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Adaptation in single units in visual cortex: The tuning of aftereffects in the spatial domain

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Abstract

Cat striate cortical neurons were investigated using a new method of studying adaptation aftereffects. Stimuli were sinusoidal gratings of variable contrast, spatial frequency, and drift direction and rate. A series of alternating adapting and test trials was presented while recording from single units. Control trials were completely integrated with the adapted trials in these experiments.

Every cortical cell tested showed selective adaptation aftereffects. Adapting at suprathreshold contrasts invariably reduced contrast sensitivity. Significant aftereffects could be observed even when adapting at low contrasts.

The spatial-frequency tuning of aftereffects varied from cell to cell. Adapting at a given spatial frequency generally resulted in a broad response reduction at test frequencies above and below the adapting frequency. Many cells lost responses predominantly at frequencies lower than the adapting frequency.

The tuning of aftereffects varied with the adapting frequency. In particular, the strongest aftereffects occurred near the adapting frequency. Adapting at frequencies just above the optimum for a cell often altered the spatial-frequency tuning by shifting the peak toward lower frequencies. The fact that the tuning of aftereffects did not simply match the tuning of the cell, but depended on the adapting stimulus, implies that extrinsic mechanisms are involved in adaptation effects.

Keywords: Adaptation aftereffects, Spatial-frequency tuning, Contrast, Visual cortex

Introduction

Single-unit studies in cat visual cortex have demonstrated that central neurons, like peripheral neurons, adapt to prolonged stimulation. Retinal adaptation occurs in the luminance domain, while visual cortical cells adapt in the contrast (Ohzawa et al., 1985; Dean, 1983; Albrecht et al., 1984), direction (Vautin & Berkley, 1977; von der Heydt et al., 1978; Hammond et al., 1985; Saul & Daniels, 1985; Marlin et al., 1988), and spatial-frequency (Maffei et al., 1973; Movshon & Lennie, 1979; Albrecht et al., 1984; Hammond et al., 1985) domains. Visual cortical adaptation occurs in the temporal-frequency domain as well (Saul & Cynader, 1989). Adapting stimuli induce aftereffects: one minute of stimulation with a high-contrast grating drifting across a cell's receptive field reduces the cell's responsiveness and contrast sensitivity to test gratings of similar spatial frequencies moving at the same rate in the same direction.

Movshon and Lennie (1979) remarked that aftereffects in the spatial-frequency domain were specific to the adapting stimulus. That is, responses to test stimuli were reduced most severely when the adapting and test stimuli had the same spatial frequency. However, Movshon and Lennie (1979) only reported results from two adapting and test frequencies which were more than an octave apart. The tuning of aftereffects has been investigated in considerable detail in psychophysical experiments. Narrow, bandpass tuning in the spatial-frequency domain is a key result in the psychophysical literature (Blakemore & Campbell, 1969). We wanted to establish similarly detailed tuning functions for single units. We also wanted to verify and extend Movshon and Lennie's result. Several studies have indicated that the tuning of aftereffects was specific, but did not demonstrate that this specificity matched the adapting stimulus. Instead, aftereffects have been shown to be strongest when the adapting and test stimuli were of the optimal spatial frequency for the cell (Maffei et al., 1973; Albrecht et al., 1984; Hammond et al., 1985). Optimal adapting stimuli clearly induce the strongest aftereffects, and the greatest response reduction can occur where the initial response is greatest. Intrinsic mechanisms in the cell (Vautin & Berkley, 1977), such as fatiguing of the spike-generation components of the membrane, could account for specific aftereffects if the specificity matched the cell's own tuning. However, if aftereffects could be shown

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to be tuned differently from the cell's tuning, then extrinsic mechanisms (Vautin & Berkley, 1977) would be necessary to explain adaptation effects.

In this article, we discuss the tuning of aftereffects in the spatial-frequency domain. We include additional results for two reasons: (1) to compare the aftereffects observed under our experimental protocol with the results of conventional methods; and (2) for their relevance to considerations of mechanisms of adaptation. The accompanying article (Saul & Cynader, 1989) describes the tuning of adaptation aftereffects in the temporal-frequency domain, and discusses the directional characteristics of adaptation.

Methods

Physiological preparation

Single-unit recordings were obtained from twelve adult cats. Anesthesia was induced by intravenous injection of sodium thiopental. 0.2 mg atropine was administered intravenously to reduce salivation. A tracheal tube was inserted and the animal was placed in a stereotaxic apparatus. Electrodes were placed across the chest to monitor heart waveform and rate. A rectal thermometer controlled a heating pad which maintained body temperature near 38°C. The scalp was split and skull screws were inserted above the right hemisphere as EEG electrodes. All surgical wounds and pressure points were infiltrated with 0.25% Marcaine.

A small craniotomy was made over the central visual-field representation of area 17 in the left hemisphere. The dura remained intact until a glass-coated platinum–iridium electrode was driven through into the visual cortex. Histological reconstructions showed that most penetrations ran ventromedially down the medial bank, sampling all layers.

Gallamine triethiodide (15 mg) was given intravenously to induce muscle relaxation, and the animal was artificially respired with N_2O and O_2 (70:30). Gallamine triethiodide was infused at a rate of 10 mg/kg/h with 5% dextrose in saline, along with 1 mg/kg/h of pentobarbital sodium. End-tidal CO_2 was monitored by a Beckman gas analyzer and the stroke volume of the respiration pump was adjusted to obtain CO_2 levels of about 4%.

Contact lenses were chosen by retinoscopy to focus the eyes at a distance of 100 cm. Atropine and Neosynephrine were applied to dilate the pupils and retract the nictitating membranes, respectively. All contact lenses contained 4-mm diameter artificial pupils. Lenses were cleaned daily and the eyes were infiltrated with hypertonic saline to maintain the optical quality of the corneas.

Recording and stimulation

Single cells were isolated using a window discriminator. Output pulses were fed to a computer which stored the arrival times in synchrony with stimulus generation to 1-ms resolution.

Using manually projected light slits, receptive fields were plotted and preferred orientation was estimated. Sine wave gratings were generated on an Electrohome video monitor driven by a video interface board in the computer. One eye was occluded, and the monitor was placed 1 m from the open eye, with the receptive field approximately at the center of the 14.25 × 11-deg screen, which was rotated to match the cell's preferred orientation. The gratings filled the screen except in rare cases when the length was reduced by electronic masking in order to evoke responses from strongly end-stopped cells. The contrast, spatial frequency, temporal frequency, and direction of the drifting grating could be varied. In most experiments, the contrast of the gratings, defined as $(L_{max} - L_{min})/(L_{max} + L_{min})$, could be set in integer multiples of 0.0015. The monitor was reasonably linear up to contrasts above 0.30. Spatial and temporal frequencies used were integer multiples of about 0.025 cycles/deg and 0.06 Hz, respectively.

Experiments consisted of alternating "adapting" and "test" trials. The parameters of both adapting and test gratings were randomly varied across sets of adapting and test conditions. A "run" consisted of a choice of these sets of parameters. Over the course of a run, all combinations of adapting and test pairs were presented several times. Figure 1 illustrates the structure of a run. Except where noted, test trials were 5-s long, while adapting trials lasted 15 s. A pause of 100 ms was inserted before every trial. When many stimulus conditions were required, long runs were attempted. We found that single units could often be held throughout 2–6 h runs (and occasionally for over 20 h), but many cells were lost before sufficient data were compiled. Runs were terminated if the physiological state of the animal changed noticeably, or if another unit contaminated the original cell's isolation. The window discriminator was adjusted to provide narrow limits for acceptance of spikes in order to avoid such contamination, with the consequence that some spikes may have been missed, particularly at the end of response bursts when spike amplitudes dropped. We doubt that this problem could bias our results (Vautin & Berkley, 1977) since spike amplitudes only seemed to decrease during bursts, not as a result of adapting.

Data Analysis

Responses reported in the Results, unless otherwise noted, are derived from the 5-s test trials. The activity evoked by each test condition was calculated separately for test trials which followed the different adapting conditions. For example, responses to a particular test stimulus were obtained following zero-contrast adapting trials and following high-contrast adapting stimulation (Fig. 1). The results consist primarily of such comparisons between responses evoked by a given stimulus contingent on the parameters of the immediately prior stimulation. In particular, we will compare "adapted" and "control" responses. Adapted responses are the results of test trials following relatively high-contrast adapting trials, whereas control responses were those following zero- or low-contrast adapting trials. Note that control responses were recorded during trials which were completely interleaved with trials which provided the adapted responses (Fig. 1).

The total number of spikes during a trial was divided by 5 s to obtain the mean firing rate during each trial. First-harmonic responses at the temporal frequency of the stimulus were also computed. In this paper, we consider only mean firing rates. (Over long runs, first-harmonic response amplitudes tended to be diminished, presumably because of slight eye position drifts. Spatial-frequency tuning assessed by the first-harmonic component of responses was shifted toward lower frequencies.)

