Visual Adaptation: Physiology, Mechanisms, and Functional Benefits

Adam Kohn
Department of Neuroscience, Albert Einstein College of Medicine, Bronx, New York

Kohn A. Visual adaptation: physiology, mechanisms, and functional benefits. *J Neurophysiol* 97: 3155–3164, 2007. First published March 7, 2007; doi:10.1152/jn.00086.2007. Recent sensory experience affects both perception and the response properties of visual neurons. Here I review a rapid form of experience-dependent plasticity that follows adaptation, the presentation of a particular stimulus or ensemble of stimuli for periods ranging from tens of milliseconds to minutes. Adaptation has a rich history in psychophysics, where it is often used as a tool for dissecting the perceptual mechanisms of vision. Although we know comparatively little about the neurophysiological effects of adaptation, work in the last decade has revealed a rich repertoire of effects. This review focuses on this recent physiological work, the cellular and biophysical mechanisms that may underlie the observed effects, and the functional benefit that they may afford. I conclude with a brief discussion of some important open questions in the field.

INTRODUCTION

Both perception and the response properties of neurons are affected by sensory history. This dependence on experience is seen over a wide range of time scales, from effects arising during development to those driven by the most recent sensory input. A particularly striking and easily studied form of plasticity is that caused by adaptation—the sensory experience of the preceding tens of milliseconds to minutes. The perceptual effects of adaptation can be demonstrated with Fig. 1. Prolonged viewing of a high contrast vertical pattern (Fig. 1*A*) reduces the perceived contrast of a similar pattern in a subsequently viewed test stimulus (Fig. 1*B*). Similarly, the response properties of visual neurons change during adaptation and responses to subsequent stimuli are altered as well. Here I review our current understanding of the neurophysiology, mechanisms, and proposed benefits of this rapid plasticity.

Adaptation is of interest for two distinct reasons. First, its effects occur rapidly enough to contribute to moment-to-moment sensory processing. Recent sensory history forms an immediate temporal context in which perceptual experience is interpreted. The neural effects of adaptation are a realization and encoding of this context in the brain and thus represent a fundamental component of sensory information processing. Second, adaptation is a useful tool for studying general issues of plasticity. Although each form of plasticity (e.g., perceptual learning or reorganization after injury) is likely to involve distinct effects and mechanisms, there are a number of shared questions as well. These include understanding the functional goals of plasticity, linking neuronal effects to changes in perception, learning how different stages of processing adjust to the environment, and exploring how plasticity at one stage impacts responses in another. These issues form the themes of this review.

Address for reprint requests and other correspondence: Dept. of Neuroscience, Albert Einstein College of Medicine, 1410 Pelham Parkway South, Rm. 821, Bronx, NY 10461 (E-mail: akohn@aecom.yu.edu).

Contrast adaptation

In principle, the visual system could adjust to recent sensory input independently at each processing stage or perhaps whenever a large number of presynaptic signals are pooled (Baccus and Meister 2004). Alternatively, it could implement effects early in the processing stream and pass this altered representation to downstream areas. Studies of how adaptation affects neuronal contrast tuning have revealed that the visual system uses a combination of both approaches.

Perceptually, adaptation with a high-contrast stimulus causes a reduction in the apparent contrast of a test stimulus (Fig. 1) (see Graham 1989 for review). Because low-contrast stimuli evoke weak responses in visual neurons, reduced perceived contrast would be expected to arise from reduced neuronal responsiveness. This neuronal effect, in turn, could arise either from a divisive or subtractive reduction in firing rate (which would reduce the cells' dynamic range and thus represent a form of deleterious fatigue) or from a reduction in contrast sensitivity (which would involve a rightward shift of the contrast response function; Fig. 2*A*). Recordings in primary visual cortex (V1) show evidence for each of these effects, but the reduction in contrast sensitivity is predominant (Albrecht et al. 1984; Bonds 1991; Crowder et al. 2005; Ibbotson 2005a; Dean 1983; Movshon and Lennie 1979; Ohzawa et al. 1982, 1985; Sclar et al. 1989). This readjustment of sensitivity centers the cells' dynamic range about the recently encountered average contrast (i.e., that of the adapter).

The reduction in perceived contrast is strongest for test stimuli that have the same orientation as the adapter (Blakemore and Campbell 1969; Blakemore and Nachmias 1971). This can be confirmed by comparing the effect of the vertical (Fig. 1*A*) and horizontal (Fig. 1*C*) adapters; the latter has little effect on the perception of the vertical test stimulus (Fig. 1*B*). In addition, adaptation is effective, although weakened, when the adapter and test stimulus are presented to different eyes (Blakemore and Campbell 1969; for neuronal correlates, see Maffei et al. 1973; Marlin et al. 1991). Both orientation specificity and interocular transfer suggest a cortical locus for the perceptual effect because orientation tuning and binocular responses first occur in V1. Initial recordings in the cat lateral geniculate nucleus (LGN) were consistent with this conclusion, revealing little or no change in contrast sensitivity or firing rate after adaptation (Derrington et al. 1984; Maffei et al. 1973; Movshon and Lennie 1979; Nelson 1991b; Ohzawa et al. 1985; Shou et al. 1996).

