Contrast response characteristics of long-range lateral interactions in cat striate cortex

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Single-cell responses in visual cortex to a target falling within their receptive field can be modified by collinear flanking stimuli concurrently presented outside the receptive field. Here, we report the presence of four types of contrast-dependent lateral effects: (1) facilitation at low target contrasts and suppression at high contrasts, (2) facilitation that increases with contrast, (3) suppression that increases with contrast, and (4) suppression at low contrasts with facilitation at high contrasts. We propose a *sensitivity modulation model* that accounts for all the four types of lateral effects by changes in two parameters. In this model, activation of neighboring neurons changes the sensitivities of the target neuron to both the direct feedforward input and inhibitory, divisive feedback from neighboring neurons. *NeuroReport* 12:655–661 © 2001 Lippincott Williams & Wilkins.

Key words: Contrast response function; Gabor patches; Gain control; Sensitivity modulation; Spatial summation

INTRODUCTION

Neurons in the visual cortex are excited by visual stimuli presented in a restricted part of the visual field, a region known as the classical receptive field (CRF). Responses to a stimulus presented within the CRF (target) are often modified by another stimulus concurrently presented outside the CRF (flanker) that, when presented alone, fails to elicit a response. The modulatory effect of the flanker on the response elicited by the target is known to depend on many factors, such as the orientation difference between the target and flanker [1–6] and differences in luminance contrast [6–9]. Flanker modulation usually operates most strongly for stimuli that are of the same orientation inside and outside the CRF [1–7]. Both suppressive [1,2,4–6,8–10] (response reduction) and facilitative [2,3,5,7–9] (response enhancement) interactions have been reported.

More recently, several groups have noted that the sign of the interaction (suppressive or facilitative) depends on the relative luminance contrast inside and outside the CRF [6–9]. Polat *et al*. [7] reported that collinear flankers comprising two Gabor patches tended to facilitate the response to a near-threshold Gabor target presented inside the CRF. Similar effects have been reported for disk/annulus arrangements of drifting gratings [8,9].

Here we show the presence of four types of contrast-dependent interactions between neural responses elicited by collinearly arranged Gabor patches, including the previously reported type (i.e. facilitation at low contrasts followed by suppression at high contrasts [7–9]). We present a novel contrast gain control model that can account for all the four interaction patterns using only variations in single excitatory and inhibitory gain parameters.

MATERIALS AND METHODS

Single-unit activity was recorded from six cats prepared for terminal experiments. The cats were anesthetized (N_2O:O_2:CO_2, 75:22.5:2.5, supplemented with continuous i.v. infusion of ~2 mg/kg/h pentobarbital) and immobilized by continuous i.v. infusion of either 10 mg/kg/h Flaxedil or 0.2 mg/kg/h Pavulon. Single-unit discharges were registered with tungsten-in-glass microelectrodes [11] (exposed tip 13–16 μm, impedance 1–3 M at 1 kHz). The electrodes were implanted in the posterolateral gyrus near the projection of the area centralis (stereotaxic coordinates: P 4 mm, L ~1.5 mm). Other details are given elsewhere [7,12]. The location, size and orientation preferences of the CRF of the isolated neurons were first determined by a moving bar of light or a flashing light slit. The spatial frequency and temporal frequency preference of the neuron were then determined by using contrast-reversing Gabor patches of the appropriate size.

The target pattern was a Gabor patch (Fig. 1a) with optimal parameters superimposed on the CRF of the neuron. The target was contrast-reversed at the neuron's preferred temporal frequency. Target contrast was varied between 0.5 and 80% and the contrast of the two flankers was constant at either 50 or 80%, depending on the

