

Factors Governing the Adaptation of Cells in Area-17 of the Cat Visual Cortex

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Abstract. Neurons in area 17 of the cat visual cortex adapt when stimulated by drifting patterns of optimal orientation, spatial frequency and temporal frequency (Ohzawa et al. 1982; Albrecht et al. 1984; Ohzawa et al. 1985). A component of this adaptation has been attributed to a contrast gain-control mechanism, rather than to neural fatigue, and results in enhanced differential sensitivity around the adapting contrast level (Ohzawa et al. 1982; Albrecht et al. 1984; Ohzawa et al. 1985). Experiments described here suggest that neural response rate, the directional selectivity of the cell, and the temporal frequency of the stimulus, are the principal determinants of adaptation, irrespective of other stimulus parameters such as contrast, velocity, or spatial frequency. The present results can nevertheless accommodate the results of previous studies of adaptation, and additionally provide scope for the resolution of apparent contradictions between results from psychophysical and neurophysiological studies of adaptation.

Introduction

Recent studies have demonstrated that the response of neurons in area 17 of the cat visual cortex declines following prolonged exposure to drifting grating patterns of optimal orientation, spatial frequency and temporal frequency (Ohzawa et al. 1982; Albrecht et al. 1984; Ohzawa et al. 1985). This reduction is in part due to a compression of the cell's dynamic range, indicative of neural fatigue or habituation, but a component has also been attributed to a contrast gain-control mechanism. The observed adaptation induces a shift of the neural response range leading to enhanced differential sensitivity around the adapting contrast level (Albrecht et al. 1984; Ohzawa et al. 1985). While cortical cells are noted to vary considerably in the degree of

adaptation they exhibit, cells of the lateral geniculate nucleus consistently fail to demonstrate changes in sensitivity like those observed in cortical cells (Ohzawa et al. 1982, 1985).

Curiously, psychophysical experiments employing prolonged exposure to high contrast grating patterns suggest that while contrast sensitivity is depressed around the adapting spatial frequency and orientation (Gilinsky 1968; Pantle and Sekuler 1968; Blakemore and Campbell 1969) a concomitant improvement of differential contrast, spatial frequency or orientation discrimination around the adapted levels is not found (Barlow et al. 1976).

The present study attempts to control for a variety of stimulus parameters and cortical cell attributes to address the question of what regulates the decrease in neural response seen during prolonged exposure to drifting grating patterns. Since it has previously been shown that this decline is at least in part due to adaptive gain control, we will refer to the effect as adaptation. Visual cortical cells in cat and monkey exhibit varying degrees of response selectivity for visual stimuli across a number of dimensions. The tuning of the response functions are, however, rarely less than an octave in bandwidth. The momentary response of a cell may thus be simultaneously influenced by a number of stimulus dimensions, where it is not known to what extent these different parameters are confounded in the cell's unidimensional output. One conceivable strategy whereby a cell might avoid prolonged periods of response saturation, or understimulation, is to let response rate itself govern adaptation. Alternatively, considering the tuned character of cortical cell response, one can imagine a system in which adaptation depends upon the level(s) of particular stimulus parameters. For one example, although not related to adaptation, Albrecht and Hamilton (1982) have demonstrated that stimulus contrast and not cell response appears to control the compression and

saturation of the contrast response functions of cat and monkey striate cells measured at different spatial frequencies.

The present experiments utilize a multiple regression analysis of stimulus parameters, receptive field characteristics, and neural response. Our results suggest that one component of adaptation is indeed linked to neural response, but that stimulus temporal frequency, and the degree of directional selectivity are also significant determining factors. Parameters such as cortical cell layer, stimulus spatial frequency or velocity, did not account for a significant proportion of the total adaptation. Of particular interest was the result that stimulus contrast did not appear to affect adaptation when its unique effects were isolated from other experimental parameters. The results may nonetheless be viewed as consistent with those obtained where adaptation was studied as a function of adapting contrast (Ohzawa et al. 1982; Albrecht et al. 1984; Ohzawa et al. 1985). In addition, the present results suggest a possible resolution of the dilemma posed by the apparently contradictory physiological and psychophysical assessments of the benefits of contrast adaptation, and are discussed with respect to their possible connection with motion-after-effects.

