Effects of surround suppression on response adaptation of V1 neurons to visual stimuli

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Abstract: The influence of intracortical inhibition on the response adaptation of visual cortical neurons remains in debate. To clarify this issue, in the present study the influence of surround suppression evoked through the local inhibitory interneurons on the adaptation effects of neurons in the primary visual cortex (V1) were observed. Moreover, the adaptations of V1 neurons to both the high-contrast visual stimuli presented in the classical receptive field (CRF) and to the costimulation presented in the CRF and the surrounding nonclassical receptive field (nCRF) were compared. The intensities of surround suppression were modulated with different sized grating stimuli. The results showed that the response adaptation of V1 neurons decreased significantly with the increase of surround suppression and this adaptation decrease was due to the reduction of the initial response of V1 neurons to visual stimuli. However, the plateau response during adaptation showed no significant changes. These findings indicate that the adaptation effects of V1 neurons may not be directly affected by surround suppression, but may be dynamically regulated by a negative feedback network and be finely adjusted by its initial spiking response to stimulus. This adaptive regulation is not only energy efficient for the central nervous system, but also beneficially acts to maintain the homeostasis of neuronal response to long-presenting visual signals.

Keywords: Surround suppression; V1 neurons; Response adaptation; Cat

Viewing a long-presenting visual stimulus with specific patterns (e.g., orientation, motion direction and spatial frequency) often inhibits or perturbs perception of a subsequent test stimulus with similar attributes. This phenomenon is termed visual adaptation, and has attracted considerable attention since the 1960s (Clifford et al, 2007; Dao et al, 2006; Greenlee & Heitger, 1988; Hua et al, 2009; Kohn, 2007; Maffei et al, 1973; Marlin et al, 1988; Movshon & Lennie, 1979; Smith & Hammond, 1985). Since visual adaptation shows both evident interocular transfer and specificity to adapted stimulus attributes, it is generally regarded as a physiological process occurred in the cortical level, especially in the primary visual cortex (V1) (Duong & Freeman, 2007; Howarth et al, 2009), although subcortical neurons also exhibit a weak adaptation to visual stimulus (Brown & Masland, 2001; DeBruyn & Bonds, 1986; Smirnakis et al, 1997).

The neuronal mechanisms of adaptation to visual stimuli are still in debate (Hua et al, 2009; Kohn, 2007; Liu et al, 2013). The contrast gain control mechanism, which suggests a somatic afterhyperpolarization due to an increasing potassium ion current triggered by sodium ion influx during prolonged stimulation (Carandini & Ferster, 1997; Sanchez-Vives et al, 2000a; Sanchez-Vives et al, 2000b), cannot interpret the specificity of adaptation to stimulus attributes. Synaptic mechanisms can fully account for stimulus-specificity of adaptation

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but lack of consistent experimental evidences. Some studies highlight the roles of excitatory synaptic depression in the adaptation process (Chung et al, 2002; McLean and Palmer, 1996; Reig et al, 2006; Vidyasagar, 1990). Some suggest that the changes of inhibitory synaptic activities may contribute to the adaptation effects (Hua et al, 2009; Yang et al, 2003). Others propose that adaptation may be caused by a network mechanism concerning a relative weight of recurrent excitation and inhibition in local neural circuitry (Teich & Qian, 2003). An important factor underlying these discrepancies is that previous studies fail to directly assess the correlations of the changes of neuronal response adaptation and the changes of local excitation / inhibition. Studies on microiontophoresis found that administrations of glutamate, gamma-aminobutyric acid (GABA) and GABA receptor's antagonists fail to change the adaptation strength of visual cortical neurons (DeBruyn & Bonds, 1986; Vidyasagar, 1990). However, it is premature to conclude that inhibition is not involved in the adaptation process because: i) the actual effects of iontophoretic drug delivery may be challenged if drug diffusion time, diffusion range and synaptic spatial alignment were concerned; ii) regulatory mechanisms from inhibitory synapses other than GABAergic ones may exist in adaptation (Ego-Stengel et al, 2002; McLean & Palmer, 1996; Waterhouse et al, 1990). Moreover, we recently found that relative to young adults, the adaptation of V1 neurons in the aged brain with compromised intracortical inhibition is actually enhanced (Hua et al, 2009).

