Adaptation to visual stimulation modifies the burst firing property of V1 neurons

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Abstract: The mean firing rate of visual cortical neurons is reduced after prolonged visual stimulation, but the underlying process by which this occurs as well as the biological significance of this phenomenon remains unknown. Computational neuroscience studies indicate that high-frequency bursts in stimulus-driven responses can be transmitted across synapses more reliably than isolated spikes, and thus may carry accurate stimulus-related information. Our research examined whether or not adaptation affects the burst firing property of visual cortical neurons by examining changes in the burst firing changes of V1 neurons during adaptation to the preferred visual stimulus. The results show that adaptation to prolonged visual stimulation significantly decreased burst frequency (bursts/s) and burst length (spikes/burst), but increased burst duration and the interspike interval within bursts. These results suggest that the adaptation of V1 neurons to visual stimulation may result in a decrease of feedforward response gain but an increase of functional activities from lateral and/or feedback connections, which could lead to a reduction in the effectiveness of adapted neurons in transmitting information to its driven neurons.

Keywords: Visual adaptation; Burst firing; Neurons; Primary visual cortex; Cat

When exposed to prolonged stimulation, a neuron’s response decreases gradually to a minimum firing level, a phenomenon called adaptation. Adaptation in the visual pathway is generally considered a property of visual cortical cells, though recent investigation showed that subcortical neurons also display a weaker adaptation to visual stimulation (Shou et al, 1996; Smirnakis et al, 1997; Baccus & Meister, 2002), and the primary visual cortex may undergo pattern-specific adaptation (Duong & Freeman, 2007). The mechanisms that underlie visual adaptation are still the subject of debate. Four possible hypotheses have been postulated, including contrast gain control (Carandini & Ferster, 1997; Sanchez-Vives et al, 2000), depression of excitatory synapses (Chung et al, 2002), strengthening of inhibitory synapses (Thomson & Deuchars, 1997) and neural network mechanism (Teich & Qian, 2003). However, these hypotheses do not provide sufficient evidence to explain adaptation properties reported by psychophysical experiments, likely because these proposals are based solely on analyses of mean firing rate, which cannot decode all the information (such as timing code) in the response of neurons to visual stimuli.

Additionally, the biological significance of visual adaptation is also unclear, though different views have been suggested (Kohn, 2007). A common concern within the proposed views is whether or not adaptation changes the efficiency of neuronal information encoding and perceptual discrimination. Several authors report that contrast adaptation may enhance contrast discrimination and information transmission (Greenlee & Heitger, 1988; Sharpee et al, 2006), while others found that the contrast...
discrimination thresholds remained unchanged after adaptation (Maattanen & Koenderink, 1991). Still others argue that Greenlee and Heitzer and Maattanen and Koenderink reach different conclusions because of respective differences in viewing conditions (Abbonizio et al, 2002). To resolve these disputes, we assessed the capacity changes of neurons in information encoding and transmitting during adaptation.

Recent studies in computational neuroscience indicate that a neuron’s response is composed of two firing modes with different roles in information processing (Krahe & Gabbiani, 2004; Oswald et al, 2004). One is an isolated firing mode with a single action potential, or spike, separated by long interspike intervals (>20 ms). The other is called high-frequency burst firing (BF), which is a sequence of action potentials separated by short interspike intervals (<20 ms, mostly several ms). Because burst firing can induce two or more excitatory postsynaptic potentials (EPSPs) within a short time frame, and these EPSPs are more likely to excite the postsynaptic neuron, most of authors believe that burst firing can facilitate synaptic transmission more reliably than isolated firing, and thus carry faithful stimulus-specific information (Cattaneo et al, 1981; Eggermont & Smith, 1996; Lisman, 1997; Krahe & Gabbiani, 2004). In a certain sense, isolated firing could be considered as noise inherent in the neural circuits (Lisman, 1997).

An in vivo investigation has shown that burst firing has a significantly higher effectiveness than single isolated spike in eliciting a spike from a driven neuron, and that longer bursts are more effective than shorter ones (Snider et al, 1998). Furthermore, burst length (spikes/burst) and burst frequency (bursts/s) are modulated by changes of local intracortical inhibition. Iontophoretic application of GABA could significantly shorten burst length, whereas administration of bicuculline, an antagonist of GABA\textsubscript{A} receptor, increased burst length of the cell’s response (DeBusk et al, 1997).