Recent work has revealed, however, that the contrast sensitivity of retinal ganglion cells (RGCs) is altered both during and after adaptation (Baccus and Meister 2002; Brown and Masland 2001; Chander and Chichilnisky 2001; Kim and Rieke 2001; Smirnakis et al. 1997; Rieke 2001). First, high-contrast stimuli reduce the integration time of RGCs Baccus
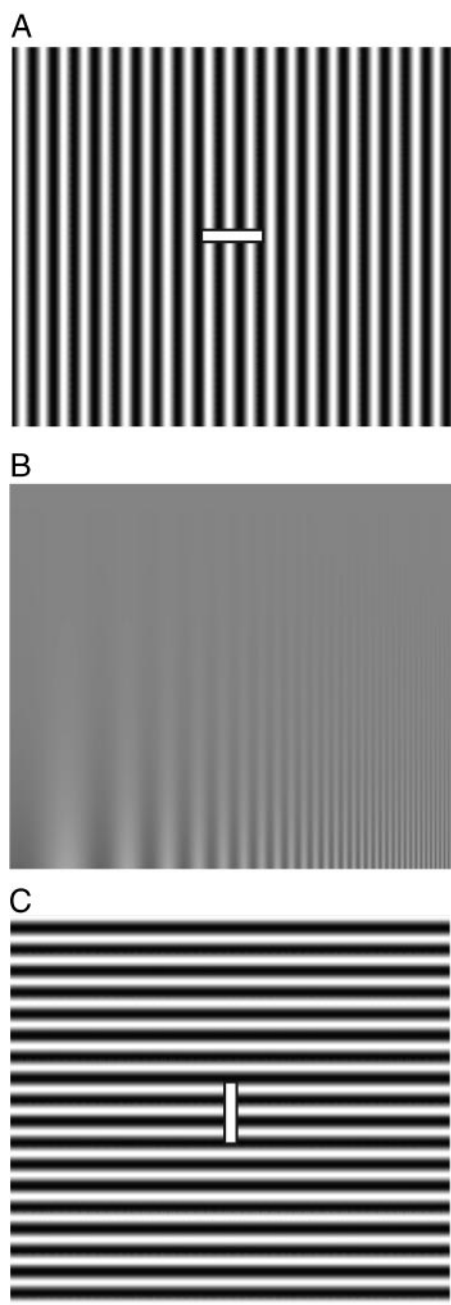
FIG. 1. Perceptual reduction in apparent contrast. Slowly move your eyes back and forth (to prevent retinal afterimages) along the white bar at the center of *A* for 30 s and then transfer your gaze to the test image in *B*. A low-contrast portion of the image (top) should briefly appear invisible. Adaptation with the pattern in *C* does not reduce sensitivity to the test pattern, demonstrating the orientation specificity of the effect. Note that the aftereffect is also spatial frequency specific: lower (left) and higher (right) spatial frequencies in the test image are not affected by adaptation with *A*.

and Meister 2002; Chander and Chichilnisky 2001; Kim and Rieke 2001), an efect that occurs within 200 ms of stimulus onset. Such rapid dynamics suggest that this effect may be closely related, if not identical to, the retinal gain control described by Shapley and Victor (1978). Second, the firing rate of RGCs declines gradually after the onset of a high-contrast stimulus and the response to subsequent low-contrast stimuli is strongly reduced. These effects are similar to those observed in

cortex and strongly suggest that retinal effects could contribute to perceptual contrast adaptation.

Motivated by these in vitro retinal studies, Solomon et al. (2004) examined contrast adaptation in the LGN of anesthetized primates. They found that parvocellular cells are largely unaffected by adaptation, but the contrast sensitivity of magnocellular cells is strongly reduced. The effect on magnocellular cells is present in simultaneously recorded S-potentials, indicating that it is inherited from the retina. The change in sensitivity is particularly strong after adaptation with stimuli of high temporal frequency (e.g., 25 cycle/s), consistent with reports that cortical effects are more profound for rapidly drifting gratings (Maddess et al. 1988; Saul and Cynader 1989b). That the adaptation affects magno- but not parvocellular cells offers a potentially useful tool for studying the relative strength of input from these two pathways to a cortical cell or area (Solomon et al. 2004).

Unlike light adaptation, which occurs entirely in the retina (Shapley and Enroth-Cugell 1984), contrast adaptation thus involves effects both in the retina and in the cortex. This raises the possibility that each subsequent stage of visual processing may adjust independently to the prevailing contrast level. Kohn and Movshon (2003) tested this possibility in area MT, an area involved in visual motion processing that is downstream from
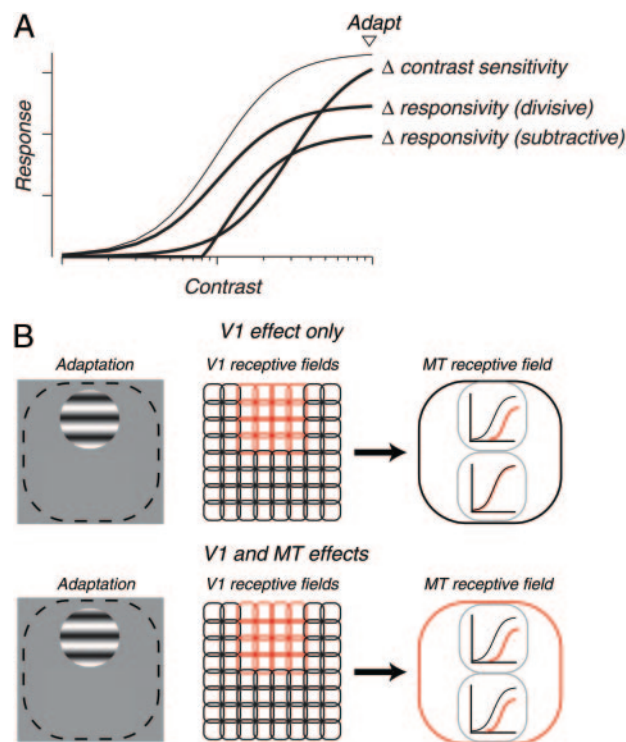
FIG. 2. Effects of adaptation on contrast sensitivity. *A*: neuronal responses to stimuli of increasing contrast typically have a sigmoidal shape (thin line). High contrast adaptation (adapter contrast indicated by arrowhead) can alter the contrast response function (thick lines) in 1 of the 3 indicated ways: a change in contrast sensitivity, a divisive change in responsivity or a subtractive shift of the function. *B*: measuring spatial specificity of adaptation in MT. If adaptation alters contrast sensitivity of primary visual cortex (V1) but not MT cells (*top row*), a small adapter (*left*) will affect a subpopulation of V1 cells whose receptive fields (RFs) overlie the adapted location (red, *middle*). A change in the contrast response function will be seen in the adapted region of an MT RF (*right*) but not at a second location. An effect of adaptation on V1 and MT will result in an effect that transfers at least partially between subregions (*bottom row*).