Methods

1 Procedure

Recording procedures and histology were as previously described (Bullier and Henry 1979) but details of anaesthesia are provided here. During preparatory surgery the cats were anaesthetized with 35 mg kg⁻¹ Ketalar and 3 mg / kg Rompun, and a long lasting local anaesthetic (2% Marcaine) was injected around any wounds. At this time tracheal and venous cannulae were inserted, and a bilateral cervical sympathectomy was done to minimise eye movements (Rodieck et al. 1967). During recording the anaesthesia was a 70 : 30 mixture of N₂O:O₂, supplemented with Nembutal (1 mg kg⁻¹ h⁻¹) (Hammond 1978) and anaesthetic level was assessed by monitoring heart rate and EEG. Paralysis was maintained by a continuous infusion of pancuronium bromide (Pavulon: 0.4 mg kg⁻¹ h⁻¹) and gallamine triethiodide (Flaxedil: 5 mg kg⁻¹ h⁻¹) in Depolyte [Intervet (Australia) Pty. Ltd.]. Anaesthetic ointment (5% Xylocaine) was applied to pressure points. The animals were not revived from anaesthesia but instead were put down via intravenous injection of Nembutal.

Cells with receptive fields within 10deg of the area *centralis* were isolated and their properties were first qualitatively assessed using handheld stimuli. Receptive field profiles were subsequently quantified using

drifting light and dark lines of the preferred orientation. Cells were classified as simple (S) or complex (B, C) according the criteria of Henry (1977). All quantitative experiments used stimuli generated on a CRT display system with a mean luminance of 70 cd m⁻² (Maddess and Laughlin 1985).

The spatial frequency characteristics of cells were determined by an automated procedure which measured the constant-contrast tuning function. The spatial contrast of drifting sinusoidal gratings was raised from 0 to between 7.5 and 20% (depending on contrast threshold) for 1 s out of 4 s. The short duty cycle was employed to minimize adaptation effects. The mean spike rate during the 1 s interval was taken as the response measure. Spatial tuning was assessed at several temporal frequencies to insure the preferred spatial frequency was stationary (Tohlhurst and Movshon 1975).

Further tests utilized peak initial spike rate to establish the range of stimuli for subsequent tests of adaptation. Each trial consisted of elevating contrast from 0 to a given test level for 6.7 s out of every 23 s. In these tests the peak initial rate was determined on-line by examination of response histograms smoothed by convolution with a 16 bin wide (104 ms) rectangular window. The recorded histograms were not filtered. The maximum value of the smoothed histogram occurring in the first second following the onset of the test contrast was defined as the peak. For off-line analysis and figure production (Fig. 1) response histograms were smoothed by convolution with a Gaussian function with a halfwidth chosen by previously defined criteria (Maddess 1986). Preliminary tests indicated that an inter-trial interval of 23 s was sufficient for cells to recover to near unadapted response levels. The contrast and temporal frequency of a grating of optimal spatial frequency were varied until 3 contrasts and 3 temporal frequencies were selected for the experiment. Contrasts were spaced at octave intervals such that the intermediate contrast produced a near saturated response. The temporal frequencies were likewise separated by 1 to 2 octaves, chosen such that the upper and lower frequencies produced less than optimal but near equal responses. Variations in the temporal bandwidths of cells resulted in 2 or 4 temporal frequencies occasionally being used.

Once test stimuli were selected, data were collected in blocks of 8 or 16 trials. Trials for the differing temporal frequencies and contrasts were randomly interleaved. After 24-32 trials had been obtained for each condition, the matrix of tests was expanded to encompass 1 or 2 spatial frequencies an octave above and below the original. The expanded-matrix experiment was continued until the cell was lost or failed to give reproducible results. The strategy of expanding

the matrix of tests, as opposed to testing all conditions from the outset, insured enough data on adaptation as a function of contrast, temporal frequency and cell response was collected even if the cell could not be held longer than 4 or 5 h. The inclusion of additional spatial frequencies permitted the assessment of the independent effects of velocity and temporal frequency. The accumulation of data for the expanded matrix usually required 8-10 h per cell.

2 Data Analysis

The analysis to be described was a conventional multiple-regression analysis (Wiesberg 1980). The fitting was achieved by using a software package designed to fit Generalized Linear Models (McCullagh and Nelder 1983). Many similar software packages are available and the reader can consult Dobson (1983) for a list of similar software as well as an introduction to linear modelling.

