Theories of Pattern Adaptation in the Retina

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Pattern adaptation, namely the adaptation of neural sensitivity to spatial correlations in the visual input, is well documented in the visual cortex. For example, the response to a grating stimulus of a given orientation wanes under prolonged stimulation, without suppression of the response to the orthogonal orientation. Such pattern-specific adaptation is almost universally explained as resulting from the depression or fatigue of neurons selective for the orientation present in the sustained stimulus. This view ascribes pattern adaptation to the cortex and discounts the possibility of similar sophisticated adaptation at earlier stages in the visual pathway, because these are devoid of pattern-tuned cells.

Recent experiments demonstrate that pattern adaptation occurs as early as in the retina [1]. For example, under stimulation by a flickering vertical grating, ganglion cells partially lose their sensitivity to inputs with vertical spatial correlations while retaining sensitivity to inputs with horizontal correlations. This adaptation phenomenon and the ensuing recovery occur on a time scale of a few seconds. How do ganglion cells achieve pattern-specific adaptation in the absence of pattern-tuned presynaptic cells? To detect spatial correlations, ganglion cells must compare inputs originating from distant points on the retina. Thus the explanation must involve a presynaptic network, rather than purely local adaptive mechanisms. Here, we discuss two competing models that may explain pattern adaptation in ganglion cells. They combine known properties of synaptic dynamics with identified aspects of retinal circuitry.

Model 1 was sketched in previous work [1], and relies upon correlation-based strengthening of amacrine-to-ganglion cell synapses. These synapses pick up correlations in the visual input and deliver a proportionate amount of inhibition to postsynaptic ganglion cells, thus counter-acting excitation in a pattern-specific manner. The principle amounts to a form of anti-Hebbian learning that serves to reject prominent patterns in the input. Model 2 is introduced in the present work. It relies on the activity-dependent depression of bipolar-to-ganglion cell synapses. Many bipolar terminals receive afferents from an amacrine cell; the so-formed diad synapses are excitatory, but their strength is modulated by inhibition. If the inhibitory input is anisotropic in space, for example if only one or a few amacrine cells contribute to it, then the diad synapse can act as a ‘mini pattern-detector’, which adapts due to synaptic depression.

Both models are formulated in terms of simple nonlinear differential equations involving few parameters: a gain and a time scale. From these equations, one can derive the adaptive evolution of the ganglion cell’s receptive field as a function of the visual input. This treatment recommends new experiments that could distinguish the two synaptic mechanisms based on ganglion cell recordings alone. We illustrate this point with an example experiment.

The models discussed here may be relevant to other instances of correlation adaptation, beyond retinal processing, and they extend the repertoire of neural mechanisms that can perform this important computation.