Interocular Transfer of Adaptation in the Primary Visual Cortex

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Prolonged viewing of an unchanging pattern causes adaptation, which can be demonstrated by visual aftereffects such as the tilt and waterfall illusions. In normal observers, these typically exhibit interocular transfer (IOT), being observed when the adapting and test stimuli are shown to different eyes. Convergence of inputs from both eyes upon binocular neurons only occurs in the primary visual cortex (V1), and adaptation is substantially a cortical phenomenon. However, little is known about a physiological substrate of IOT in V1 and how it relates to the binocularity of neurons and local ocular dominance (OD) column architecture. We employed optical imaging to obtain OD maps in cat V1 and recorded from single neurons at targeted penetration sites to quantify their adaptation by drifting gratings when adapter and test stimulus were presented either to the same or to the opposite eyes. In contrast to earlier reports, clear IOT of adaptation was observed for binocular as well as monocular neurons; at population level, its strength amounted to 55%. Moreover, the position of the cells with respect to OD column borders had no significant effect on the strength of IOT. IOT does not appear to strongly depend on conventional binocularity of neurons.

Keywords: aftereffect, binocular, monocular, optical imaging, orientation, V1

Introduction

Prolonged viewing of an unchanging visual pattern affects its perception as well as that of subsequently viewed patterns in a characteristic fashion known as pattern or contrast adaptation. It leads to reduced contrast sensitivity and a decrease in perceived contrast of adapting and test stimuli (Blakemore and Campbell 1969; Blakemore et al. 1973; Georgeson 1985; Hammett et al. 1994; Ross and Speed 1996; Snowden and Hammett 1996). At the same time, the perception of other stimulus attributes like size, orientation, color, and speed of motion is also altered. These effects are stronger the more similar adapting and test patterns are (Blakemore and Campbell 1969; Blakemore and Nachmas 1971; Dealy and Tolhurst 1974; Berkley 1990). This also pertains to the eyes to which adapting and test stimuli are presented; nevertheless, for a wide range of stimuli, adaptation is observed when the adapting stimulus is shown to one eye and the test stimulus subsequently to the other eye. This phenomenon is called interocular transfer (IOT). Adaptation-based effects that exhibit IOT include the tilt illusion (Campbell and Maffei 1971; Movshon et al. 1972; Mitchell and Ware 1974), the motion aftereffect (MAE) (Lehmkuhle and Fox 1976; O’Shea and Crassini 1981), as well as contrast threshold elevation (Blakemore and Campbell 1969; Gilinsky and Doherty 1969; Bjorklund and Magnussen 1981), with an estimated magnitude of IOT typically ranging between 50% and 80% of the effect observed when the adapter and the test stimulus are shown to the same eye (SE). Because it is generally assumed that the strength of any aftereffect is dependent on the proportion of adapted neurons out of the total population, the magnitude of IOT may give an indication of the percentage of binocular neurons at the cortical site of adaptation (Blake et al. 1981).

Neural correlates of contrast adaptation were first described in the primary visual cortex (V1): neurons exhibit a decrease in firing rate during continuous high-contrast stimulation (Maffei et al. 1973; Vautin and Berkley 1977). In order to elicit the same level of response as prior to adaptation, a test stimulus has to be elevated in contrast by an amount, which depends on the contrast of the adapting stimulus (Ohzawa et al. 1982, 1985). Although contrast adaptation has also been observed in the retina (Chander and Chichilnisky 2001) and the lateral geniculate nucleus (LGN) (Shou et al. 1996; Solomon et al. 2004), it is generally weak compared with that in the cortex, and pattern-specific contrast adaptation is a cortical process (Duong and Freeman 2007). Although some binocular interactions occur in the LGN (Xue et al. 1987; Sengpiel et al. 1995), V1 is the earliest stage at which excitatory input from the 2 eyes converges onto individual, binocular neurons. This is therefore the likeliest point early in the visual pathway at which a physiological substrate of IOT might be found. Indeed, early psychophysical studies suggested that stereoblind subjects with conditions that are known to drastically reduce the proportion of binocular neurons in V1 (such as strabismus or anisometropia) display little or no IOT of the tilt aftereffect (Movshon et al. 1972; Mitchell and Ware 1974), the threshold elevation effect (Ware and Mitchell 1974), or the MAE (Mitchell et al. 1975). However, both the threshold elevation effect (Hess 1978) and the suprathreshold tilt and MAEs (Mohn and van Hof-Van Duijn 1983) were later found to show residual IOT in some stereoblind observers, whereas some subjects with normal stereo thresholds exhibited greatly reduced or no IOT (Mohn and van Hof-Van Duijn 1983). These results cast some doubt over a straightforward causal link between cortical binocularity and IOT of adaptation.