V1. Adaptation in MT leads to a threefold change in contrast sensitivity with little change in peak firing rate. To distinguish a direct effect of adaptation on MT from the simple inheritance of effects occurring early in the visual system, Kohn and Movshon (2003) measured the consequence of adapting a restricted subregion of an MT receptive field (RF; Fig. 2*B*). Intracellular recordings have shown that changes in V1 contrast sensitivity involve postsynaptic hyperpolarization (discussed in more detail in the following text), so a direct effect of adaptation on MT cells would reduce the response evoked by test stimuli presented at both adapted and unadapted locations within the RF. In MT, however, adaptation with a small stimulus affects only stimuli presented to the same spatial subregion of the RF. This suggests that changes in MT contrast sensitivity occur prior to the spatial integration of input within MT, presumably being inherited from the early visual system where RFs are small. Thus while contrast adaptation affects both the retina and V1 directly, not all stages of processing adapt independently to contrast.

Specificity of adaptation effects

A hallmark property of adaptation is that the strength of both perceptual and physiological effects depend on the similarity between the test stimulus and the adapter. At the neuronal level, this specificity means that the tuning of single neurons is altered by adaptation. Recent studies have shown that how tuning is altered depends on the cortical area investigated and on the adaptation paradigm used.

Early studies showed that V1 responses to preferred stimuli are reduced after adaptation with preferred but not opposite ("null") or orthogonal stimuli (Giaschi et al. 1993; Hammond et al. 1988; Marlin et al. 1988; Vautin and Berkley 1977; von der Heydt et al. 1977; see also Barlow and Hill 1963). These observations established that adapters that evoke a more robust response generally result in stronger effects. For an effective adapter, the reduction in V1 responsiveness is strongest for test stimuli that are well-matched to the adapter, in part because contrast sensitivity is reduced most for such stimuli (Albrecht et al. 1984; Crowder et al. 2005; Movshon and Lennie 1979). As a result, an adapter presented on the tuning curve flank causes a local reduction in responsiveness and a repulsive shift (i.e., away from the adapter) in the cell's preference (Fig. 3*A*). Repulsive shifts have been observed in V1 tuning for spatial frequency (Saul and Cynader 1989a), temporal frequency (Saul and Cynader 1989b; see also Ibbotson 2005b), and often (Dragoi et al. 2000, 2002; Felsen et al. 2002; Muller et al. 1999; Nelson 1991a) but not always (Kohn and Movshon 2004; see also Dragoi et al. 2001) for orientation.

The specificity underlying these reported changes in V1 tuning is for simple stimulus attributes such as orientation and spatial and temporal frequency. Perceptual aftereffects show similar specificity for these attributes (e.g., Fig. 1) but also for stimulus correlations or conjunctions. For example, in the McCollough effect, adaptation to pairs of colored gratings causes colorless test gratings to appear tinged with the opposite color to the similarly oriented component of the adapter (McCollough 1965). In a simple correlate for such contingent adaptation, Carandini et al. (1997a, 1998) showed that adaptation with superimposed preferred and orthogonal gratings
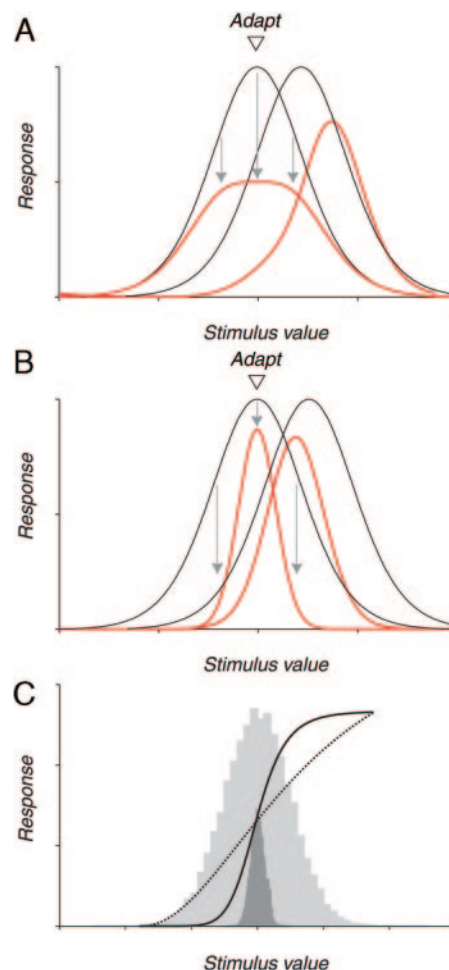

