Contrast and Temporal Frequency-Related Adaptation in the Pretectal Nucleus of the Optic Tract

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In mammals, many cells in the retina-geniculate-cortical pathway adapt during stimulation with high contrast gratings. In the visual cortex, adaptation to high contrast images reduces sensitivity at low contrasts while only moderately affecting sensitivity at high contrasts, thus generating rightward shifts in the contrast response functions (contrast gain control). Similarly, motion adaptation at particular temporal frequencies (TFs) alters the temporal tuning properties of cortical cells. For the first time in any species, this paper investigates the influence of motion adaptation on both the contrast and TF responses of neurons in the retina-pretectal pathway by recording from direction-selective neurons in the nucleus of the optic tract (NOT) of the marsupial wallaby, Macropus eugenii. This species is of interest because its NOT receives almost all input directly from the retina, with virtually none from the visual cortex (unlike cats and primates). All NOT cells show changes in their contrast response functions after adaptation, many revealing contrast gain control. Contrast adaptation is direction-dependent, preferred directions producing the largest changes. The lack of cortical input suggests that contrast adaptation is generated independently from the cortex in the NOT or retina. Motion adaptation also produces direction-selective effects on the TF tuning of NOT neurons by shifting the location of the optimal TF. Cells that show strong adaptation to contrast also tend to show large changes in TF tuning, suggesting similar intracellular mechanisms. The data are discussed in terms of the generality of contrast adaptation across mammalian species and across unconnected brain regions within the same species.

INTRODUCTION

This paper shows that exposure to moving grating patterns alters the contrast and temporal frequency response functions of highly direction-selective neurons in the pretectal nucleus of the optic tract (NOT) of the marsupial wallaby, Macropus eugenii. These effects resemble those seen in the visual cortex of other species (e.g., cats, Ohzawa et al. 1985). The wallaby pretectum is a retina-recipient region of which the NOT forms a large component (Ibbotson et al. 2002). The wallaby pretectum also contains other nuclei such as the olivary pretectal nucleus, as is the case in eutherian mammals (e.g., Hutchins and Weber 1985; for review, Ibbotson and Dreher 2005). The wallaby NOT contains direction-selective neurons that are optimally responsive to wide-field visual motion, as naturally generated by self-movement. Stimulation and lesion studies have confirmed that the NOT has an essential role in driving horizontal optokinetic nystagmus (OKN) in eutherian mammals (e.g., Collewijn 1975b; Schiff et al. 1988), while evidence from eye rotation and electrophysiological experiments point toward a similar role in the wallaby (Hoffmann et al. 1995). Recordings have been made from the NOT in marsupials (opossum: Volchan et al. 1989; wallaby: Hoffmann et al. 1995; Ibbotson et al. 1994) and in several eutherian species (e.g., cat: Hoffmann and Schoppmann 1981; ferret: Klauer et al. 1990; monkey: Hoffmann et al. 1988; Iig and Hoffmann 1996; Mustari and Fuchs 1990; rabbit: Collewijn 1975a). The NOT has a highly conserved physiology despite significant differences in input structure between species.

In marsupials, direct retinal inputs arise almost exclusively from the contralateral eye (opossum: Vargas et al. 1998; wallaby: Ibbotson et al. 2002), and there is little if any input from the visual cortex (Ibbotson et al. 2002; Pereira et al. 2000). In monkeys, retinal input comes from both eyes (Telkes et al. 2000), and there is a very large input from the visual cortex, which is ∼10-fold larger than the retinal input (Distler et al. 2002; Iig and Hoffmann 1993). The visual cortex also provides a major input to the NOT in cats (Schoppmann 1981). This paper shows that contrast adaptation occurs in the wallaby NOT, but the connectivity suggests that the effects most likely arise from retinal circuitry. This is of interest because much speculation has previously suggested that contrast adaptation is a solely cortical phenomenon (Maffei et al. 1973; Movshon and Lennie 1979; Ohzawa et al. 1985). However, recent comparative studies in both primates and salamanders have shown that contrast adaptation may, at least partially, arise in the retina (e.g., Baccus and Meister 2002; Chander and Chichilnisky 2001). Evidence also suggests that adaptive effects can be observed in other subcortical brain structures, such as the macaque dorsal lateral geniculate nucleus (Solomon et al. 2004). This work provides further comparative data suggesting that contrast adaptation is not limited only to the cortex.

Studies have revealed that contrast adaptation may have a beneficial effect on visual processing by shifting the contrast response functions of cells such that they are maximally sensitive to contrast changes close to the prevailing contrast in the environment (e.g., Ohzawa et al. 1985). This mechanism is thought to provide an important function for the visual system because it allows the system to operate optimally in a wide range of visual environments (Ibbotson 2005). It is likely that contrast adaptation at the inputs to motion detectors allows them to function more effectively, and this could explain adaptive effects observed in the highly motion-selective NOT. It has also been shown that adapting to specific temporal frequencies of motion can adjust the tuning properties of cat
cortical cells (Saul and Cynader 1989a,b) and some motion-sensitive insect neurons (Maddess and Laughlin 1985). In the insect, this mechanism was argued to have beneficial effects on the coding of image speed (also see, Clifford et al. 1997; for a review, Ibbotson 2005).