In our initial model [see (2) Results] we categorized several continuous variables, such as contrast or angular velocity, into classes, and the classes were numbered according to the magnitude of the values in each category. Thus, temporal frequency class 2 represented higher temporal frequencies than temporal frequency class 1 and so on. We therefore occasionally refer to these categorized variables as "relative" variables, because of the classes' hierarchical organization, and to the continuous variables as "absolute" variables.

The multiple regression allowed assessment of the separate effects of the various experimental variables on adaptation. The approach allowed the categorized variables or "classes" described above to be fitted as dummy variables or "factors" (McCullagh and Nelder 1983). Following fits to the response data, plots of class estimates against midpoints of class intervals provided insights into possible functional forms for the relationship between the response variable(s) and possible explanatory variables. Thus, the rationale for fitting the variables as factors (categorized variables) becomes clear, i.e., fits to factors can suggest the functional form of relatively complex relationships between predictor variables and the response. One may then test models where the continuous variables are fitted with analytic functions suggested by the fits to categorized data. In our original model linear regressions were at first applied to the uncategorized continuous variables but other analytic functions suggested by fits to factors were also tried and were rejected.

The object of the analysis was to find the most parsimonious model which could explain the response variance. In all cases the significance level chosen was $p = 0.05$. We also checked that the significance of terms

in our models was invariant to the order in which the terms were fitted.

A summary of the ranges covered by the classes is given below. The 10 angular velocity classes spanned the range 0.48 to 46 deg/s, or 6.6 octaves. The 10 temporal frequency classes covered 1.2 to 19.2 Hz, or 4 octaves. Spatial frequencies were between 0.156 and 2.5 cyc/deg spanning 4 octaves. Ocular dominance corresponded to the 7 classes of Hubel and Wiesel (1962) where 7 = ipsilateral and 1 = contralateral. There were 6 classes for directional selectivity defined by the relative percentile response to to-and-fro motion of a moving bar i.e. 1=100:0, 2=90:10, . . . , 6 = 50:50. Laminal position within the striate cortex was determined through electrode tract reconstruction. The three classes of "relative" contrast correspond to the three criterion contrasts described above, that is, a near saturating contrast, and contrasts an octave higher and lower.

Results

Of 38 complex (B + C) and 14 simple (S) cells for which spatial tuning was determined, the smaller matrix of test conditions was completed for 17 complex and three simple cells; the larger matrix was completed for six complex and one simple cell. The sample was biased toward complex cells because in general the spike rates of simple cells were insufficient to allow the required volume of quantitative data to be accumulated, even within 8-10 h.

As noted previously, in off-line analysis histograms were smoothed by convolution with a Gaussian (see Methods). Examples of response profiles illustrating the range of those collected in our sample are shown in Fig. 1. The shapes of these profiles suggest three effects. First, when sufficient recovery time between trials is allowed (i.e. 23 s), large declines in response rate during a trial (6.7 s) are visible in some cells. As mentioned previously, these response changes have been shown to be in part due to adaptive gain control (Albrecht et al. 1984; Ohzawa et al. 1985). Second, some cells demonstrate little adaptation at low temporal frequencies but adapt conspicuously at higher temporal frequencies, even when contrast and all other test conditions, including peak initial spike rate, are the same (Fig. 1a and b). Finally, other cells show marked adaptation at all temporal frequencies with relatively minor enhancement at high frequencies (Fig. 1c). These representative histograms summarize the principal findings of this study, and are confirmed by quantitative statistical analysis.

In agreement with previous reports (Albrecht et al. 1984), satisfactory approximations of response decay throughout the 6.7 s stimulus interval could not be

