

Brightness–Color Interactions in Human Early Visual Cortex

Dajun Xing,¹ Ahmed Ouni,² Stephanie Chen,² Hinde Sahmoud,² James Gordon,^{2,3} and Robert Shapley²

¹State Key Laboratory of Cognitive Neuroscience and Learning & IDG/McGovern Institute for Brain Research, Beijing Normal University, Beijing 100875, People's Republic of China, ²Center for Neural Science, New York University, New York, New York 10003, and ³Psychology Department, Hunter College, New York, New York 10065

The interaction between brightness and color causes there to be different color appearance when one and the same object is viewed against surroundings of different brightness. Brightness contrast causes color to be desaturated, as has been found in perceptual experiments on color induction and color-gamut expansion in human vision. However, it is not clear yet where in the cerebral cortex the brightness–color interaction that causes these major perceptual effects is located. One hypothesis is that brightness and color signals are processed separately and in parallel within the primary visual cortex V1 and only interact in extrastriate cortex. Another hypothesis is that color and brightness contrast interact strongly already within V1. We localized the brightness–color interaction in human V1 by means of recording the chromatic visual-evoked potential. The chromatic visual-evoked potential measurements decisively support the idea that brightness–color interaction arises in a recurrent inhibitory network in V1. Furthermore, our results show that the inhibitory signal for brightness–color interaction is generated by local brightness contrast at the boundary between target and surround, instead of by the luminance difference between the interior of the color target and its large background.

Key words: brightness; color; cVEP; inhibition; visual cortex; visual perception

Introduction

The interaction between brightness and color can change the color appearance of an object very markedly. For example, an object is most colorful, most saturated with color, when there is color contrast between the object and its surroundings but also when there is no brightness contrast. Perceived color saturation can be washed out almost completely when there is a large brightness contrast between an object and its surroundings. Examples of the power of increased brightness contrast to reduce color saturation are given in Figure 1A. Figure 1A (middle, red and green rectangles) are approximately equally luminant with their surroundings and therefore are maximally colorful. The same color rectangles placed on the black surroundings in Figure 1A (bottom) are much less colorful. As demonstrated in Figure 1A (top), the effect of brightness contrast is mainly happening at the edges of objects; in Figure 1A (top), we added thin black bands around the edges of the color targets placed on surroundings equal in luminance to the rectangles, and the color was almost as desaturated as on the full black surround in the bottom panel.

Such brightness–color effects can be observed in natural images; for instance, Figure 1B shows that the color appearance of natural visual objects, such as flowers, can be strongly affected by brightness contrast with their surroundings. Recently, precise psychophysical experiments were designed to measure the desaturation of human color perception by brightness contrast (Faul et al., 2008; Bimler et al., 2009); we performed similar experiments (see below).

However, it is not clear yet where in the cerebral cortex such perceptually significant brightness–color interaction takes place. One hypothesis, the classical view of color and brightness derived from ideas about color encoding, originally proposed by Hering in the 19th century (Hurvich and Jameson, 1957), is that brightness and color signals are processed separately and independently and in parallel (Fig. 2A) in early vision and only interact at some high perceptual level well beyond the primary visual cortex (V1). Another hypothesis is that color and brightness contrast interact as early as V1 (Fig. 2B) (Johnson et al., 2008). Here we show that the second hypothesis is correct by means of recording the human chromatic visual-evoked potential (cVEP). The behavior of cVEP from V1 decisively supports the idea that brightness–color interaction arises in a recurrent network in V1, in an inhibitory manner. Furthermore, the results provide direct electrophysiological evidence that color–brightness interactions take place very locally at the edges between a color target and its surroundings. Recurrent inhibition in local cortical circuits has been called a canonical cortical computation, and here we show that this canonical computation implemented in V1 cortex has a strong influence on color perception.

Materials and Methods

All observers in both psychophysics and EEG experiments gave informed consent to participate in this study. The experiments were conducted in

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Correspondence should be addressed to Dr. Dajun Xing, State Key Laboratory of Cognitive Neuroscience and Learning & IDG/McGovern Institute for Brain Research, Beijing Normal University, No. 19, XinJieKoWai ST, HaiDian District, Beijing, 100875, People's Republic of China. E-mail: dajun_xing@bnu.edu.cn.