This work is the first to study adaptation to both contrast and temporal frequency (TF) in a noncortical visual pathway, namely the retina–pretectal system. Adaptation in this pathway shows the generality of adaptation in different visual brain areas and suggests that adapting to the prevailing visual environment is important for coding motion signals relevant for eye movement control. Evidence from humans has shown that adaptation to moving gratings can influence the magnitude of subsequent horizontal ocular following responses, which are partially driven by cells in the NOT (Ibbotson and Maddess 1994; Maddess and Ibbotson 1992). The wallaby results presented here may therefore have general implications for processing in mammalian visual systems beyond this specific species.

METH O D S

Recordings were made from seven wallabies (Macropus eugenii); five animals were male. They were prepared for microelectrode recording as described earlier (Ibbotson et al. 1994). Briefly, animals were anesthetized with an intramuscular injection of 30 mg/kg ketamine (Parnell) and 1.5 mg/kg xylazine (Rompun, Bayer). Anesthesia was continued with periodic intravenous injections of thiopentone (Pentothal, Boehringer-Ingelheim) into the tail vein. The wallabies were intubated with a 4-mm endotracheal cannula and placed in a stereotaxic apparatus. Paralysis was initiated by intravenous injection of suxamethonium. Body temperature was maintained at 37°C with an electric blanket. All procedures were approved by the animal ethics committee of the Australian National University and followed the guidelines of the Australian National Health and Medical Research Council.

Recordings were made from single units in the NOT using tungsten-in-glass microelectrodes. Extracellular responses were amplified, passed through a Schmidt trigger, and collected on a computer as spike arrival times. The locations of the recording sites were marked with electrolytic lesions at the bottom of each track using DC current (2 μA for 5 s). Animals were killed with an overdose of pentobarbitone sodium and perfused via the heart with 10% formol saline solution and then 4% paraformaldehyde in saline. The locations of the electrolytic lesions and electrode tracks were evident in all animals, confirming that the recording sites were in the NOT (for examples of NOT anatomy in wallabies, see Ibbotson et al. 1994, 2002).

Visual stimul i

The stimuli were achromatic, luminance modulated, drifting sine-wave gratings presented on a display monitor (CCID7551, Barco Industries) and were generated by a computer-controlled video display driver (AT Vista, True Vision). The refresh rate of the monitor was 97.75 Hz, and each frame contained 480 lines (640 pixels/line). The screen luminance was 45 cd/m². Gratings could be positioned at any orientation and moved back and forth along the spatial frequency vector. The gratings could be moved at TFs between 0.38 and 24.32 Hz. The screen subtended 90 × 67°. All experiments began by identifying the optimal motion direction, approximate center of the receptive field, and the optimum spatial frequency and TFs. Gratings were presented to the contralateral eye in a circular aperture, surrounded by a gray field with mean grating luminance. Stimulus aperture sizes were selected such that moving patterns generated robust responses and clear temporal tuning characteristics without any obvious signs of saturation close to the optimum TF. In most cells, the aperture sizes were large (>30° diameter). However, in a small number of cells, it was necessary to use small apertures of only 10° diameter to prevent response saturation in the control data, i.e., responses that flattened out for a wide range of TFs or contrasts. The aperture size was the only parameter used to reduce saturation; all other stimulus parameters were optimal for each cell.

Adaptation protocol

Response functions were measured by moving the grating for 1 s in the preferred direction. The test sequence consisted of 1 s of motion (control), a 5-s rest period (blank screen), and an adapting motion for 10 s, followed by a short interval of 100 ms where the grating was stationary. The 1-s test stimulus was then presented, followed by a 20-s rest period where the image was a gray field of mean luminance. In some cells, the controls were run in separate trials before and after the adaptation protocol to make sure that changes did not occur in the controls as a result of the adaptation protocol. For these trials, 1-s test periods were interspersed between 5-s rest periods. Adapting gratings moved at one of several constant drift rates. The spatial frequency in most experiments was 0.25 cpd, but for some cells sensitive to very low image speeds, it was necessary to use ≤1.0 cpd to produce optimal response magnitudes. Spatial frequencies were never changed between adapting and test phases. Contrast response functions were measured using the same procedure except that the grating contrast was varied in the test phase rather than the drift rate of the gratings. All contrasts are expressed as Michelson contrasts (Luminance max − Lmin)/max + Lmax), and the tests ranged from 0 to 0.9. The drift rates in the adapting and test phases were the same and were chosen as the optimum based on prior tests. The adapting contrast used in the experiments was usually 0.24, but other values were also used. A least eight trials were run for each adaptation condition. Spontaneous activities were calculated by averaging the firing rates measured in blank-screen periods at intervals during the experiments, always following the 20-s rest periods.