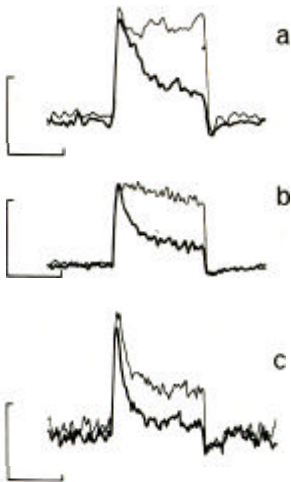


Fig. 1a-c. Smoothed and averaged response histograms from three cells which illustrate the range of results obtained in this study. The response histograms were generated using grating stimuli in which spatial frequency and contrast were constant. The stimuli in each case differed only in temporal frequency, where the two different frequencies were matched (or nearly so) for peak initial spike rate $R(i)$. In each case the higher temporal frequency (bold line) produced a larger response decline throughout the interval. The degree of response decline observed for the low temporal frequency stimulus varies from nearly none (**a**) to about 50% (**c**). Between-cell variation such as this is partly accounted for by the directional selectivity of the cell. Striate cortical layer, cell type, contrast, spatial frequency and temporal frequency for each stimulus pair are: **a** 4a, C-cell, 15% 0.78 cyc/deg, 9 Hz and 3 Hz. **b** 4b, C-cell, 40%, 0.31 cyc/deg, 19.2 Hz and 4.8 Hz. **c** 6, C-cell, 15%, 0.31 cyc/deg, 14.4 Hz and 2.4 Hz. The scale bars are: 40 impulses s^{-1} (ordinate) and 5 s (abscissa). Each histogram pair is respectively the average of 60, 80, and 32 interleaved trials

obtained using simple analytic functions. Response decline was therefore indexed from two points the peak initial spike rate: $R(i)$, and the final rate, $R(f)$, achieved after 6.7 s of stimulation.

Having selected $R(f)$ and $R(i)$ as indices one would like to examine the ratio $R(f)/R(i)$ as the response variable for statistical analysis. Preliminary investigation showed the distribution of these ratios to be positively skewed (to the right), and the data displayed heterogeneous variance (heteroskedasticity), that is, the variance in $R(f)$ was related to $R(i)$. The relation between $R(f)$ and $R(i)$ appeared to be multiplicative rather than an additive. The heterogeneous variance and the multiplicative nature of the relation suggested that a logarithmic transformation of both $R(f)$ and $R(i)$ would also linearize the relationship between $R(i)$ and $R(f)$ permitting the use of straightforward multiple regression analysis. As will be seen later the heterogeneous variance reflected the fact that the systematic effects produced by some test parameters acted on the dependent variable $R(f)$ in a multiplicative fashion.

We examined the possibility of a direct proportionality between $R(f)$ and $R(i)$ as a null hypothesis and by fitting them to the following model

$$\ln(R(f)) = B + C(\ln R(i)) . \quad (1)$$

Note that Eq. (1) with $C=1$ is equivalent to considering the logarithm of the ratio $R(f)/R(i)$ as the response variable for statistical analysis. This model of initial and final responses proved to be highly significant ($F(1,147) = 153.5$). Figure 2 shows that the coefficient C above was not 1, as initially assumed, but 0.802 ± 0.090 ($p=0.05$) indicating a power function relationship between $R(i)$ and $R(f)$.

In general, larger initial spike rates produce proportionately smaller final response rates, suggesting a component of response driven adaptation. In other words, large spike rates, irrespective of origin, tend to result in more adapted final responses after a fixed period. The high significance of the response model (1) was partly due to the necessary covariation of $R(i)$ and $R(f)$, e.g., cells with larger initial responses tended to give relatively larger final responses.

Equation 1 was actually part of a larger initial regression model of the form:

$$\ln(R(f)) = B + C \ln(R(i)) \quad (= \text{response model})$$

- + possible effects of
 - absolute contrast
 - 3 relative contrast classes
 - cell class (S, B, C)
 - striate cortical layer
 - 7 ocular dominance classes
 - 10 spatial frequency classes
 - absolute spatial frequency
 - 6 directional selectivity classes
 - absolute angular velocity
 - 10 relative angular velocity classes
 - absolute temporal frequency
 - 10 relative temporal frequency classes. (2)

Those parameters described as absolute represent the continuous variables to which linear regressions were initially applied. The classes are described in Methods and fits to these as factors allowed significant determinants of $R(f)$ to be modelled even if they showed no simple trend.