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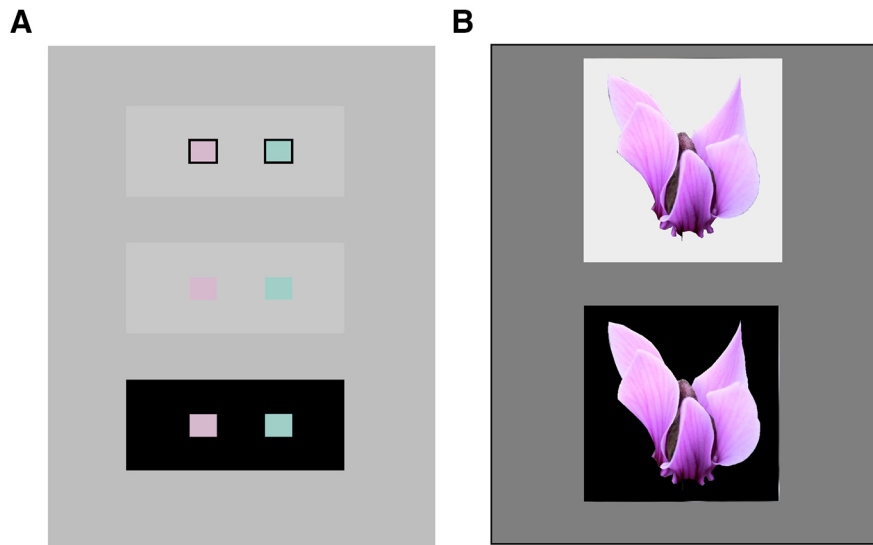


Figure 1. *A*, Color appearance is strongly affected by surrounding context. The red targets all have the same physical wavelength spectrum, but the perceived color saturation is greatest when brightness contrast between target and local surround is near zero in the middle panel; likewise for the green targets. In addition, a thin black line surrounding the color target strongly affects the color appearance as is apparent in the top. *B*, Color appearance of natural images also is greatly influenced by luminance contrast between target and surroundings. An image of a cyclamen flower, copied from the Internet, placed on a light gray background that is approximately equiluminant with the flower image (above) and on a black background (below), to illustrate the effect of luminance contrast on color appearance of the flower's petals.

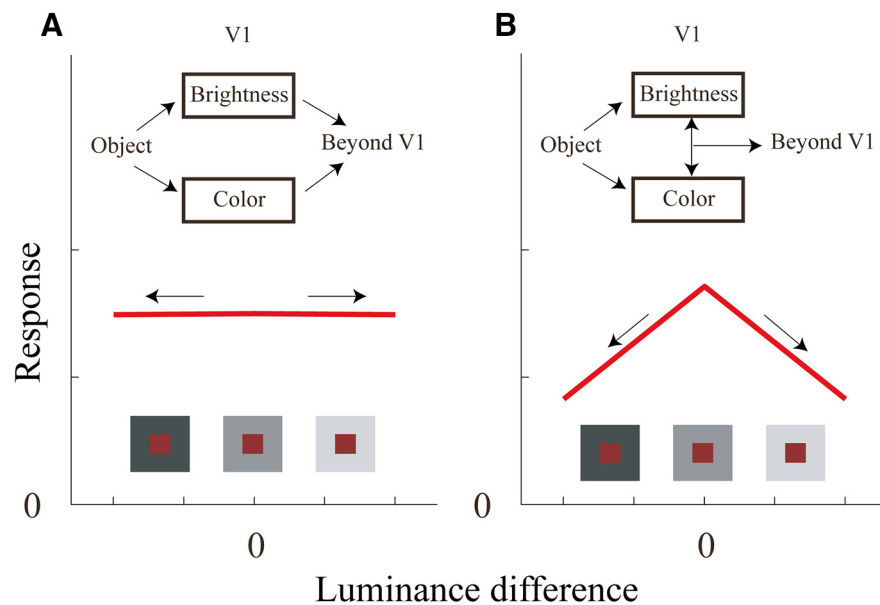


Figure 2. Two hypotheses for brightness–color interaction and their response prediction. *A*, Demonstration of the parallel-channels hypothesis. According to this hypothesis, the population responses should be constant regardless of the brightness contrast. *B*, Demonstration of the mutual-inhibition hypothesis. According to this hypothesis, the population responses should be decreasing as the brightness contrast (luminance difference) deviates from zero.

accordance with the principles embodied in the Declaration of Helsinki and were approved by the Hunter College/City University of New York and the New York University Institutional Review Boards.

Psychophysics experiments

Participants. Five observers (four females and one male between the ages of 20 and 23) participated in this experiment. Color vision was assessed with Ishihara plates, and all observers were color normal.