The spiking response of a Vl neuron to a high-contrast stimulus placed within its classical receptive field (CRF) can be suppressed by a simultaneously presented stimulus within the surrounding nonclassical receptive field (nCRF), especially by the one with the similar orientation, motion direction and spatial frequency (Cavanaugh et al, 2002b; Haider et al, 2010; Series et al, 2003; Webb et al, 2005). This phenomenon, termed surround suppression, is induced by the increased activation of local inhibitory interneurons that are driven chiefly by the lateral horizontal connections and / or the feedback from higher visual cortical areas (Bair et al, 2003; Durand et al, 2007; Haider et al, 2010; Li & Freeman, 2011; Series et al, 2003; Smith et al, 2006). Therefore, the local inhibition (Akasaki et al, 2002; Fu et al, 2010; Walker et al, 2000) on the surround-suppressed neurons can be regulated by the varying stimulus size outside the CRF.

In this study, grating stimuli of different sizes were presented outside the CRF to evaluate the effects of intracortical inhibition on the response adaptation of V1 neurons.

MATERIALS AND METHORDS

Animals

Four healthy young adult cats (2−3 years old) were examined ophthalmoscopically prior to experimentation to confirm that no optical or retinal problems impaired their visual function. All experiment procedures were performed strictly in accordance with the guidelines published in the NIH Guide for the Care and Use of Laboratory Animals.

Electrophysiological recording preparation

All cats were prepared for acute *in vivo* single-unit recording using a previously described method (Hua et al, 2010; Hua et al, 2009; Hua et al, 2006; Meng et al, 2013). Briefly, anesthesia was induced by injection 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. Glucose (5%)-saline (0.9%) solution containing a mixture of urethane (20 mg/h/kg) and gallamine triethiodide (10 mg/h per kg of body weight) was infused intravenously by a syringe pump to keep the animal anesthetized and paralyzed. Pupils were maximally dilated with atropine (1%) eye drops, and contact lenses (zero power) were used to protect the corneas from dryness. Neosynephrine (5%) was applied to retract the nictitating membranes. Artificial respiration was performed, and expired pCO_2 was maintained at approximately 3.8%. Anesthesia level was closely evaluated during the experiment by continuously monitoring the animal's heart rate (180−220 pulses/min) and electrocardiogram (ECG) throughout the experiment.

V1 was partly exposed (8 mm posterior to the earbar, 4 mm lateral to the midline) by removing the skull and dura over V1 (area 17) with the aid of a light microscope (77019, Reward, China). The small hole over V1 was filled with 4% agar saline solution prior to electrophysiological recording. The optic discs of the two eyes were reflected onto a movable transparent tangent screen positioned 57 cm from the animal's eyes and overlapped with a CRT monitor (resolution 1024×768, refresh rate 85 Hz) for visual stimuli presentation. The

area centralis of each eye was precisely located according to the position of the optic discs reflected onto the tangent screen (Bishop et al, 1962). After all the preparations were completed, single-unit recordings were performed using a glass-coated tungsten microelectrode (with an impedance of 3−5 MΩ) which was advanced by a hydraulic micromanipulator (Narishige, Japan). When the experiment was finished, the distance of each recorded cell's receptive field from the retinal central area was measured and calculated as visual acuity (1°/cm).

Visual stimuli and recording procedures

Visual stimuli were drifting sinusoidal gratings, which were generated in MATLAB with the aid of extensions provided by the high-level Psychophysics Toolbox (Brainard, 1997) and low-level Video Toolbox (Pelli, 1997). Once a cell's visually-evoked response was detected, the cell's receptive field center was preliminarily determined using bars of light emitted from a hand pantoscope and then precisely located by consecutively presenting a series of computer-generated flashing bars of light on the CRT. The cell's preferred stimulus attributes, including orientation, motion direction, spatial and temporal frequency were determined by comparing the cell's response to a series of grating stimulus packages. Then, the cell's responses to grating stimuli with optimal attributes but different sizes were recorded to build the response-stimulus size tuning curve (Figure 1A). We fitted the size tuning curve with a function described in previous papers (Cavanaugh et al, 2002a; Tailby et al, 2007):

$$R(x) = \frac{k_c \mathrm{erf}\,(x/w_c)^2}{1 + k_s \mathrm{erf}\,(x/w_s)^2} \quad (1)$$

Where, x is stimulus size, $R(x)$ is the neuronal response to a stimulus with size x, k_c and w_c are the gain and spatial extent of the center mechanism, k_s and w_s are the gain and spatial extent of the surround mechanism, erf is the error function.