To date, only 2 electrophysiological studies have investigated a possible neural correlate of IOT of adaptation. Using drifting gratings, in a paradigm suitable to evoke a MAE, the strength of IOT at the level of neuronal responses in cat V1 was found to be closely related to the degree of binocularity (Hammond and Mouat 1988). In addition, Maffei et al. (1986) described IOT in almost all simple cells and a third of the complex cells that exhibited adaptation in split-chiasm cats; this transfer was found to be mediated by the corpus callosum. Here we employ optical imaging of intrinsic signals to obtain ocular dominance (OD) and orientation preference maps in cat V1, and we then record from single neurons at targeted penetration sites to quantify their adaptation by drifting gratings when adapter and...
Materials and Methods

Data presented here were obtained from 19 cats bred in a closed laboratory colony and ranging in age from 2 to 6 months. All procedures were approved by local ethical review and carried out under license from the UK Home Office in accordance with regulations on animal experimentation (Animals [Scientific Procedures] Act 1986) and the European Communities Council Directive 86/609/EEC. Efforts were made to minimize animal suffering and to reduce the number of subjects used.

Surgery and Optical Imaging

Anesthesia was induced by intramuscularly (i.m.) administration of ketamine (20–40 mg/kg) and xylazine (4 mg/kg). Atropine (0.2 mg/kg i.m.) was given to reduce mucus secretion. A tracheotomy was performed, and the animals were incubated and placed on heating blanket in a stereotactic frame. Subjects were artificially ventilated with a N2O:O2 mixture of 60:40 and isoflurane (2–3% during surgery and 1–1.5% during data collection). End tidal CO2 (3.5–4.0%), rectal temperature (37.5–38.0 °C), electrocardiogram (150–200 bpm), and electroencephalogram were monitored throughout the experiment and adequate measures taken if any of the values diverged from the described target values. Atropine and phenylephrine were administered to the eyes, which were fitted with gas permeable contact lenses, to protect them and to focus the animal’s vision onto the stimulus display.

An intravenous catheter was inserted in one of the hind legs for administration of drugs and for a continuous infusion of 4% glucose in saline at a rate of 3 mL/kg/h; the infusion solution also contained dexamethasone (Dexafort, Intervet, Milton Keynes, UK; 0.2 mg/kg/h) for prevention of eye movements. The cortex was illuminated with red light at 700 nm. Intrinsic signals were obtained primarily from layers 2/3. The imaged area subtended about 33 cm, on which stimuli were displayed by a visual stimulus generator (RSI, Boulder, CO). Single-condition responses (averages of 48–64 trials per eye) were averaged over 8 trials. The protocol for adaptation and stimulation was illustrated in Figure 1. Following a 15-min interval in which the animal viewed a blank screen (in order to allow for recovery from adaptation), the protocol was repeated with adaptation of the other eye. The control orientation tuning curve and 2 adaptation protocols were recorded over a total period of 1 h. This resulted in 3 orientation tuning curves for each eye, the nonadapted control, one with the dominant eye (DE) adapted, and one with the nondominant eye (NE) adapted. In all the following analyses, the PO (eliciting the largest response) is referred to as PO, and all other orientations are given relative to PO. The level of spontaneous activity in any of the adapted or control conditions was defined as the mean response to a 1.5 s test stimulus, which consisted of a blank screen of the same mean luminance as the gratings and was randomly interleaved with them; this was subtracted from the response to the grating stimuli.