Apparatus. Square 1.15° foveal chromatic target stimuli with a luminance of 20 cd/m² were surrounded contiguously by achromatic surrounds. Surround luminances ranged from 0 to 40 cd/m². All stimuli

were presented using a Dell p780 monitor set at a refresh rate of 100 Hz. The full screen display was comprised of 1025 × 768 pixels. The screen was viewed at a distance of 1 m. Stimuli were calibrated with a Photo Research Spectrascan 670 spectroradiometer. Red and green chromatic stimuli (dominant wavelengths of 612 and 535 nm) were each presented at a medium value of color excitation purity (CEP = 0.15). CEP is defined as the quotient of the distance in CIE *xy* color space from the white point to the stimulus, divided by the distance from the white point to the spectrum locus of the stimulus' dominant wavelength.

Saturation scaling procedure. We used five surround luminances (0, 10, 20, 30, and 40 cd/m²); note that the 20 cd/m² surround luminance was equiluminant to the 20 cd/m² chromatic stimuli. Participants used saturation scaling to indicate their perception of the relative color saturation of the color target when it was located on the different surrounds. The saturation scaling technique, first introduced by Hurvich and Jameson (1957, 1959), asks the observer to describe the percentage of the total sensation (achromatic plus chromatic) that can be attributed to color. In other words, a total absence of hue would be indicated by 0% saturation, whereas a target that is completely saturated and lacking of any achromatic sensation would be 100% saturation. Saturation scaling has been shown to be a rapid and reliable method for comparing relative hue quantity (Gordon et al., 1994). The chromatic stimuli were each rated five times in each of the five surrounds. Stimuli were presented for 3 s with a 20 cd/m² gray background on during the 15–20 intertrial interval. This procedure minimized carryover sensory effects. Scaling values for five repeats of each presentation were averaged.

cVEP experiments

Participants. Thirteen observers (eight males and five females), ranging in age from 17 to 70 years (mean age = 32 years, median = 23 years), participated in the cVEP experiments. Data from one of the 13 subjects were excluded for further analysis due to bad quality of VEP signals. Another subject only participated in the cVEP experiment for the color red. The participants in the cVEP experiments were different from those in the psychophysics experiments. Color vision was assessed with HRR Pseudo-Isochromatic Plates. All observers were color normal. Observers had normal (at least 20 of 25) or corrected-to-normal visual acuity at the viewing distance of 114 cm.

Stimuli. A Venus system (Neuroscientific)

was used for presenting stimuli and recording electrophysiological responses. A Nokia monitor (Multigraph 447×, 90 Hz frame rate) set to a mean luminance of 20 cd/m² was used as a stimulus display. The display size was 20 × 20 cm, giving a field size of 10 × 10° at the viewing distance of 114 cm. Targets were viewed binocularly and were calibrated with a Photo Research PR670 Spectrascan Radiometer/Photometer.

The stimulus pattern was a set of 13 1° squares: one square at fixation was surrounded by a circular array of 12 squares. The center of each peripheral square was set 4° from the center of the central square. The luminance of all squares was 20 cd/m². In each experiment, the color

Table 1. CIE *xy* values and the color excitation purities of the red and green stimuli used in Figure 4

	<i>x</i>	<i>y</i>	Excitation purity
Reds	0.4587	0.3373	0.4024
	0.4050	0.3423	0.2406
	0.3814	0.3411	0.1690
	0.3606	0.3388	0.1055
	0.3349	0.3360	0.0272
Greens	0.2986	0.5764	0.5948
	0.3012	0.5312	0.4841
	0.3092	0.4634	0.3170
	0.3135	0.4011	0.1647
	0.3236	0.3305	0.0125

squares were modulated (rectangular wave in time) periodically at a temporal frequency of 0.5 Hz. Each stimulus cycle consisted of a transition from gray to equiluminant color (0.5 s on) and a transition back to gray (color temperature 5800°K, degrees Kelvin; 20 cd/m²; 1.5 s on). The 2 s period was repeated 30 times for run durations of 60 s. In a given run, all stimulus squares were the same color. CIE *xy* values of the red and green stimuli (dominant wavelengths of 614 and 543 nm, respectively) and their CEPs are given in Table 1. Both red and green color points are on straight lines in CIE space that run from the white point to their respective dominant wavelengths on the CIE spectrum locus. The equivalent color temperature of the white point was 5800°K (degrees Kelvin). In each run, the 10° × 10° background in which the checks were embedded was set to one of the following luminance values: 0, 10, 20, 30, and 40 cd/m². In the experiments for Figure 6, the stimulus squares were surrounded by a thin black band (6') separating them from the gray surround.

cVEP recording. Gold-cup electrodes were placed along the midline of the scalp according to the international ten–twenty system (Klem et al., 1999): active electrode at Oz (near the occipital pole), reference electrode at Cz (vertex of the head), and ground electrode at Pz (midway between Oz and Cz). The EEG, recorded in synchrony with the stimulus, was amplified (20,000), bandpass filtered (0.1–100 Hz), digitized at a rate of 180 Hz (two samples per frame of the Nokia display), and stored in a computer for analysis.