FIG. 3. Effects of adaptation on neuronal tuning. *A*: adaptation has been found to alter V1 neuronal tuning by reducing responses to stimuli similar to the adapter most strongly, as indicated by the longer downward arrows for the adapted value than for offset values. Flank adaptation causes a repulsive shift in tuning. Tuning before adaptation is shown in black; after adaptation, in red. *B*: in MT, adaptation affects responses at the adapted value least, resulting in narrower tuning (center pair of tuning curves) and attractive shifts in preference (offset pair of tuning curves). *C*: tuning curve slope depends on the distribution of stimuli used to drive the cell. A high variance distribution (light gray histogram in the background) results in shallower tuning (dotted line); a low variance distribution (dark gray histogram) results in steeper tuning (solid line).

reduces V1 responses to the resultant plaid pattern more than to either of its constituent gratings. More recently, adaptation with temporally or spatially correlated stimuli has been shown to lead to a pattern-specific reduction in responsiveness in the salamander retina (Hosoya et al. 2005). Adaptation with oriented stimuli, for instance, results in reduced sensitivity only to stimuli of the same orientation, even though RGCs are untuned for orientation. The mechanisms proposed to underlie these retinal effects, however, are somewhat distinct from those believed to underlie adaptation effects in cat and monkey cortex (see *Mechanisms*). Specifically, they appear to arise from a precise and consistent temporal relationship between the pre- and postsynaptic cells, a situation that is observed in cortex more typically with protocols that are designed to evoke spike-timing dependent plasticity (e.g., Fu et al. 2002). The degree to which the pattern specificity of cortical effects arises in the retina is thus unclear but deserves further study.

We know most about the specificity of adaptation early in the visual system, but a number of recent studies have investigated this issue in MT (Kohn and Movshon 2003, 2004; Krekelberg et al. 2006b; Petersen et al. 1985; Priebe and Lisberger 2002; Priebe et al. 2002; van Wezel and Britten 2002; see also Xu et al. 2001). MT is an attractive target for adaptation studies because the basic properties of its cells have been well-characterized, its activity has been strongly linked to perception (Born and Bradley 2005), and adaptation to visual motion has profound perceptual consequences (e.g., the motion aftereffect) (Mather et al. 1998). As in V1, adaptation in an MT cell's preferred direction reduces responsiveness, whereas adaptation in the null direction results in maintained (Kohn and Movshon 2003; Priebe et al. 2002; van Wezel and Britten 2002) or enhanced (Petersen et al. 1985) responses. Effective adapters, however, alter MT tuning in a manner *opposite* to effects observed in V1: responses to test stimuli well-matched to the adapter are least affected, but other test stimuli evoke much weaker responses after adaptation (Fig. 3*B*) (Kohn and Movshon 2004; Krekelberg et al. 2006b). As a result, adaptation in the preferred direction of an MT cell results in narrower tuning and flank adaptation results in attractive shifts in preference. These changes can explain the strong repulsion of perceived direction of motion after adaptation (the direction aftereffect), which is not predicted by effects in lower cortical areas (Jin et al. 2005; Kohn and Movshon 2004).

Whether changes in MT tuning are typical of effects in extrastriate cortex is unknown because the adaptation properties of other extrastriate areas have not been explored thoroughly. Brief adaptation has been shown to result in weaker effects in V2 than in V1 (Nelson 1991a), but more prolonged adaptation results in similar changes in contrast sensitivity in the two areas (Crowder et al. 2005). In primate V4, adaptation alters tuning so that it becomes more directional (Tolias et al. 2005). Finally, adaptation in inferotemporal cortex results in weaker responses to repeated but not to novel test stimuli (Baylis and Rolls 1987; Miller et al. 1991; Ringo 1996; Sawamura et al. 2006). Some of this loss of responsiveness persists even after the presentation of a number of intervening stimuli, so it may represent a form of more long-term memory (Miller and Desimone 1994). In general, these studies have established that responses to test stimuli similar to the adapter are reduced but not how tuning curve shape is altered by adaptation.

All of the V1 and extrastriate studies discussed in the preceding text employed a traditional adapt-test design with a single stimulus serving as the adapter, a paradigm also typically used in psychophysics. Several recent studies have reported novel effects using a different approach: measuring cells' tuning and responsiveness during the dynamic presentation of different ensembles of stimuli. This approach was adopted by many of the previously discussed retinal studies, which used the temporal modulation of full-field luminance stimuli to investigate contrast adaptation. In the fly visual system, Brenner et al. (2000) used this approach to show that the speed tuning of the H1 neuron adjusts to match the range of speeds in a stimulus ensemble. When H1 is probed with a sequence of stimuli chosen from a low variance distribution (i.e., a narrow range of speeds), tuning is steep; when exposed to a high variance distribution, tuning is substantially shallower (Fig. 3*C*). Adaptation can thus stretch or compress the range of

stimuli over which the cell's responsiveness is modulated (i.e., it can change tuning curve slope) (see also Bair and Movshon 2004; Dean et al. 2005; Nagel and Doupe 2006). A related approach was used by Sharpee et al. (2006), who showed that neural filters in cat V1 differ during and after exposure to a dynamic sequence of natural scenes or filtered white-noise stimuli. Spatial frequencies that are common in a particular ensemble become less effective over time, enhancing the information relayed by the neuron about rare spatial frequencies. The reduced sensitivity of V1 neurons to common but not rare spatial frequencies is consistent with the specificity of traditional adaptation for stimulus spatial frequency (Movshon and Lennie 1979; Saul et al. 1989a).