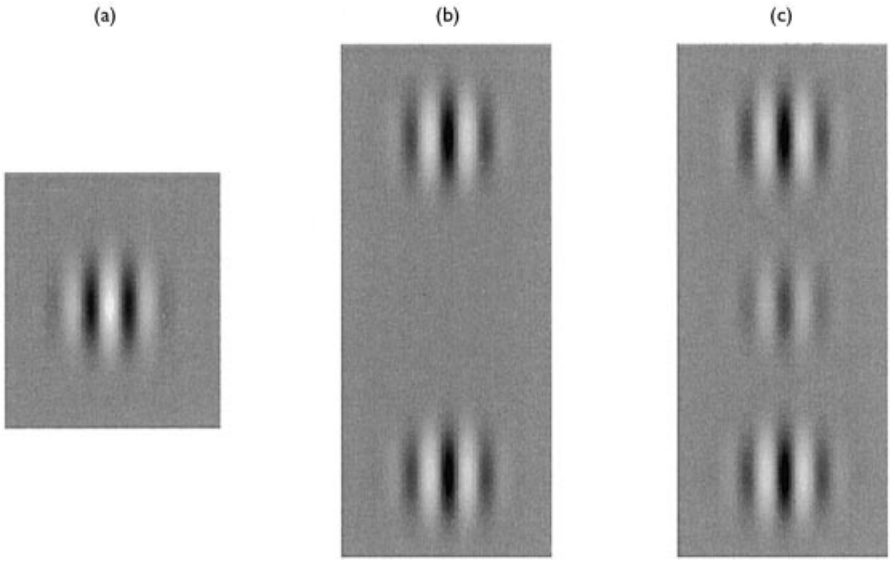

Fig. 1. Examples of the experimental stimuli: (**a**) target alone, (**b**) target plus flankers and (c) flankers alone.

neuron's contrast threshold for the target. The contrast threshold was estimated from the contrast–response function in two steps. An initial estimate in the swept-contrast method [13] was obtained by increasing target contrast in 10 equally spaced logarithmic values from low to high during a stimulation period of 10 s. The slope of the first linear portion of the contrast–response curve was linearly extrapolated to the zero amplitude. Then, stimulating at 10 contrast values around the initial estimate, we obtained another contrast–response curve which covered a *narrower* range of contrasts than before. Finally, threshold was determined by executing the same linear extrapolation on the second curve. The estimated threshold always falls to the left of the point at which the response rises above noise. Non-significant response amplitudes were excluded from the threshold estimation procedure and also all subsequent analyses [7].

Two collinear flanking Gabor patches were placed above and below the target pattern along the line defined by the preferred orientation of the cell (Fig. 1b). The centers of the flankers were located at four or more wavelengths of the Gabor carrier spatial frequency away from the center of the target. The flanker Gabor patches were of the same spatial frequency, temporal frequency, and orientation as those used to drive the target inside the CRF and the carriers have the same spatial phase. The flankers did not activate the cell when presented alone (Fig. 1c).

Contrast–response functions for the target alone and those for the combination of the target plus collinear flankers were measured by sweeping the contrast of the target Gabor patch in 10–24 equal logarithmic steps, from well below the cell's firing threshold to well above it in trials that lasted 10–30 s. Target alone, flanker alone and target plus flanker trials were presented in random order under computer control.

The amplitude and phase of the cell's response at the first and second harmonics of the stimulus frequency were determined by an adaptive filter. The filter had a memory length of 1 s, which was sufficiently short to track the time varying response *vs* contrast. Amplitude and phase values were coherently averaged in the frequency domain over all records collected under the same stimulus conditions. The classification of simple cells and complex cells was based on the average ratio of their first harmonic and second harmonic amplitudes across all records. A cell was classified as a simple cell if its average first harmonic amplitude was greater than its second harmonic; otherwise it was considered to be complex. Model fits were thus made to the amplitude function from the first harmonic for simple cells and from the second harmonic for complex cells.

RESULTS

The lateral interaction effects we have observed are of four types. An example of the flanker effect from each cell type is shown in Fig. 2. In each panel, the smooth curves are fits from the *sensitivity modulation model* described below. In type I cells (Fig. 2a), the collinear flankers increase the target response at low target contrasts, while they decrease the target response at high target contrasts. The two target contrast response functions, with and without flankers, thus show a *cross-over* of the sign of the lateral interaction with increasing target contrast, as reported previously [7–9]. In type II cells (Fig. 2b) the flankers produce only an increment in target response as target contrast increases and there is no suppression at low contrasts. We refer this effect as *expansive facilitation*. Type III, or *expansive suppression*, cells (Fig. 2c) show a suppression effect that increases with target contrast with no facilitation at low target contrasts. Previous studies [8,9] showed that expansive suppression is common (about 50% of cells) with annular surrounds, but we found that it is less frequent for collinear flankers (about 10% occurrence). Finally, type IV cells display an expansive facilitation at high target contrasts but show a suppressive flanker effect at low target contrasts (Fig. 2d). This behavior (reverse cross-over) is opposite to that seen in type I.