Cells were classified as simple or complex based upon the modulation of the poststimulus time histogram responses to gratings of optimal spatial frequency. A discrete Fourier transform was performed, and the ratio between the F1 component (response...
modulation) and the $F_0$ component (mean response) was calculated. If the ratio of $F_I/F_0$ was greater than 1, then the cell was classified as simple; otherwise as complex (Skottun et al. 1991).

An ocular dominance index (ODI) was calculated by dividing the total response to all orientations in the left eye by the sum of responses to the right eye. Cells were then placed into 7 OD categories (Hubel and Wiesel 1962) as follows: if $ODI > 10$ or $< 0.1$, the cell was considered monocular (ODI or OD7, respectively); if $4 < ODI < 10$ or $0.1 < ODI < 0.25$, the cell was classified as OD2 or OD6, respectively; if $1.5 < ODI < 4$ or $0.25 < ODI < 0.67$, the cell was classified as OD3 or OD5, respectively; and finally, if $0.67 < ODI < 1.5$, the cell was considered binocular with equal input from both eyes (OD4).

A Gaussian function was fitted to the orientation tuning curves in order to obtain quantitative measures of their characteristics (Carandini and Sengpiel 2004):

$$f(o) = R_{max} \exp \left(-\frac{(o - o_w)^2}{2o_w^2}\right) + b,$$

where $R_{max}$ is the maximal response, $o_w$ the preferred orientation, $o_w$ the tuning width, and $b$ the baseline activity.

The direct adaptation effect through each eye was quantified by calculating the response reduction (in percent) of test stimulus responses after adaptation in the SE relative to those before, that is, under control conditions. In order to be able to quantify IOT of adaptation for both binocular and monocular cells, we devised the following formulas:

1. For all cells, the IOT of DE adaptation was calculated by dividing the reduction in DE response following adaptation in the NE by that observed for DE adaptation:

$$IOT_{DE} = \frac{DE_{test after NEadap}}{DE_{test after DEadap}}$$

2. The IOT of NE adaptation was determined in an analogous fashion, for cells that showed a significant NE response (i.e., excluding monocular and near-monocular cells).

$$IOT_{NE} = \frac{NE_{test after NEadap}}{NE_{test after DEadap}}$$

This definition of IOT at the level of individual cells differs slightly from that commonly used to describe the strength of IOT in psychophysical studies, where aftereffects in the SE and the OE are compared following adaptation in one eye. Although this formula cannot be applied to monocular cells (where the NE response is zero or near zero even before adaptation), it can be used at the population level, allowing us to more directly compare physiological and perceptual measures of IOT.

Results

For a total of 59 V1 cells, the entire set of control and postadaptation response curves through both eyes was obtained, allowing quantitative analysis of IOT. The OD of these cells was determined based upon the 7 OD category classification system introduced by Hubel and Wiesel (1962). Of those 59 cells, 18 were either strictly monocular (OD groups 1 and 7) or showed, but, a weak response through one eye (OD classes 2 and 6). Following adaptation, this response was usually indistinguishable from spontaneous activity. For these cells, therefore, direct adaptation as well as IOT of adaptation was quantified only for DE test stimulus responses. The remaining 41 cells displayed more or less balanced responses to monocular stimulation of either eye (23 cells in OD groups 3 and 5, 18 cells in OD group 4).

Binocular Cells

V1 neurons with clear responses through both eyes typically exhibited direct adaptation as well as IOT of adaptation regardless of which eye was adapted and which eye was presented with the test stimuli. An example of a cell with almost identical monocular responses through both eyes (ODI = 1.04; OD group 4) is shown in Figure 2. When the right (slightly dominant) eye was adapted with a grating of optimal orientation (PO = 0°), the response in that eye to the PO was reduced by 36.2% from 90.2 spikes/s to 57.6 spikes/s; when the left eye was adapted, the right-eye response was reduced by 21.6% to 70.7 spikes/s (Fig. 2A). This amounted to an IOT of 59.7% (see Materials and methods, eq. 2). Conversely, left-eye responses were reduced after left-eye adaptation by 56.7% and after right-eye adaptation by 14.1% (Fig. 2B), implying an IOT of 24.9% (see Materials and methods, eq. 3).