In summary, the visual system has a rich repertoire of ways in which it adjusts to recent stimulus history. These effects include repelling tuning curves away from the adapter (in V1), attracting tuning toward the adapter (in MT), changing sensitivity to stimulus conjunctions, and altering tuning curve slope based on the range of recently encountered stimuli. These diverse findings raise important challenges for mechanistic explanations of how effects arise and for the functional benefit that they provide. Before exploring these topics, I will briefly review the role of adaptation duration.

Time scales of adaptation

Changes in neuronal response properties occur after adaptation as brief as tens of milliseconds or as prolonged as many seconds. Attempts to determine the time scale over which effects arise have often involved measuring the decay in firing rate during the presentation of the adapter. Measurements on brief time scales reveal a rapid decay (e.g., an exponential decay with a time constant of ~30 ms) (Priebe et al. 2002), whereas more prolonged adaptation reveals a slower process (e.g., time constant of 2–60 s) (Giaschi et al. 1991; Hammond et al. 1988; Ohzawa et al. 1985; Vautin and Berkeley 1977). That the decay in firing rate can be described by multiple exponential processes suggests that its dependence on adaptation duration may be better described by a power-law relationship (Drew and Abbott 2006).

Regardless of this distinction, to a first approximation, adaptation effects appear qualitatively similar on a wide range of time scales with more prolonged adaptation resulting in stronger effects. For instance, the horizontal shift in the contrast response function, observed after prolonged adaptation (80 s) (Ohzawa et al. 1985) occurs in a weaker form after brief adaptation (50 ms) (Bonds 1991). Similarly, repulsive shifts in V1 tuning occur after adaptation as brief as several tens or hundreds of milliseconds (Dragoi et al. 2002; Felson et al. 2002; Muller et al. 1999) or lasting as long as 20 min (Dragoi et al. 2000, 2001). In the most systematic exploration of the role of adaptation duration to date, Dragoi et al. (2000) found repulsive shifts in V1 orientation tuning after adaptation ranging in duration from 10 s to 10 min with the latter giving rise to the strongest effects. Studies of well-known phenomena such as the tilt and motion aftereffect have also found that adaptation duration determines the strength not the nature of perceptual effects (e.g., Magnussen and Johnsen 1986; Mather et al. 1998).

Although the statement that more prolonged adaptation leads to stronger effects is a useful rule of thumb, there are both

physiological and perceptual reports of distinct effects following very brief (<100 ms) adaptation. For instance, Priebe and colleagues studied the decay of firing rate that occurs in MT cells within tens of milliseconds of stimulus onset (Lisberger and Movshon 1999; Priebe and Lisberger 2002; Priebe et al. 2002). The reduced responsiveness induced by a brief motion pulse (e.g., 64 ms) at one location affects the response to a second pulse presented elsewhere in the RF, indicating an effect intrinsic to MT. This result is opposite to that observed after more prolonged adaptation for which effects are spatially specific within the RF (Kohn and Movshon 2003), although differences in the stimuli used in the two studies may also contribute to the different findings. On the perceptual level, very brief adaptation (27 ms) induces a robust aftereffect for object shape, even for test stimuli that do not overlap the adapted location. Orientation-specific aftereffects are negligible after such brief adaptation (Suzuki 2001, 2005). Together these studies indicate that brief adaptation may preferentially target extrastriate areas. This does not, however, imply that all effects of prolonged adaptation occur early in the visual system: complex aftereffects such as repulsive shifts in face perception (Leopold et al. 2001; Webster et al. 2004) occur after prolonged adaptation and almost certainly involve high-level neurons.

Adaptation with stimulus ensembles involves effects occurring over a range of time scales (Fairhall et al. 2001), but most of the change in tuning is present within 100 to 1,000 ms of a change in distribution variance and increases only slightly in strength with longer adaptation (Fairhall et al. 2001; Nagel and Doupe 2006). Such rapid and profound effects resemble more closely "gain control" mechanisms such as retinal contrast gain control (Shapley and Victor 1978, 1981) or contrast normalization in cortex (Carandini et al. 1997b; Smith et al. 2006). The situation is further complicated by the demonstration that variance-dependent changes in model neurons can occur with fixed parameters (Bair and Movshon 2004; Borst et al. 2005; Yu and Lee 2003). That is, tuning can appear altered simply because two distributions reveal different aspects of a cell's static receptive field (Borst et al. 2005; Yu and Lee 2003) or because they recruit different sets of hardwired presynaptic inputs (Bair and Movshon 2004; Nagel and Doupe 2006). Whether the sensitivity of neuronal tuning to stimulus variance is truly a form of plasticity—that is, an experience-driven process that develops over time—remains to be determined. The effect of adaptation with ensembles of natural stimuli (Sharpee et al. 2006), on the other hand, arises particularly slowly (over minutes) suggesting it may represent a distinct form of plasticity.

A final consideration is that the time scale of adaptation may not be fixed. Just as the ability to induce long-term synaptic plasticity is itself plastic (i.e., "metaplasticity") (Abraham and Bear 1996), how quickly the visual system adjusts to the environment may depend on how frequently the environment changes. Specifically, frequent changes in stimulus statistics appear to result in a more rapid adjustment of neuronal sensitivity (Fairhall et al. 2001). The time scale of recovery from adaptation is also not a passive process with fixed dynamics. Perceptual aftereffects, which typically last for several seconds after tens of seconds of adaptation, can be stored for prolonged periods if subjects are not exposed to a test stimulus immediately after adaptation (Mather et al. 1998; Thompson and

Movshon 1978). These findings strongly suggest that adaptation causes an active recalibration of sensitivity based on experience.