Fitting algorithms

Contrast response functions in both the control and postadaptation conditions were fit with the following sigmoidal function using a least squares fitting algorithm

\[
R = R_{\text{max}} \left(1 + \frac{C^a}{C^a + C_{\text{SO}}}\right) + m
\]

R is the response, \(R_{\text{max}}\) is the maximum response, C is the contrast, \(C_{\text{SO}}\) is the contrast generating a response that is halfway between \(R_{\text{max}}\) and the spontaneous activity, the latter represented by m. A change in \(R_{\text{max}}\) indicates a change in the response gain of a cell, and a change in \(C_{\text{SO}}\) indicates a shift in the contrast response function along the contrast axis. It was found that the value n was little affected by adaptation. Given this finding and following the fitting methods of Kohn and Movshon (2003), the value of n was forced to assume a single value for the adapted and unadapted conditions, which was optimized jointly for both conditions.

The TF response functions were fit using a least squares fitting algorithm with a skewed Gaussian (Priebe et al. 2003)

\[
R = R_{\text{max}} e^{-\frac{(\log(TF_{\text{opt}}))}{\tau}} + m
\]

R is the response at each speed, \(R_{\text{max}}\) is the fitted maximum spiking
amplitude, $TF_{opt}$ is the preferred TF, $c$ is the tuning bandwidth, $d$ is a parameter controlling the skew of the curve, and $s$ is the stimulus TF. The value $m$ is the spontaneous activity. A change in $R_{\text{max}}$ following adaptation is indicative of a reduction in absolute firing rate, whereas a shift in $TF_{opt}$ suggests a change in the position of the entire TF tuning function. Once a curve had been generated, the TF at which the fitted curve crossed the response level halfway between the spontaneous activity and $R_{\text{max}}$ was measured and referred to as $TF_{50}$. The value of $TF_{50}$ indicates any differential shifts in the inclining phase of the TF response function.

For all fits, the $R_{\text{max}}$ value was constrained so that it could not fall outside $\pm 15\%$ of the maximum measured response. It was found that this constraint prevented the $R_{\text{max}}$ value from occurring at contrasts significantly higher than unity or the peak TF ($TF_{opt}$) value occurring at unrealistically high values. The $C_{50}$ values never exceeded unity contrast. $R^2$ values were calculated for all fits, with the $R^2$ values ranging from 0.81 to 0.99 (mean $R^2 = 0.89$, SD = 0.10).

RESULTS

Extracellular recordings were made from 42 single units in the nucleus of the optic tract. These neurons generally have high spontaneous activities usually in the range of 20–50 spikes/s (Price and Ibbotson 2002). Preferred direction motion increases the firing rate, whereas anti-preferred motion inhibits the background firing rate. In this paper, only responses to preferred direction motion are considered, but adapting motion directions were varied. Wallaby NOT neurons can be divided into two categories in the unadapted state: fast cells and slow cells (Ibbotson and Price 2001). Fast cells are optimally stimulated at relatively high TFs but low spatial frequencies and slow cells at low TFs and high spatial frequencies. The dividing line between the two populations occurs at a speed of 4°/s (TF/spatial frequency). The adaptation results are shown separately for fast and slow cells, but there were no obvious differences between the adaptive properties of the two cell types.

Influence of adaptation on contrast responses

Figure 1 shows peristimulus time histograms (PSTHs) obtained from an NOT neuron in a range of control and adapting conditions. In all plots, the mean spontaneous activity is shown as a straight horizontal line through the graph. Increases in spiking rate are shown as bars above the spontaneous rate, whereas decreases below spontaneous are represented by bars below that value. The left column shows responses to image motion in the cell’s preferred direction at five contrasts with no prior adaptation (motion starts at 1 s and ends at 2 s). The right column shows the responses to the same five contrasts, but the test stimulus is preceded by 10 s of image motion in the preferred direction with a grating of $C = 0.24$ (adaptation phase). Only the tail end of the adapting phase of the stimulus is shown in Fig. 1. It is evident that in the control condition the response increases with increasing stimulus contrast. This is also the case in the adapted condition, but responses are greatly attenuated for contrasts $< 0.3$.

Figure 2A shows the mean responses above spontaneous in Fig. 1 plotted as functions of log stimulus contrast. The window used to measure stimulus magnitude was from 100 ms after the test stimulus started moving to 100 ms after it stopped (i.e., 1.1–2.1 s in Fig. 1). For this cell, the five unadapted controls form an approximate straight line on a semi-log plot (Fig. 2A, solid line). However, following adaptation, the responses to low contrasts are attenuated such that the contrast response function appears sigmoidal. Even with no further quantitative analysis, it is clear that the point where the response is 50% of maximum ($C_{50}$) occurs at a far higher contrast following a period of adaptation, whereas the maximum response at $C = 0.6$ has hardly changed.