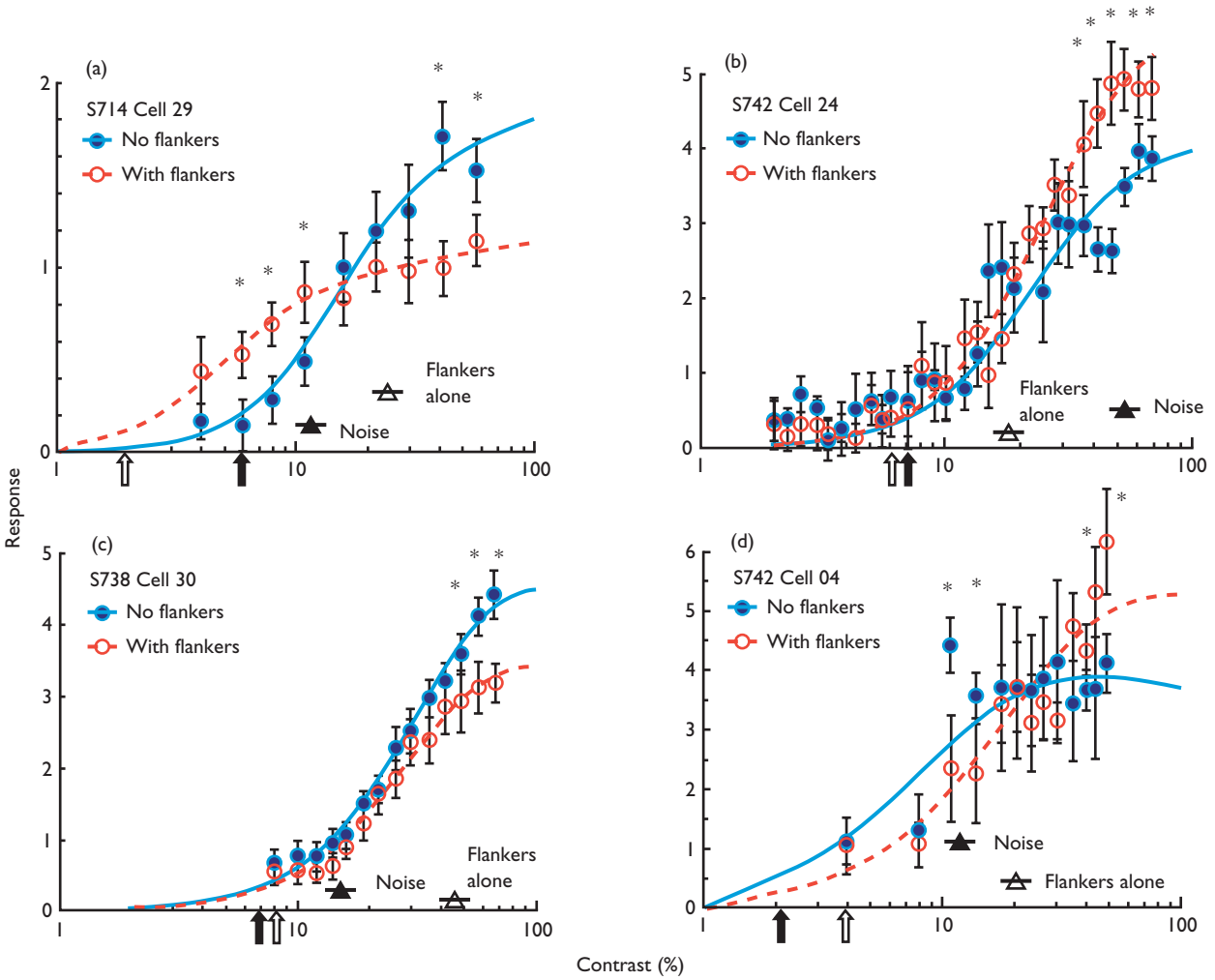

Fig. 2. Four types of lateral interactions. (**a**) Contrast-response function of a complex cell that shows cross-over effects, i.e. facilitation at low contrasts and suppression at high contrasts (Type I). The goodness of fit is $\chi^2(12) = 0.5105$, $p > 0.99$. A *t*-test at 8% contrast, where the largest difference was seen between with and without flankers conditions in the lower half of the functions, shows the flanker has a significant facilitative effect at low contrasts ($t(14) = 2.1764$, $p = 0.0234$, < 0.05). (**b**) A complex cell whose response function shows an expansive facilitation effect (Type II). That is, the flanker suppression increases with increasing target contrast. The goodness of fit is $\chi^2(22) = 2.4108$, $p > 0.99$. (**c**) A simple cell whose response function shows an expansive suppression effect (Type III). That is, the flanker suppression increases with increasing target contrast. The goodness of fit is $\chi^2(22) = 0.8034$, $p > 0.99$. (**d**) A complex cell response function that shows a cross-over effect, i.e. suppression at low contrasts and facilitation at high contrasts. The goodness of fit is $\chi^2(22) = 3.0583$, $p > 0.99$. A *t*-test at 12% contrast shows the flanker has a significant suppressive effect at low contrasts ($t(16) = 3.0275$, $p = 0.0040$, < 0.05). In all panels, solid and open arrows denote, respectively, the estimated threshold of cell without and with the presence of the flankers. The noise level (denoted as closed triangle) is the cell response to a blank screen. The response to flanker alone is denoted as open triangle. *denote where the two functions are significantly different from each other ($p < 0.05$ or stronger by *t*-test)