Mechanisms

Of all adaptation effects, the cellular mechanisms underlying changes in neuronal contrast sensitivity are perhaps the best understood. In cortex, whole cell recordings have shown that adaptation results in a strong somatic afterhyperpolarization but little change in synaptic input (Carandini and Ferster 1997; see also Harris et al. 2000). This afterhyperpolarization is due primarily to the activation of a sodium-gated potassium channel, triggered by the sodium influx that occurs with synaptic input and the generation of action potentials (Sanchez-Vives et al. 2000a,b). Depolarizing current injection can also activate this channel, resulting in changes similar to those following visual adaptation. In the retina, intrinsic mechanisms in ganglion cells—namely, sodium channel inactivation—also contribute to changes in contrast sensitivity (Kim and Rieke 2003), although effects on presynaptic bipolar cells (Rieke 2001) and at bipolar to ganglion cell (Manookin and Demb 2006) synapses have also been implicated. Interestingly, the time scale over which sodium channels recover from inactivation depends on the duration of the preceding depolarization, which would allow this mechanism to contribute to a range of physiological effects (Toib et al. 1998).

A second mechanism possibly recruited by adaptation is synaptic depression due to the depletion of vesicles from the presynaptic terminal. In vitro studies have reported profound synaptic depression which involves two processes, one that arises and recovers over hundreds of milliseconds and another over seconds (Abbott et al. 1997; Finlayson and Cynader 1995; Markram and Tsodyks 1997; Varela et al. 1997). Although theoretical studies have shown how synaptic depression can explain certain neuronal aftereffects (Chance et al. 1998), to date only one study has provided evidence that this mechanism is important in vivo. Specifically, Chung et al. (2002) showed that responses in rat barrel cortex decline more strongly during repetitive sensory drive than those in the thalamus due to rapid depression of thalamocortical synapses (Gil et al. 1997; Stratford et al. 1996). Unlike studies in cat visual cortex, Chung et al. (2002) found no evidence for changes in the intrinsic properties of cortical cells (e.g., hyperpolarization). The importance of synaptic depression in the rat barrel system may be related to the low spontaneous and evoked firing rates of the relevant neurons. In the cat LGN, high spontaneous firing rates causes substantial thalamocortical synaptic depression, leaving little room for further change after adaptation (Boudreau and Ferster 2005). Similarly, corticocortical synapses in cat V1 undergo substantial depression only when ongoing activity is artificially suppressed (Reig et al. 2005).

Because evidence for the depression of excitatory synapses is limited, an obvious alternative is a change in the strength of synaptic inhibition (Barlow 1990; Barlow and Foldiak 1989; Dealy and Tolhurst 1974; Ohzawa et al. 1985; Wainwright et al. 2002). Barlow and Foldiak (1989) suggested that "anti-Hebbian" plasticity at inhibitory synapses—that is, the strengthening of inhibition between cells that are simultaneously active—could explain repulsive shifts in V1 tuning. In the retina, exactly such a strengthening of inhibitory input from

amacrine cells to RGCs may explain pattern-specific changes in RGC responsiveness (Hosoya et al. 2005). In cortical brain slices, synapses between pyramidal and some types of inhibitory cells facilitate during repetitive drive, which could cause an increase in the strength of recurrent inhibition (Thomson and Deuchars 1997). The available in vivo evidence, however, suggests that changes in inhibition do not underlie the cortical effects of adaptation. Iontophoretic application of bicuculline methiodide, which blocks GABA$_A$-mediated synaptic inhibition, does not alter the effect of adaptation in V1 (Debruyn and Bonds 1986; McLean and Palmer 1996; Nelson 1991c; Vidyasagar 1990) and enhances effects in the LGN (Yang et al. 2003). In MT, neurons receive strong inhibitory input when presented with a stimulus drifting opposite to their preferred direction (Qian and Anderson 1994; Snowden et al. 1991), providing a direct opportunity to evaluate how adaptation affects synaptic inhibition. Using compound stimuli composed of a preferred and null grating, Kohn and Movshon (2003) showed that null adaptation reduces the strength of opponent inhibition. This result is most easily explained by a reduction in the contrast sensitivity of the cells that provide the inhibition and is inconsistent with the suggestion that adaptation potentiates inhibition.

Although there is little direct evidence for synaptic changes in cortical ionotropic inhibition and excitation after adaptation, it would be premature to conclude that synaptic mechanisms play no role. For instance, metabotropic receptors are often activated only during periods of intense or repetitive activity and can remain active for hundreds of milliseconds to many seconds, thus having onset and offset kinetics appropriate for underlying a range of adaptation effects (Kohn and Whitsel 2002). The role of metabotropic receptors has gone largely unstudied, although McLean and Palmer (1996) reported that a metabotropic glutamate receptor antagonist could block the cortical effects of adaptation. GABA$_B$ agonists also appear to enhance the effects of adaptation in the LGN (Yang et al. 2003). Given their prevalence and kinetics, the role of metabotropic receptors in adaptation deserves closer examination.