Figure 2B shows the response of another NOT cell before and after adaptation with a moving grating at $C = 0.24$. This graph presents the responses at 12 tested contrasts and thus reveals the entire contrast response function measured from 0 to 90% contrast. As for all cells, when the spike rate of the neuron was plotted as a function of log-contrast, a sigmoidal contrast response function was evident (Fig. 2B, solid line). Following adaptation with a grating moving in the preferred direction at $C = 0.24$, the cell also shows a sigmoidal contrast response function, but the responses at low contrasts are clearly attenuated (Fig. 2B, dashed line). The data points in these graphs have been fitted with sigmoidal curves (see METHODS). Following adaptation, the $C_{50}$ value is pushed to much higher values (Fig. 2B, dashed line). Note that while responses to low contrasts are clearly attenuated following adaptation, the responses to high contrasts are largely unaffected. In other cells, the response functions following contrast adaptation retained a
similar shape to the control, but there was a general reduction in response magnitude for all contrasts (Fig. 2C).

Scatter plots present the $C_{50}$ values (Fig. 3A) and $R_{\text{max}}$ values (Fig. 3B) before and after adaptation for 26 fast cells (circles) and 16 slow cells (triangles). Each plot shows a diagonal line that represents the equality point where control and postadaptation values are the same. Cells fell along a continuum between those where the shift in $C_{50}$ was large but the reduction in $R_{\text{max}}$ was small and those where the reduction in $R_{\text{max}}$ was large but the shift in $C_{50}$ was small. All cells showed either a change in $C_{50}$, $R_{\text{max}}$, or both. There were no obvious differences between the fast and slow cell populations. It is important to note that the $R_{\text{max}}$ value is only the response component and does not include the spontaneous activity (Fig. 3). Most $R_{\text{max}}$ values were between 20 and 60 spikes/s. Lower values were recorded in some cells where the diameter of the stimulus aperture throughout testing was 10°. Small stimuli were used in these cells because they showed very large increases in response with small increases in stimulus size, such that their responses saturated very easily. Efforts were made to reduce saturation in the controls so that any changes that were observed could be attributed to adaptation rather than to other nonlinearities.

To establish the directional characteristics of the adaptation, tests were conducted in which the adapting grating moved in each cell’s anti-preferred direction but test gratings moved in the preferred direction. Adaptation in the anti-preferred direction always led to significant reductions in each cell’s spontaneous activity. Scatter plots of $C_{50}$ and $R_{\text{max}}$ before and after anti-preferred motion reveal little difference between the two conditions (Fig. 3, C and D). It is concluded that contrast adaptation is highly direction-selective, such that preferred direction motion generates adaptive effects, whereas anti-preferred motion does not produce consistent adaptive effects.

Influence of adaptation on TF responses

The TF response functions of NOT cells were measured for gratings moving at a range of TFs in the preferred direction using the same adapting and test times as those used in the

**FIG. 2.** A: mean responses measured in the time windows shown in Fig. 1. Solid line (circles) shows unadapted responses, and dashed line (triangles) shows responses after adaptation at $C = 0.24$. B and C: unadapted (solid lines, circles) and adapted responses (dashed lines, triangles) to a range of contrasts following adaptation at a contrast of $C = 0.32$ for 2 other neurons. Zero on the ordinate is the mean spontaneous activity of each cell. Data in B and C have been fit with the function described in METHODS. Error bars are SD.

**FIG. 3.** Fitted parameters ($C_{50}$ and $R_{\text{max}}$) measured for control (ordinate) and after adaptation (abscissa) in 42 cells. **Left:** effects for preferred direction adaptation (A: $C_{50}$; B: $R_{\text{max}}$). **Right:** effects for anti-preferred adaptation (C: $C_{50}$; D: $R_{\text{max}}$). In all plots, circles are fast cells ($n = 26$) and triangles are slow cells ($n = 16$). All $R_{\text{max}}$ values are response minus spontaneous activity.
contrast experiments. Spatial frequencies were always held constant during adapting and test phases. Optimum spatial frequencies and TFs were determined in initial tests, and the adapting drift rate was usually chosen to be at or below the optimum TF (for exceptions, see Fig. 8).

Figure 4 shows PSTHs from one neuron (the same cell as in Fig. 1) in the test condition (left column) and after motion adaptation (right column). The time configurations of the plots are identical to those in Fig. 1. The cell responded maximally for motion at 3.2 Hz without prior adaptation. This cell was optimally responsive to a 1-cpd grating, so its optimum speed tuning was 3.2°/s, classifying it as a slow cell. Following adaptation at 3.2 Hz, the responses in the test phase changed (Fig. 4, right column) such that responses were attenuated for low TFs but enhanced for higher TFs (4.8–6.4 Hz). Plotting the responses in the control condition as a function of TF revealed a triangular response function (Fig. 5A, solid line, filled circles). Following adaptation at 3.2 Hz, the response function shifted to higher TFs, the optimum now being 4.8 Hz (Fig. 5A, dashed line, filled triangles).