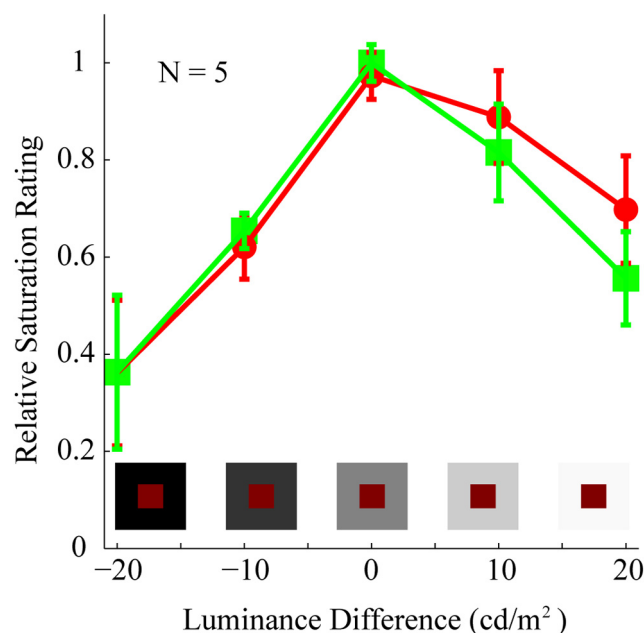
Analysis. Responses were averaged across the 30 presentations. The cVEP usually had a single negative going wave followed in some observers by a positive rebound (Crognale, 2002; Crognale et al., 2013). Response strength was quantified by calculating the rms power of the response waveform around the baseline, averaged over the 0.5 s period of visual stimulation.

Results

Psychophysics

Because it is so important for color perception, we sought to replicate prior results by Faul et al. (2008) and Bimler et al. (2009) on color appearance on neutral backgrounds of different brightnesses. Color saturation scaling was performed to quantify the degree of saturation change induced by increasing amounts of luminance contrast. In these trials, observers rated the relative saturation of the chromatic target while the target was displayed on one of five achromatic backgrounds. The chromatic targets appeared red or green on neutral backgrounds. They remained a fixed chromaticity and luminance throughout the trials.

We found that, when a color target appeared with an achromatic surround that had a high luminance contrast with the target, the apparent saturation of the color was decreased. Thus, we replicated the result that, as luminance contrast increased (when either the luminance of the surround increases or decreases relative to the luminance of the target), the apparent color saturation of the target gradually decreased (Faul et al., 2008; Bimler et al., 2009). Figure 3 shows the average of the normalized ratings of

**Figure 3.** Psychophysics of color suppression by luminance contrast. Group average ($N = 5$ observers) of saturation scaling of a target's color as a function of luminance contrast between the target and the neutral surround. The color of the data points and connecting curves represent the color used in the experiments (red and green, respectively).

color saturation of red and green targets (see Methods and Materials). It is evident that maximum color saturation was perceived when luminance contrast was minimal. An average of 21% luminance contrast in either the negative or positive direction produced 25% reduction of the scaled color-saturation value.

cVEPs and color cells in V1

We investigated the link between perceived color saturation and responses of neurons in the visual cortex by using VEPs evoked by the onset of pure-color stimuli in the human EEG, the cVEP, to reveal brain mechanisms of color vision (Murray et al., 1987; Rabin et al., 1994; Crognale, 2002; Souza et al., 2008; Martinovic et al., 2011; Crognale et al., 2013). Evidence that the cVEP reflects V1 activity is as follows. Prior work on VEPs as we recorded them, over occipital cortex along the midline, indicates that the signals recorded come from V1 cortex (Onofrij et al., 1995; Nakamura et al., 2000; Kulikowski et al., 2002). The cVEP does not vary with attention, a result that strongly suggests it is evoked early in cortical visual processing (Highsmith and Crognale, 2010). Furthermore, normal cVEPs have been recorded in cases of cerebral achromatopsia where color appearance was lost and lesions were observed in ventromedial extrastriate cortex, but V1 responses to color were unaffected by the lesion (Victor et al., 1989; Crognale et al., 2013). The combined evidence from source localization, lack of attentional effects, and cerebral achromatopsia indicates that the cVEP is an index of V1 responses to color.

