta burst stimulation (TBS) applied repeatedly to the dLGN lasts for at least the hour of recording time prior to recovery of animals from anaesthesia. (c) Interactions between LTP and SRP can be tested by inducing LTP in just one hemisphere (black) and comparing VEP amplitudes with those recorded in the other, control hemisphere (open) before or after SRP. (d) VEP amplitude was significantly greater in the LTP hemisphere (black) than the control hemisphere (open) whether VEPs were sampled 1 day later or 5 days later, indicating the impact of LTP on responsiveness to visual stimuli. (e) Although VEPs are significantly greater in amplitude in the TBS hemisphere than in the control hemisphere there is less SRP in this hemisphere as a consequence, so that VEP amplitude is no longer significantly different after 5 days of repeated presentation of the same grating stimulus. (f) In the reverse experiment, after SRP has been saturated to one familiar orientation (blue), TBS-induced LTP significantly impacts only those VEPs driven by a novel orientation (red), indicating that SRP occludes the effects of LTP on the VEP. (b–f) are reproduced from [87]. (g) A schematic describing the simple interpretation of these results shows that feed-forward TC plasticity forms templates of strengthened synapses for familiar stimuli (blue) in thalamo-recipient layer 4 that overlap minimally with patterns driven by other, novel oriented stimuli (red). LTP indiscriminately potentiates large numbers of synapses impacting response to all stimuli (black), whether familiar or novel. Asterisks denote significance of $p < 0.05$. 