Examples of the effect of preferred direction adaptation on the TF response functions of two other neurons are shown in Fig. 5, B and C. In the control, the spike rates of the cells increased steadily with increasing TF to the $R_{max}$ after which the spike rate fell away again as TF increased. Following adaptation to drifting gratings moving at or below the peak unadapted TF, the response properties of the neurons fell along a continuum between those in which the attenuation primarily occurred at low TFs (Fig. 5B) or adaptation led to a general attenuation of response gain for all TFs (Fig. 5C). The responses for the cells in Fig. 5, B and C, have been fitted with a skewed Gaussian (see METHODS). In the cells that showed differential adaptation to TF, the combination of attenuated low TF responses, rightward shifts in TF tuning, and small changes in $R_{max}$ often led to rightward shifts in TF.

To quantify these changes for the cell population, the post-adaptation values for $R_{max}$, $TF_{opt}$, and $TF_{50}$ are plotted as functions of the respective control values in the form of scatter
As with the contrast data, the value of $R_{\text{max}}$ is the response component only and does not include the spontaneous activity of the cells. The values for fast and slow cells are shown in Figs. 6 and 7, respectively. For both cell types, values of $R_{\text{max}}$ ranged from 15 to 50 spikes/s (Figs. 6, A and D, and 7, A and D). Values of $R_{\text{max}}$ are the same or lower in the postadaptation condition (i.e., above the line in Figs. 6A and 7A). Values of $T_{\text{Fopt}}$ were either unchanged or showed rightward shifts to higher TFs (i.e., below the line in Figs. 6B and 7B). Changes in $T_{\text{F50}}$ showed quite large variations across the cell population (Figs. 6C and 7C). Many cells showed very little change in $T_{\text{F50}}$, whereas others showed quite large rightward shifts along the TF axis. There were no obvious differences between fast and slow cells.

To establish the directional characteristics of the changes to TF tuning, tests were conducted in which the adapting grating moved in each cell’s anti-preferred direction but test gratings moved in the preferred direction. Scatter plots of $R_{\text{max}}, T_{\text{Fopt}},$ and $T_{\text{F50}}$ before and after adaptation revealed little difference between the two conditions for most cells (Figs. 6, D–F, and 7, D–F). We conclude that adaptation-related changes in TF tuning are highly direction-selective, such that preferred direction motion generates adaptive effects, whereas anti-preferred motion does not produce consistent adaptive effects.

All data up to this point have focused on adaptation at TFs at or below the peak TF tuning (e.g., Fig. 5, B and C, vertical dashed lines). More thorough examinations were conducted on 12 neurons in which the influence of motion adaptation at each cell’s peak TF and at one-half and twice the peak value were measured (e.g., 1.6, 3.2, and 6.4 Hz). To present this data succinctly, ratios were calculated as follows: $R_{\text{max}}$(after adaptation)/$R_{\text{max}}$(before adaptation) and $T_{\text{F50}}$(after adaptation)/$T_{\text{F50}}$(before adaptation). These ratios were selected because in both cases no adaptation gives values of unity, whereas normal adaptation (i.e., $R_{\text{max}}$ decreased, $T_{\text{F50}}$ increased) will give values below unity. Values above unity will show a reversal of the usual adaptation effects (i.e., $R_{\text{max}}$ increased, $T_{\text{F50}}$ decreased). The $R_{\text{max}}$ and $T_{\text{F50}}$ ratios for the 12 cells are shown in Fig. 8. The greatest change in $R_{\text{max}}$ always occurred after adaptation at the peak TF (Fig. 8A). Similarly, the largest rightward shift in $T_{\text{F50}}$ always occurred for adaptation at the peak TF (Fig. 8B). There was no evidence that adaptation at TFs above the optimum TF caused a leftward shift in the tuning functions. In other words, adaptation did not cause repulsion along the TF axis.

**Comparing contrast and TF results**

Figure 9A shows a scatter plot of $C_{50}$ before and after adaptation for all the fast and slow cells combined. These data are presented alongside a scatter plot showing the $T_{\text{F50}}$ before and after motion adaptation in the same cells (Fig. 9B). In both plots, the open symbols represent cells that showed a significant change in $C_{50}$ ($t$-test, $P < 0.01$). The comparison reveals that cells that show rightward shifts in their contrast response functions also tend to show rightward shifts in their TF response functions, suggesting a link between the two effects.
DISCUSSION

Contrast adaptation

This paper shows for the first time in any species that directional neurons of the pretectal NOT demonstrate contrast adaptation effects similar to those observed in the primary visual cortex. Adaptation to motion in the preferred direction causes some cells to show a reduction in firing rate to all tested contrasts, suggesting a fatigue-like effect. However, many cells show a clear rightward shift in the tuning function along the contrast axis. The rightward shift occurs because responses to low contrasts are attenuated, whereas those to high contrasts remain largely unaffected. This effect is commonly discussed as a type of contrast gain control (e.g., Ohzawa et al. 1982, 1985). In this view, adaptation adjusts the sensitivity of cells to the range of contrasts that they have experienced in the recent past (for review, Ibbotson 2005). Consequently, the dynamic range of the cells is optimized to detect changes in contrast close to the prevailing level, rather than being spread thinly across a wide range of contrasts. Such a mechanism might improve the performance of the visual system by providing robust input signals that are matched to the prevailing contrasts in the visual environment.