Synaptic mechanisms of adaptation are commonly believed necessary because of the stimulus specificity of neuronal effects. If adaptation only led to somatic hyperpolarization, all test stimuli would be expected to evoke weaker responses rather than only those similar (or dissimilar in MT) to the adapter. In addition, adaptation with stimuli that fail to evoke a response in the recorded cell (Bonds 1991; Crowder et al. 2005; Ohzawa et al. 1985; but see also Maffei et al. 1973 and Saul and Cynader 1989a) or during GABA-induced suppression of cortical activity (Vidyasagar 1990) can have substantial effects, even though activity-dependent mechanisms intrinsic to the postsynaptic cell would not be recruited. However, given that neurons are embedded in recurrent networks, neither stimulus specificity nor the efficacy of nonexcitatory adapters requires a synaptic mechanism. Because the response of a cell to a given stimulus is driven by other responsive neurons, hyperpolarization of these presynaptic cells would give rise to a stimulus-specific reduction in responsiveness. The efficacy of adapters that fail to evoke a response in the recorded cell can be explained by the hyperpolarization of broadly tuned presynaptic cells. Although hyperpolarization of presynaptic cells is a somatic mechanism, it does result in reduced synaptic input to the recorded cell. This is in apparent disagreement with intra-

cellular recordings that find that synaptic input is maintained during adaptation (Carandini and Ferster 1997; Sanchez-Vives et al. 2000a). This discrepancy can be explained by postsynaptic hyperpolarization, which would increase the driving force for synaptic events and thus mask a reduction in the absolute but not relative amplitude of input provided by the adapter (Nowak et al. 2005). Consistent with specificity arising from presynaptic input in a recurrent network, Dragoi et al. (2001) found that repulsive shifts in V1 tuning were prominent in orientation map pinwheels, where neurons receive input from cells with a broad range of preferences, and largely absent in iso-orientation domains.

The importance of considering network mechanisms is further highlighted by a modeling study by Teich and Qian (2003). These authors found that both attractive and repulsive shifts in tuning can occur in a recurrent model of V1 circuitry, depending on which synapses are altered. A reduction in excitatory input to the adapted cell results in narrow tuning and attractive shifts in tuning (as seen in MT), whereas a reduction in both excitation and inhibition (with the latter predominating) can result in repulsive shifts in preference (as seen in V1). Although the effects in the Teich and Qian model were implemented by changing synaptic weights, similar effects could be caused by hyperpolarization of the presynaptic neurons. Similar models have been used to investigate the propagation of plasticity through the visual system. In a computational study of attention-induced changes in responsiveness, Compte and Wang (2005) found that implementing attentional effects in V1 could result in either attractive or repulsive shifts in downstream receptive fields, depending on the relative strength of recurrent excitation and inhibition in downstream networks. These models may help explain how adaptation can alter tuning differently at successive stages of processing.

Functional benefits

The clearest example of a beneficial effect of adaptation is undoubtedly light adaptation in the retina (Shapley and Enroth-Cugell 1984). Confronted with light intensity that varies over roughly ten orders of magnitude, we are able to discriminate relatively small changes in luminance because of retinal mechanisms that alter sensitivity to match prevailing conditions. Functionally, this is observed in the output of the retina as a horizontal shift in the luminance-response relationship of RGCs. The horizontal shift in the contrast response function of cortical neurons is remarkably similar to this effect. It allows cortical cells to use their limited dynamic range to encode a narrow range of contrasts with adaptation altering sensitivity to match the prevailing contrast level. Adaptation should thus lead observers to discriminate better between contrasts that are similar to the recently encountered mean. The psychophysical evidence for improved contrast discrimination, however, is weak. Improvements were found in some studies (Abbonizio et al. 2002; Greenlee and Heitger 1988) but not others (Barlow et al. 1976; Maatanen and Koenderink 1991). A recent comprehensive study concluded that discriminability could improve but that the effect is extremely sensitive to the details of the adaptation and the discrimination task (e.g., monocular versus binocular viewing) (Abbonizio et al. 2002).

The idea that adaptation re-centers tuning around prevailing stimulus conditions so as to improve discriminability has also

been proposed for both repulsive (Muller et al. 1999) and attractive shifts in preference (Krekelberg et al. 2006b). As with contrast, the psychophysical evidence for improved discriminability is weak: discrimination thresholds for stimuli similar to the adapter are either unaffected or only slightly enhanced by adaptation (Dragoi et al. 2002; Hol and Treue 2001; Krekelberg et al. 2006a; Regan and Beverly 1985; but see also Clifford and Wenderoth 1999; Kristjansson 2001). The strongest effect appears to be a strong reduction in discriminability for test stimuli offset from the adapter, either in orientation, direction or speed (see Clifford 2002 for review). The poor link between changes in the tuning of single neurons and discriminability may be because of other factors that influence performance. First, response variability is as important as response magnitude in determining performance (Green and Swets 1966; Stocker and Simoncelli 2006). Second, the neurons that contribute most to a particular task may be difficult to determine and may change when tuning or response strength is altered (Butts and Goldman 2006; Pouget et al. 2003). Third, sensory decisions involve a population of cortical cells the performance of which is strongly influenced by correlated variability (Abbott and Dayan 1999). To understand how adaptation affects population coding, and thus perception, knowledge of its effect on response variability and correlation is essential.