In this research, we examined the changes of burst firing property of V1 neurons during adaptation to prolonged visual stimulation, attempting to find clues to the mechanisms that may mediate visual adaptation process as well as biological implications of adaptation.

MATERIALS AND METHODS

Subjects

Subjects for this study were four young adult cats (2−3 years old). Each subject was a healthy domestic cat with the history of age and healthcare properly documented by veterinarians. Subjects were examined ophthalmoscopically before experiment to confirm that no optical or retinal problems impaired their visual function. All experiment procedures were done strictly in accordance with the guidelines published in the NIH Guide for the care and use of Laboratory Animals.

Extracellular recording procedures

All subjects were prepared for extracellular single-unit recording using a procedure previously described (Hua et al, 2006; Hua et al, 2009; Hua et al, 2010). Briefly, anaesthesia was induced by injection (i.m) of ketamine HCl (40 mg/kg, im) and xylazine (2 mg/kg, im). After intubation of intravenous and tracheal cannulae, the cat was immobilized in a stereotaxic apparatus with ear, eye and bite bars. Pupils were maximally dilated with atropine (1%), and appropriate contact lenses were used to protect the corneas. Neosynephrine (5%) was administered to retract the nictitating membranes. Glucose (5%)-saline (0.9%) solution containing a mixture of urethane (20 mg/hr/kg body weight) and gallamine triethiodide (10 mg/hr/kg body weight) was infused intravenously to keep the animal anesthetized and paralysed. Expired pCO\textsubscript{2} was maintained at approximately 3.8%.

The level of anaesthesia was closely assessed throughout the experiment by continuously monitoring the animal’s heart rate (about 180−220 pulses/min) and ECG to ensure that the animals were not responding to pain. The skull and dura over V1 (area 17) were removed with a fine surgery under light microscope. Single-unit recordings were performed using a glass-coated tungsten microelectrode (with an impedance of 3−5 MΩ) which was driven by a hydraulic micromanipulator (Narisighe, Japan). The small hole over V1 was filled with 4% agar solution in saline and sealed with wax. After the preparation was complete, the optic discs of the two eyes were reflected onto a movable transparent tangent screen positioned 57 cm from the retina and overlapped with the CRT monitor used for stimulus presentation. The area centralis of each eye was located prior to physiological recording based on the position of the optic discs reflected onto the tangent screen (Bishop et al, 1962).

Visual stimuli

Visual stimuli were drifting sinusoidal gratings shown on a CRT monitor (resolution 1 024×768, refresh rate 85 Hz) positioned 57 cm from the animal’s eyes. The program to generate the stimuli was written in MATLAB, using the extensions provided by the high-level Psychophysics Toolbox (Brainard, 1997) and low-level VideoToolbox (Pelli, 1997). Once a single unit was isolated, the cell’s receptive field center was carefully located by consecutively presenting a series of computer-generated light spots on the CRT. By comparing the neuron’s response to a series of stimulus package, we determined the optimal stimulus orientation, motion direction, temporal and spatial frequency and stimulus size for each cell. Then, the cell’s response to a prolonged (30 s) stimulation with optimal stimulus was measured, and that longer bursts are more effective than shorter ones (Snider et al, 1998). Furthermore, burst length (spikes/burst) and burst frequency (bursts/s) are modulated by changes of local intracortical inhibition. Iontophoretic application of GABA could significantly shorten burst length, whereas administration of bicuculline, an antagonist of GABA\textsubscript{A} receptor, increased burst length of the cell’s response (DeBusk et al, 1997). In this research, we examined the changes of burst firing property of V1 neurons during adaptation to prolonged visual stimulation, attempting to find clues to the mechanisms that may mediate visual adaptation process as well as biological implications of adaptation.

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recorded, which was used to assess the neuronal response properties during adaptation. Each stimulus was presented monocularly to the dominant eye and repeated 4-6 times with a 3-minute interval between each trial for functional recovery of the recorded cell. Before each stimulus was presented, spontaneous activity was acquired during a 10 s period while a mean luminance was shown on the screen.

The contrast of each stimulus used was set at 100%. The mean luminance of the display was 19 cd/m², and the environmental ambient luminance on the cornea was 0.1 lux.