The contrast adaptation observed in the NOT is likely to occur in the motion detector input circuitry presynaptic to the recorded cells (Ibbotson and Clifford 2001a,b). This suggestion arises from two observations. First, Ibbotson et al. (1998) showed that a test stimulus must be presented in the same region of an NOT cell’s receptive field as the prior adaptation for adaptive effects to be observed. Such a finding argues against the effect being related to changes in the membrane potential of the recorded cell itself, because, if it were, adaptation in any region of the receptive field should influence the responses of all other regions of the receptive field (Kohn and Movshon 2003). Second, neurons in the wallaby NOT can be

FIG. 8. Influence of adaptation on $R_{max}$ (A) and $TF_{50}$ (B) at 3 TFs for 12 NOT cells. Because peak TF for each cell varied, data are plotted against the TF scale factor. Peak TF for each cell is TF scale factor 1 (circles); peak TF/2 is TF scale factor 0.5 (triangles); peak $TF \times 2$ is TF scale factor 2 (diamonds).

FIG. 9. Comparison of $C_{50}$ (A) and $TF_{50}$ (B) for 35 neurons following preferred direction motion adaptation. Open symbols in both plots are those that show significant differences between $C_{50}$ in the control and adapted conditions. Cells that show significant rightward shifts in $C_{50}$ also show rightward shifts in $TF_{50}$. Data from fast and slow cells have been combined.
divided into two categories based on their spatiotemporal tuning properties (Ibbotson and Price 2001; Ibbotson et al. 1994). Slow cells prefer relatively low TFs but high spatial frequencies, whereas fast cells prefer high TFs and low spatial frequencies. The spontaneous activities of these neurons are altered after a period of motion in very characteristic ways. After preferred direction motion, the spontaneous activities of fast cells are transiently inhibited, whereas the spontaneous rate is transiently elevated after anti-preferred motion: opposite-sign after-effects (Price and Ibbotson 2002). Some slow cells can show small opposite-sign after-effects, but most have the reverse pattern. That is, after preferred direction motion, the spontaneous rate is transiently elevated and vice versa: same-sign after-effects (Price and Ibbotson 2002). Recordings were obtained from 16 slow cells and 26 fast cells in this study. Cells that showed changes in their contrast and TF tuning functions after adaptation were found among both cell types, suggesting that the effects are not restricted only to cells where adaptation generates a hyperpolarization of the membrane potential (i.e., fast cells). Contrast adaptation has been shown to be at least partially generated by a hyperpolarization of the recorded cell’s membrane potential in the cat primary visual cortex (Carandini et al. 1998). Slow cells in wallaby NOT show adaptation despite motion stimulation, causing same-sign after-effects, which implies an afterdepolarization of the membrane potential. These observations support the notion that the adaptation occurs before the NOT.

Where could the adaptation occur? The NOT in the marsupial wallaby receives a large direct input from the contralateral eye and a very small input from the ipsilateral eye (Ibbotson et al. 2002). Areas 17 and 18 of the visual cortex provide no obvious input to the NOT (Ibbotson et al. 2002), unlike the case in the eutherian cat (Schoppmann 1981) and monkey (Distler et al. 2002; Ilg and Hoffmann 1993). Unpublished observations on a small number of cells (n = 3) examined contrast adaptation in the wallaby cortex as part of studies of other cortical properties (Ibbotson and Mark 2003). In these three cortical cells, clear contrast adaptation was observed, which resembled the rightward shifts in contrast tuning seen in the NOT (Fig. 2, A and B). While the sample size is not sufficient to make major comment, the fact that NOT and cortical neurons show contrast adaptation, but the two areas do not appear to be connected (Ibbotson et al. 2002), suggests that contrast adaptation may be generated independently in two brain regions in the same species or that both derive the effect from the retina.

It was generally accepted that contrast adaptation is a cortical phenomenon (Maffei et al. 1973; Movshon and Lennie 1979; Ohzawa et al. 1985). However, recent experiments have shown that contrast adaptation does influence the responses of cell’s in salamander and monkey retinas (Baccus and Meister 2002; Chander and Chichilnisky 2001) and the magnocellular layers of the lateral geniculate nucleus in macaques (Solomon et al. 2004). Increases in stimulus contrast progressively and reversibly attenuate light responses in both salamander and monkey retinal ganglion cells, suggesting that a portion of the contrast gain alterations observed in the cortex arise from retinal mechanisms. It is therefore quite feasible that wallaby NOT shows contrast adaptation as a result of mechanisms in the retina. This information would then feed into the NOT and perhaps be further processed such that the effect becomes direction-selective (Ibbotson et al. 1998). We do not know if the retinal inputs to wallaby NOT are direction-selective, but this is the case in rabbits (Oyster et al. 1972) and cats (Hoffmann and Stone 1985).