As a first approximation, the random variation in $\ln(R(f))$ was assumed to be normally distributed. Preliminary analyses and subsequent model checking or "diagnostics" (Weisberg 1980) revealed the above model was appropriate. In particular the residuals

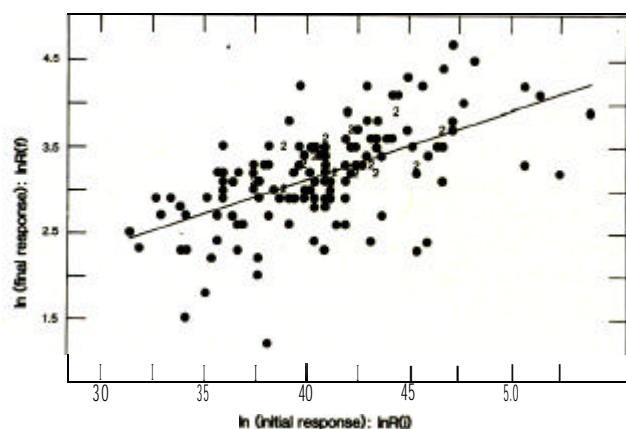


Fig. 2. A scatter plot of the $\ln(R(f))$ on $\ln(R(i))$ displaying the regression line of (1). The slope of the line, i.e. the coefficient C in (1), is not unity but 0.802 ± 0.090 ($p=0.05$) indicating a power function relationship between the initial response, $R(i)$, and the final response, $R(f)$, i.e. (3) in the text. Thus, a component of response driven adaptation is suggested by this power function of $R(i)$ because larger initial responses will lead to relatively smaller final response rates after a fixed period. The units of $R(i)$ and $R(f)$ are impulses per second

from (2) had constant variance and were normally distributed, reinforcing our justification for the log-transformation.

The interpretation of (1) and (2) can be clarified by rewriting the final model in terms of simple neural response.

$$R(f) = AR(i)^{0.802} \quad (3)$$

where the gain (A) depends on the effects of all the significant parameters of (2). Here it is easier to see that for a particular value of A , larger initial spike rates produce proportionately smaller final response rates, suggesting that one component of response reduction (adaptation) is response-driven. In the search for the significant determinants of A , from (2) above, most of the parameters incorporated in the model proved non-significant; significant predictors of cell adaptation were restricted to the factors: directional selectivity ($F(5,142) = 11.8$), and temporal frequency ($F(8,134) = 9.8$). The final model including only the nonlinear (power function) response model and the effects of directional selectivity and temporal frequency accounted for 65.2% of the total variance. The values of A from the final model are plotted in Fig. 3a and b.

Directional selectivity was able to explain much of the between-cell variance, but no simple trend between degree of directional selectivity and susceptibility to adaptation emerged (Fig. 3). It is possible that some factor not measured by us is correlated with our directional selectivity classes or that a range of adaptive behaviours exists for cells of the same directional selectivity class. Models where ocular dominance was

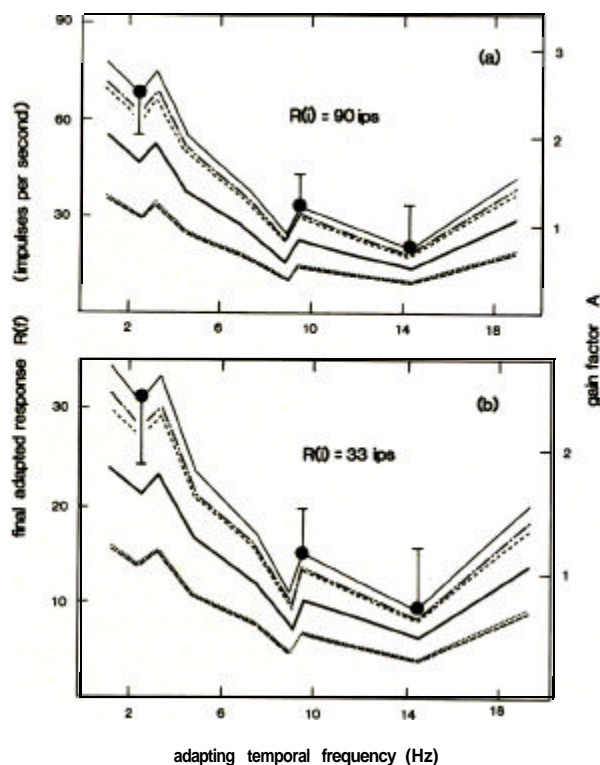


Fig. 3a and b. Panels a and b are predictions, based on the final model (3) of the expected response after 6.7 s, $R(f)$, for the initial peak responses of: **a** 90 impulses s^{-1} and **b** 33 impulses s^{-1} . For all directional selectivity classes increased temporal frequency gives increased adaptation. In both (a) and (b) the expected values of the gain factor A as well as the predicted $R(f)$ are given as a function of adapting temporal frequency. Each curve is for a directional selectivity class. Some 95% confidence limits illustrate that the directional selectivity classes are significantly different at low temporal frequency, while the trend towards higher final rates at very high temporal frequencies (> 15 Hz) is not significant. From top to bottom the curves for the directional selectivity classes are in the order 1, 3, 5, 4, 2, 6. The directional selectivity classes are defined in Methods