Data analysis

After the signal was amplified with a microelectrode amplifier (Nihon Kohden, Japan) and differential amplifier (Dagan 2400A, USA), action potentials were fed into a window discriminator with an audio monitor. The original voltage traces were digitized using an acquisition board (National Instruments, USA) controlled by IGOR (WaveMetrics, USA), and saved for later analysis. The response of a cell to a drifting sinusoidal grating was defined as the mean firing rate (spontaneous activity subtracted) corresponding to the time of stimulus modulation, which was used to draw the orientation, temporal and spatial frequency tuning curves. Preferred orientation was calculated as previously described (Schmolesky et al, 2000; Hua et al, 2006; Hua et al, 2010). The optimal temporal and spatial frequency of each cell were determined respectively by comparing the cell’s mean firing rate to high contrast (100%) grating stimuli with different temporal and spatial frequencies, and selecting the temporal and spatial frequency with the maximum response.

To determine if neurons included in this study exhibited distinct adaptation to visual stimulation, the adaptation index (AI) was calculated respectively from each cell’s response to the prolonged optimal stimulus and defined as the mean firing (spontaneous activity subtracted) of the cell’s average response during the last 5 s of stimulus adaptation, a period when the neuron’s response retained a stable minimum level, to the average response during the initial 5 s of adaptation (Figure 1A). The smaller the AI, the stronger the adaptation of the cell. Cells with adaptation index ≥1 were treated as showing no adaptation to visual stimulation and excluded from our data analysis.

Two or more adjacent spikes with ISI (interspikes interval) ≤20 ms were classified as a burst. A: Voltage trace of a cell’s response during absence (0-15 s) and presence (15-45 s) of visual stimulation. A spike above the broken horizontal line is counted as an action potential; B: Burst time stamp in the cell’s response during the initial 5 s of adaptation. The mean BF (burst frequency), IBI (interbursts interval), BL (burst length), BD (burst duration) and ISI (interspikes interval) was 7.2 burst/s, 0.135 s, 2.72 spikes/burst, 18.6 ms and 6.4 ms respectively; C: Burst time stamp in the cell’s response to the last 5 s of stimulation. The mean BF, IBI, BL and ISI was 1.8 burst/s, 0.501 s, 2.33 spikes/burst, 26.8 ms and 12.9 ms respectively.

To disclose how visual adaptation influences burst firing of a cell’s response, we compared burst firing properties of each cell’s response at the initial 5 s and last 5 s of prolonged visual stimulation. We used a simple method to define bursts as strings of spikes with only two parameters: a minimum number of spikes per burst and a maximum interspike interval (ISI) (Turnbull et al, 2005). In this study, we set the minimum number of spikes per burst as 2, which was consistent with previous studies on the burst identification of visual cortical and subcortical neurons in cats (Mandl, 1993; DeBusk et al, 1997). According to the probability for two neighboring spikes to integrate (Traub & Miles, 1991; Barbieri et al, 2001), we combined both short and long ISI bursts (DeBusk et al, 1997) in data analysis and defined a maximum ISI as 20 ms. After the bursts have been identified, the mean burst frequency (BF: bursts/s), interbursts interval (IBI: interval between neighboring bursts), burst length (BL: spikes per burst), burst duration (BD: time interval from the first spike to the last
spike within a burst) and interspikes interval (ISI) within bursts were acquired (Figure 1B,C).

All values were expressed as mean±SD. Variations of BF, IBI, BL, BD and ISI in the neuronal response between different periods of adaptation were assessed using paired t-test or analysis of variance (ANOVA).

RESULTS

A total of 151 cells from 4 young adult cats were studied (Table 1), comprised of 65 simple cells and 86 complex cells. All cells had receptive fields within 8 degrees from the central area of the dominant eye. Measured from the mean firing rate of neuronal response, all studied cells showed an evident adaptation to prolonged visual stimulation (30 s), as indicated by the adaptation index ranged from 0.11-0.97. Because simple and complex cells showed no significant difference in adaptation index (F(1,149)=0.298, P=0.586), data from the two types of cells were combined for statistical analysis.