Motion-specific adaptation

Many NOT cells show changes not only to their contrast tuning but also to their TF tuning following motion adaptation. The physiological results confirm a close link in the retino-pretectal pathway between contrast and motion adaptation. Psychophysical studies on humans, which presumably reflect processing in the retino-geniculate-cortical pathway, have shown that speed and contrast are not independently coded (e.g., Muller and Greenlee 1998). For example, adaptation to moving patterns decreases perceived contrast (Blakemore et al. 1973; Hammett et al. 1994), whereas stimulus contrast influences perceived speed over a wide range of contrasts (Stone and Thompson 1992; Thompson 1982; Thompson et al. 1996). It might therefore be the case that the adaptation mechanisms generating changes to contrast coding also influence speed coding and that these mechanisms are common to both the retino-pretectal and retino-geniculate-cortical pathways.

The change in the TF response functions of NOT cells following motion adaptation could represent a form of TF-related gain control. Certainly the fact that the maximum firing rate at optimum TFs changes very little but the optimum TF and TF$_{50}$ shift to the right in many cells following adaptation suggests that these cells are not being fatigued by adaptation. The rightward shift in TF tuning functions tends to release the cells from saturation at values at or below the unadapted optimum TF and in so doing increases the sensitivity of the cells to changes in TF close to the adapting value. This combined with a very small change in R$_{\text{max}}$ in those cells that show rightward shifts in TF$_{50}$ is suggestive of an active speed-related gain control mechanism. Saul and Cynader (1989a,b) examined the influence of motion adaptation on responses of neurons in area 17 of the cat primary visual cortex. They recorded responses to motion before and after adaptation to moving gratings. They found that, in the spatial domain, adapting at a given spatial frequency resulted in a broad reduction in responsiveness at spatial frequencies above and below the adapting frequency, often with a differential loss of sensitivity at low spatial frequencies (Saul and Cynader 1989a).

In the temporal domain, adaptation generally shifted the preferred TFs of the cat cells. The most common result was that adaptation at a particular TF led to the maximal response attenuation at that same frequency, thus altering the shape of the overall tuning function (Saul and Cynader 1989b). The changes in TF tuning in cat area 17 were direction dependent: after preferred direction adaptation, response attenuation was greater at frequencies equal to or above the adapting frequency; after anti-preferred adaptation, attenuation was greatest at frequencies equal to or below the adapting level. These effects have similarities to the wallaby data but also differences. In the cat, adaptation was evident after anti-preferred motion, whereas there was little evidence of adaptive effects in the wallaby cells. The difference may be species-related or could reveal differences between processing in different visual pathways. Area 17 is not specialized only for motion-processing,
whereas the NOT in both species is a motion-specific region (Hoffmann and Schoppmann 1981; Ibbotson et al. 1994). Perhaps different adaptation mechanisms have developed to allow optimum processing for the selective roles of these brain regions.

**Functions**

Wallabies are known to be highly visual animals and to be day-and-night active (for discussion, see Ibbotson and Mark 2003), so it would be expected that their visual systems need to operate over wide ranges of contrast and luminance. A major function of the NOT is to detect horizontal retinal slip, which would usually be caused by the head rotating about its vertical axis, and to send this information to the motor centers in the brain stem (Collieijn 1975a,b; Hoffmann 1989; Yakusin et al. 2000). These directional signals can be combined with vestibular signals to drive appropriate image-stabilizing ocular following during head and body rotation. Ocular following responses of this type have been shown to occur in wallabies during wide-field visual stimulation with a panoramic stimulus (Hoffmann et al. 1995). Maddess and Ibbotson (1992) showed, in humans, that prior exposure to moving patterns while the eyes fixated a stationary target attenuated the speed of the early components of subsequent ocular following (also see Ibbotson and Maddess 1994). These results suggest at first glance that motion adaptation has a negative effect on the control system that drives ocular following. However, further experiments suggested that, under certain conditions where sudden speed changes were imposed on the adaptive stimulus, ocular following could be enhanced by motion adaptation (Maddess and Ibbotson 1992). No behavioral evidence is available from the wallaby to show the effects of motion adaptation on subsequent ocular following responses. However, the present neural data suggest that horizontal ocular following would be influenced in the wallaby following prolonged exposure to moving patterns.