When the monocular responses through the 2 eyes were less well balanced (OD groups 3 and 5), then DE responses tended to be more effectively adapted through that eye than through NE adaptation, whereas NE responses were reduced equally strongly regardless of whether the adapting stimulus was presented to the DE or the NE (see below for an analysis of population responses).

Monocular Cells

A typical example of a V1 neuron strongly dominated by one eye (in this case, OD2) is shown in Figure 3. This cell was strongly direction selective and had a PO of 225°. When adapted with a grating of the PO shown to the DE, the PO response was reduced by 57.4%. On the other hand, when adapted with the same grating presented to the NE (which by itself only caused a weak excitatory response of 4.1 spikes/s), the PO response to stimulation of the DE was also reduced very substantially, by 35.2%. IOT of adaptation in this case amounted to 61.3% (see Materials and methods, eq. 2). It is worth noting that both DE and NE adaptation caused a moderate widening of the orientation tuning curves recorded. For this neuron, the control tuning curve had $o_w = 12.6°$. After DE adaptation, $o_w$ was 19.9°, and after NE adaptation, it was 20.4°.

Population Responses to DE and NE Adaptation

In order to assess the overall effect of adapting through either DE or NE on DE and NE responses, we calculated population average response curves (Fig. 4). Monocular control responses were normalized to the PO response of the DE and plotted for that eye (filled circles) and the other NE (open circles). Following adaptation with a grating at PO, presented to either the DE (triangles) or the NE (squares), both population average DE eye responses (filled symbols) and NE responses (open symbols) showed a substantial reduction.
we plotted the ratio of adapted/nonadapted response ratio versus ODI (Fig. 5C,D). We found that in none of the 4 conditions was there a significant correlation between adaptation-induced reduction in response and binocularity. This is particularly noteworthy for the 2 conditions in which OEs were adapted and tested (Fig. 5B,C). Linear regression of adapted/nonadapted response ratio versus ODI yielded $R^2 < 0.04$ in all 4 conditions. In other words, neither SE adaptation nor IOT of adaptation increased with the binocularity of the recorded neurons. The median response ratios for the DE responses were 31.5% following DE adaptation and 45.7% following NE adaptation (response reductions of 68.5% and 24.5%, respectively). We fitted Gaussian functions (see Materials and methods) to the population average tuning curves to assess orientation tuning widths at the population level were slight. In other words, although the strongest response reduction (in absolute terms) occurred at the optimum orientation, responses at other orientations were also reduced to an extent that was similar in relative terms, thereby maintaining orientation tuning. DE responses had a tuning width of 22.1° in the control condition, 19.4° after DE adaptation, and 22.5° after NE adaptation. Similarly, NE control responses had a tuning width of 22.8°, which was reduced slightly to 21.1° after DE adaptation and increased slightly to 24.5° after NE adaptation.

**IOT versus Binocularity**

In order to address the question whether the strength of IOT is correlated with the degree of binocularity of individual neurons, we plotted the ratio of adapted DE responses (compared with the control condition) following either DE or NE adaptation against their OD for all 59 cells in our sample (Fig. 5A,B). While a reliable determination of control and adapted responses through the NE was naturally restricted to the more binocular neurons (OD groups 3, 4, and 5), we similarly plotted DE and NE adaptation effects for the NE responses against those cells’ OD (Fig. 5C,D). We found that in none of the 4 conditions was there a significant correlation between adaptation-induced reduction in response and binocularity. This is particularly noteworthy for the 2 conditions in which OEs were adapted and tested (Fig. 5B,C). Linear regression of adapted/nonadapted response ratio versus ODI yielded $R^2 < 0.04$ in all 4 conditions. In other words, neither SE adaptation nor IOT of adaptation increased with the binocularity of the recorded neurons. The median response ratios for the DE responses were 31.5% following DE adaptation and 78.4% following NE adaptation (response reductions of 68.5% and 21.6%, respectively). The median response ratios for the NE responses were 45.7% following NE adaptation and 50.2%
following NE adaptation (response reductions of 54.3% and 49.8%, respectively).