Data from related conditions were sometimes pooled. For

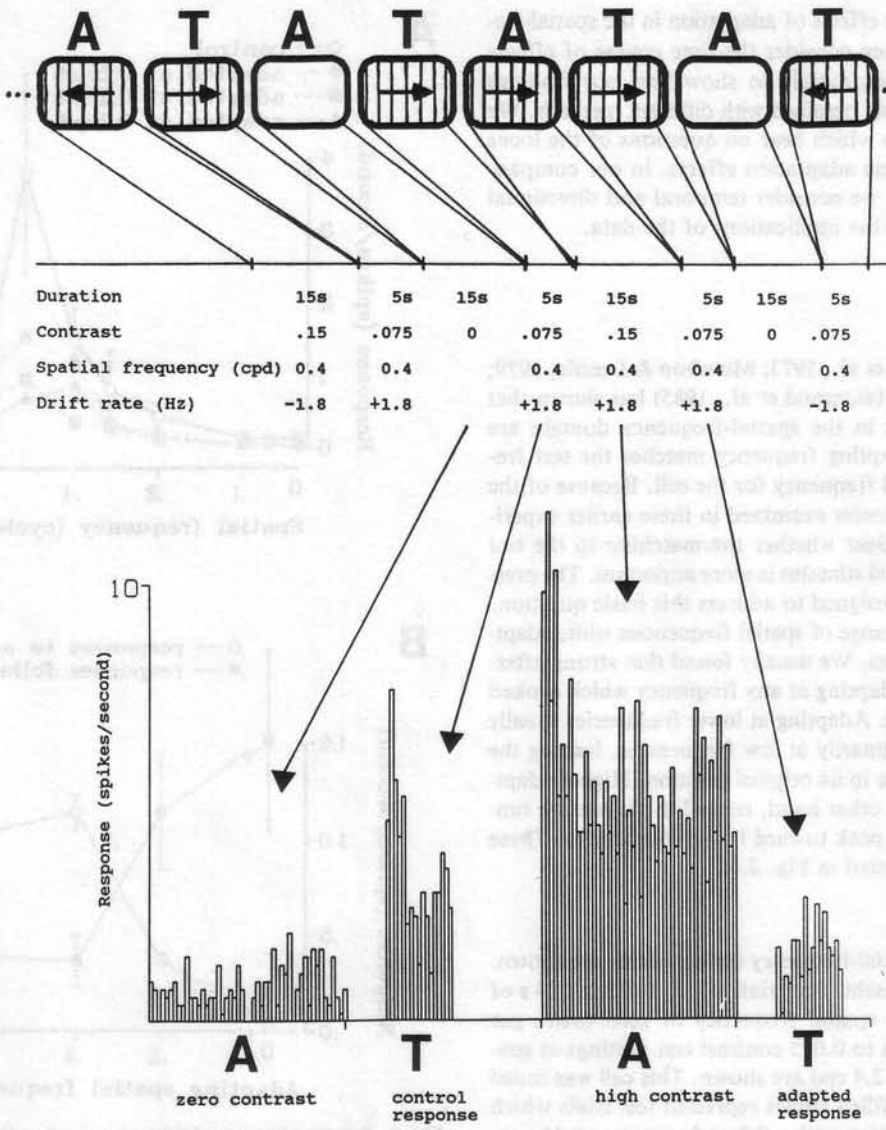

Fig. 1. Each run comprised a series of alternating "adapting" (A) and "test" (T) trials. The parameters of each stimulus were chosen randomly from predetermined sets of conditions. In the run illustrated in part here, adapting trials lasted 15 s, had a spatial frequency of 0.4 cpd, contrasts of either 0 or 0.15, and drifted in either direction at 1.8 Hz. Test trials lasted 5 s, had a contrast of 0.075, a spatial frequency of 0.4 cpd, and drifted in either direction at 1.8 Hz. Several hundreds of such trials were presented in a run. Responses to each test stimulus were then analyzed contingent on the preceding adapting condition. Control trials were those test trials which followed zero- or low-contrast adapting trials, while adapted responses were evoked by test stimuli which followed high-contrast adapting trials. The response histograms shown are averages over 12 or 15 trials.

instance, contrast was often varied during a run, but trials at several contrasts might be grouped together for analysis of responses to other stimulus parameters such as spatial frequency. We found that aftereffects were often bidirectional (Saul & Cynader, 1989) and for this reason combined directions as well when appropriate. Rather than choose which conditions to pool *a posteriori*, as a rule we either combined all conditions or none across a stimulus dimension.

Averages and standard errors were computed from samples of at least 5 trials. As a measure of the strength of aftereffects, we computed the difference between the control and adapted mean responses, divided by the square root of the sum of the squared standard errors. This *t*-score allows the significance of the response decrement to be assessed. More importantly, it provides a quantitative comparison of the aftereffects at different frequencies, so that the tuning properties of adaptation can be examined objectively. One of the advantages of the *t*-score over the simple difference in response is that the large response at the optimal frequency does not necessarily lead to large aftereffects. Other normalizations, such as the ratio of responses, were found inadequate because they often gave clearly spurious results.

Results

Recordings were obtained from 47 visual cortical cells in 12 adult cats. Typically, several runs were attempted on each cell. In total, 67 runs were successfully completed. In every one of these runs, selective adaptation effects were observed.

We first describe the effects of adaptation in the spatial-frequency domain. We then consider the time course of effects and contrast adaptation, mainly to show how our findings relate to results previously obtained with different methods. We will also present results which bear on questions of the locus and mechanisms of these adaptation effects. In our companion paper in this issue, we consider temporal and directional properties, and discuss the implications of the data.

Spatial frequency

Previous work (Maffei et al., 1973; Movshon & Lennie, 1979; Albrecht et al., 1984; Hammond et al., 1985) has shown that adaptation aftereffects in the spatial-frequency domain are strongest when the adapting frequency matches the test frequency and the optimal frequency for the cell. Because of the limited range of frequencies examined in these earlier experiments, it has been unclear whether the matching to the test stimulus or to the optimal stimulus is more important. The present experiments were designed to address this basic question.

We tested cells at a range of spatial frequencies while adapting at several frequencies. We usually found that strong aftereffects resulted from adapting at any frequency which evoked a response from the cell. Adapting at lower frequencies usually decreased responses primarily at low frequencies, leaving the peak of the tuning curve in its original position. Higher adapting frequencies, on the other hand, tended to flatten the tuning curve and shift the peak toward lower frequencies. These observations are illustrated in Fig. 2.

Individual examples

In Fig. 2A, four spatial-frequency tuning curves are shown. The control curve represents test trials which followed 15 s of blank stimulation at a spatial frequency of zero cycles per degree (cpd). Responses to 0.075 contrast test gratings at spatial frequencies from 0–2.4 cpd are shown. This cell was tuned to about 0.6 cpd. The filled circles represent test trials which followed 15 s of stimulation with a 0.2-cpd grating at 0.15 contrast. Responses are below control levels at all frequencies but 1.6 cpd, although the differences are small in a statistical sense (t values are all less than 2.0). Adapting at 0.4 cpd produced the responses indicated by the filled squares, which are depressed at all frequencies. However, the peak remains at 0.6 cpd. Finally, adapting at 0.8 cpd (filled triangles) reduced the responses for 0.6-cpd gratings to a level below that for 0.4 cpd gratings. Such a shift in the peak of the spatial-frequency tuning curve was often seen when adapting at frequencies above the optimum.

In Fig. 2B, the average responses to test stimuli which followed each of the adapting conditions are plotted (filled circles). These responses are complementary to the responses evoked by each of the adapting conditions (open circles), illustrating that optimal stimuli induce the strongest aftereffects. That is, the more spikes evoked during an adapting trial, the fewer spikes could be evoked during the subsequent test trial. However, aftereffects do not depend solely on the responses evoked by adapting stimuli. The 0.4-cpd and 0.8-cpd adapting stimuli evoked similar responses but induced differently *tuned* aftereffects (Fig. 2A).

The spatial-frequency tuning characteristics of adaptation aftereffects showed great variability from cell to cell. Often,

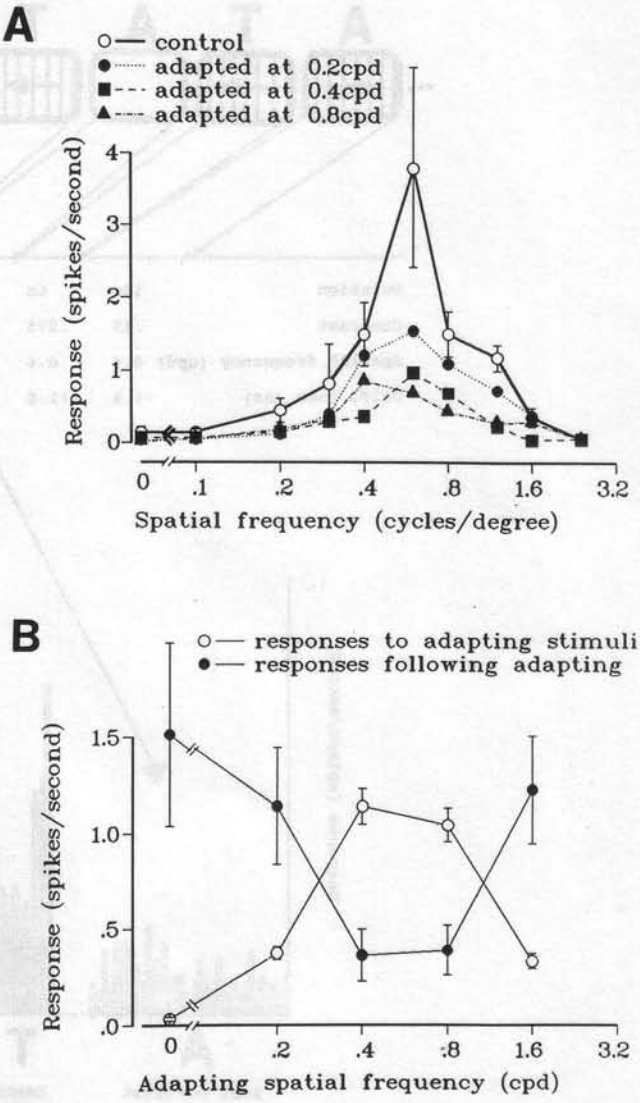

Fig. 2. Results of an experiment on a simple cell. Ten spatial frequencies were tested, from 0–2.4 cpd. Test contrast was 0.075. Adapting trials were 15 s long at a contrast of 0.15, and spatial frequencies of 0, 0.2, 0.4, 0.8, and 1.6 cpd. A, tuning curves are shown for test trials which followed adapting trials at 0 (control), 0.2, 0.4, and 0.8 cpd. The tuning curve for the 1.6 cpd adapting condition is omitted for clarity, as are the error bars for the adapted responses. Each point represents an average over 5–22 trials. B, open circles indicate responses evoked during the adapting trials. Filled circles plot the averaged responses to test trials which followed each of these five adapting conditions. These averages include trials pooled across all spatial frequencies tested.

frequencies lower than the adapting frequency were more affected than higher frequencies. An example is shown in Fig. 3. The adapting stimulus had a spatial frequency of 0.3 cpd (arrow), but test responses were reduced primarily at lower frequencies. These cells tended to be complex cells preferring lower spatial frequencies. This asymmetry was often present, but many cells showed aftereffects at frequencies both above and below the adapting frequency. However, no cells showed reductions in responses only at higher frequencies. Therefore, the envelope of adaptation aftereffects in the cortical population should reflect this asymmetry. We confirmed that a slight

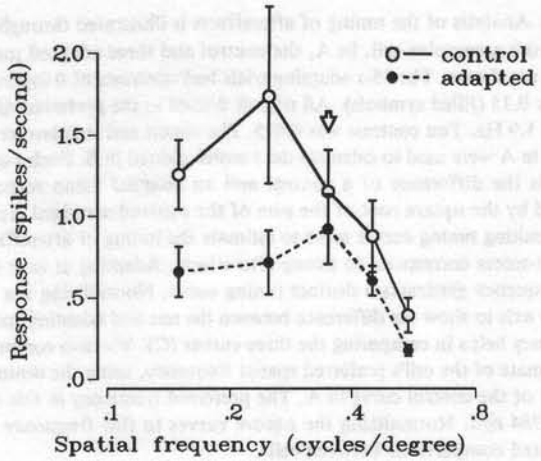

Fig. 3. Spatial-frequency tuning curves from a complex cell. Adapting and test gratings drifted in both directions and have been combined, as have test contrasts from 0.0015 to 0.192. Adapted responses followed 30 s of stimulation with a 0.3 cpd grating (indicated by arrow) at contrasts of 0.096 and 0.192, while control responses followed 30 s of low contrast (0.003) stimulation.

bias toward stronger aftereffects at low frequencies existed in the population data in the following manner.