A second proposed benefit is that adaptation improves the detectability or discriminability of novel or rare stimuli. In the simplest scenario, novelty detection can be accomplished by suppressing responses to frequent or persistent stimuli, leaving those to novel stimuli relatively enhanced (Dragoi et al. 2002; Hosoya et al. 2005; Sharpee et al. 2006; Ulanovsky et al. 2003). Novelty detection is an attractive idea because it can be viewed as an extension of the general predictive coding strategy of the visual system, which improves efficiency by encoding the environment as differences in stimulus strength in space (e.g., the center-surround organization of RGC receptive fields) or in time (Attneave 1954; Barlow 1961; Srinivasan et al. 1982). In a direct demonstration of the relevance of this concept for adaptation, modeling work has shown that the recruitment of slow hyperpolarizing conductances makes cells less sensitive to slow fluctuations in input and emphasizes rapidly varying or recent input (Wang et al. 2003). On the other hand, using plasticity to enhance performance for completely novel stimuli is arguably a questionable strategy: the nervous system should become more sensitive to subtle variations in common stimuli rather than to stimuli that seldom appear. This is what occurs with light adaptation, which does not lead to greater sensitivity to or discrimination between particularly dim or bright features but instead to improved discriminability of differences in light intensity about the mean. In addition, plasticity that arises on longer time scales typically enhances the representation and discriminability of frequent stimuli not rare ones (e.g., Recanzone et al. 1993). Finally, there is only weak perceptual evidence for changes in detection thresholds for novel stimuli (Graham 1989; but see also De Valois 1977) although there may well be improvements in discriminability between such stimuli (Clifford et al. 2001; Dragoi et al. 2002; but see also Westheimer and Gee 2002).

A final proposal, also motivated by efficient coding principles, is that changes in tuning after adaptation serve to improve representational efficiency (Barlow 1990; Barlow and Foldiak

1989; Sharpee et al. 2006; Wainwright 1999). When a particular stimulus or conjunction of stimuli is common, efficiency is improved by decorrelating the response of the activated cells. Decorrelation can be achieved either by repelling the preference of activated neurons (as suggested by Barlow and Foldiak) or by making those neurons more narrowly tuned, both make the signals provided by the cells less redundant. A clear benefit of reduced redundancy is metabolic savings. Given that action potentials are metabolically expensive (Laughlin 2001; Lennie 2003), having fewer active neurons and weaker responsiveness after adaptation may be of significant value. Whether more efficient representations lead to changes in performance—for example, in detection and discriminability—is unclear.

In summary, the effects of adaptation should allow the visual system to make use of regularities in the environment (i.e., persistent or recurring stimuli) to perform better. The differences among proposals concern whether performance should be enhanced for stimuli like the adapter or very different from it or whether the improvement involves a more abstract notion of efficiency. The proposed benefits should ultimately be evident in improved perceptual performance; because there is limited psychophysical data showing improvement—particularly given the robust physiological effects—the benefit of adaptation remains unclear.

Open questions

Over the past three decades neurophysiological studies have elucidated how recent sensory experience affects neuronal response properties in the retina, the LGN, V1, and extrastriate cortex. Effects have been measured over a wide range of time scales, from tens of milliseconds (that might occur during inter-saccade fixation) to minutes (as typically explored psychophysically). Despite this substantial progress, most of the fundamental questions about adaptation remain unanswered.

First and foremost, the functional benefit of most adaptation effects is unclear. A better understanding of how adaptation affects population coding may provide some insight. Adapting with stimulus ensembles may also be important because such dynamic input may activate mechanisms not recruited (or not activated in the same manner) by the traditional adapt-test paradigm using prolonged exposure to single stimuli. This possibility is supported by recent findings that tuning is sensitive to ensemble variance, although psychophysical studies using a similar paradigm are needed to explore whether the reported neuronal effects are paralleled by changes in human performance. Finally, a better knowledge of effects at different stages of visual processing, particularly extrastriate areas closely linked to perception, is crucial.

In measuring plasticity in extrastriate cortex, it will be important to determine the relative influence of effects inherited from earlier areas and arising directly from local cells or circuits. The relative importance of low- and high-level effects may depend on the duration and characteristics of the adapter (e.g., stimuli that provide weak, broadly distributed input to early cortex may preferentially affect extrastriate areas). Tracing the effects of adaptation through the sensory hierarchy is important for interpreting imaging studies that increasingly use adaptation as a tool to assign computations to distinct cortical regions (Grill-Spector and Malach 2001; Krekelberg et al.

2006a). Comparing plasticity at multiple levels of the visual system may also prove a useful way to measure and evaluate cortical population coding. Because adaptation distorts the sensory representation in the early visual system, measuring the impact downstream could provide important insight into how this altered representation is read out.

A complete understanding of adaptation requires, of course, an understanding of the mechanisms that underlie it. As is the case for many computations in sensory cortex, the biophysical underpinnings of neuronal effects remain murky. Given the wide range of cellular and synaptic phenomena that operate on the same time scales as adaptation, it is likely that many distinct mechanisms are involved. The slow dynamics of many metabotropic receptors and their intracellular signaling cascades are well-matched to the measured changes in neuronal response properties. Molecular tools for interfering with specific receptor systems (Callaway and Sanes 2006) will hopefully provide insight into both the function of these receptors and to the mechanisms of adaptation.

Finally, the growth in our knowledge of how adaptation alters the neuronal representation of the sensory world will have to be integrated with our understanding of other forms of plasticity, such as perceptual learning (Fahle and Poggio 2002) and recovery of function after peripheral or central injury. To date, each form of plasticity has been treated independently, both in terms of the effects emphasized (e.g., changes in tuning or in cortical maps) and the functional benefits provided. Although the nervous system may change in different ways on different time scales, all plasticity presumably functions to allow the brain to make use of statistical regularities in the environment to improve performance (Barlow 1997). As a result, there should be some relationship between effects observed at different time scales, and we should seek to understand how temporary changes—such as those following adaptation—are transformed into more permanent effects. An intriguing possibility is that adaptation leaves a short-term memory trace that guides subsequent long-term synaptic plasticity (Gutfreund and Knudsen 2006).

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