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Evidence that the Adaptive Gain Control Exhibited by Neurons of the Striate Visual Cortex is a Co-operative Network Property

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Abstract

Evidence is provided that the adaptation of cells of the striate visual cortex is mediated by a highly derived signal from a network of neurons. In particular, the adaptation process does not depend on the response level of the cell. We investigated which visual stimuli govern adaptation. We also studied adaptation while perturbing inhibitory and excitatory inputs these neurons. These data suggest that co-operative phenomena may play an important role in regulating the temporal dynamics and gain of motion perception.

1 Introduction and Results

Neurons of the striate cortex have been shown to exhibit an adaptive gain control in response to moving images [e.g. 1,2,3] which is not expressed in the geniculate inputs to the cortex [e.g. 4]. This adaptation is readily seen as a gradual waning of the response over several seconds as the control mechanism reduces gain. More recently this gain control has been shown to be controlled not by the response level of the cell but by the temporal frequencies generated by the moving visual stimulus [5,6]. Thus, if temporal frequencies are high even weak stimuli can rapidly adapt the cells while stimuli which evoke very vigorous responses will not adapt the cells if presented at low stimulus frequencies (Fig. 1). In particular, even very high contrast stimuli, evoking near saturating responses can fail to adapt the cell (thin lines, Fig 1 a,b).

We have expanded on this initial result by performing experiments in which adaptation was assessed in response to a range of contrasts and spatial and temporal frequencies. Other factors which might play a

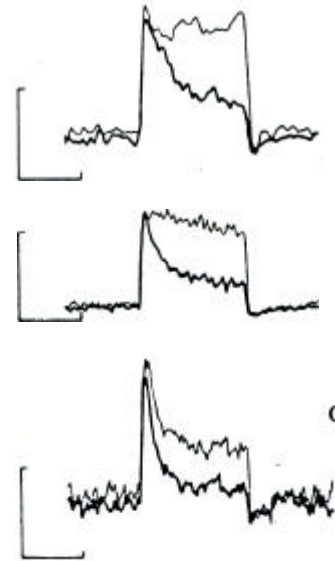


Figure 1: Pairs of response histograms from 3 cat striate cortical cells which illustrate that initial unadapted response level does not determine the rate of adaptation. Each histogram has 3 phases: (1) response to no grating (0% contrast), (2) response to a moving grating, (3) a recovery phase in which the contrast was again set to 0%. The thick and thin lines denote the response of the cells to a slowly (thin) and quickly (thick) moving sinusoidal grating. In each case the initial response has been set to be equal by adjustment of the gratings' contrast. In every case the more rapidly drifting grating causes much greater changes in sensitivity over the 6.5 seconds of motion even though the responses to the slower moving stimuli are driving the cells to near saturation. The calibration bars indicate 5s (abscissa) and 40 impulses s^{-1} (ordinate), 0 impulses s^{-1} is indicated by the level of the abscissa scale bar. The cells and stimulus conditions were: a layer 4a complex cell, 14.4 and 2.4 Hz, 0.78 c.p.d., contrast 15%; b layer 4b complex cell, 19.2 and 4.8 Hz, 0.31 c.p.d., contrast 40%; c layer 6 complex cell, 14.4 and 2.4 Hz, 0.31 c.p.d., contrast 15%. Simple cells adapt in the same way. The total trial time was 23s, only a segment containing the 6.5s period of image motion is shown here.