Many investigators have found that color computations in V1 are based on the activity of two kinds of cone-opponent neurons: single- and double-opponent cells (for review, see Shapley and Hawken, 2011). Single-opponent cells respond to large areas of color. Double-opponent cells respond to color patterns, textures, and color boundaries (Johnson et al., 2001; Schluppeck and Engel, 2002; Friedman et al., 2003). The VEP responses we measured were time-locked to the color modulation of a target that was constant in luminance at all times. The equiluminant color modu-

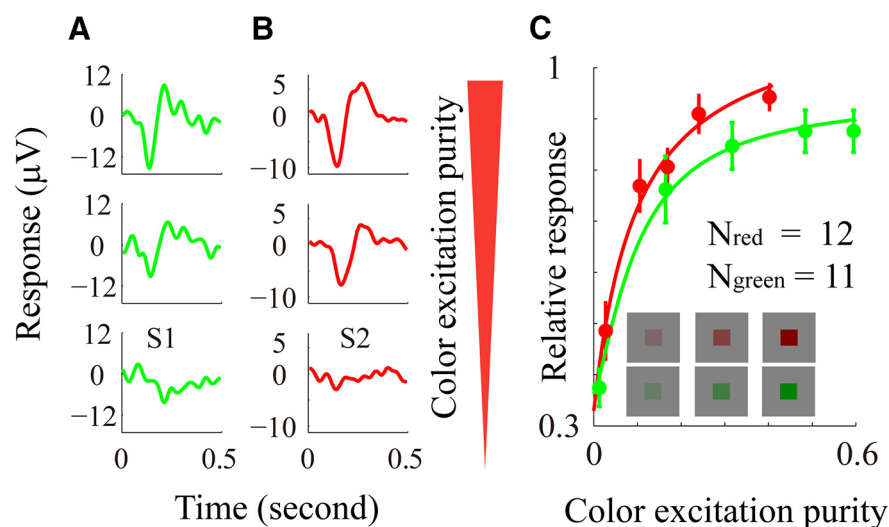


Figure 4. The cVEP grows with color excitation purity. CEP is defined as distance in *xy* color space from the white point to the stimulus relative to the distance from white point to the spectrum locus of the stimulus' dominant wavelength. **A**, The waveforms of an observer's cVEPs to green color targets of different CEPs. Top, CEP = 0.59. Bottom, CEP = 0.01. **B**, The waveforms of another observer's cVEPs to red color targets of different purities. Top, CEP = 0.40. Bottom, CEP = 0.03. **C**, Population-averaged magnitudes of cVEPs as functions of red and green CEP. Vertical bars represent SEM. The data were fitted by Naka–Rushton functions.

lation of targets in our experiments produced cVEPs that must have been influenced by single- and double-opponent cells because they are the only V1 cells that respond to color modulation (Johnson et al., 2001; Schluppeck and Engel, 2002). To allow comparisons across observers and to use the entire cVEP signal not just the peak, we used the rms power of the cVEP waveform over the 0.5 s of stimulus presentation as a measure of cVEP amplitude. cVEPs were graded in amplitude with color saturation (color excitation purity) of the stimuli (Fig. 4), confirming earlier results (Croganale, 2002; Souza et al., 2008). In Figure 4, the population average is taken from the data of 11 of 13 participants for green and 12 of 13 participants for red (the 13th subject was excluded because of bad quality of the VEP signals). The 11 participants whose data are included are the averages of those of the 13 who ran in all stimulus conditions, and we only obtained cVEP data for red from the 12th observer. The gradation of cVEP amplitude with the color purity of stimuli for which there was no luminance modulation supports the idea that the cVEP represents the population response of color-sensitive neurons in V1.

Neuronal mechanisms and competing hypotheses

Previous psychophysical work (Gordon and Shapley, 2006; Faul et al., 2008; Bimler et al., 2009) suggested that the explanation of the color–brightness phenomenon illustrated in Figure 1 is that the cortical mechanisms for color and brightness perception are mutually inhibitory. Specifically, we hypothesized that, at the neural representation of edges in the cerebral cortex, there is mutual inhibition between neural mechanisms for color and brightness in a “winner-take-all” network (Fig. 2B) (Gordon and Shapley, 2006). Perception is ruled by the strongest signal for boundary identification. The perceptual importance of edges is indicated in Figure 1A (top) where a black band drawn around the targets greatly reduces the saturation of the color targets on the approximately equiluminant surround (Faul et al., 2008).