We tested the statistical significance of the lateral effects using a two-way ANOVA with factors of flanker present/absent and target contrast. The contrast response functions were considered to show a lateral effect only if the main effect of the flankers or the interaction between flanker and target contrast was significant at the $\alpha = 0.01$ level. The ANOVA is not a powerful test for non-linear functions like ours, but it is a conservative approach for determining the lateral effects. Type I and type IV cells showed facilitation and suppression, respectively, at low target contrasts. In these the effect was established with a *t*-test ($\alpha = 0.05$) at the target contrast that showed the greatest difference between the lower half of the two contrast–response functions.

Table 1 summarizes the occurrence frequency of four types of the lateral effect observed in the present population of 99 cells. In total, 37.4% of cells showed type I flanker effects (cross-over) and 29.3% cells showed a type II effect (expansive facilitation). There were 10.1% cells in Type III (expansive suppression) and 8.1% cells in Type IV (reverse cross-over). There were 15 cells (15.2%) showing no lateral effects. Within each of the five categories, the ratio of simple cells and complex cells were not notably different.

Model: The most salient feature of the flanker effect is that, no matter whether the flankers suppress (as in Type I and III) or facilitate (as in Type II and IV) the neural

Table 1. The sampling incidences of the four types of flanker effects with the distinction of simple and complex cells.

	Simple cell ($n = 46$)	Complex cell ($n = 53$)	Total ($n = 99$)
Type I (cross-over)	34.8%	39.6%	37.4%
Type II (expansive facilitation)	30.4%	28.3%	29.3%
Type III (expansive suppression)	13.0%	7.6%	10.1%
Type IV (reverse cross-over)	6.5%	9.4%	8.1%
No effect	15.2%	15.1%	15.2%

response at high target contrasts, the magnitude of the effect increases with target contrast. This result, combined with the fact that the flankers alone produce no response in the target cell, suggests that the lateral interaction acts like a *multiplicative* factor on target physical contrast. Our model incorporates this idea within currently available contrast normalization models of striate cell responses [14–16].