Ibbotson et al. (1998) suggested that visual motion adaptation might have a beneficial effect in the eye movement control system because the temporal resolution of neurons was improved following motion adaptation, i.e., the sensitivity to speed changes was increased (also see Clifford et al. 1997; Ibbotson and Mark 1996). This conclusion is supported by these data, which reveal that some cells are released from saturation at TFs close to the adapting TF, without loss of spiking capacity at the optimum TF. Other cells show a distinct shift in their optimum TF, again with little reduction in \( R_{max} \). These effects could be said to resemble a form of speed-related gain control that is similar to contrast gain control. Therefore cells are better able to resolve changes in image speed by using their entire spiking capacity to code a more restricted range of speeds (Maddess and Laughlin 1985; Maddess et al. 1991).

Experiments on the visual system of a fly have shown effects that resemble speed-related gain control. Maddess and Laughlin (1985) presented moving patterns to a uniquely identifiable direction-selective neuron in the fly optic lobe (H1). They found that the speed response function in the unadapted state increased almost linearly when response was plotted as a function of log-speed (response range: 100–300 spikes/s for speeds of 3–80°/s). When the speed response function was measured following adaptation at a speed of 58°/s, the function shifted rightward such that a linear relationship was obtained on the semilogarithmic plot (response range, 80–220 spikes/s for speeds of 20–100°/s). The results show that the cell generated a smaller \( R_{max} \) value (300–220 spikes/s) and shifted its speed sensitivity rightward to match the prevailing adaptation speed in a manner similar to some cells in wallaby NOT.

**Mechanisms**

The observation that cells showing contrast gain control also show lateral shifts in their TF tuning suggest a potential link between the effects. It is therefore reasonable to suggest that alterations in the TF tuning are simply related to the alterations in contrast sensitivity. The differential attenuation to low contrast stimuli following adaptation may be the result of an increase in the threshold required to initiate spikes (Carandini and Ferster 1997). In this scheme, for unadapted conditions, high contrasts produce such strong stimulation that the spiking output saturates, but low contrasts produce graded responses that are closely correlated with the intracellular activity (Carandini et al. 1998). Following adaptation, a membrane hyperpolarization increases the threshold required to initiate spikes. Spiking responses to high contrasts appear largely unchanged because the cells are still close to saturation, if not still fully saturated. However, intracellular activity generated by low contrast stimuli is no longer sufficient to generate spikes, leading to a rightward shift of the contrast response function, as observed from the spiking output. The same change in spiking threshold would presumably influence responses to other stimulus parameters. Therefore low TFs, which normally produce low spiking rates, might also be expected to show attenuated spiking activity, whereas responses to optimum TFs would be unaffected.

One possible mechanism by which an NOT cell’s membrane potential could be adjusted during adaptation is if local interneurons, which exist as part of an adapting neural network (e.g., Carandini and Ferster 1997), provide an inhibitory input. However, evidence from rats, cats, and monkeys shows that GABAergic inputs to the large direction-selective neurons in the NOT are rare or absent (Horn and Hofmann 1987). This suggests that it is unlikely that local interneurons have a role in altering membrane thresholds during adaptation. However, in marsupial opossums, there is evidence of inhibitory interactions between the NOTs in each hemisphere, suggesting that GABA may be present in that marsupial species (Periera et al. 1995). Even so, connections between the nuclei are unlikely to be a source of adaptive processes and are thought to relate primarily to the provision of ipsilateral input to each NOT (Ibbotson et al. 2002).

While a simple explanation for adaptation involving membrane hyperpolarization is tempting, it cannot easily explain the rightward shifts in \( T_{opt} \) which were observed in some NOT cells. Clifford and Langley (1996) suggested that adaptation of the temporal delay filters, which are an inherent component of motion detectors, might explain some of the motion-related adaptive effects observed in insect nervous systems (Maddess and Laughlin 1985). Similar claims were later made for neurons in mammalian motion detectors (Clifford et al. 1997; for a review, Clifford and Ibbotson 2003). However, adaptive temporal filters should lead to very large rightward shifts along the TF axis, whereas these data show only modest rightward shifts. The theory suggests that halving
the delay filter time constant should double $TF_{opt}$ (Clifford et al. 1997). Given that motion detection requires multiple processing steps, it is likely that adaptation might influence several stages in the motion processing pathway. It is therefore quite possible that modest adaptation occurs at the temporal delay filter stage, thus shifting $TF_{opt}$ to slightly higher values. This specifically motion-related adaptation would presumably combine with contrast-related effects occurring at earlier stages in the NOT input circuitry.

Summary

This paper has shown that neurons in the pretectal NOT of the marsupial wallaby adapt after exposure to moving gratings. The adaptation in many cells attenuates future responses to low contrasts while having less effect on the maximum response at high contrasts. These results suggest a form of contrast gain control, as observed previously in cortical neurons in cat and monkey. Cells also show changes to their TF tuning functions after motion adaptation, the maximum adaptation occurring when cells are stimulated at their peak unadapted TF. All adaptive effects were direction-selective, the preferred direction of motion producing the strongest effects. Because the primary visual cortex of the wallaby has few, if any, direct connections with the NOT, it is likely that the adaptation observed here occurs independently of the cortex or that adaptation in the NOT and cortex arises in the retina.

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References


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