**IOT at Population Level**

In order to better relate the strength of IOT at the single-cell level to that at the perceptual level in human observers, we analyzed IOT in terms of the effect of adaptation in one eye on neuronal responses through the OE versus the SE. We asked how the response of all cells to the optimal stimulus presented to one eye before adaptation compared with the responses to the same stimulus following adaptation of either the SE or the OE. The 3 distributions of neuronal responses are shown in Figure 6. Although the histograms show obvious overlap, the differences between all 3 distributions are highly significant \( P < 0.001 \) for all 3 comparisons, Wilcoxon test). The median response before adaptation was 25.30 spikes/s. After adaptation through the SE, this value dropped to 8.33 spikes/s (32.9% of the nonadapted response or a reduction by 67.1%), and after adaptation through the OE, the median response was 15.91 spikes/s (62.9% of the nonadapted response or a reduction by 37.1%). In other words, IOT of adaptation, as typically defined in human psychophysics, amounted to 55.3% (see Materials and methods, eq. 4).

**IOT versus Cortical Depth and Cell Type**

We did not observe any obvious trend in terms of the strength of IOT of adaptation with respect to the cortical depth at which cells were recorded. Cells displaying strong IOT therefore do not appear to be limited to any particular cortical layer. Although the majority of 15 simple cells were found at depths that likely corresponded to layer 4 (600–1000 \( \mu m \)), their average IOT was not significantly different from that of the 44 complex cells (44.4% vs. 35.0% for the DE; \( P = 0.18 \), \( t \)-test).

**OD Maps and IOT**

Although there appeared to be no strong link between a cell’s OD and the degree of IOT displayed (Fig. 6), we considered it possible that the location within a cortical map of OD, as obtained by optical imaging, might play a role if IOT of adaptation depended on network properties of cells in the vicinity of the recorded ones. We therefore determined the OD of 16 penetration sites on the basis of the imaged OD maps as follows. Because maps represented the ratio of left-eye responses, summed up across all orientations, divided by right-eye responses, summed up the same set of orientations, a pixel value of 1.0 was taken to signify a balanced binocular response (OD group 4). Actual pixel values were range fitted on a 256-step gray scale (see Materials and methods) such that a pixel value of 1.0 was represented by a grayscale value of 128. The whole 256-step range was then divided equally into 7 OD categories. Whereas the ODI of individual neurons recorded at the 16 sites correlated fairly well with the OD categories derived from the images \( R^2 = 0.24 \); Fig. 7C), the level of IOT did not vary systematically with the OD of the penetration site (Fig. 7D). At all sites, a wide scatter of neurons showing either very little or very pronounced IOT was observed.

**Discussion**

We found clear evidence of IOT of pattern motion adaptation in the majority of cat V1 neurons. For binocular neurons, this is in agreement with earlier physiological reports (Hammond and Mouat 1988). However, contrary to those reports as well as to expectations based on psychophysical observations on stereo-blind humans (Mitchell et al. 1975), IOT was frequently observed for monocular or near-monocular cells when the adapting stimulus was shown to the NE, which in itself elicited little or no response, and the correlation between a cell’s OD and IOT of adaptation was weak. Furthermore, the strength of IOT was not in any obvious way linked to cell type, cortical layer, or location of recording sites within the cortical OD map.
The Neural Substrate of Adaptation and IOT

Although contrast adaptation has been observed both in the retina (Chander and Chichilnisky 2001) and the LGN (Shou et al. 1996; Solomon et al. 2004), these are unlikely to be the physiological origin of the cortical adaptation effects studied here because of their lack of pattern specificity (Duong and Freeman 2007).