fit instead of directional selectivity were almost as good at explaining between-cell variance, and it appeared that cells receiving strong ipsilateral input showed the greatest adaptation. Models incorporating ocular dominance or directional selectivity had the same number of degrees of freedom, providing no formal way to test one against the other. Models employing ocular dominance instead of directional selectivity, however, accounted for much less of the total variance, and so ocular dominance was rejected on this basis. Further investigation might revive ocular dominance as a significant determinant of adaptation.

The trend seen for temporal frequency, however, was clear: high temporal frequencies produced much smaller final responses than lower temporal frequencies. Figure 3 shows predictions from the model

where each panel (a, b) illustrates a particular initial response and each curve represents one class of directionally selective cell. The graphed quantity is the predicted final rate for a particular adapting temporal frequency. The two initial rates represent the upper and lower standard deviations of $\ln(R(i))$ from the data. The fact that the curves look so similar indicates that over the range of initial responses presented (33-90 impulses s^{-1}), the effects of temporal frequency and directional selectivity are far greater than those of the response-driven adaptation.

The response histograms in Fig. 1 illustrate the predictions of the model. Cells such as those in Fig. 1a and b show little response decline at low temporal frequencies, but, even though the initial spike rates and adapting contrast are the same, these same cells show large decreases in response when temporal frequency is higher (cf. Fig. 3 directional selectivity classes 1, 3, 5). By contrast, some cells such as that of Fig. 1c show large response decreases at all temporal frequencies but nonetheless an even smaller final response at high temporal frequencies (cf. Fig. 3 directional selectivity classes 2, 4, 6).

As in previous adaptation studies, simple and complex cells did not distinguish themselves (Ohzawa et al. 1982; Albrecht et al. 1984; Ohzawa et al. 1985) nor were significant differences found between cells from different striate cortical layers (Ohzawa et al. 1985).

Discussion

In the present study a component of response-driven adaptation is suggested by the nonlinear relationship found between initial and final response rates. A large amount of differential adaptation, however, is determined by temporal frequency and the directional selectivity of a particular cell. The effect that temporal frequency and directional selectivity have on the adaptation of cortical cells has not previously been isolated. Rather, adaptation has generally been ascribed to spatial contrast level, which in our study produced a non-significant influence. The present results suggest that adaptation formerly attributed to contrast might be reinterpreted as resulting from a component of response-driven adaptation, the magnitude of which is governed by a gain factor largely determined by temporal frequency and directional selectivity. Adaptation so produced would possess the appearance of being controlled by spatial contrast to the extent that increases in contrast level increase the amplitude of cell response. By analogy, factors such as spatial frequency or orientation might also be expected to produce adaptation-like effects to the extent they likewise control cortical cell response rate.

Shapley and Victor (1978) demonstrated a contrast and temporal frequency dependent effect in the cat retina which they termed a "retinal contrast gain-control". It is natural to ask how it might express itself in the cortex. The effect causes increases in ganglion cell gain when high temporal frequencies are presented at moderate to high stimulus contrast. The retinal effect is seen most strongly in Y-cells (Shapley and Victor 1978) and this is replicated in "Y" or transient cells (Sclar 1987) of the lateral geniculate nucleus (LGN) which receive their input from Y retinal ganglion cells (Cleland et al. 1971a, b; Kaplan and Shapley 1984). Given that the retinal gain control increases Y-cell response over the same temporal frequency range as that for which we observe stronger adaptation in the cortex, the possibility arises that the sensitivity changes seen in the cortex are driven by the "Y" input from the LGN. Thus, the retinal effect of Shapley and Victor might supply the temporal frequency dependence of cortical cell adaptation.