We performed two normalization procedures to the results from each cell, illustrated for a complex cell in Fig. 4.

1. Each pair of control and adapted tuning curves (Fig. 4A) gave rise to a plot of *t*-score *vs* frequency (Fig. 4B). The adapted mean responses were subtracted from the control mean responses, and the difference was divided by the square root of the sum of the squared standard errors. These *t*-scores were computed for each adapting condition at each test frequency. The *t*-score serves as an index of the strength of aftereffects; assumptions about the distribution of responses would need to be studied in order to apply this index in statistical tests. Strong aftereffects produced *t*-scores above 3. Weak aftereffects gave *t*-scores less than 1.
2. These plots were then centered on either (a) the adapting frequency (Fig. 4C) or (b) the preferred frequency (Fig. 4D). That is, the abscissa was scaled using the number of octaves separating the test frequency from either the adapting or preferred frequency. Thus, in Fig. 4C, the points obtained from adapting at 0.6 cpd (circles) lie an octave above the points obtained from adapting at 1.2 cpd (triangles). The preferred frequency was estimated as the weighted average of the test frequencies, where the weights were the control responses.

This cell showed some of the strongest aftereffects observed. Each of the adapted points in Fig. 4A is well below its corresponding control point. This is reflected in the *t*-values in Fig. 4B, which are all above 2.5. At the lowest test frequency, the lowest adapting frequency (0.6 cpd—filled circles and dotted line) induced stronger aftereffects than did the other two adapting frequencies. At the highest test frequencies, responses were reduced to similar levels by each of the adapting frequencies, but adapting at 0.6 cpd was the least effective. The highest adapting frequency, 1.2 cpd (filled triangles), had relatively weak effects at the lower test frequencies. The tuning of aftereffects shown in Fig. 4B shows a progressive shift with adapting frequency in the frequency at which aftereffects are maximized. By normalizing the frequency axis to the adapting frequency (Fig. 4C), this progressive shift is reduced. Aftereffects were strongest near the adapting frequency, giving a peak around 0. From the control curve in Fig. 4A, the preferred spatial frequency was estimated as 0.78 cpd. Centering and scaling the data of Fig. 4B around this value yielded Fig. 4D. This last normalization simply allows data to be compared across cells having different spatial tuning.

Population results

Data from 34 cells are combined by these means in Fig. 5. The horizontal axis is spatial frequency in octaves above or below (negative values) the adapting frequency (A) or the preferred frequency (B). The vertical axis is the *t*-score, which estimates the strength of the adaptation aftereffect. Almost all of the 534 points have *t* values above zero, as adapted responses were almost always reduced from control levels. No *t*-scores less than -2.0 were obtained (that is, no significant enhancement of responses was seen). Significant aftereffects (using the *t*-test with $P > 0.95$; at least 8 degrees of freedom) were obtained from at least 47% of the points. Adaptation reduced responses in cortical cells over a broad range of spatial frequencies. Aftereffects continued to appear at frequencies more than two octaves below the preferred frequency, and four octaves below the adapting frequency.

Although aftereffects were evident at all frequencies, they were stronger and more reliable around the adapting and preferred frequencies. Figures 5C and 5D show the tuning of aftereffects in the population. Each point in these plots represents the average over those points in Figs. 5A and 5B falling into 1 octave-wide bins. The average *t*-score over the population peaks at the adapting frequency and at the preferred frequency. The spatial-frequency tuning of aftereffects is asymmetric. Aftereffects were stronger at frequencies 1, 2, or 3 octaves lower than the adapting frequency than at corresponding higher frequencies (Fig. 5C). Similarly, aftereffects were on the average much stronger 1 or 2 octaves below the preferred spatial frequency than at 1 or 2 octaves above (Fig. 5D). (We limited the range of spatial frequencies tested to those within the cell's response range. Inclusion of low or high frequencies to which a cell does not respond would produce artifacts when compiling these population data, since aftereffects cannot be observed in the absence of response. Figure 5B therefore shows very few points more than two octaves higher than the preferred frequency.)

The tuning of aftereffects is tied to both the adapting and optimal spatial frequency. The two curves in Figs. 5C and 5D are highly dependent on each other, since adapting frequencies were chosen on each side of the preferred frequency. The more regular shape of the curve in Fig. 5C suggests that the adapting frequency is the more important determinant of the tuning of aftereffects. Results from individual cells, as illustrated in Fig. 4, support this suggestion. The relative dependence on adapting *versus* optimal stimulus can be seen in another way by replotting Fig. 5D for cases where the adapting frequency was either below or above the optimal frequency. The 534 points were separated into 3 sets: (I) the adapting frequency was more than half an octave below the optimal frequency (194 points); (II) the adapting frequency was within a half octave of the optimal frequency (236 points); and (III) the adapting frequency was more than half an octave above the optimal fre-

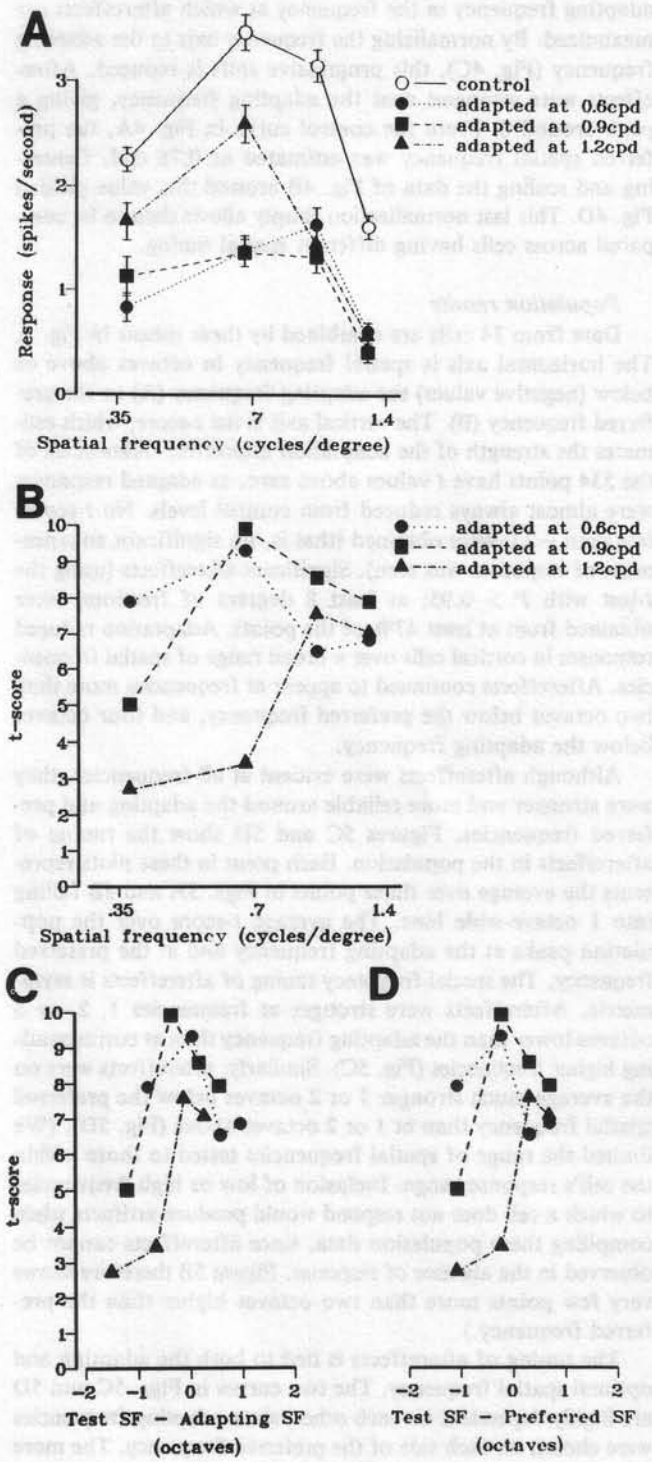

Fig. 4. Analysis of the tuning of aftereffects is illustrated through the data from a complex cell. In A, the control and three adapted tuning curves are shown. The 15-s adapting trials had contrasts of 0 (open circles) or 0.15 (filled symbols). All stimuli drifted in the preferred direction at 1.9 Hz. Test contrast was 0.075. The means and standard errors shown in A were used to calculate the *t*-scores plotted in B. Each *t*-score point is the difference of a control and an adapted mean response divided by the square root of the sum of the squared standard errors. The resulting tuning curves serve to estimate the tuning of aftereffects. Large *t*-scores correspond to strong aftereffects. Adapting at each spatial frequency generates a distinct tuning curve. Normalizing the frequency axis to show the difference between the test and adapting spatial frequency helps in comparing the three curves (C). We also computed an estimate of the cell's preferred spatial frequency, using the center of gravity of the control curve in A. The preferred frequency in this case was 0.784 cpd. Normalizing the *t*-score curves to this frequency (D) facilitated comparisons between cells.

quency (104 points). The points were binned and averaged in each case as in Fig. 5. The middle set showed the strongest aftereffects by far, as adapting near the preferred frequency always reduces responses maximally. The average *t*-score when testing near the optimal adapted frequency was 3. The tuning curve for this case was very sharp, and showed a slight asymmetry with lower test frequencies showing stronger aftereffects than higher frequencies. In Fig. 6, we show the average *t*-scores induced by cases I and III. Figure 6 shows that adapting at lower spatial frequencies (square symbols) induced stronger aftereffects at low test frequencies. Adapting at higher frequencies (circles) was less effective overall; in particular, higher test frequencies were no more affected by adapting at nearby frequencies than by adapting at much lower frequencies. Higher adapting frequencies induced aftereffects mainly near the optimal frequency, as illustrated in Figs. 2–4. Although aftereffects were not as strong as in case II in general, the lowest test frequency showed significantly stronger aftereffects from case I than from case II (mean of $2.03 \pm .04$ for case I *vs* mean of $1.61 \pm .05$ for case II).