From the fitting curve, we acquired three test stimulus sizes (a, b, c; Figure 1A), at which the cell's response reached maximum, half of the maximum and minimum value on the right side of the fitting curve, respectively. Size a is the optimal stimulus size that only stimulates the cell's CRF, but not induces surround suppression. Stimulus with size b and c can co-stimulate both CRF and nCRF, but may also evoke medium and maximum surround suppression, respectively.

The contrast of each stimulus was set at 100%. The mean luminance of the display was 19 cd/m^2, and the environmental ambient luminance on the cornea was 0.1 lux.

Data acquisition and analysis

Action potentials of the recorded cells were amplified with a microelectrode amplifier (Nihon Kohden, Japan) and a differential amplifier (Dagan 2400A, USA), and then fed into a window discriminator with an audio monitor. The original voltage traces (Figure 1C, E, G) were digitized by an acquisition board (National Instruments, USA) controlled by IGOR software (WaveMetrics, USA), and saved for on- or off-line analysis. A cell's response to a grating stimulus was defined as the mean firing rate (spontaneous response subtracted) corresponding to the time of stimulus presentation, which was used to acquire the curves of tuning response to stimulus orientations, temporal and spatial frequencies. The optimal orientation of each cell was obtained as previously described. The optimal temporal and spatial frequency were determined respectively by comparing the cell's response to high contrast (100%) grating stimuli with different temporal and spatial frequencies, and selecting the temporal and spatial frequency with the maximum response.

The adaptation index (AI) was defined as the ratio of the cell's mean response during plateau period of adaptation to visual stimulation, a period when the cell's response reached a stable minimum value, to the mean initial response of the cell (Figure 1D, F, H). The change of AI with different stimulus sizes was plotted for each studied cell (Figure 1B). The smaller the AI is, the stronger the adaptation of the cell becomes. In order to assess the impact of surround suppression on the response adaptation, several neurons that did not exhibit surround suppression to visual stimuli presented in its nCRF were excluded from our data analysis. All studied neurons had a receptive field within 8° from the central area of the dominant eye.

All values were expressed as mean±*SE*. Variations between different stimulus sizes and subjects were assessed using analysis of variance (ANOVA) or *t*-test.

RESULTS

A total of 61 V1 cells from four young male adult cats were analyzed in this study (Table 1). All cells