Table 1 Changes of burst firing properties of V1 neurons during adaptation to prolonged visual stimulation in four cats

<table>
<thead>
<tr>
<th></th>
<th>Cat1</th>
<th>Cat2</th>
<th>Cat3</th>
<th>Cat4</th>
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</thead>
<tbody>
<tr>
<td>CN</td>
<td>26</td>
<td>56</td>
<td>34</td>
<td>35</td>
</tr>
<tr>
<td>AI</td>
<td>0.59±0.22</td>
<td>0.65±0.18</td>
<td>0.54±0.21</td>
<td>0.49±0.17</td>
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<tr>
<td>Burst firing during the initial 5s of adaptation</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>BF</td>
<td>4.54±2.10</td>
<td>5.85±3.48</td>
<td>5.17±2.58</td>
<td>4.80±1.62</td>
</tr>
<tr>
<td>IBI</td>
<td>0.24±0.09</td>
<td>0.21±0.10</td>
<td>0.22±0.09</td>
<td>0.23±0.08</td>
</tr>
<tr>
<td>BL</td>
<td>4.86±0.69</td>
<td>5.30±0.88</td>
<td>5.14±0.79</td>
<td>5.09±0.97</td>
</tr>
<tr>
<td>BD</td>
<td>40.7±21.2</td>
<td>47.3±24.5</td>
<td>46.1±21.0</td>
<td>44.5±20.6</td>
</tr>
<tr>
<td>ISI</td>
<td>8.31±4.40</td>
<td>8.70±3.87</td>
<td>8.85±3.42</td>
<td>8.90±4.25</td>
</tr>
<tr>
<td>Burst firing during the last 5s of adaptation</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BF</td>
<td>2.85±1.62</td>
<td>3.85±2.07</td>
<td>2.95±1.62</td>
<td>2.34±1.16</td>
</tr>
<tr>
<td>IBI</td>
<td>0.41±0.29</td>
<td>0.31±0.16</td>
<td>0.47±0.50</td>
<td>0.48±0.35</td>
</tr>
<tr>
<td>BL</td>
<td>4.29±0.68</td>
<td>4.88±0.93</td>
<td>4.61±0.97</td>
<td>4.49±0.86</td>
</tr>
<tr>
<td>BD</td>
<td>53.6±16.3</td>
<td>55.1±25.0</td>
<td>59.1±19.8</td>
<td>53.8±20.5</td>
</tr>
<tr>
<td>ISI</td>
<td>12.5±3.36</td>
<td>11.3±4.66</td>
<td>13.1±4.17</td>
<td>12.2±4.57</td>
</tr>
</tbody>
</table>

CN and AI denote cell number and mean adaptation index of V1 cells in each cat.

BF, IBI, BL, BD and ISI represent the mean burst frequency (bursts/s), interbursts interval (s), burst length (spikes/burst), burst duration (ms) and interspikes interval (ms) of neuronal response in each cat.

Effects of adaptation on burst and isolated firing

To determine if burst firing and isolated firing of V1 cells were equally affected by adaptation, we compared the effects of adaptation on isolated spikes and burst spikes in the same neuron. Paired t-test showed that adaptation resulted in a significant reduction of both burst spikes and isolated spikes (all P<0.0001) (Figure 2A). The ratio of burst spikes to total spikes during the last 5 s of adaptation was significantly decreased as compared with that during the initial 5 s (P<0.01) (Figure 2B). However, the ratio of isolated spikes to total spikes during the last 5 s of adaptation was significantly increased when compared with that during the initial 5s (P<0.01) (Figure 2B). Therefore, adaptation of V1 neurons to prolonged visual stimulation decreased its burst firing more specifically than isolated firing.

Burst frequency and interbursts interval

To uncover how adaptation affected burst firing, we examined first the effects of adaptation on burst occurrence frequency (BF) of neuronal response by comparing the mean BF of V1 cells during the initial 5 s (15–20 s) and the last 5 s (40–45 s) of adaptation to prolonged visual stimulation. Most cells (64.2%) had a BF larger than or equal to 4 bursts/s during the initial 5 s of adaptation, whereas only a small part of cells (28.5%) had a BF larger than or equal to 4 bursts/s during the last 5 s of adaptation (Figure 3A). The mean BF of V1 cells during the last 5 s of adaptation to visual stimulation was significantly lower than that during the initial 5 s (paired t-test, P<0.000001). Therefore, adaptation to visual stimulation greatly decreased BF of V1 cells.