Figure 3 shows a schematic diagram of our model. The CRF of a simple cell (shown in the lower right corner) acts approximately like a linear operator. The sensitivity profile of a simple cell is usually modeled as a wavelet such as a Gabor patch or a difference of Gaussian [17]. The output of the linear operator to an input image L(x,y) is

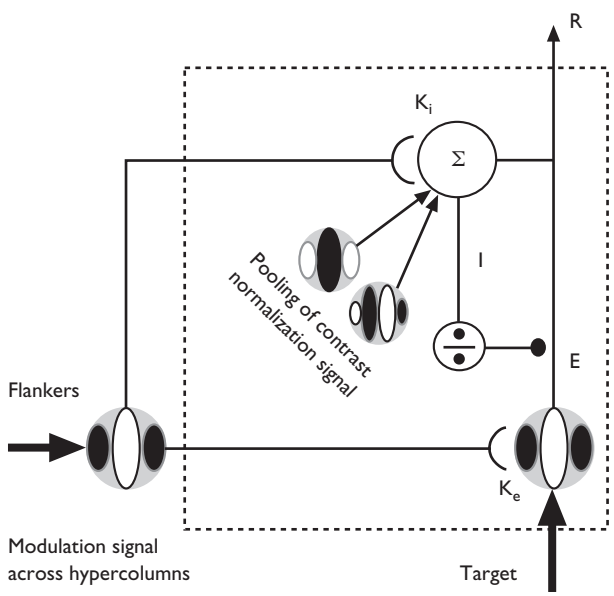

Fig. 3. The sensitivity modulation model. Within the same hypercolumn, enclosed here by a dotted line, the cell response and the interactions among cells are described by contrast normalization models [14–16]. The receptive field of the target cell acts as a linear filter. The initial excitation (E) is the contrast of the target pattern weighted by cell's sensitivity to that pattern. The initial excitations of all relevant cells are pooled together to form the contrast normalization signal (I). The final response is the initial excitation raised by a power and then divided by the normalization signal plus a constant. The flanking cells from other hypercolumns send fibers to contact both the target cell and the intermediate cells that pools normalization signals. The signal from the flanking cells changes the sensitivities of the contacted cells. See the text for further details.

$$E_j = \int_{-\infty}^{\infty} F_j(x, y) \times L(x, y)dxdy \quad (1)$$

where $F_j(x,y)$ is the sensitivity profile of the j-th cell. If the image is a periodic pattern with contrast C, eqn 1 can be simplified to

$$E_j = S_{e,j} \times C \quad (2)$$

where $S_{e,j}$ is a constant called the excitatory sensitivity of the j-th cell. The CRF of complex cells can be modeled as a combination of rectified CRFs of relevant simple cells [15]. The output of the CRF of the j-th complex cell to a periodic image can also be written as

$$E_j = \Sigma_k[S'_{e,k} \times C] = S_{e,j} \times C \quad (2)'$$

where [ ] denotes a rectification operation and $S'_{e,k}$ is the sensitivity of the k-th linear operator that contributes to the CRF of the j-th complex cell.

The initial response of the cell is the linear operator output raised to a power to capture the transducer non-linearity [16]. According to the contrast normalization models [15,16], this initial response is raised by another power and pooled together with the initial response of other relevant cells to produce the contrast normalization signal. The *contrast normalization signal* to the j-th cell is

$$I_j = \Sigma_m(S'_{e,m} \times C^p)^q = S_{i,j} \times C^{pq} \quad (3)$$

where $S_{i,j} = \Sigma_m\ (S'_{e,m})^{pq}$ is called the inhibitory sensitivity of the j-th cell. This contrast normalization signal is then fed back to the cell to produce the final response. The final response is the initial response divided by the contrast normalization signal plus a constant. That is, the final response R_j of j-th cell is

$$R_j = E_j^p/(I_j + \sigma') \quad (4)$$

where σ' is an additive constant.

Unlike the current contrast normalization models, which implement the network interaction by means of pooling contrast normalization signals across cells (eqn (3), we propose here that the lateral interaction between cells modulates both the excitatory and the inhibitory sensitivities of the target cell. The lateral cells thus affect the recorded cell's sensitivity to both feedforward inputs as well as feedback inputs. Rather than increasing or decreasing the magnitude of either feedforward or feedback signals, the lateral cells change the gains of the target cell to both signals. This operation has the same effect as changing both the excitatory and inhibitory sensitivity of the cell to the target. Thus, the response of the cell under lateral influence is

$$\begin{aligned} R_j &= (K_e \times E_j)^p/(K'_i \times I_j + \sigma') \\ &= (K_e \times S_{e,j} \times C)^p/(K'_i \times S_{i,j} \times C^{pq} + \sigma') \end{aligned} \quad (5)$$

