Contrast adaptation by adjusting neurotransmitter release probability in a hypercolumn model of visual cortex

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It has been proposed (Adorján & Obermayer 1999) that contrast adaptation is at least partly due to the change of neurotransmitter release probability at the thalamocortical synapses. The model there consists of one layer of recurrently connected excitatory cortical neurons corresponding to one orientation column in cortex that receives geniculate input from sinusoidally modulated Poisson processes with identical phase. Afferent and recurrent connections are endowed with depressing synapses (Tsodyks & Markram 1997). The adaptation to different levels of contrast is achieved via slowly changing the probability of neurotransmitter release at the feed-forward synapses in a way that the mutual information between input and output firing rate is maximized.
Our goal now is to translate the above ideas into a biologically more relevant framework. Here we extend this model to a full orientation hypercolumn of coupled excitatory and inhibitory conductance-based integrate and fire neurons. The LGN input to this hypercolumn is modeled as a two-dimensional grid of sinusoidally modulated Poisson processes. Each cortical neuron has a certain orientation prefference and has connections coming from the LGN according to its receptive field (ON regions) as well as recurrent connections to its neighbours in orientation space. The LGN input is broadly tuned with an aspect ratio between 2 and 3. We use a narrow distribution of excitatory and an only somewhat broader distribution of inhibitory connections to achieve contrast invariant orientation tuning (Somers, Dragoi & Sur 2002). Our choice of inhibitory tuning thus is consistent with estimates of cortical orientation currents (Anderson, Carandini & Ferster 2000). Each afferent synaptic connection is explicitly modelled as depressing, each recurrent connection as non-depressing, depressing or facilitating synapse. To derive a learning rule for the neurotransmitter release probability we again make the hypothesis that the mutual information between a cortical neurons output firing rate and its geniculate input firing should be maximal. For the output firing rate we use an approximation of the contrast response function of the model. Specifically we simulate a neuron in the full recurrent network and vary the release probability of the afferents. The corresponding mean output firing rate is fitted by a function of the stationary afferent EPSC, which is used to model the input-output relationship of a neuron in our hypercolumn. The mutual information between the input and output distribution is thus maximised by performing gradient ascent on this approximated output firing rate.
First we note that contrast saturation in our model is due to the depressing afferent connections (Carandini, Heeger & Senn 2002). Second we show that the experimental findings of contrast adaptation (Carandini & Ferster 1997) can be reproduced by regulating neurotransmitter release at the afferent excitatory but not recurrent connections. Specifically different temporal components of the membrane potential and spike response at the optimal and near optimal orientations in our model show qualitatively the same behavior as in the experiments. Fig. 1 summarizes the results of the preffered orientation response for adaptation at the afferents, while the recurrent connections utilize a fixed amount of neurotransmitter. Fig. 1 can be compared to experimental findings of e.g. (Carandini & Ferster 1997). Clearly from Fig. 1 it can be seen that the F1 component of the subthreshold membrane potential adapts differently from the average (DC) component. It has been shown (Adorján & Obermayer 1999) that in the limit of week recurrent connections the low frequency components of the membrane potential do not adapt. Again we argue that the small adaptation effect of the F1 component of the membrane potential visible in Fig. 1 is due to the backprojection of F1 firing. On the other hand the F1 component of the firing adapts strongly, because the average membrane potential decreases, which leads to a smaller part of the membrane potential above threshold. We note that the absolute value of membrane potential hyperpolarization in our model is less than the one observed in experiment.
We also find a phase advance relative to the driving stimulus with increasing contrast as well as with adaptation (Fig. 1), that is with reduction in release probability, as has been experimentally observed (Saul 1995). Two different mechanisms in the synaptic depression model (Tsodyks,

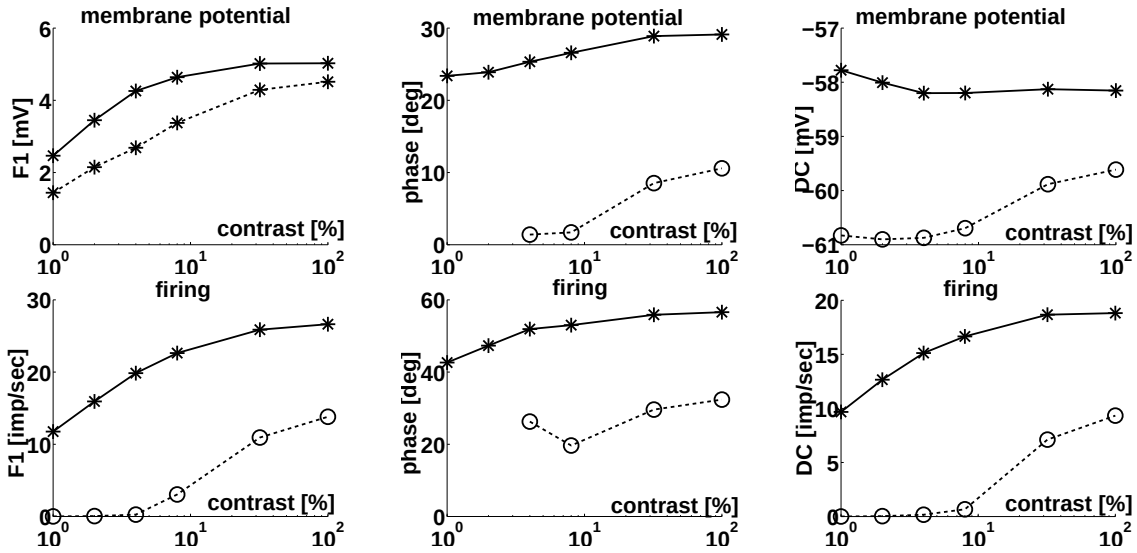

Figure 1: Simulation results of the preffered orientation response of the full hypercolumn model; the transmitter release probability of the geniculo-cortical synapses has been slowly adapted, whereas release probability of the recurrent connections has remained fix. The F1 component, the phase of the F1 component, and the average of the subthreshold membrane potential (top) and the firing (bottom) of the preffered orientation response are plotted as a function of stimulus contrast after adaptation to 1% (solid line) and to 32% (dashed line) contrast stimuli. All simulations are averaged over three independent trials with different afferent connections and different realizations of the Poisson process for the geniculate-firing.

Pawelzik & Markram 1998) account for these dynamical effects, namely the dependence of the effective time constant of synaptic depression on presynaptic firing frequency *and* neurotransmitter release probability.

We conclude that our biologically more realistic model of one hypercolumn in the input layer of V1 reproduces all findings of the previous model and shows that the hypothesis of adaptation of probability of neurotransmitter release is able to explain important experimental observations related to contrast adaptation.

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