The interbursts interval (IBI) change of V1 cells during adaptation to prolonged visual stimulation was also examined. The majority of cells (81.5%) had an IBI shorter than 0.3 s during the initial 5 s of adaptation, whereas more than half of cells (57.6%) had an IBI equal to or longer than 0.3 s during the last 5 s of adaptation.
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The mean IBI value of V1 cells during the last 5 s of adaptation was significantly larger than that during the initial 5 s (paired t-test, \( P<0.00001 \)).

\[ \text{Figure 3B} \]

The mean IBI value of V1 cells during the last 5 s of adaptation was significantly larger than that during the initial 5 s (paired t-test, \( P<0.00001 \)). Consistent with our expectations, however, the ISI of V1 neurons was significantly lengthened during adaptation to prolonged visual stimulation. The majority of cells (71.5%) displayed a mean ISI value smaller than 12 ms during the initial 5 s of adaptation, whereas most cells (61.6%) had a mean ISI equal to or larger than 12 ms during the last 5 s of adaptation (Figure 3C). The mean ISI value of V1 neurons during the last 5 s of adaptation (12.1±4.3 ms) was significantly greater than during the initial 5 s of adaptation (8.7±3.9 ms) (paired t-test, \( P<0.000001 \)). Therefore, a reduced BL of V1 cells during visual adaptation was likely caused by an increased ISI within burst.

\[ \text{Figure 4B} \]

Burst length, burst duration and interspikes interval

Previous studies showed that burst length (BL) is closely related to stimulus information encoding and synaptic transmission reliability (DeBusk et al., 1997). To examine how visual adaptation influences visual signal processing of visual cortical cells, we compared the BL of V1 neurons during the initial 5 s and the last 5 s of adaptation to visual stimulation. As indicated in Figure 4A, more than half of cells (55%) had a BL value equal to or larger than 6 spikes/burst during the initial 5 s of adaptation, while a smaller portion of cells (33.8%) had a BL value equal to or larger than 6 spikes/burst during the last 5 s of adaptation. Paired t-test showed that the mean BL of V1 neurons during the last 5 s of adaptation was significantly smaller than that during the initial 5 s of adaptation (\( P<0.0001 \)). Accordingly, we concluded that visual adaptation decreased BL of neuronal response in V1.

A decreased BL in the neuronal response during adaptation could be caused by shortened burst duration (BD) or an increased interspikes interval (ISI) or both. To assess these possibilities, we compared the BD and ISI of V1 neurons during the initial 5 s and the last 5 s of adaptation to visual stimulation. Contrary to what we expected, the BD of V1 neurons actually increased during adaptation. Nearly half the cells (46%) exhibited a BD less than 60 ms during the initial 5 s of adaptation to visual stimulation, while most cells (72.8%) had a BD value equal to or larger than 60 ms during the last 5 s of adaptation (Figure 4B). The mean BD of V1 neurons during the last 5 s of visual adaptation (55.4±21.4 ms) significantly longer than that during the initial 5 s of adaptation (45.2±22.2 ms) (paired t-test, \( P<0.0001 \)).

\[ \text{Figure 4B} \]

Consistent with our expectations, however, the ISI of V1 neurons was significantly lengthened during adaptation to prolonged visual stimulation. The majority of cells (71.5%) displayed a mean ISI value smaller than 12 ms during the initial 5 s of adaptation, whereas most cells (61.6%) had a mean ISI equal to or larger than 12 ms during the last 5 s of adaptation (Figure 4C). The mean ISI value of V1 neurons during the last 5 s of adaptation (12.1±4.3 ms) was significantly greater than during the initial 5 s of adaptation (8.7±3.9 ms) (paired t-test, \( P<0.000001 \)). Therefore, a reduced BL of V1 cells during visual adaptation was likely caused by an increased ISI within burst.

\[ \text{Figure 4C} \]

DISCUSSION

Adaptation mechanisms

Visual cortical neurons show a reduced firing rate to prolonged visual stimulation. The underlying neural basis for this, however, remains unresolved (Kohn, 2007; Hua et al., 2009). Based on analyses of the mean firing...
rate of neuronal response, previous studies proposed several models to explain the adaptation phenomenon (Kohn, 2007). The contrast gain control mechanism (Carandini and Ferster, 1997; Sanchez-Vives et al, 2000), which suggested a strong somatic afterhyperpolarization due to an increased potassium ion current triggered by sodium ion influx during prolonged stimulation, cannot interpret the specificity of visual adaptation to the test stimulus. Excitatory synaptic depression can account for stimulus-specificity of adaptation; however, in vivo experiments lack convincing evidence to support this point of view (Chung et al, 2002; Nowak et al, 2005; Reig et al, 2006).