where K_e and K'_i are sensitivity modulation factors from the cells activated by flankers. When there is no lateral stimulus presented, K_e and K'_i is set to 1 such that the response function can be reduced to eqn (4). For our

purposes, eqn (5) can be further simplified by rearranging the parameters. That is:

$$R_j = M \times (K_e \times C)^p / ((K_i \times C)^{pq} + \sigma) \quad (6)$$

where $M = S^p_{e,j}/S_{i,j}$, $K_i = (K'_i)^{1/pq}$, and $\sigma = \sigma'/S_{ij}^{1/pq}$. This arrangement has several benefits. First of all, it reduces the number of the parameters in the model. Second, if $q = 1$, the response function has the same hyperbolic form used in the earlier literature [18] with M corresponding to the maximum response and σ to the semi-saturation constant. A non-unity value of q is required if we want to fit over-saturation [19] (i.e. the cell response decreases with target contrast after reaching a maximum response) of the contrast response function. Both K_e and K_i are required to account for all types of lateral interactions.

Figure 4 illustrates how different values of K_e can influence the response function, while K_i is held constant and *vice versa*. In this exercise, K_e was varied from 0.7 to 2.0 in half-octave steps, while K_i was held constant at 1.0 (Fig. 4a) and *vice versa* (Fig. 4b). It is obvious that K_e is required to account for the facilitation effect while K_i is required for the inhibition. Different combinations of K_e and K_i values, which are multiplicative factors expressed in a ratio scale, thus produce the four possible types of lateral interactions. The model fits the data well. The smooth curves in Fig. 2a–d are fits of this model. We used a χ^2 test to the goodness-of-fit and found all but three out of 99 cells fitted have a χ^2 value $\alpha < 0.1$ level. The three cells that have poor model fits show an irregular response function that is difficult to fit by a smooth function.

Except for the two sensitivity modulation factors, K_e and K_i, the fitted values of all other parameters are about the same across all the four types of lateral effects. Table 2 lists the mean and one standard error of K_e and K_i values for each of the four lateral effect types shown in Fig. 2a–d. Both K_e and K_i differ dramatically between type I lateral effects and all other three types. K_e and K_i are both much larger for cells with type I lateral effects than any other types, type IV showing the smallest values. In type I cells, K_i is twice larger than K_e, while in type II cells K_e roughly equals K_i. K_i for type II is about unity, suggesting that the feedback normalizing signal is almost free of the lateral effects. K_e for type III is also near unity, likewise suggesting that the feedforward signal is almost free of the lateral effects. These results imply that the cells with type II effects are less sensitive, as compared to type I cells, to inhibitory lateral inputs, while moderately sensitive to excitatory lateral inputs. In short, type I cells show strong lateral effects on both excitatory and inhibitory inputs, the latter being stronger than the former (Fig. 2a). The type II (Fig. 2b) and III (Fig. 2c) effects are more an increase in excitatory and inhibitory input at a moderate level, respectively (K_e and K_i, about 1). Type IV shows a substantial reduction in both excitatory and inhibitory sensitivities (Fig. 2d), receiving weak lateral effectss on both feedforward and feedback inputs. The difference between simple cells and complex cells in all lateral interaction categories is rather small, suggesting that they are subjected to similar long-range interaction processes. Likewise, the cells with type III effects are more sensitive to inhibitory lateral inputs.

Table 2. Mean (± s.e.) values of the two sensitivity modulation parameters, excitatory K_e and inhibitory K_i, in terms of cell types (i.e. simple *vs* complex cells) and flanker effect types (type I, II, III and IV).

	K_e	K_i
Type I (cross-over)	2.44 ± 0.42	4.79 ± 1.40
Type II (expansive facilitation)	1.15 ± 0.10	0.95 ± 0.11
Type III (expansive suppression)	0.94 ± 0.14	1.36 ± 0.36
Type IV (reverse cross-over)	0.76 ± 0.10	0.77 ± 0.14

DISCUSSION

We have found that there are four types of lateral effects in cat striate cortex: facilitation at low contrasts followed by