There is an alternative to the mutual-inhibition hypothesis: the parallel-channels hypothesis (Fig. 2A). Suppose that color saturation is encoded as the population activity profile across all V1 cells (Wachtler et al., 2003). The alternative hypothesis

assumes that there is no interaction between color-responsive and color-nonresponsive (achromatic) V1 neurons; these two groups form parallel, independent channels (Livingstone and Hubel, 1984). The parallel-channels hypothesis is a good description of color processing in the thalamus (in the lateral geniculate nucleus) where parvocellular and magnocellular channels are literally in parallel (Kaplan, 2014). The parallel-channels hypothesis assumes that the population code in V1 is read out in higher cortical areas (McKeefry and Zeki, 1997; Hadjikhani et al., 1998; Mullen et al., 2007). For instance, the explanation of the appearance of the color targets in Figure 1, according to the parallel-channels view, is that, when brightness contrast increases, V1 color-blind nonopponent cells at the edge boundaries become more activated without affecting the responses to color contrast by V1 double-opponent cells. The resulting population profile then would resemble that produced by a target with

lower chroma and would be read out at a higher cortical level as lower saturation. However, population coding also is compatible with the mutual-inhibition hypothesis; the net result of V1 brightness–color interactions could affect the population distribution of activity within V1 that is interpreted at higher cortical levels.

Our experiments decided between the mutually inhibitory and parallel-channels hypotheses by measuring the response of the color-selective population with cVEPs. The parallel-channels theory predicts that the cVEPs we measured should not change with increasing brightness contrast between target and surround (Fig. 2A). The mutual-inhibition hypothesis predicts a drop in cVEP amplitude with increasing brightness contrast (Fig. 2B). There was a drop in cVEP amplitude for both positive and negative brightness contrast in 12 of 13 observers (see Materials and Methods) we studied, clearly supporting the mutual-inhibition hypothesis. Representative cVEP waveforms from two observers illustrate the drop in cVEP amplitude with brightness contrast between color target and surround, averaged across observers. (We only included the 11 subjects who participated in cVEP experiments for both red and green color.) One of the 12 included observers only participated in the cVEP experiment for red (Figure 5B). Consistently, the largest cVEP was obtained when brightness contrast between target and surround was zero. These results are highly consistent with the prediction of the mutual-inhibition hypothesis: color responses are inhibited by brightness strength. Furthermore, our observations of suppression of the cVEP recorded with posterior electrodes indicate that the effect was mainly in V1 because, if V1 responses were unaffected and instead there was suppression in V4 or in LOC cortex, we would not expect to see such large reductions in the cVEP.

Importance of edges

The perceptual importance of neuronal activity near the edges of targets (Krauskopf, 1963; Faul et al., 2008) is illustrated in Figure 1A (top) by the reduced perceived color saturation of targets on

equiluminant surrounds when the targets have thin black bands drawn around them. We found analogous reductions of cVEPs when observers viewed color-modulated targets that had black bands around them (Fig. 6), even when the surround outside the black band was equiluminant with the target. This experiment was run on two observers (Fig. 6) who were members of the larger group that contributed data to Figure 5.

Discussion

The strong effect of brightness contrast on color appearance has been studied previously with human psychophysics. For instance, it has long been known that brightness contrast affects color induction from a colored surround into an achromatic target (Kirschmann, 1891). Kirschmann's third Law stated that chromatic induction was greatest when brightness contrast was minimal. Our own psychophysical measurements of color induction, based on the same technique of saturation scaling used in the present paper, confirmed that perceived color saturation in chromatic induction was maximal when brightness contrast was minimal (Gordon and Shapley, 2006). A very different kind of experiment also suggests that there is a suppressive effect of brightness contrast on color: masking with luminance gratings can raise the threshold of detectability of pure-color gratings (Switkes et al., 1988). The phenomenon we studied in this paper is the effect of brightness contrast on the color appearance of colored targets as in Figure 1. This phenomenon is related to Kirschmann's third Law, but it is different in the following way. In color induction experiments, the target is a neutral color (mid-gray). The region surrounding the target is colored and induces a complementary color in the target. In the experiments in this paper, the targets were colored regions surrounded by regions that were neutral in color (different gray levels from black to white). But as in color induction, the appearance of color was subdued when there was brightness contrast at the boundary between target and surroundings (Fig. 1).

Another important effect on color appearance is the phenomenon of gamut expansion, discovered by Brown and MacLeod (1997; see also Brou et al., 1986). Gamut expansion means that the color saturation of a target is much greater when it is viewed on an equiluminant uniform gray surround than when the same color target is surrounded by a high-contrast checkerboard of colored squares. In a very powerful set of experiments, Faul et al. (2008) found that the phenomenon of color gamut expansion discovered by Brown and MacLeod (1997) was caused mainly by brightness contrast at the borders of the color targets with their surroundings and that color contrast near to or remote from the