Therefore, adapting at frequencies lower than the optimum induced aftereffects which matched the adapting stimulus, whereas adapting at frequencies higher than the optimum induced aftereffects which more closely matched the cell's tuning. Since aftereffects were asymmetric, however, one might expect that adapting at higher frequencies would result in the strongest aftereffects occurring at lower frequencies, near the preferred frequency.

Reproducibility

Although the spatial-frequency tuning of aftereffects was broad in some cells and narrow in other cells, the tuning of aftereffects in a single cell was highly reproducible, as judged by the several occasions when we could repeat a run successfully. An example of two similar runs performed about 3 h apart is shown in Fig. 7A. The activity in the second run was lower than during the earlier run, but the behavior was quite similar. Adapting at 0.175 cpd, just above the optimal frequency for this cell, induced broadly tuned aftereffects. In Fig. 7B, we illustrate the results of two consecutive runs in which frequency-selective adaptation was observed. This cell was adapted at two spatial frequencies. Adapting at the lower frequency (filled triangles) induced aftereffects only at low test frequencies, in both runs. Adapting at the higher frequency (filled squares) depressed responses more generally, but was more effective at higher than at lower test frequencies, as shown by the *t*-score data. Most of the variability in the tuning of adaptation aftereffects seemed to reflect cell-to-cell differences, rather than variability in the behavior of a given cell.

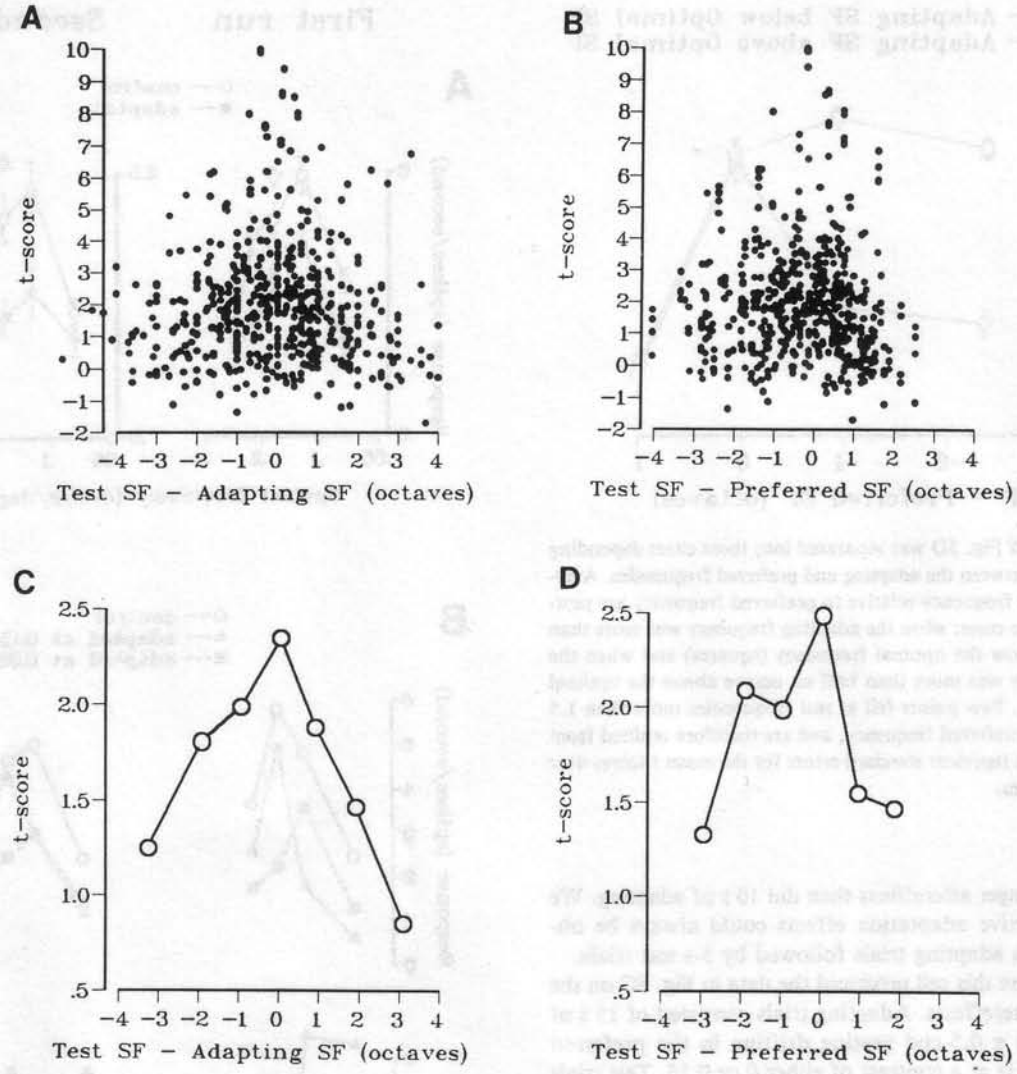

Fig. 5. Population data on the spatial-frequency tuning of aftereffects. Results from 34 cells were analyzed as in Fig. 4. Each *t*-score was paired with its corresponding spatial frequency relative to either the adapting or preferred frequency. In each of the plots in A and B, 534 such points were compiled. Note that test frequencies were necessarily within a few octaves of the preferred frequency, whereas the adapting and test frequencies could fall at opposite ends of a cell's response range. The points in A and B were grouped into bins and their coordinates were averaged, to produce C and D. The seven bins have boundaries of −2.5, −1.5, −0.5, 0.5, 1.5, and 2.5. For D the last two bins were combined into one bin for all frequencies more than 1.5 octaves above the preferred frequency. Standard deviations were less than 0.3 in the *t*-score coordinate and less than 0.1 in the frequency coordinate. Each bin contained between 29 and 148 points.

Time course

The strength of adaptation aftereffects depends on how long the adapting stimulus is presented. The duration of adapting stimulation also influences how long aftereffects persist. Although previous studies have often relied on 60 s or more of adapting stimulation, our results depended on presenting many more trials. Brief adapting trials proved to be effective in eliciting aftereffects. Even 10 s of stimulation induced strong aftereffects. Indeed, the strength of aftereffects seemed to saturate as the duration of adapting stimuli surpassed 10 s.

Figure 8 illustrates the effects of varying the duration of the adapting stimulation, and the persistence of the aftereffects. This complex cell was adapted for 0, 5, 10, 15, and 30 s at 0.15 contrast. Figure 8A shows the responses evoked by the 5 adapting conditions, and the responses subsequently evoked by the single test condition used (0.075 contrast, 0.5 cpd, 1.4 Hz), which was presented 100 ms after each adapting trial. Each adapting trial actually lasted 30 s, but the contrast was zero for the first 30, 25, 20, 15, or 0 s. All trials were interleaved. Twenty trials were presented in each of the 5 conditions. The spontaneous firing rate was only 0.1 spikes/s, while evoked activity approached 10 spikes/s. In Fig. 8B, the open circles indicate the response magnitudes evoked by the test stimulus following adapting stimulation of 0, 5, 10, 15, and 30-s durations. Increasing the duration of adapting stimulation produced a progressive decline in test responses, with the greatest decline occurring between 5 and 10 s. Adapting for 15 or 30 s induced

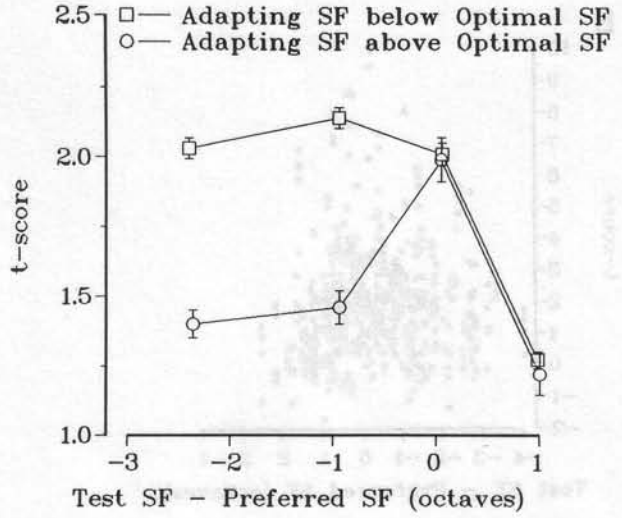

Fig. 6. The data of Fig. 5D was separated into three cases depending on the difference between the adapting and preferred frequencies. Average *t*-scores *vs* test frequency relative to preferred frequency are plotted for two of these cases: when the adapting frequency was more than half an octave below the optimal frequency (squares) and when the adapting frequency was more than half an octave above the optimal frequency (circles). Few points fell at test frequencies more than 1.5 octaves above the preferred frequency, and are therefore omitted from the plot. Error bars represent standard errors for the mean *t*-scores over the data in each bin.

only slightly stronger aftereffects than did 10 s of adapting. We found that selective adaptation effects could always be observed using 15-s adapting trials followed by 5-s test trials.

Another run on this cell produced the data in Fig. 8C on the persistence of aftereffects. Adapting trials consisted of 15 s of stimulation with a 0.5-cpd grating drifting in the preferred direction at 1.4 Hz at a contrast of either 0 or 0.15. Test trials lasted 13 s, but the contrast was zero for the initial 0, 1, 2, 4, or 8 s. Following this initial delay, the contrast was set at 0.075 for 5 s, after which the contrast was returned to 0. The responses evoked by each of these test conditions are shown, contingent on whether the preceding adapting trial had a contrast of 0 (control) or 0.15 (adapted). Responses were reduced when test stimuli were presented soon after adapting stimuli. Delays of 4 or 8 s before presentation of the test stimulus permitted responses to return to near control levels. Precise estimation of the persistence of aftereffects was impossible using 5-s test trials (using briefer trials would hinder comparison with our other results). For this and other reasons, the effect of the duration of adapting stimulation on the persistence of aftereffects could not be reliably gauged. Figure 8C suggests that 15 s of adapting stimulation induced aftereffects persisting for 6–9 s, given that responses were averaged over the 5-s test duration.