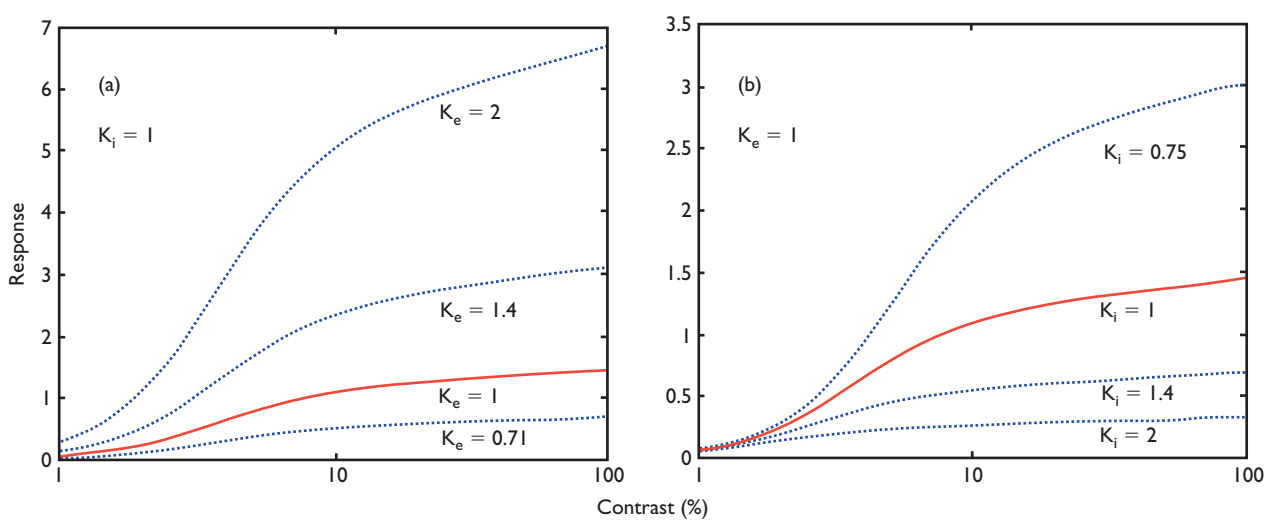

Fig. 4. The effects of two sensitivity modulation parameters on contrast-response function. (**a**) The parameter K_i is fixed at 1.0, while K_e varies from 0.7 to 2.0 at a half-octave step. Increasing K_e produces a facilitation effect in the response function and this facilitation increases with an increase in contrast. (**b**) The parameter K_e was fixed at 1.0, while K_i was varied from 0.7 to 2.0 in half-octave steps. Increasing K_i has an opposite effect from that of increasing K_e. The suppression effect by a greater K_i also increases with contrast.

suppression at high contrasts (type I, see also [7]), a facilitative effect that increases with target contrast (type II), a suppression effect that increases with target contrast (type III) and suppression at low contrasts followed by facilitation at high contrasts. We model lateral interaction across hypercolumns as a sensitivity modulation process affecting the impact of both feedback and feedfoward inputs. This model captures the basic features of all types of lateral effects, with a change in the two sensitivity modulation factors, K_e and K_i.

Our data provide a challenge to other models of striate-cell responses and network interactions. For example, the contrast normalization model [14–16], as described above, implements network interactions in terms of a pooling of contrast normalization signals. Thus, the presence of a lateral stimulus just adds another term to the contrast normalization pool. If the flanker contrast is held constant, as in our experiment, the effect of the flankers becomes negligible at high target contrasts because of the relatively large effects of the target itself (feedforward input). The response function with the flankers should move closer to the response function without flankers as target contrast increases. These predictions contradict the current data which have shown that lateral effects increase with target contrast in all the four types of cells (Fig. 2a–d).

The local circuit model proposed by Somers *et al.* [9] suggested a response function that based on hyperpolarizing, rather than divisive inhibition. In their model, each neuron receives both excitatory and inhibitory inputs. The neuron has a lower threshold for excitatory than for inhibitory inputs. The neuron subtracts the inhibitory input raised to a power q from the excitatory input raised to a power p to produce the final response. To obtain a sigmoidal contrast response function, q is set larger than p. The accelerating part of the contrast response function at low contrasts is due to the excitation acting unopposed by inhibition. When contrast increases beyond the threshold for inhibition, inhibition begins to take effect. Since $p < q$, the inhibition eventually catches up with the excitation and bends the response function over. Somers *et al.* [9] showed that their model, with appropriately chosen parameters such as increasing the excitatory and the inhibitory gains of the cells, could explain the type I lateral interaction effects. However, this model can never explain the type II effects. In order to obtain the sigmoid response function, inhibition has to outrun excitation at high contrasts. However, to account for the type II effect, excitation has to outrun inhibition. These contradicting requirements are beyond the scope of the local circuit model.