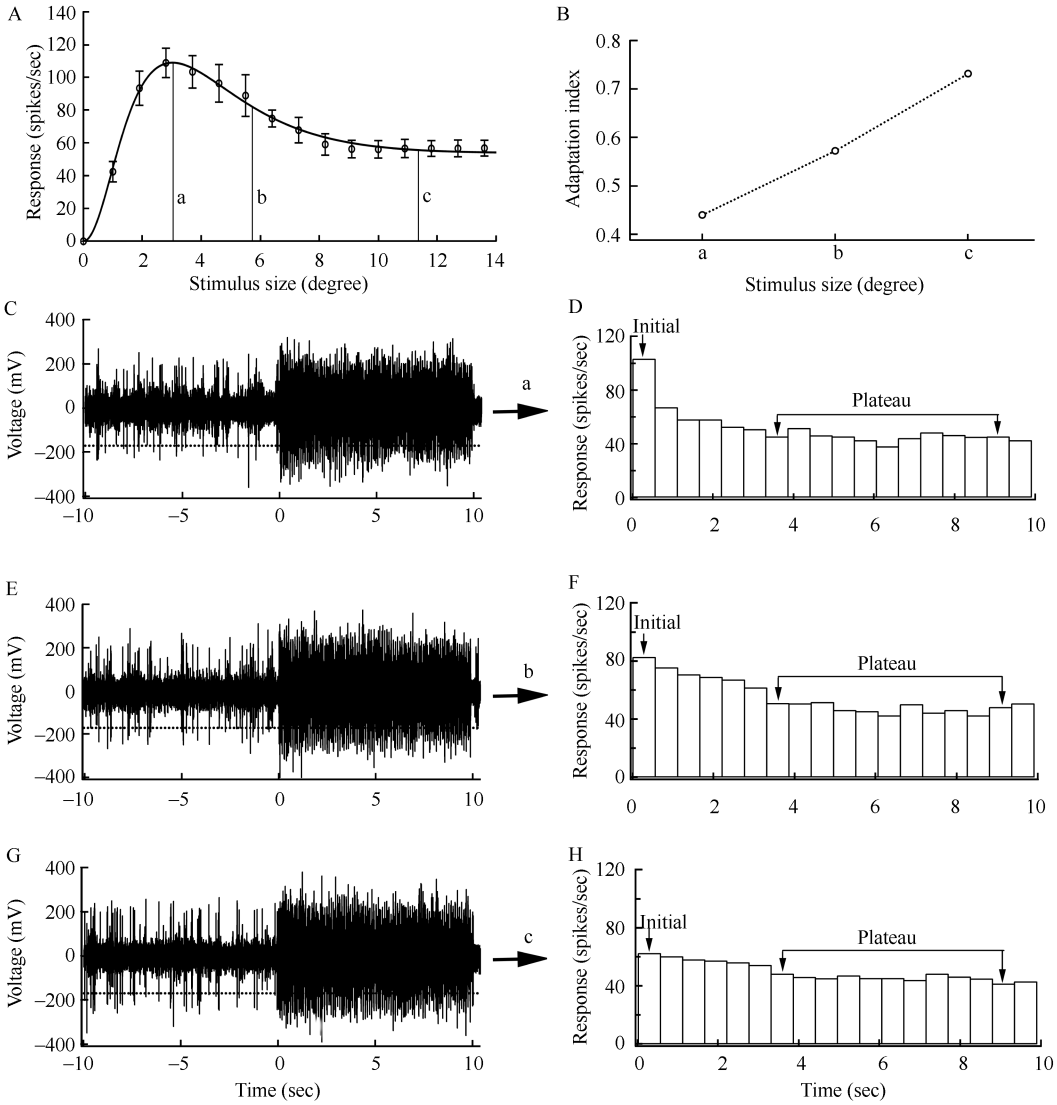

Figure 1 Response adaptation of a sample V1 cell to visual stimuli

A: The response-size tuning curve fitted with equation 1. B: AI changes with stimulus sizes. The AI at stimulus sizes a, b and c for this sample cell was 0.44, 0.57 and 0.73 respectively. Subsequently, the cell's response to prolonged stimulation (90 stimulus cycles) were recorded with three stimulus sizes, respectively, which were used to assess the cell's response adaptation changes with the magnitude of surround suppression. Each stimulus was presented monocularly to the dominant eye and repeated 4−6 times with a 3-minute interval between adjacent trials for the cell's functional recovery. Before each stimulus was presented, spontaneous activity was acquired during a 10 s period while a mean luminance was shown on the CRT. C, E and G: Voltage traces of the sample cell's response to 90 cycles of preferred visual stimuli with size a, b and c respectively, which were employed to evaluate the cell's response adaptation strength. Spontaneous activity was obtained during the first 10 s period while mean luminance was shown on the screen. The dashed horizontal line in each voltage trace indicated the threshold for action potential counting. D, F and H: PSTHs show the cell's average response (counted across each 5 stimulus cycles, with spontaneous activity subtracted) changes as a function of time. Spikes in the first bar were defined as the average initial response, and the mean spikes from the 7th to the 17th bar as an average response, a period when the cell's response decreased to a stable minimum level.

showed an evident adaptation to prolonged visual stimuli (90 stimulus cycles) as indicated by the AI value ranged from 0.104 to 0.760.

Changes of neuronal response adaptation with the stimulus size outside the CRF

The comparison of mean AI of studied neurons with three stimulus sizes (a, b and c) showed that the surround suppression effects on the adaptation strength of neuronal response to visual stimuli from weak to strong were a, b and c, respectively. The ANOVA analysis showed significant differences in the averaged AI value of all the studied neurons with three different stimulus

Table 1 Mean adaptation index of V1 neurons at different stimulus sizes in each cat

Subject	Cell number (*n*)	Adaptation index at different stimulus size		
		a	b	c
Cat1	19	0.33±0.021	0.41±0.028	0.45±0.028
Cat2	17	0.33±0.021	0.40±0.020	0.48±0.025
Cat3	13	0.28±0.034	0.36±0.036	0.46±0.033
Cat4	12	0.18±0.018	0.22±0.025	0.29±0.032

a, b and c: the stimulus size at which the cell's response reached maximum, half of the maximum and minimum value on the right side of the response-stimulus size fitting curve, respectively.

