Modeling Learning and Adaptation Induced Plasticity of Orientation Tuning in V1

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Hubel and Wiesel demonstrated that primary visual cortex (V1) is the first stage in the visual pathway where cells respond to the orientation of a stimulus. However, there is still controversy over how this property arises. Feed-forward models propose that orientation tuning arises from the alignment of inputs from the LGN to V1. The modified feed-forward model (MFM) is an updated version that includes anti-phase intracortical inhibition to maintain contrast invariance. By contrast, the recurrent model (RM) proposes that LGN inputs are only broadly tuned for orientation and are sharpened through recurrent intracortical connections. We have recently shown that although the MFM is applicable to simple cells, the RM produces complex cells when cells with different receptive field phases are included in the model. We have also developed a modified recurrent model (MRM) which adds anti-phase inhibition to the RM in order to preserve a cell’s receptive field phase. However, the MRM still sharpens a broadly tuned feed-forward input through a recurrent cortical mechanism. Thus, the question still stands: do simple cells achieve sharp orientation tuning through a feed-forward mechanism (such as the MFM) or through a recurrent intracortical mechanism (such as the MRM)?

One fruitful way to address this issue is to examine these models from the standpoint of plasticity. Orientation tuning has recently been shown to be plastic in two different paradigms. Learning an orientation discrimination task has been shown to 1) sharpen orientation tuning curves near the learned orientation and 2) modestly broaden tuning curves far from the learned orientation, while adaptation has been shown to 1) broaden tuning curves near the adapted orientation, 2) modestly sharpen tuning curves far from the adapted orientation, and 3) skew tuning curve peaks away from the adapted orientation. We have previously modeled all of these changes using the RM. Here, we compare the MFM, the RM, and the MRM in order to assess how well each model can reproduce the plasticity data. Learning and adaptation in the orientation domain have both been found to lead to a suppression of neural activity for cells whose preferred orientation is near the learned or adapted orientation. Suppression of firing may be either pre-synaptic (e.g., via synaptic vesicle depletion) or post-synaptic (e.g., via receptor desensitization). For the MFM, the RM, and the MRM, we explore the effects of pre and post-synaptic reductions in both excitatory and inhibitory cells in order to assess how well each of the three orientation models may account for the published learning and adaptation data. We find that the RM and the MRM are better able to do so than the MFM. Two key features are found to be required for plasticity: 1) a feed-forward input from the LGN that is broadly tuned for orientation, and 2) recurrent intracortical connections that sharpen the feed-forward tuning. These two features are prominent in the RM and MRM and absent in the MFM. Our work suggests that 1) complex cells that achieve sharp orientation tuning in a recurrent fashion (such as in the RM) will show all aspects of learning and adaptation-induced plasticity, and 2) the extent to which the MRM verses the MFM is applicable at the simple cell level may be assessed by the degree of observed plasticity in simple cells.