Contrast

We employed low contrasts in this study for several reasons: we wanted to avoid fatiguing cells; aftereffects seemed most reliable at lower contrasts; the response–contrast relation is most linear between contrasts of 0.05 and 0.20 (Dean, 1981); stimulus nonlinearities were minimized at lower contrasts; and psy-

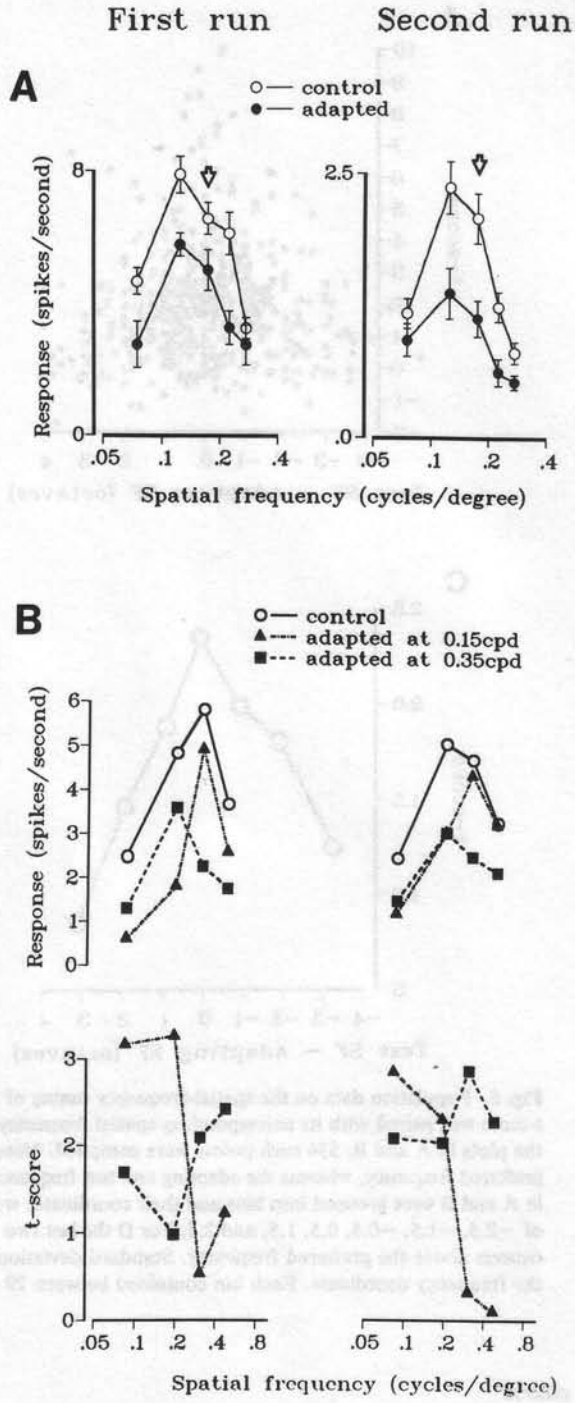

Fig. 7. Two examples of the reproducibility of the results. A, spatial-frequency tuning curves from adapted (filled circles) and control (open circles) trials are shown for two consecutive runs on a complex cell. Responses are averages over 7–22 trials at drift rates of 1.2, 2.4, and 3.6 Hz in the preferred direction, at a contrast of 0.096. Adapted trials followed 10 s of stimulation with 0.175 cpd gratings at a contrast of 0.15, at drift rates of 1.2, 2.4, and 3.6 Hz (first run) and 1.2, 2.4, 3.6, 4.8, 6.0, and 7.2 Hz (second run) in the preferred direction. B, two consecutive runs from another direction-biased complex cell. The test contrast was 0.06. Adapting trials lasted 10 s, had contrasts of 0.0015 (control) or 0.15 (adapted), and spatial frequencies of 0.15 cpd (triangles) or 0.35 cpd (squares). All stimuli drifted in the preferred direction. The responses are shown, as well as the *t*-scores between the control and each of the adapted curves.

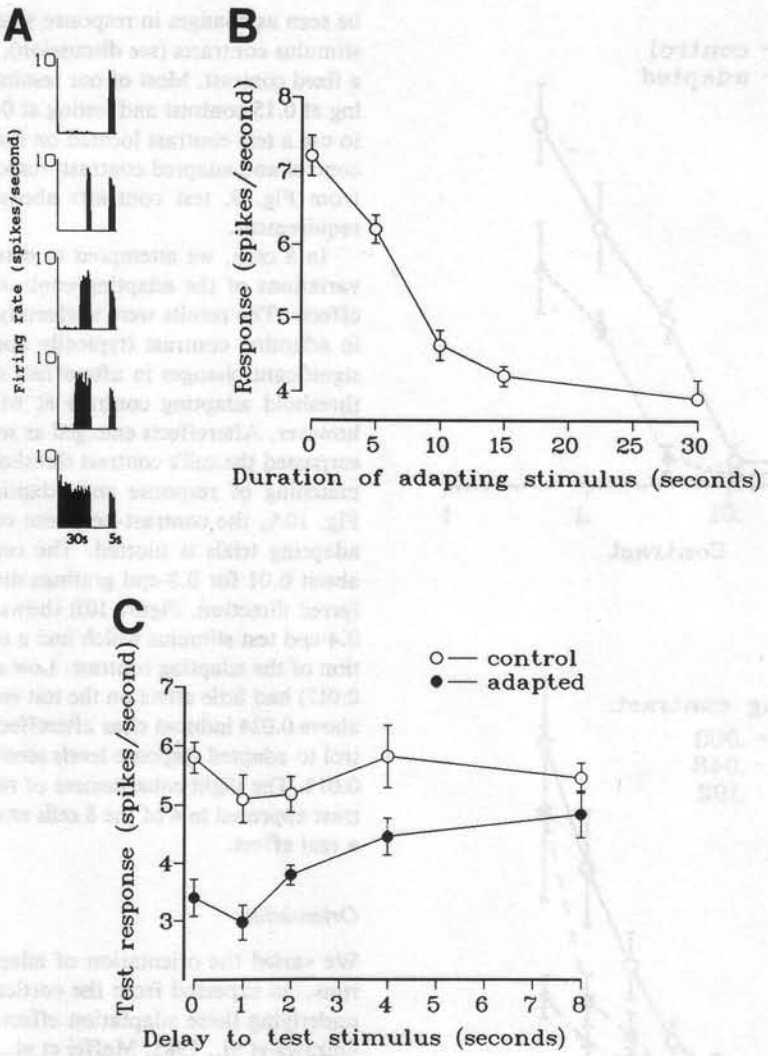

Fig. 8. The time course of adaptation was explored in this complex cell. A, adapting gratings (0.15 contrast, 0.5 cpd, 1.4 Hz) were presented for the last 0, 5, 10, 15, or 30 s of 30-s adapting trials. All test trials were identical: they lasted 5 s, and had a contrast of 0.075, a spatial frequency of 0.5 cpd, and a drift rate of 1.4 Hz in the preferred direction. Each adapting/test pair was presented 20 times. Peristimulus time histograms are shown for the 5 adapting conditions and for the subsequent test condition. Test trials began 100 ms after the end of the preceding adapting trial. Histograms are separated for clarity. B, the mean firing rate of the test responses in A is plotted against the duration of prior adapting stimulation. Error bars represent plus and minus one standard error. C, in another run on this same cell, 15-s adapting trials were used, while the time delay before the onset of the test grating was varied. Adapting trials had contrasts of either 0 (control, open circles) or 0.15 (adapted, filled circles). Test contrast was 0.075. Test gratings did not appear for the first 0, 1, 2, 4, or 8 s of the 13-s test trials. The test grating was always present for 5 s, after which the contrast was returned to zero for the remainder of the trial.

chophysical studies generally employ low contrasts. By testing at low contrasts, low adapting contrasts could be used to produce significant aftereffects.

We varied the contrast of the sinusoidal grating stimuli between 0 and 0.30, while fixing the mean luminance at about 4 cd/m^2. At the contrasts used, cells showed no evidence of response saturation, but responded well to optimal stimuli. In many runs, both adapting and test contrasts were varied. The data were then plotted as separate contrast–response curves for each adapting contrast (Fig. 9).

As previously described (Dean, 1983; Albrecht et al., 1984; Ohzawa et al., 1985), contrast–response functions were shifted laterally and sometimes rotated by adapting at high contrasts. Figure 9 shows examples of these effects. The entire curve was shifted along the contrast axis in Fig. 9A, so that the contrast threshold was elevated and the response to any given contrast was diminished by a fixed amount. The open circles and solid line represent the responses following low contrast (0.003) adapting stimulation. The control contrast threshold lies at about 0.01. The responses following adapting at a contrast of 0.096 are illustrated by the filled triangles and dashed line. The adapted contrast threshold was about 0.03. The adapted contrast–response curve was shifted about one-half log unit to the right of the control curve, or about 5 spikes/s downward. The slopes of the control and adapted curves are similar, however.

Some neurons showed changes in the slope of the contrast–

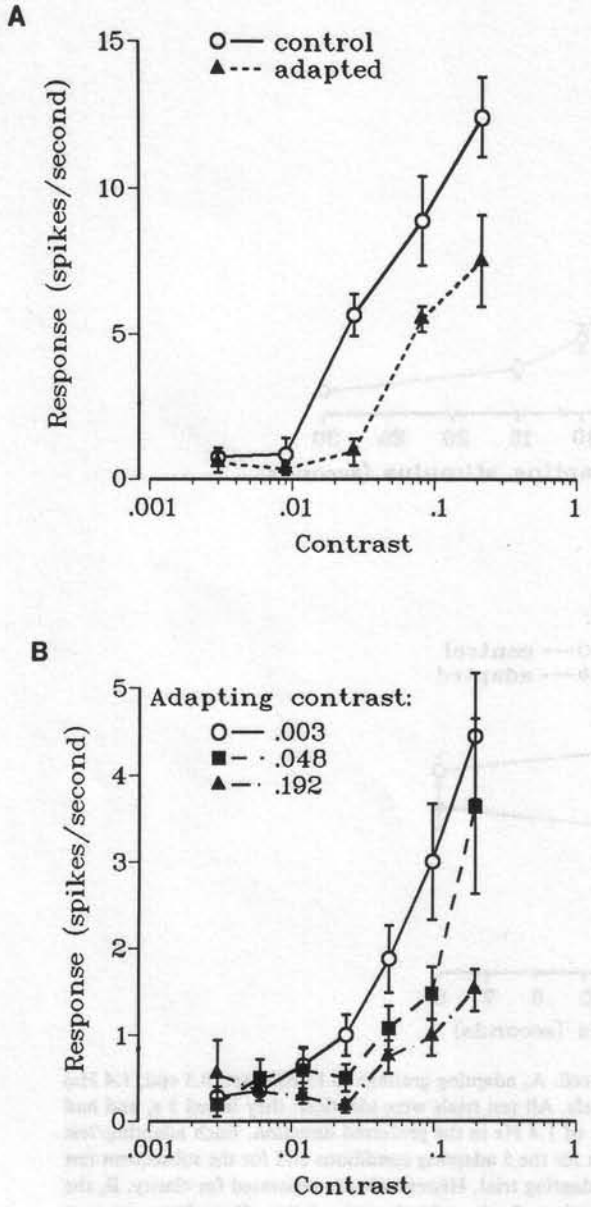

Fig. 9. Contrast-response functions from a simple cell. A, the 30-s adapting trials had contrasts of either 0.003 (control) or 0.096 (adapted). All stimuli had spatial and temporal frequencies of 0.225 cpd and 2.4 Hz. B, in this run, the adapting contrast varied among 0.003, 0.048, and 0.192. The responses plotted are derived from 8–24 trials pooled across spatial frequencies from 0.075–0.375 cpd. Adapting stimuli had a spatial frequency of 0.15 cpd, and lasted 30 s.

response function (i.e. the contrast gain) at suprathreshold contrasts. When the adapting contrast was low, responses to high contrasts were not diminished in some cases (Fig. 9B, filled squares). In other words, in these cases responses were only affected around threshold contrast, or alternatively, since the adapting contrast was near threshold, the adaptation effects were specific to the adapting contrast. At higher adapting contrasts, a downward rotation of the contrast-response curve was often seen (Fig. 9B, filled triangles). Contrast sensitivity and contrast gain were both reduced in these cases.