The finding of IOT of adaptation among monocular neurons is yet more evidence that adaptation is not the result of some kind of fatigue that would be expected to be proportional to the cells’ suprathreshold spiking response (Vidyasagar 1990). Work by Carandini and Ferster (1997) has shown that the major effect of an adapting stimulus (both in the presence and in the absence of a test stimulus) is tonic hyperpolarization, the strength of which depends on the nature of the adapting stimulus. For example, adaptation with an orthogonal-to-optimum grating does not evoke much hyperpolarization (Carandini et al. 1998) and therefore causes little reduction in the response to an optimally oriented test stimulus. Conversely, tonic hyperpolarization affects the response to any subsequently presented test stimulus, optimal or non-optimal. This may explain why in our study pattern motion adaptation displayed little eye specificity (such that in most cells it was observed regardless of which eye was presented with the adapting and the test stimuli). Similarly, although adaptation was stimulus specific in that adaptation was strongest when adapter and test shared the same orientation and direction of drift, clear adaptation was also found when test stimuli were of a nonpreferred direction or, to a lesser extent, orientation.

Sanchez-Vives and colleagues found that the hyperpolarization associated with adaptation is at least partially intrinsic to the recorded cells because it can be mimicked by an intracellular current injection, which then causes a decrease in the spike response to either sinusoidal current injection or grating stimuli. They further identified Na\(^+\)-activated and Ca\(^{2+}\)-activated K\(^+\) currents as playing an important part in the cellular process underlying adaptation (Sanchez-Vives et al. 2000a, 2000b). Because the hyperpolarization is in principle independent of spiking activity, the pronounced reduction of

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**Figure 5.** Relationship between IOT of adaptation and OD for all 59 cells. The 4 plots show the effect of adapting either DE or NE on the ratio of responses to the PO through each eye following adaptation over the control response without adaptation (the smaller this ratio the stronger the adaptation). Effects of adaptation on responses through the DE are presented in (A, B) and effects of adaptation on responses through the NE are presented in (C, D). The latter includes only those cells in OD groups 3, 4, and 5 (ODI ≤ 4.0). For each cell, the response ratio is plotted against its ODI. Dotted lines in each panel indicate best fit of a linear regression. \(R^2\) values for the regression are indicated within each panel. Note that for cells with ODI <1.0 (dominated by the ipsilateral eye), the reciprocal of the actual ODI is plotted.
DE responses caused by adaptation through the NE in many monocular cells may have been induced by subthreshold NE activity. Certainly, many cells that would be considered monocular when tested in the conventional way, that is, with monocular stimuli presented to each eye, display binocular interactions when stimulated binocularly, thus revealing both excitatory and inhibitory inputs from the silent eye (Ohzawa and Freeman 1986a, 1986b). Therefore, we would predict that the level of hyperpolarization produced by our adaptation protocol may be correlated with the strength of IOT.

Alternatively, IOT may be explicable on the basis of inhibitory interactions between left- and right-eye-dominated neurons, either via horizontal connections within one hemisphere of V1 or via callosal interhemispheric connections. The latter has been suggested by Maffei et al. (1986) on the basis of their study of IOT in split-chiasm cats, where many V1 neurons displayed IOT, including several which appeared to respond only to the ipsilateral eye. No IOT was found after callosotomy, but conversely, cats with intact chiasm exhibited IOT of adaptation even after callosotomy, indicating an intrahemispheric mechanism. However, intracortical inhibition has to be ruled out as the substrate of adaptation in V1 (albeit not necessarily as the substrate of IOT) because blockade of γ-aminobutyric acidergic inhibition does not affect the magnitude of adaptation (DeBruyn and Bonds 1986; Vidyasagar 1990).

Although the studies by Carandini and Ferster (1997) and Sanchez-Vives et al. (2000a, 2000b) shed light on the cellular substrate of cortical contrast adaptation, they do not explain all the psychophysical observations, in particular the stimulus specificity of adaptation. It has been hypothesized that adaptation represents a normalization of neuronal responses, based on integration of inputs deriving from most or all cells within a certain range (Heeger 1992). In that respect, it is surprising that the degree of IOT in V1 neurons does not appear to depend on their position within the cortical OD map. However, this finding adds to earlier evidence that the orientation selectivity of (monocular) adaptation in V1 is similarly independent of the location of neurons within the orientation preference map (Sengpiel and Bonhoeffer 2002). It is of course possible that the stimulus specificity of adaptation is generated by integrating inputs from only a subset of cells within a local network. Such selective connectivity between (groups of) cells with specific stimulus preferences may also explain location or stimulus-specific adaptation effects observed by others (Dragoi et al. 2000, 2001).