sizes ($F_{(2, 183)}$=25.7, P<0.0001). These differences were independent of subjects ($F_{(6, 183)}$=0.38, P>0.5), although the mean AI exhibited a significant variance from cat to cat ($F_{(3, 183)}$=22.4, P<0.0001) (Figure 2A). The mean AI of each individual cat was found significantly different with different stimulus sizes (cat1: $F_{(2, 57)}$=6.12, P<0.01; cat2: $F_{(2, 51)}$=9.472, P<0.001; cat3: $F_{(2, 39)}$=6.927, P<0.01; cat4: $F_{(2, 360)}$=4.935, P<0.05). The mean AI at stimulus size b was significantly less than that at stimulus size c (t-test, P<0.0001), whereas, was significantly larger than that at stimulus size a (t-test, P<0.0001) (Figure 2B, C), indicating that the neuronal response adaptation decreased with the increase of surround suppression. These results suggest that the response adaptation of V1 neurons to visual stimuli is negatively correlated with the surround suppression that was modulated by the stimulus size.

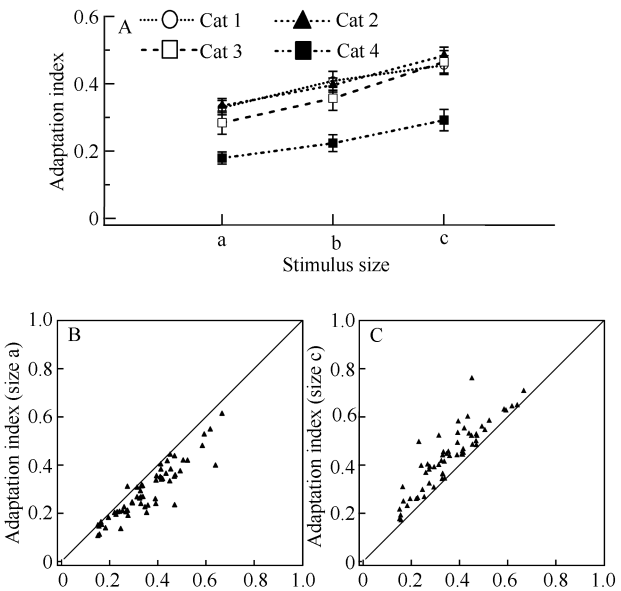

Figure 2 Average AI changes of studied neurons with different stimulus size

A: mean AI at different stimulus sizes of individual cat; B and C: AI at stimulus size a *v.s.* size b and size c *v.s.* size b of all the cells from all cats, respectively.

Changes of neuronal response with the stimulus size

The increase or decrease of AI could result from a change of the initial response (IR) of neurons to visual stimuli, a change of the plateau response (PR), the response during the plateau period of adaptation, or any combination thereof. As such, we compared the IR (the mean response to the first five cycles of visual stimuli) and PR (the mean response of visual stimuli cycles from the 36th to the 85th, which represents a minimal and stable response after adaptation) of V1 neurons to prolonged visual stimuli with different stimulus sizes, respectively.

ANOVA analysis showed significant differences in the averaged IR of the studied neurons with different stimulus sizes ($F_{(2, 183)}$=111.207, P＜0.0001＝. These differences were independent of subjects ($F_{(6, 183)}$= 0.536, P>0.5), although the mean IR varied significantly from cat to cat ($F_{(3, 183)}$=14.633, P<0.0001) (Figure 3A). The mean IR of each individual cat also showed significant differences at different stimulus sizes (cat1: $F_{(2, 57)}$= 21.810, P<0.0001; cat2: $F_{(2, 51)}$=75.240, P<0.0001; cat3: $F_{(2,39)}$= 23.203, P<0.0001; cat4: $F_{(2, 36)}$=20.203, P<0.0001). The mean IR at stimulus size b was significantly