Because the effects of adaptation on contrast sensitivity can be seen as changes in response when one chooses appropriate stimulus contrasts (see discussion), we generally tested cells at a fixed contrast. Most of our results were obtained from adapting at 0.15 contrast and testing at 0.075 contrast. The goal was to use a test contrast located on the linear portion of both the control and adapted contrast-response curves. As can be seen from Fig. 9, test contrasts above about 0.04 satisfied this requirement.

In 8 cells, we attempted to determine the effects of small variations of the adapting contrast on the strength of aftereffects. The results were uniformly noisy. Only large changes in adapting contrast (typically about 0.5 log units) revealed significant changes in aftereffect strength. We estimated the threshold adapting contrast at which aftereffects appeared, however. Aftereffects emerged as soon as the adapting contrast surpassed the cell's contrast threshold. Figure 10 illustrates this matching of response and adapting contrast thresholds. In Fig. 10A, the contrast-response curve derived from the 15-s adapting trials is plotted. The cell's contrast threshold was about 0.01 for 0.8-cpd gratings drifting at 1.8 Hz in the preferred direction. Figure 10B shows the responses evoked by a 0.4-cpd test stimulus which had a contrast of 0.024, as a function of the adapting contrast. Low adapting contrasts (less than 0.012) had little effect on the test responses. Adapting contrasts above 0.024 induced clear aftereffects. The transition from control to adapted response levels seemed to occur at a contrast of 0.012. The slight enhancement of responses near threshold contrast appeared in 4 of the 8 cells examined, and could represent a real effect.

Orientation

We varied the orientation of adapting and test gratings in 7 runs. As expected from the cortical locus of the mechanisms underlying these adaptation effects (Vautin & Berkley, 1977; Ohzawa et al., 1985; Maffei et al., 1986; Marlin et al., 1986), orientation-specific aftereffects were always observed. An example is presented in Fig. 11. The responses to test gratings presented at four orientations are plotted in Fig. 11A. The 0- and 180-deg points are the same, and represent the preferred orientation. Adapting at the preferred orientation induced a strong response suppression (filled circles), whereas adapting 90 deg away (open squares) had little effect. We show the *t*-scores obtained from comparing control and adapted curves in Fig. 11B. Adapting at the preferred orientation resulted in large *t*-values, especially when testing the preferred orientation. On the other hand, adapting at the orthogonal orientation resulted in *t*-scores less than 2.0, except at the adapted, nonpreferred orientation. This suggests that aftereffects matched the adapting orientation. We also adapted at 45 deg and 135 deg in this run (not shown). The aftereffects were weak and did not exhibit clearly systematic tuning, although they were strongest at the adapting orientations.

Masking

Masking the excitatory receptive field and stimulating only the surround during adapting trials led to mixed results (Maffei et al., 1973; Ohzawa et al., 1985). In some cases, masking while adapting seemed equivalent to zero-contrast adapting. That is, the presence of the mask rendered the stimulus ineffective in inducing adaptation aftereffects. In some runs, aftereffects

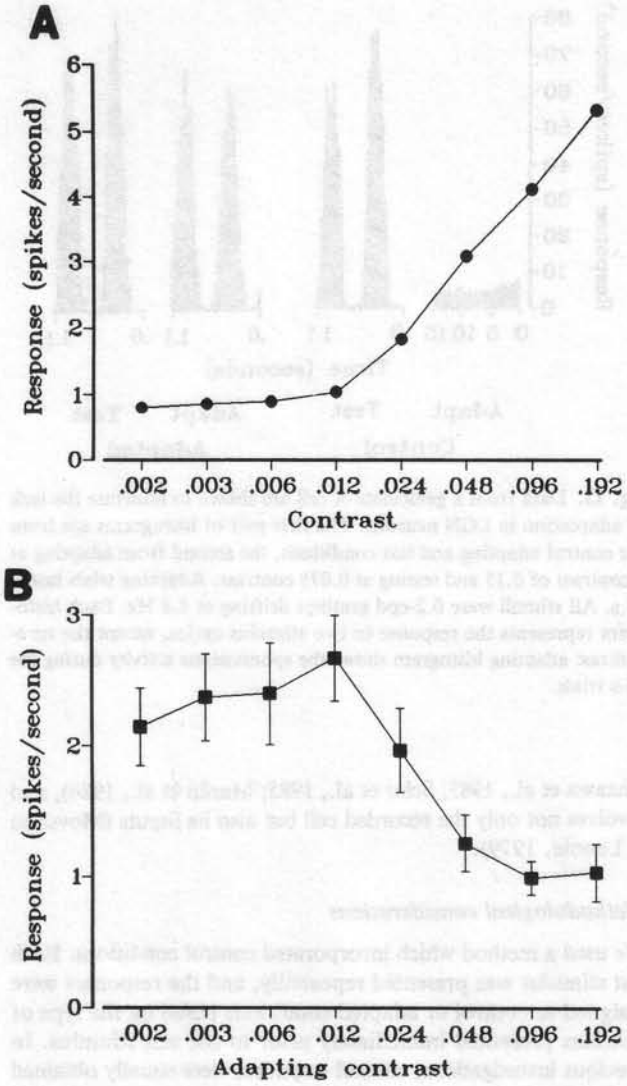

Fig. 10. Results of an experiment on a simple cell in which adapting contrast was varied in octave steps. A, the responses evoked by the adapting stimuli themselves show that the contrast threshold was about 0.01 for the 0.8-cpd adapting stimulus. B, the responses to a 0.4-cpd test stimulus are shown as a function of the contrast of the preceding adapting stimulus.

appeared despite effective masking (effective meaning evoked activity was abolished during the adapting trials). Figure 12 is an example from a simple cell. Masking the field eliminated the responses evoked by the adapting grating, but test responses were nonetheless smaller than in the control condition. Compared to the aftereffects seen without masking, however, adapting the surround alone had little effect.

Subcortical recordings

We have sampled only a few geniculate cells, but in contrast to our finding that every cortical cell adapts to the stimulation used in these experiments, no geniculate adaptation has been seen. An example is shown in Fig. 13. The response of this LGN X cell was unaffected by adapting with high-contrast gratings of any of five spatial frequencies used. This provides

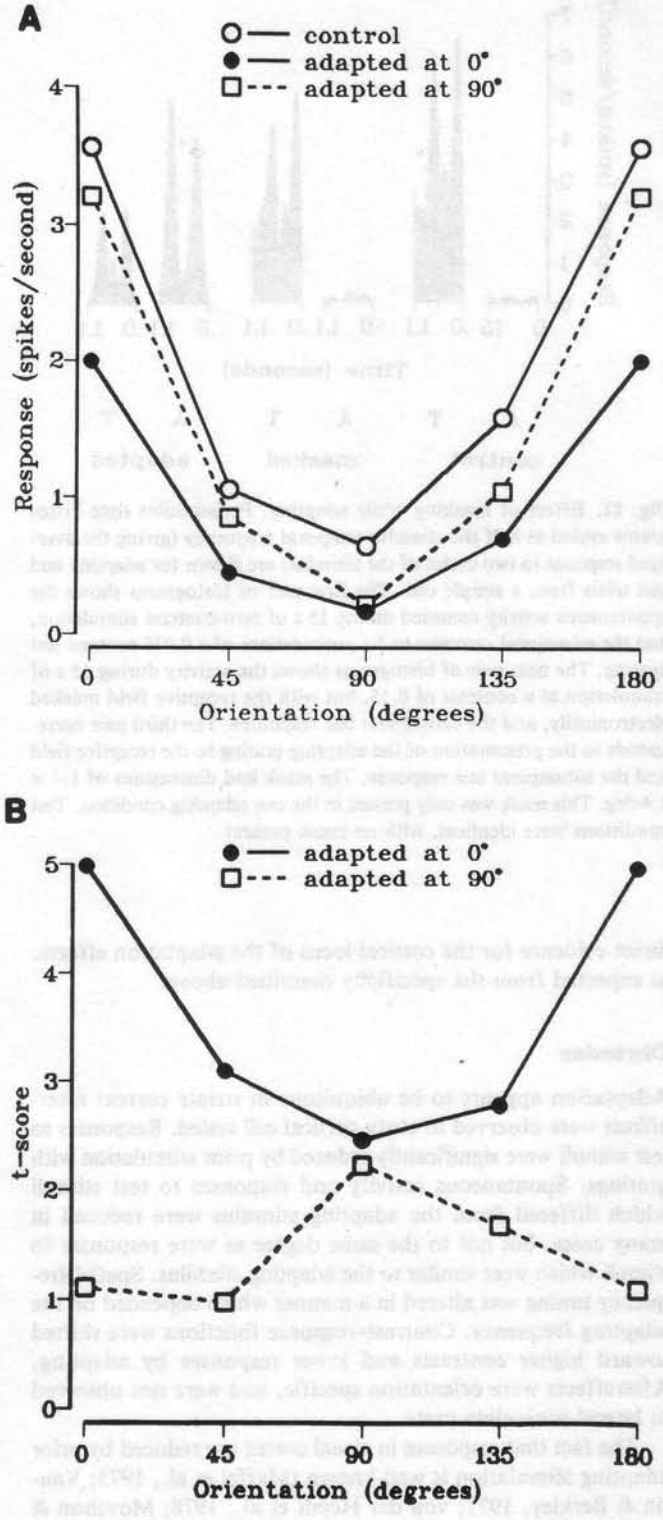

Fig. 11. Orientation tuning curves from a complex cell. A, responses in the two directions at each orientation have been combined, as have the four spatial frequencies tested (0.3–1.0 cpd) and the two adapting spatial frequencies (0.5 and 0.8 cpd). The 180-deg point is identical to the 0-deg data. Adapting trials consisted of 13 s of stimulation with gratings of 0.15 contrast (plus 1 s before and after at zero contrast). Test trials also had 1-s periods at zero contrast before and after 3 s of stimulation at 0.045 contrast. These 1-s periods of blank screen were inserted to eliminate artifacts from the process of redrawing the gratings. B, the *t*-score data obtained from comparing each of the adapted tuning curves to the control tuning curve are plotted.