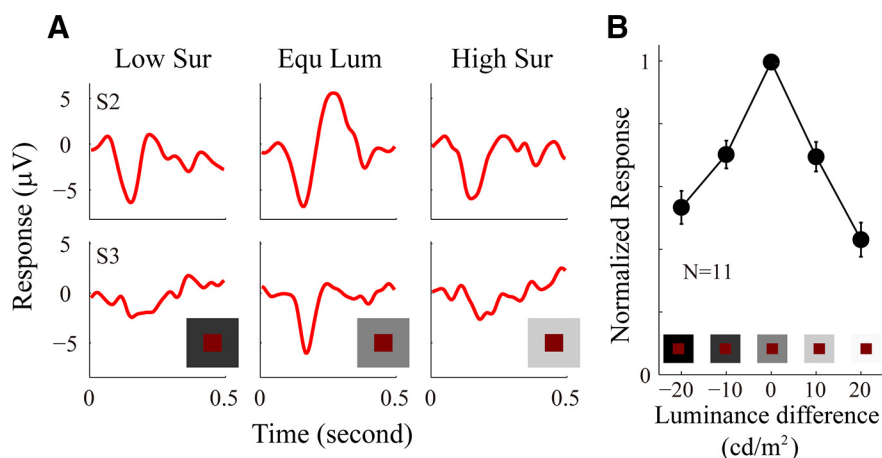


Figure 5. cVEP reduced by increased brightness contrast between color target and its achromatic surround. **A**, The waveforms of two observers' cVEPs to red color targets (luminance was set at 20 cd/m²) with different surround luminances. Left, The background luminance was set at 10 cd/m², and the background luminance on the right was set at 30 cd/m². Middle, The equiluminance condition; both target and background had luminance of 20 cd/m². **B**, cVEP reduced by increased brightness contrast between color target and achromatic surround — population average. Over 11 observers, magnitudes of cVEPs were strongest when target (including both red and green color targets) and background had the same luminance (equiluminance). The cVEP was weaker when brightness contrast between target and background increased. This is true for both red and green color targets. Vertical bars represent SEMs.

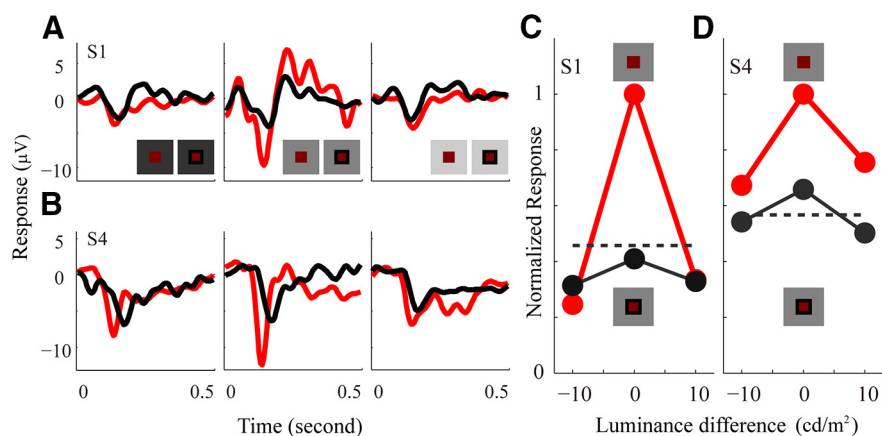


Figure 6. The effect of brightness context on the cVEP was strongly affected by a thin black line (6') surrounding the color target. **A**, The waveforms of an observer's cVEPs to a red target (luminance, 20 cd/m²) with different surround luminances (10, 20, and 30 cd/m²) were strongly affected by a thin (6') black surrounding line. Waveforms without black line are drawn in red, and those with black line are drawn in black. **B**, The result from another observer. **C**, **D**, Summarization of the change of cVEP magnitudes before and after a thin line was added around the color target. Black dashed line indicates the cVEP magnitude when surround luminance was set at 0 cd/m².

target contributed little to gamut expansion. Thus, gamut expansion and the desaturation by surrounds of high brightness contrast (as in Fig. 1) are probably the same phenomenon. Bimler et al. (2009) studied brightness–color interactions with scaling methods and found results essentially the same as what we show in Figure 3. The results on cVEPs in this paper were designed to uncover the neural locus and mechanism of brightness–color interactions in human vision.

Our experimental data on cVEPs (Figs. 5 and 6) indicate that the mechanism of perceptual brightness color interaction is inhibition in human primary visual cortex.