As previously noted, psychophysical experiments have failed to demonstrate the consequences which might be expected to result from the operation of electrophysiologically-demonstrated adaptive gain control mechanisms in the cortex; *vis.* no improvement in the relative discrimination of contrast, spatial frequency, or orientation around adapted values of these parameters has been shown to occur (Barlow et al. 1976). The present results suggest, however, that improved temporal frequency discrimination (or contrast etc.) might be observed as a function of adapting temporal frequency. Interestingly, a recent information-theoretic analysis of adaptation to contrast using information theory (MacKerras et al. 1988) indicates the typical benefits would be small (0.1 to 0.2 bits per pixel), suggesting little physiological value would accrue from a gain control mechanism linked to the level of stimulus contrast.

The relationship we observe between cell adaptation, directional selectivity and stimulus temporal frequency may implicate the participation of directionally-selective motion detection mechanisms, since many such mechanisms respond in proportion to the temporal frequency of moving gratings (van Santen and Sperling 1985). Indeed, adaptive changes in an invertebrate visual interneuron (H1) itself a temporal frequency dependent motion detector, have been shown to be regulated by stimulus temporal frequency (Maddess and Laughlin 1985; Borst and Egelhaaf 1987). The existence of directionally-selective elements in the human visual system is psychophysically demonstrated by direction specific adaptation and the motion-after-effect (for reviews see Sekuler 1975; Sekuler et al. 1978). Since it is the temporal frequency of periodic patterns which controls the magnitude of

the motion-after-effects (Pantle 1974; Johnston and Wright 1983; Wright and Johnston 1985), and which, as shown here, also controls the gain of response driven adaptation, it is tempting to suggest a relationship between these phenomena. These factors taken with the interocular transference of motion-after-effects (Barlow and Brindley 1966) suggest a cortical gain control mechanism sensitive to and regulated in part by the temporal frequency of moving stimuli. In fact it may be a mechanism of this type which underlies the Weber behaviour revealed in psychophysical studies of velocity discrimination (McKee 1981; Nakayama 1981; Van Doorn and Koenderink 1983).

Lorenceau (1987) has recently done psychophysical studies on the time-course of recovery from adaptation to drifting gratings as a function of the spatiotemporal parameters of the stimulus. The rate of recovery and the amount of residual sensitivity loss were significantly and systematically related to the temporal frequency of the adapting stimulus, such that higher temporal frequencies were more effective than lower frequencies at producing adaptation. Spatial frequency was not a significant determinant of recovery. The parallel with the present work is clear and intriguing.

Finally, the present results suggest that cortical cells possess temporal frequency subchannels. That is, for equal initial response rates, cells nevertheless adapt in accord with their temporal frequency input, in other words they can distinguish their input based on information not contained in the mean spike rate alone. Subchannel specific adaptation has been observed in studies of spatial frequency adaptation in single cortical neurons (Movshon and Lennie 1979; Albrecht et al. 1984).

Acknowledgements. We would like to thank A. James for helpful discussions and G. H. Henry for numerous suggestions and support. T. Maddess was supported in part by a Canadian Medical Research Council Fellowship.