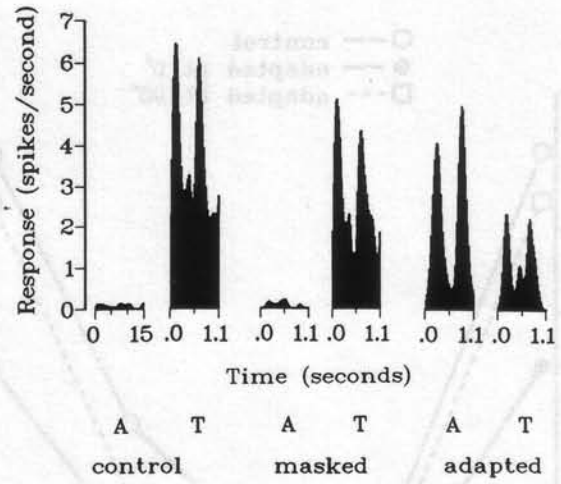

Fig. 12. Effect of masking while adapting. Peristimulus time histograms cycled at half the stimulus temporal frequency (giving the averaged response to two cycles of the stimulus) are shown for adapting and test trials from a simple cell. The first pair of histograms shows the spontaneous activity recorded during 15 s of zero-contrast stimulation, and the subsequent response to 5-s presentations of a 0.075 contrast test grating. The next pair of histograms shows the activity during 15 s of stimulation at a contrast of 0.15, but with the receptive field masked electronically, and the subsequent test responses. The third pair corresponds to the presentation of the adapting grating to the receptive field and the subsequent test response. The mask had dimensions of 1.4 × 1.4-deg. This mask was only present in the one adapting condition. Test conditions were identical, with no mask present.

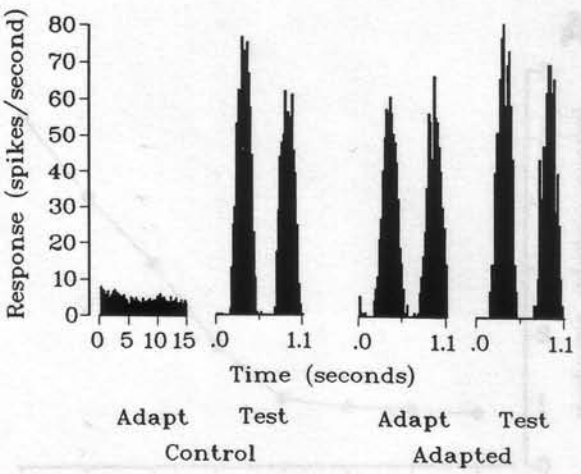

Fig. 13. Data from a geniculate X cell are shown to illustrate the lack of adaptation in LGN neurons. The first pair of histograms are from the control adapting and test conditions, the second from adapting at a contrast of 0.15 and testing at 0.075 contrast. Adapting trials lasted 15 s. All stimuli were 0.2-cpd gratings drifting at 1.8 Hz. Each histogram represents the response to two stimulus cycles, except the zero-contrast adapting histogram shows the spontaneous activity during the 15-s trials.

direct evidence for the cortical locus of the adaptation effects, as expected from the specificity described above.

Discussion

Adaptation appears to be ubiquitous in striate cortex: aftereffects were observed in every cortical cell tested. Responses to test stimuli were significantly reduced by prior stimulation with gratings. Spontaneous activity and responses to test stimuli which differed from the adapting stimulus were reduced in many cases, but not to the same degree as were responses to stimuli which were similar to the adapting stimulus. Spatial-frequency tuning was altered in a manner which depended on the adapting frequency. Contrast–response functions were shifted toward higher contrasts and lower responses by adapting. Aftereffects were orientation specific, and were not observed in lateral geniculate units.

The fact that responses in visual cortex are reduced by prior adapting stimulation is well-known (Maffei et al., 1973; Vautin & Berkley, 1977; von der Heydt et al., 1978; Movshon & Lennie, 1979; Movshon et al., 1980; Ohzawa et al., 1982; Dean, 1983; Albrecht et al., 1984; Ohzawa et al., 1985; Sclar et al., 1985; Saul & Daniels, 1985; Hammond et al., 1985; Hammond et al., 1986; Maffei et al., 1986; Marlin et al., 1988). Our investigation of this phenomenon provides more extensive data about the spatial-frequency tuning of aftereffects, and reinforces several proposals relevant to understanding the mechanisms of adaptation. First, as found by Vautin and Berkley (1977), adaptation affects every neuron. Second, the locus of adaptation lies in visual cortex (Maffei et al., 1973, 1986; Ohzawa et al., 1985; Sclar et al., 1985; Marlin et al., 1986), and involves not only the recorded cell but also its inputs (Movshon & Lennie, 1979).

Methodological considerations

We used a method which incorporated control conditions. Each test stimulus was presented repeatedly, and the responses were assigned to control or adapted conditions based on the type of stimulus presented immediately prior to the test stimulus. In previous investigations, control responses were usually obtained prior to adapting, with retests after some recovery period to ensure that the changes seen after adapting could be associated with the adapting stimulation. Our control trials were instead completely interleaved with the adapted trials.

Previous descriptions of aftereffects (Movshon & Lennie, 1979; Hammond et al., 1985; Marlin et al., 1986) have often relied on responses to the onset of test stimulation. We took care to ascertain that the observed aftereffects did not only occur in a transient fashion, for instance as an artifact of a change in stimulus presentation. Response levels were always estimated over a 5-s duration. Responses were clearly reduced throughout the 5-s test trials. The data were reanalyzed ignoring the first 250, 500, or 1000 ms after stimulus onset. The aftereffects were always as profound during the last few seconds of the test trials as during the initial portion. A delay of 100 ms was always present prior to test trials. A further delay of 1–2 s did not reduce aftereffects following 15 s of adapting.

Investigating the tuning of adaptation requires the presentation of many stimuli. The variability of cortical cells during physiological recording presents a danger that spontaneous variability could be interpreted as changes induced by adaptation. Interleaving stimuli reduces this danger considerably. Since all of the changes we observed were in the direction of reduced responses following adapting with moving gratings, random variability cannot account for the results.

Aftereffects were observable in all cells. Some investigators have reported that some cells fail to adapt (Maffei et al., 1973, 1986; Ohzawa et al., 1985; Marlin et al., 1988). The low contrasts used in this study probably contribute to the universality of aftereffects, since aftereffects could be very small at higher test contrasts. Adaptation had strong effects on neuronal responses near threshold test contrasts, however. This physiological result may reflect the psychophysical observation that adaptation degrades detection (i.e. threshold) but not discrimination (i.e. suprathreshold) performance (Wilson & Regan, 1984; Regan & Beverly, 1985).

Some studies have emphasized that the effects of adaptation should be examined in terms of contrast sensitivity, rather than response reduction (Movshon & Lennie, 1979; Albrecht et al., 1984). Consistent objective measurements of contrast thresholds proved far more difficult than the alternative of measuring simple response levels, which we therefore adopted. By appropriate choices of contrast levels, one should be able to transform from contrast to response. By adapting at a contrast of 0.15 and testing at 0.075 contrast, for example, we were confident that most stimuli presented would be above threshold and below saturation in both the unadapted and adapted states. The linearity of contrast–response functions at the contrasts used implies that aftereffects measured by response magnitudes reflect changes in contrast sensitivity and contrast gain. This is because linear functions are described by two parameters, slope (gain) and intercept (sensitivity). When only responses at a fixed contrast are measured, sensitivity and gain changes are unfortunately confounded. We confirmed that adaptation always increases thresholds, and generally lowers gains (Dean, 1983; Albrecht et al., 1984).

Dealy and Tolhurst (1974) showed that psychophysical contrast thresholds for the emergence of aftereffects matched the detection threshold for the adapting stimulus. This result was used to argue for an inhibitory mechanism underlying adaptation. We looked at the magnitude of aftereffects as a function of adapting contrast. Although the effects were unreliable at low contrasts, extrapolation showed that the threshold for the emergence of adaptation aftereffects was the contrast threshold of the cell for responding to the adapting grating.

Rather than adapting at a fixed level of absolute contrast, it may be more appropriate to adapt at contrast levels which are some fixed multiple of the threshold contrast for the adapting stimulus (Keck et al., 1976). For example, if a cell has a higher contrast threshold for one direction of stimulus motion than the other, when adapting in the nonpreferred direction one should adapt at a higher contrast. This would have the effect of making the responses to the adapting stimuli similar. We prefer again, for practical reasons, to compare adapting stimuli in terms of the responses they evoke, rather than in terms of equivalent contrasts. Adapting stimuli can evoke equivalent responses yet induce distinctly tuned aftereffects, as was shown in Fig. 2.

Adaptation in the contrast and spatial-frequency domains

Contrast–response functions were shifted toward higher contrasts, and sometimes rotated downwards, by adapting at high contrasts. Such changes to contrast sensitivity and contrast gain have been observed repeatedly in adaptation experiments (Ohzawa et al., 1982, 1985; Dean, 1983; Albrecht et al., 1984). One interpretation of the downward rotation is that adaptation produces a divisive scaling, or a decrease in contrast gain, as opposed to a subtractive scaling of contrast–response (Dean, 1983). Alternatively, adaptation can be seen as a rescaling of the contrast axis, because of the fact that contrast and response are monotonically related. Contrast adaptation has been described as contrast gain control (Ohzawa et al., 1985), with the implication being that neurons regulate their responses to match their dynamic range to the domain of ambient contrasts. That is, the contrast axis is rescaled to reflect the recent history of stimulus contrasts. This same process of regulation might occur in the spatial-frequency domain. Because spatial-frequency tuning is not monotonic, the rescaling is slightly more complicated. However, as long as tuning curves remain inverted U-shaped, they can be interconverted by rescaling the frequency axis. If spatial-frequency tuning curves developed a notch from adapting with a sine wave grating, one could not explain the effect in terms of rescaling of the frequency axis. We did not observe such narrowly tuned aftereffects, however, and our observations are therefore consistent with a mechanism which acted to rescale the spatial-frequency axis.