**Relation of Physiological Evidence of IOT to Psychophysics**

The overall strength of IOT among the population of neurons we studied (the response reduction through OE adaptation amounting to 55% of the reduction through SE adaptation) compares well with perceptual measures of IOT of the MAE in humans, although the latter tend to be slightly higher. Mitchell et al. (1975) found that the magnitude of the MAE in the eye contralateral to the adapted one was on average 73% of the effect in the adapted eye. Lehmkuhle and Fox (1976) reported 76% transfer when the nonadapted eye had viewed a uniform field of the same mean luminance as the adapting grating shown to the other eye and 52% when it had simply been occluded; these results were later confirmed by Timney et al. (1996). O'Shea and Crassini (1981) reported 66% IOT of the MAE. Despite a slightly lower level of IOT and some overlap in the distributions of neuronal responses in the adapted and nonadapted conditions (Fig. 6), our study revealed highly significant IOT of adaptation among V1 neurons. This is likely to contribute, if not to account for, the phenomenon observed perceptually.

**Relation of IOT to Cortical Binocularity**

Based on the assumption that the strength of visual aftereffects is related to the proportion of neurons adapted by the inducing stimulus, the magnitude of IOT is thought to indicate the percentage of binocular neurons responding to the test stimulus at the cortical site of adaptation (Blake et al. 1981). However, our results cast doubt on such a straightforward relationship. Monocular cells frequently exhibit clear IOT of adaptation and, conversely, some binocular cells do not.

Whereas it was initially thought that strabismic humans do not exhibit IOT of adaptation phenomena such as the MAE (Mitchell et al. 1975), subsequent studies have shown IOT of the MAE in some subjects (Wade 1976; Hess 1978; O'Shea et al. 1994), but not of the contrast threshold elevation aftereffect (Levi et al. 1980). IOT of the MAE is asymmetric in normal subjects with clear dominance of one eye (Mitchell et al. 1975) and in strabismic subjects (Wade 1976), such that it is generally greater after adaptation of the DE and testing of the NE than vice versa; the amount of asymmetry was found to correlate inversely with the amount of IOT (Keck and Price 1982). These findings agree reasonably well with our physiological data, showing greatest symmetry of IOT in binocular neurons with balanced input from both eyes (OD group 4) and a stronger transfer from the DE to the NE for cells in OD groups 3 and 5.

Whether or not a neuron shows binocular interaction in the sense that stimulation through one eye modulates the responses evoked by simultaneous stimulation of the other eye may be more pertinent to IOT than conventional binocularity. This might help to explain why IOT has been found in some human observers who are likely to be lacking
conventionally binocular V1 neurons. Based on their observation that dichoptic masking is similar in subjects with normal and abnormal binocular vision, occurring even when the masking grating was presented to the strabismic and/or amblyopic eye (Levi et al. 1980), suggested that strabismus and/or amblyopia disrupted the normal excitatory interactions between the 2 eyes but left cortical inhibitory binocular connections intact. Anatomical support for this hypothesis comes from the finding that connections between left- and right-eye OD columns are reduced in V1 of strabismic cats (Löwel and Singer 1992) and monkeys (Tychsen and Burkhalter 1995). Physiologically, pronounced interocular suppression in case of binocularly presented grating stimuli is clear evidence of the persistence of inhibitory binocular connections in V1 of strabismic cats (Chino et al. 1994; Sengpiel et al. 1994) and monkeys (Sengpiel and Blakemore 1996; Smith et al. 1997). In order to clarify the relationship between cortical binocularity and IOT of adaptation aftereffects, it will be important to quantify IOT in V1 of strabismic animals.

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**Notes**

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