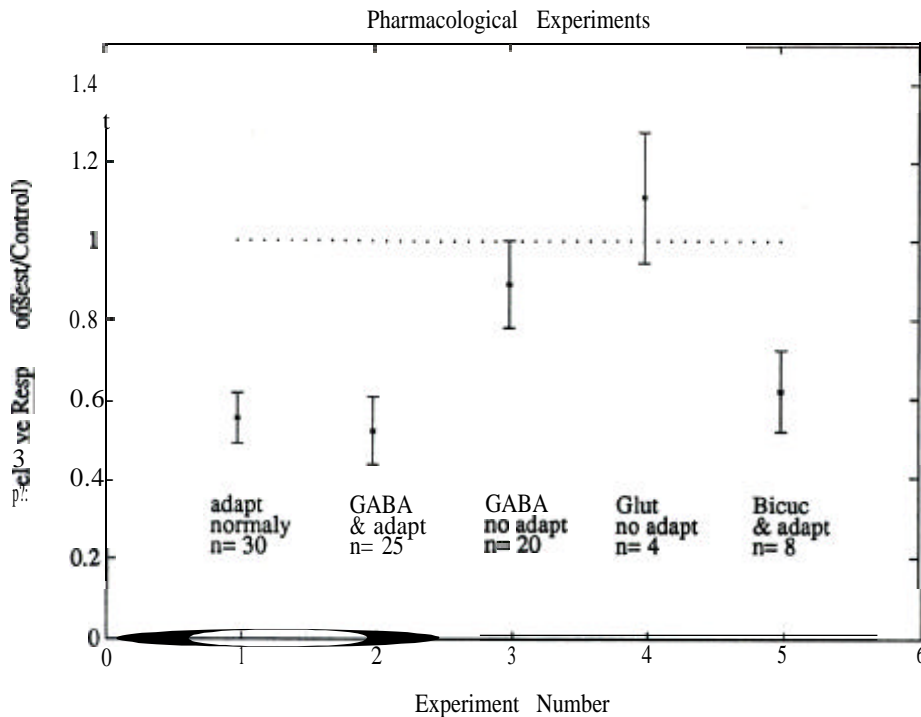


Figure 2: Summarised results from 5 experiments. In each experiment there were 3 phases: a control phase, an “adapting” phase and a test phase. The quantities shown are the ratio: test/control. Typically a cell was adapted in some way in the adapting phase and the effectiveness of that adaptation was assessed in the test phase by presentation of a standard low contrast drifting grating. (1) cells were adapted with a stimulus effective enough to reduce their response to 50% of the unadapted level. (2) cells were given the same visual adapting stimulus but simultaneously had their responses extinguished by iontophoretic application of GABA; the fact that the cells did not respond (and so could not fatigue) did not stop adaptation. (3) application of GABA in the absence of any visual stimulus leaves the cell unadapted, *i.e.* the application of GABA has no adapting effect itself. (4) an attempt is made to adapt the cell by elevation of the maintained discharge by application of glutamate (Glut); like stimulation with slowly moving stimuli in Fig. 1 iontophoretic stimulation of the cell does not adapt the cell. (5) application of the GABA antagonist bicuculine (Bicuc) fails to stop adaptation to a simultaneous adapting visual input, indicating that adaptation is not mediated by inhibition acting upon the cell. The error bars are 95 % confidence limits.

part in determining adaptation levels such as ocular dominance, directional selectivity, cell type (simple-linear, simple-nonlinear, complex: [7]) and cortical cell layer were also considered. Statistical analysis revealed that in addition to a weak response driven adaptation the primary determinants of adaptation level were stimulus temporal frequency and cell directional selectivity. The response driven adaptation, also appeared to be linked to cell direction selectivity such that more directional cells affected more by the stimulus temporal frequency. Examination of several spatial frequencies allowed us to also exclude image velocity as a factor determining adaptation. Since the response of the cells is not important in determining the state of adaptation and since the cells frequently do not exhibit the temporal tuning or motion selectivity of the mechanism driving adaptation it is tempting to suggest that the adaptation signal

represents a co-operative neural network signal given that the cell does not appear to contain the requisite information within its own response to control its adaptation. Psychophysical correlates of cortically mediated temporal frequency dependent, and contrast independent, adaptation to image motion have also been shown in humans [8].

More recently one of us [9] has used iontophoretically applied pharmaceutical agents to investigate the nature of the adaptation and its relation to cell response. Figure 2 summarises the results of these experiments. Five types of experiment were performed and in each case experimental trials were divided into an “adapting” and a test phase. In type 1, cells were adapted using sufficient contrast to reduce their responses by about 50% of their control response level (similar to the case presented in Fig. 1c).

In the remaining four types of experiment various pharmaceutical agents were iontophoretically applied in the “adapting” phase with or without adapting visual stimuli. In experiment 2, GABA was injected concurrently with the presentation of a stimulus which would normally produce a 50% reduction in cell response during the test phase. Injection of GABA almost abolished the cells’ responses during the period when the adapting visual stimulus was presented. In this case there was no opportunity for any “fatigue” or response driven adaptation to occur. Nevertheless, during the subsequent test phase, when the GABA was removed, the response was reduced by 50%. Presentation of GABA alone (experiment 3) without any adapting visual input, left cell sensitivity in the unadapted state. Injection of glutamate (Glut in Fig. 2) in the absence of visual stimulation elevated cell average response levels to that of the adapting visual stimulus. Once again cell response did not determine the adaptation level because in the absence of visual stimulation strong responses evoked by glutamate left the cell unadapted. Finally, a GABAa blocker, bicuculine (Bicucu in Fig. 2), did not prevent adaptation to a moving pattern, indicating that the adaptation process is unlikely to be driven by an inhibitory input to the particular cell.

2 Discussion

The two groups of experiments indicate that individual cell response, whether strongly driven by high contrast visual stimulation or by injection of glutamate, was ineffective in determining cell adaptation. Further injection of GABA with concurrent presentation of adapting visual stimuli, while suppressing cell response, had no effect on the resulting adaptation state, indicating that cell response has little effect on the adaptation process. The adaptation signal itself appears to be highly derived in that it is insensitive to the level of stimulus contrast and is tuned to temporal frequencies not necessarily optimal for the cell in question. Also, stimulus velocity does not appear to be important.

Another observation was that all cell layers and classes (simple and complex) appear to adapt in the same way. The observation that even layer 4 cells such as those in Fig. 1a,b (i.e. cells at the input to area-17) adapt

in a way which does not depend on factors which can modify their response, such as image contrast, indicates that the adaptation process may be occurring in a co-operative neural network, and may be reentrant [10]. We feel that the lack of any effect by bicuculine makes it unlikely that adaptation is mediated by an inhibitory input to the cell. So we are left with the possibility that the adaptation process reflects a reduction in the excitatory co-operative interactions between cells. Cross-correlation studies indicate that long-range horizontal interconnections are largely between clusters of cells having similar orientation preference and that these connections are excitatory [11]. Moreover, anatomical studies indicate that horizontal connections within the visual cortex are excitatory [12,13,14]. From the functional stand-point it is interesting that this apparently cooperative neural inter-action, in addition to synchronising activity across the cortex [e.g. 15], is strongly involved in the regulation of cell responses to the temporal frequencies produced by moving visual stimuli. These strategies may be of general image processing importance given that they also occur in other animal phyla [16,17]

3 References

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