Adaptation is primarily interesting because of its selectivity. Adapting at low contrasts sometimes affected the responses at low contrasts only. This suggests that changes in the contrast domain are selective effects. The evidence in the frequency domain for selective effects is much clearer. Although the spatial-frequency tuning of aftereffects was usually fairly broad, this tuning shifted with the adapting frequency, as first shown by Movshon and Lennie (1979). We were not able to characterize the spatial-frequency tuning of adaptation in single units to the extent that pyschophysical tuning has been described (Pantle & Sekuler, 1968; Blakemore & Campbell, 1969; Legge, 1976), but several generalizations can be stated. The strongest aftereffects occurred at the adapting and optimal frequencies. Aftereffects were stronger at lower frequencies than at higher frequencies, both absolutely and relative to the adapting frequency. When the adapting frequency exceeded the cell's optimal frequency, the peak spatial frequency could shift toward lower frequencies. In a few cases, bandpass tuning of aftereffects was observed in a narrow range around the adapting frequency. The tuning of aftereffects averaged over our sample (Fig. 5) appears to be broader than psychophysical threshold elevation curves (cf. Fig. 8 in Blakemore & Campbell, 1969). Comparing the two sets of results requires a number of uncertain hypotheses, however. Note also that aftereffects can be demonstrated in single units only over each cell's narrowband response range. Psychophysical aftereffects can be demonstrated from low frequencies up to the acuity limit.

Intrinsic vs extrinsic sources of adaptation effects

Vautin and Berkley (1977) discussed how adaptation aftereffects could arise from either processes in the recorded cell itself or from events occurring in other cells which influenced the recorded cell. They concluded that while intrinsic factors are important, extrinsic factors played a significant role. The spatial-frequency tuning of aftereffects provides further evidence supporting this view. Although spatial-frequency-specific adaptation aftereffects have been reported (Maffei et al., 1973; Movshon & Lennie, 1979; Albrecht et al., 1984; Hammond et al., 1985), these earlier investigations did not examine a range of adapting and test frequencies. Specificity of aftereffects

could be inherited from the cell's own tuning, and intrinsic mechanisms such as fatigue could induce narrowly tuned aftereffects which matched the cell's own tuning, independent of the adapting frequency. We varied the adapting frequency across the response range of the cells. The results of our more extensive testing demonstrate that the tuning of aftereffects follows the adapting frequency. Although there is a substantial dependence on the cell's own tuning, the existence of a clear dependence on the adapting stimulus means that mechanisms extrinsic to the recorded cell are involved in adaptation. These extrinsic mechanisms are simply the inputs to the cell.

Although we attempted to assess the relative importance of the adapting *vs* the optimal stimulus in determining the strength of aftereffects, the fact that each factor plays a role makes a clean separation difficult. At low adapting frequencies, aftereffects clearly matched the adapting frequency. At high adapting frequencies this match no longer held. However, the fact that low test frequencies were more sensitive to adaptation than high test frequencies suggests that aftereffects might depend primarily on the adapting stimulus even at frequencies above the optimum. Tuning of aftereffects seemed to be linked more tightly to the adapting frequency than to the optimal frequency. However, the interaction of the optimal and the adapting frequency determines the final tuning of aftereffects.

We have confirmed that the locus of adaptation does not lie outside of the cortex, by observing that aftereffects are orientation specific, and by failing to observe adaptation in geniculate neurons (Maffei et al., 1973; Vautin & Berkley, 1977; Movshon & Lennie, 1979; Ohzawa et al., 1985). However, these results do not rule out the possibility that adaptation alters transmission at the geniculocortical synapse. Other studies have additionally used the interocular transfer of adaptation aftereffects to infer that the mechanism involves cortical factors (Maffei et al., 1973; 1986; Sclar et al., 1985; Marlin et al., 1986). Adapting one eye can induce aftereffects when tested through the other eye, despite the fact that the geniculate inputs to cortex are monocular. These several lines of evidence implicate intracortical inputs in adaptation effects.

Intrinsic factors are evident in our data. The response properties of the neuron play an important role in the tuning of adaptation effects. Optimal stimuli induced the strongest aftereffects, and adapting stimuli which did not excite the cell seldom induced any aftereffect. Masking the receptive field frequently abolished adaptation aftereffects. We found that complex cells preferring lower spatial frequencies tended to lose their responsiveness to low frequencies following adapting with gratings of moderate frequencies. We also noted that the adapting contrast at which aftereffects first appeared seemed to match the contrast threshold for driving the cell. Each cell therefore plays a role in adaptation, rather than being passively influenced by events occurring in its inputs.

Nonetheless, we emphasize the evidence for extrinsic factors. Although a correlation exists between the number of spikes evoked by the adapting stimulus and the strength of the aftereffects, this correlation was far from perfect. Adapting stimuli which excited the cell equally could induce quite distinct aftereffects. Masking the excitatory receptive field and adapting only the surround, so that no activity was evoked by the adapting stimulus, could, at least on occasion, induce aftereffects. High spatial frequencies which did not drive the cell strongly seemed to induce stronger aftereffects than did lower frequencies which evoked similar responses. Most importantly, the tuning of aftereffects matched the adapting stimulus. An interaction between a cell and its inputs must therefore determine the effects of adaptation.

A fatigue hypothesis may be consistent with these results. The observed extrinsic source of adaptation effects could consist of the reduced activity in excitatory inputs from other fatigued cortical cells. The results presented in this paper constrain such a fatigue model: one must account for the observed specificity; fatigue must be a universal property of cortical, but not subcortical neurons, and must become evident even at low adapting contrasts; and the bias toward stronger effects at lower spatial frequencies must be explained.

We discuss models of adaptation further in the accompanying paper, where we incorporate our results on adaptation in the temporal frequency and direction domains. The fatigue model fails to predict the interaction between adaptation and direction selectivity. An alternative model is described which relies on mutually inhibitory interactions between cortical cells.

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References

Albrecht, D.G., Farrar, S.B. & Hamilton, D.B. (1984). Spatial contrast adaptation characteristics of neurons recorded in the cat's visual cortex. *Journal of Physiology* **347**, 713–739.

Blakemore, C. & Campbell, F.W. (1969). On the existence of neurons in the human visual system selectively sensitive to the orientation and size of retinal images. *Journal of Physiology* **203**, 237–260.

Dealy, R.S. & Tolhurst, D.J. (1974). Is spatial adaptation an aftereffect of prolonged inhibition? *Journal of Physiology* **241**, 261–270.

Dean, A.F. (1981). The relationship between response amplitude and contrast for cat striate cortical neurones. *Journal of Physiology* **318**, 413–427.

Dean, A.F. (1983). Adaptation-induced alteration of the relation between response amplitude and contrast in cat striate cortical neurones. *Vision Research* **23**, 249–256.

Hammond, P., Mouat, G.S.V. & Smith, A.T. (1985). Motion aftereffects in cat striate cortex elicited by moving gratings. *Experimental Brain Research* **60**, 411–416.

Hammond, P., Mouat, G.S.V. & Smith, A.T. (1986). Motion aftereffects in cat striate cortex elicited by moving texture. *Vision Research* **26**, 1055–1060.

Keck, M.J., Palella, T.D. & Pantle, A. (1976). Motion aftereffect as a function of the contrast of sinusoidal gratings. *Vision Research* **16**, 187–191.

Legge, G.E. (1976). Adaptation to a spatial impulse: implications for Fourier transform models of visual processing. *Vision Research* **16**, 1407–1418.

Maffei, L., Fiorentini, A. & Bisti, S. (1973). Neural correlates of perceptual adaptation to gratings. *Science* **182**, 1036–1039.

Maffei, L., Berardi, N. & Bisti, S. (1986). Interocular transfer of adaptation aftereffect in neurons of area 17 and 18 of split chiasm cats. *Journal of Neurophysiology* **55**, 966–976.

Marlin, S.G., Hasan, S.J. & Cynader, M.S. (1986). Interocular transfer of direction-selective adaptation in cat striate cortex cells. *Society for Neuroscience Abstracts* **12**, 433.

Marlin, S.G., Hasan, S.J. & Cynader, M.S. (1988). Direction-selective adaptation in simple and complex cells in cat striate cortex. *Journal of Neurophysiology* **59**, 1314–1330.

Movshon, J.A. & Lennie, P. (1979). Pattern-selective adaptation in visual cortical neurones. *Nature* **278**, 850–852.

MOVSHON, J.A., BONDS, A.B. & LENNIE, P. (1980). Pattern adaptation in striate cortical neurons. *Investigative Ophthalmology and Visual Science* (Suppl.) **21**, 193.

OHZAWA, I., SCLAR, G. & FREEMAN, R.D. (1982). Contrast gain control in the cat visual cortex. *Nature* **298**, 266–268.

OHZAWA, I., SCLAR, G. & FREEMAN, R.D. (1985). Contrast gain control in the cat's visual system. *Journal of Neurophysiology* **54**, 651–667.

PANTLE, A. & SEKULER, R. (1968). Size-detecting mechanisms in human vision. *Science* **162**, 1146–1148.

REGAN, D. & BEVERLY, K.I. (1985). Postadaptation orientation discrimination. *Journal of the Optical Society of America A* **2**, 147–155.

SAUL, A.B. & DANIELS, J.D. (1985). Adaptation effects from conditioning area 17 cortical units in kittens during physiological recording. *Society for Neuroscience Abstracts* **11**, 461.

SAUL, A.B. & CYNADER, M.S. (1989). Adaptation in single units in visual cortex: the tuning of aftereffects in the temporal domain. *Visual Neuroscience* **2**, 609–620.

SCLAR, G., OHZAWA, I. & FREEMAN, R.D. (1985). Contrast gain control in the kitten's visual system. *Journal of Neurophysiology* **54**, 668–675.

VAUTIN, R.G. & BERKLEY, M.A. (1977). Responses of single cells in cat visual cortex to prolonged stimulus movement: neural correlates of visual aftereffects. *Journal of Neurophysiology* **40**, 1051–1065.

VON DER HEYDT, R., HÄNNY, P. & ADORJANI, C. (1978). Movement aftereffects in the visual cortex. *Archives of Italian Biology* **116**, 248–254.

WILSON, H.R. & REGAN, D. (1984). Spatial-frequency adaptation and grating discrimination: predictions of a line-element model. *Journal of the Optical Society of America A* **1**, 1091–1096.