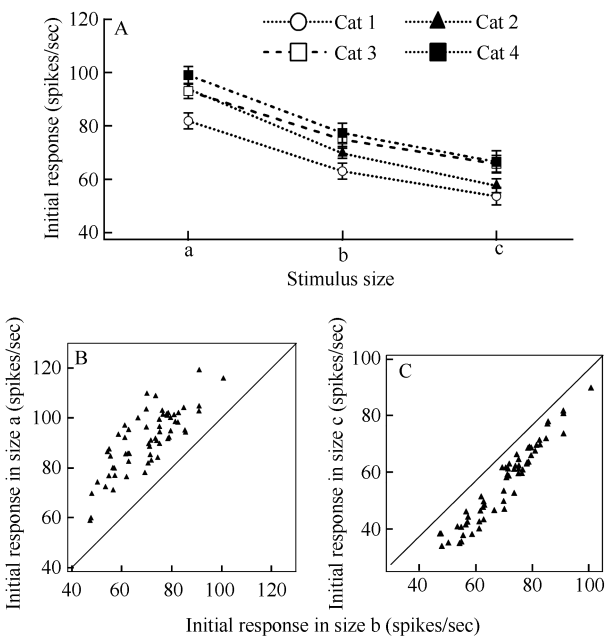

Figure 3 Average IR of neurons to prolonged visual stimuli with different sizes

A: The mean IR at different stimulus sizes of individual cat; B and C: The IR at stimulus size a *vs.* size b and size c *vs.* size b of all the cells from all cats.

larger than that at stimulus size c (*t*-test, $P<0.0001$), whereas, was significantly less than that at stimulus size a (*t*-test, $P<0.000001$) (Figure 3B, C), indicating that the IR of neurons to prolonged visual stimuli reduced greatly with the increase of surround suppression.

However, although the mean PR varied significantly from cat to cat ($F_{(2, 183)}=0.667$, $P<0.0001$), no significant differences were found in either all the studied neurons from all the cats ($F_{(2, 183)}=0.403$, $P>0.1$) or individual cat (cat1: $F_{(2, 57)}=0.466$, $P>0.5$; cat2: $F_{(2, 51)}=1.173$, $P>0.1$; cat3: $F_{(2, 39)}=0.561$, $P>0.5$; cat4: $F_{(2, 36)}=0.237$, $P>0.5$) with different stimulus sizes (Figure 4). These results indicate that the responses of neurons during the plateau period of adaptation to visual stimuli are stable and do not change significantly with the changes of surround suppression.

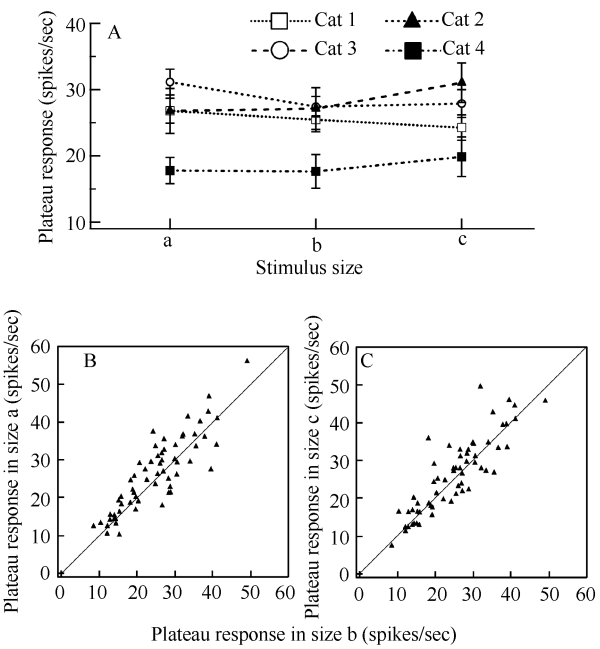

Figure 4 Average plateau response of neurons to prolonged visual stimuli with different sizes

A: The mean plateau response at different stimulus sizes for individual cat; B and C: the plateau response at stimulus size a *vs.* size b and size c *vs.* size b for all the cells from all cats.

Therefore, the response adaptation changes of the studied neurons with different surround suppression may attribute to the changes of their IR to prolonged visual stimuli, whereas, the PR maintains relatively stable.

DISCUSSION

Adaptation mechanisms

Visual cortical neurons exhibit a reduction in firing rate to prolonged visual stimulation. However, the underlying mechanisms remain in debate, and previous studies proposed several hypotheses. For example, the response adaptation is caused by the activity fatigue of the neuron because the prolonged stimulation may evoke sustained firing and the fatigued neurons respond less than they normally do (Carandini, 2000; Sekuler & Pantle, 1967). The contrast gain control mechanism suggests that adaptation leads to a strong somatic afterhyperpolarization due primarily to the activation of voltage-gated potassium channels, triggered by the sodium influx during generation of action potentials (Carandini & Ferster, 1997; Sanchez-Vives et al, 2000a; Sanchez-Vives et al, 2000b). Although the above two mechanisms can interpret the neuronal response reduction during visual adaptation, they are unfortunately unable to account for the specificity of adaptation to the adapted stimulus attributes, such as orientation.