Inhibitory synaptic strengthening is another alternative mechanism that may underlie adaptation process. Nevertheless, in vivo electrophysiology research shows that manipulating local inhibition by the iontophoretic administration of GABA or GABA\(_A\) receptor antagonist could not modify the extent of adaptation of visual cortical neurons to visual stimulation (DeBruyn & Bonds, 1986; Vidyasagar, 1990). Modeling studies proposed a network mechanism concerning a relative weight of recurrent excitation and inhibition in local circuitry (Teich & Qian, 2003; Compte & Wang, 2006), which may help clarify the above-mentioned discrepancy in the interpretation of adaptation. Unfortunately, no direct in vivo experimental evidence is available at present to elucidate this mechanism.

Burst firing, or spike train, neuronal responses contain both spike rate and spike timing information (Reich et al, 2000). For example, burst frequency (BF) and burst length (BL), which are linearly and nonlinearly proportional to the mean firing rate of neuronal response (DeBusk et al, 1997), can represent spike rate coding, whereas burst duration (BD) and the interspikes interval (ISI) within bursts can serve as determinants of spike timing (Dan et al, 1998; Usrey et al, 1998; Reich et al, 2000). Therefore, examining the burst firing properties of neurons during adaptation will provide an information in order to assess the changes in the neuronal signatures in the neural network (Snider et al, 1998). In this study, we found that both mean BD and ISI value of V1 neurons significantly increased during adaptation (Figure 4B,C), which indicates that neuronal activities from lateral or feedback connections might increase with adaptation. Additionally, we also found that the mean BF and BL of V1 neurons were significantly decreased during adaptation (Figure 3A, Figure 4A), suggesting that an inhibitory synaptic strengthening (DeBusk et al, 1997) may have also occurred. This finding was in direct contradiction to previous studies, which showed that manipulation of intracortical inhibition in adapted neurons had little effect on its adaptation strength (DeBruyn & Bonds, 1986; Vidyasagar, 1990). Likely, the decreased BF and BL of the adapted neuron are attributable to its presynaptic depression and/or postsynaptic somatic afterhyperpolarization. Based on polysynaptic connections between cortical pyramidal neurons and inhibitory interneurons (Szabados et al, 2006; Ren et al, 2007; Silberberg & Markram, 2007; Silberberg, 2008), we propose that, due to a decreasing BF and BL, the adapted neuron might gradually inactivate its inhibitory driven interneurons, which could in turn trigger more excitatory inputs from neurons in the surroundings or higher visual cortical areas to the adapted neuron by lateral or feedback connections, and thus result in an increase of BD and ISI, similar to what we found in this study.

In conclusion, adaptation of V1 neurons to visual stimulation decreased BF and BL, probably by presynaptic depression and/or postsynaptic somatic afterhyperpolarization, and thus contributed to firing rate reduction, whereas the spike timing change of the adapted neuron, as indicated by an increase of BD and ISI, might be caused by an improved neuronal activities from lateral and/or feedback connections. Therefore, an interplay of neuronal activities within local network circuitry, as indicated by reduced activities from feedforward connections and increased activities from lateral and/or feedback connections, could underlie the adaptation of V1 neurons to prolonged visual stimulation. Further investigations with in vivo multielectrode array recording and patch clamping techniques are needed to explore this speculation more fully.

**Biological implications of adaptation**

Due to limited psychophysical data, it remains unclear whether adaptation improves the detectability or discriminability of adapted stimuli (Abbonizio et al, 2002; Greenlee & Heitger, 1988; Maattanen & Koenderink, 1991) or novel stimuli (Dragoi et al, 2002; Greenlee & Heitger, 1988; Maattanen & Koenderink, 1991) or novel stimuli (Dragoi et al, 2002; Greenlee & Heitger, 1988; Maattanen & Koenderink, 1991) or novel stimuli (Dragoi et al, 2002; Greenlee & Heitger, 1988; Maattanen & Koenderink, 1991). According to current computational neuroscience research, burst firing in a neuron’s response is more effective than single spikes in eliciting a time-related response in its driven neurons, and longer bursts are more effective than shorter ones (Snider et al, 1998; Krahe & Gabbiani, 2004). In this study, we found that adaptation caused a significant reduction in burst frequency (BF) and burst length (BL), which suggested that the information transmitted by V1 neurons regarding the adapted visual stimuli actually became less reliable during adaptation. Therefore, we propose that adaptation may exacerbate the detection of adapted stimuli, but may also improve the sensitivity of the visual system to novel stimuli in the field of vision. Further psychophysical experiments are needed to clarify this issue.
References