References

- Albrecht DG, Hamilton DB (1982) Striate cortex of monkey and cat: contrast response function. *J Neurophysiol* 48: 217-237
- Albrecht DG, Farrer SB, Hamilton DB (1984) Spatial contrast adaptation characteristics of neurons recorded in the cat's visual cortex. *J Physiol* 347: 713-739
- Barlow HB, Brindley GS (1963) Inter-ocular transfer of movement after-effects during pressure blinding of the stimulated eye. *Nature* 200: 1347
- Barlow HB, Macleod DIA, van Meeteren A (1976) Adaptation to gratings: no compensatory advantages found. *Vision Res* 16: 1043-1045
- Blakemore C, Campbell FW (1969) On the existence in the human visual system of neurons selectively sensitive to the orientation and size of retinal images. *J Physiol (London)* 203: 237-260
- Borst A, Egelhaaf M (1987) Temporal modulation of luminance adapts time constant of fly movement detectors. *Biol Cybern* 56: 209-215
- Bullier J, Henry GH (1979) Laminar distribution of first-order neurons and afferent terminals in cat striate cortex. *J Neurophysiol* 42: 1251-1263
- Cleland BG, Dubin MW, Levick WR (1971a) Sustained and transient neurone in the cat's retina and lateral geniculate nucleus. *J Physiol (London)* 217: 473-497
- Cleland BG, Dubin MW, Levick WR (1971b) Simultaneous recording of input and output of lateral geniculate neurone. *Nat New Biol* 231: 191-192
- Dobson AJ (1983) An introduction to statistical modelling. Chapman & Hall, London
- Gilinsky AS (1968) Orientation-specific effects of adapting light on visual acuity. *J Opt Soc Am* 58: 13-18
- Hammond P (1978) Inadequacy of nitrous oxide/oxygen mixtures for maintaining anaesthesia in cats: satisfactory alternatives. *Pain* 5: 142-151
- Henry GH (1977) Receptive field classes of cells in the striate cortex of the cat. *Brain Res* 133: 1-28
- Hubel DH, Wiesel TN (1962) Receptive fields, binocular interaction and functional architecture in the cat's visual cortex. *J Physiol (London)* 160: 106-154
- Johnston A, Wright MJ (1983) Visual motion and cortical velocity. *Nature* 304: 436-438
- Kaplan E, Shapley RM (1984) The origin of the S (slow) potential in the mammalian lateral geniculate nucleus. *Exp Brain Res* 55: 11-116
- Lorenceau J (1987) Recovery from contrast adaptation: effects of spatial and temporal frequency. *Vision Res* 27: 2185-2191
- McCullagh P, Nelder JA (1983) Generalized linear models. Chapman & Hall, London
- McKee SP (1981) A local mechanism for differential velocity detection. *Vision Res* 21: 491-500
- MacKerras P, Bossomaier T, Maddess T, Laughlin SB (1988) Information gains from contrast adaptation (in preparation)
- Maddess T (1986) Afterimage-like effects in the motion-sensitive neuron H1. *Proc R Soc London B* 228:433-459
- Maddess T, Laughlin SB (1985) Adaptation of the motion-sensitive neuron **H1** is generated locally and governed by contrast frequency. *Proc R Soc London B* 225: 251-275
- Movshon JA, Lennie P (1979) Pattern-selective adaptation in visual cortical neurons. *Nature* 278: 850-852
- Nakayama K (1981) Differential motion hyperacuity under conditions of common image motion. *Vision Res* 21: 1475-1482
- Ohzawa I, Sclar G, Freeman RD (1982) Contrast gain control in the cat visual cortex. *Nature* 298: 266-268
- Ohzawa I, Sclar G, Freeman RD (1985) Contrast gain control in the cat visual system. *J Neurophysiol* 54: 651-667
- Pantle A (1974) Motion aftereffect magnitude as a measure of the spatio-temporal response properties of direction-sensitive analyzers. *Vision Res* 14: 1229-1236
- Pantle A, Sekuler RW (1968) Size-detecting mechanisms in human vision. *Science* 162:1146-1148
- Rodieck RW, Pettigrew JD, Bishop PO, Nikara T (1967) Residual eye movements in receptive field studies of paralyzed cats. *Vision Res* 7: 107-110
- Santen JPH van, Sperling G (1985) Elaborated Reichardt detectors. *J Opt Soc Am A* 2:300-231
- Sclar G (1987) Expression of "retinal" contrast gain control by neurons of the cat's lateral geniculate nucleus. *Exp Brain Res* 66: 589-596

- Sekuler R (1975) Visual motion perception. In: Carterette EC, Freeman MP (eds) Seeing. Handbook of perception, vol V. Academic Press, New York, pp 387-430
- Sekuler R, Pantle A, Levinson E (1978) Physiological basis of motion perception. In: Held R, Leibowitz HW, Teuber H (eds) Perception. Handbook of sensory physiology, vol VIII. Springer, Berlin Heidelberg New York, pp 67-98
- Shapley RM, Victor JD (1978) The effect of contrast on the transfer properties of cat retinal ganglion cells. *J Physiol* London 285: 275-298
- Tohlhurst DJ, Movshon JA (1975) Spatial and temporal contrast sensitivity of striate cortical neurons. *Nature* 257:674-675
- Van Doorn AJ, Koenderink JJ (1983) Detectability of velocity gradients in moving random dot patterns. *Vision Res* 23: 799-804
- Weisberg S (1980) Applied linear regression. Wiley, New York
- Wright MJ, Johnston A (1985) Invariant tuning of motion aftereffect. *Vision Res* 25:1947-1955

Received: March 11, 1988

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