Sengpiel *et al.* [8] have suggested that there are two modulation types, response-gain control (equivalent to our Type III) and contrast-gain control (equivalent to our Type I), are mediated by different cell populations, because the former is often seen among layer 4 simple cells and the latter, outside layer 4 involving both simple and complex cells. Though this simplification is attractive, it remains to be seen whether similar distinction exists between the four types of modulation shown in the present study. First, we failed to detect a correlation between the modulation type and laminar loci of recorded cells in a given type (see Table 3). Second, the relative incidence of lateral effects was different in the two studies: in ours type I (cross-over with high-contrast suppression) is predominant in about of 40% cases and type III (expansive high-contrast suppression) occurs in 10%. In the study of Sengpiel *et al.* [8], expansive suppression (response-gain control) was dominant (48%), although the contrast-gain control was observed at a moderate frequency (36%), comparable with type I here. Moreover, type II and IV modulation which shows facilitation at high contrasts, with or without cross-over behavior, was not seen by Sengpiel *et al.* [8]. The presence of this type of modulation has been suggested, though not explicitly, in our early study (Fig. 3b of [7]).

Table 3. The sampling incidences of four types of flanker effects in relation to the laminar location of cells.

	Layer 2/3	Layer 4	Layer 5/6
Type I (cross-over)	5	6	21
Type II (expansive facilitation)	3	7	17
Type III (expansive suppression)	3	2	4
Type IV (reverse cross-over)	2	0	5

Among 84 cells that show a flanker effect, recorded loci of 75 cells were identified on Nissl-stained histological sections. There is no effect of location on the types of the flanker effect (χ^2 (6) = 6.59, $p = 0.36 > 0.05$).

How can the sensitivity modulation be implemented biologically? The presentation of the flankers alone produces no spike response in the target cell, though delayed subthreshold depolarizing responses were obtained by stimulating the CRF with a large pattern [20]. Therefore, the lateral interaction cannot be directly implemented as an increment or decrement in the cell's membrane potential. Nevertheless, the presence of the flankers does change the cell's firing sensitivity to the target, consistent with voltage-dependent enhancement of EPSP [21] which may be mediated by NMDA receptor activation [22]. Dendrites of pyramidal cells rich in voltage-gated Ca^{2+} channels are excitable [23] and dendritic depolarization or spiking, when coincident with somatic spiking, may facilitate opening of NMDA channels producing a further influx of Ca^{2+} [24]. Thus, the lateral excitation and inhibition are likely to be implemented first as changes in the voltage-sensitive membrane conductance at distal dendrites of the target cell.

Furthermore, for the majority of cells recorded from cat visual cortex, NMDA iontophoresis amplify cell responses to visual input without affecting cells' spontaneous activity, while the minority of neurons showed an opposite effect [25]. This type of response-gain change is similar to the sensitivity modulation we have proposed here. Therefore, it is possible that the axon collateral fibers from neighboring cells activate the NMDA receptors both on the target cell and the cells that pool normalization signals within the hypercolumn. In either way, through activation of NMDA channels, the flankers may modulate the sensitivities of the target cell to both the direct visual inputs and to the indirect, normalization signals.

CONCLUSION

Receptive-field responses in visual cortex are often modified by collinear pattern stimuli concurrently shown outside the receptive field. The mode and magnitude of the collinear flanker modulation depend on contrast of the

target stimulus placed on the receptive field. There are four types of contrast-dependent modulation. None of the currently available mechanisms/models can explain the divergent cellular behavior consistently. A novel sensitivity modulation model was presented to account for all the four types by the selection of one *excitatory* and one *inhibitory gain parameter*. It is suggested that the multiplicative effect (global context) provided by flanking stimuli modulates the additive effect (local context) related to contrast normalization *before* it is subjected to non-linearities.

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