To date, more studies support the synaptic mechanism due to its advantage in explaining the stimulus-specificity of visual adaptation. However, debates concerning the contributions of excitation and inhibition in local circuitry to the adaptation still exist. Some studies emphasize the importance of excitatory synaptic depression in mediating the adaptation process (Chung et al, 2002; Nowak et al, 2005; Reig et al, 2006). Some suggest an involvement of inhibitory synaptic activation in the adaptation effect, and others propose a network mechanism based on recurrent excitation and inhibition models (Kohn, 2007). Current evidences on the role of local inhibition in the adaptation process are mutually inconsistent. An *in vivo* study reported that the iontophoretic delivery of $GABA_A$ receptor antagonists could significantly improve the adaptation strength of relay cells in the dorsal geniculate nucleus (LGNd) and the administration of baclofen, a $GABA_B$ receptor agonist, could decrease the adaptation strength (Yang et al, 2003). However, similar manipulation of GABA inhibition failed to alter the amplitude of visual cortical cells to visual stimuli (DeBruyn & Bonds, 1986; Vidyasagar, 1990). Interestingly, V1 neurons of aged cats showed stronger adaptation to visual stimuli than that of young adult cats (Hua et al, 2009). This enhanced adaptation of V1 neurons during aging may indirectly suggest that neuronal response adaptation is correlated with the changes of intracortical inhibition (Hua et al, 2008; Hua et al, 2006; Leventhal et al, 2003).

In the present study, we evaluated the effects of surround suppression on the adaptation strength of V1

neurons. By manipulating the levels of surround suppression using different stimulus sizes outside the CRF, we found that the amplitude of response adaptation of V1 neuron decreased significantly with the increase of surround suppression due to the decrease of the neuron's IR to the adapted stimulus. These results indicate that the surround suppression might only modify a neuron's IR but not the adaptation process and the adaptation strength depends closely on the neuron's IR to the adapted stimulus. IR decreases with the increase of surround suppression and the decrease of response adaptation, vice versa. Therefore, the response adaptation of V1 neurons may under the dynamic regulation of a negative feedback mechanism. Our results, together with several recent findings (Benucci et al, 2013; Compte & Wang, 2006; Levy et al, 2013; Liu et al, 2013) suggest that visual adaptation may depend on a network mechanism that involves an interplay between inhibitory and excitatory neurons in the local neural circuitry.

Benefits of visual adaptation

The functional benefits of adaptation remain unclear due to the inconsistent evidences suggest that adaptation sometimes decreases sensitivity for the adapting stimuli, and sometimes it changes sensitivity for stimuli very different from the adapting ones (Gepshtein et al, 2013). Some studies claimed that adaptation could improve the detectability of the adapting stimuli (Abbonizio et al, 2002; Greenlee & Heitger, 1988; Määttänen & Koenderink, 1991). Others reported that adaptation increased perception of novel stimuli in the environment while suppressing the perception of adapted stimuli (Dragoi et al, 2002; Hosoya et al, 2005; Sharpee et al, 2006). Benucci et al (2013) measured adaptation in the response of populations of V1 neurons to stimulus ensembles with markedly different statistics of stimulus orientation, and found that adaptation might act as a mechanism of homeostasis by maintaining time-averaged response quality and orientation selectivity independence across the population of neurons.

In this study, we determined the response adaptation changes of V1 neurons with different degree of surround suppression. We found that the response adaptation of V1 neurons decreased significantly with the increase of surround suppression due to the reduction of its IR to the adapted stimulus, whereas, the response of neurons during the plateau period of adaptation remained stable. These results are consistent with previous studies (Cavanaugh et al, 2002a) and suggest that V1 neurons may dynamically adjust its adaptation strength according to its initial spiking activities evoked by the adapted stimulus: adaptation enhances if initial activities are high or otherwise weakens if initial activities are low. The response of the neuron can eventually be reduced to the similar level, which is independent of the amplitude of initial response. This adaptation strategy may be critical in maintaining the homeostasis of neuronal response to long-lasting visual signals and aiding the energy efficiency/frugality of brain activities (Hua et al, 2009).

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