These data support the mutual-inhibition explanation of brightness–color interactions in perception (for the statement of the mutual-inhibition hypothesis, see Results). It is also possible, even likely, that the parallel-channels hypothesis (also presented

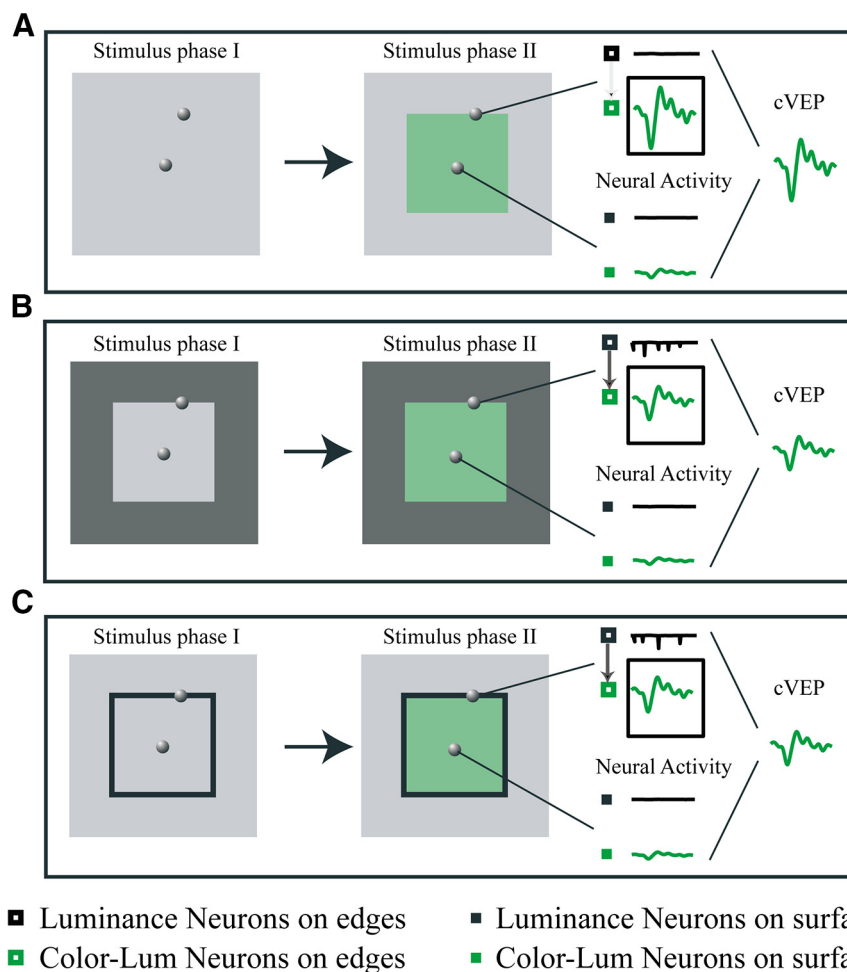


Figure 7. Proposed neural mechanism of luminance–color suppression. In the cVEP experiment, the V1 population that contributes to the cVEP is comprised of the Color-Lum neurons at the border of the square. **A–C**, Color-Lum neurons’ stimulus-locked responses are shown as green curves surrounded by black frames. When the stimulus has the same luminance as its surround, Lum neurons on the edge are inactive (**A**), and there is no suppression of the full-size cVEP. However, when a spatial luminance contrast exists at the square border due to either luminance difference between square and its surround (**B**) or high spatial frequency line (**C**), Lum neurons are activated by the stimulus due to asynchronous modulation (e.g., small eye movements or brain state fluctuation). **B, C**, Lum neurons’ responses are locked to eye movement but not to stimulus; therefore, Lum neurons’ responses (**B, C**, spikes) cannot be seen in the trial-averaged cVEP. However, such asynchronous Lum neuron responses provide a source of constant inhibition onto Color-Lum neurons. **B, C**, cVEPs are also mainly caused by Color-Lum responses; but because Lum neurons are activated, Color-Lum responses are suppressed by inhibition from Lum neurons, and the amplitudes of cVEPs are reduced.

in Results) is also partially correct and that there is population coding of color in V1 and in extrastriate cortex (Wachtler et al., 2003; Shapley and Hawken, 2011). The mutual-inhibition hypothesis is not incompatible with population coding; the net result of brightness–color interactions will shift the peak of the population distribution of activity away from the color-responding population and toward the brightness-responsive, nonopponent (Johnson et al., 2001) population. Such a shift in population distribution will be interpreted at higher cortical levels as less color. The inhibition from brightness onto color signals in V1 revealed in Figure 5 would in this way work together with population coding of color to create the desaturated appearance of color targets on brighter or darker surrounds, as in Figure 1.

The data we have presented in this paper are consistent with the idea that there is inhibitory suppression of the color-responding population by nonopponent cells that respond to brightness contrast. The converse (the mutual inhibition of brightness responding neurons by color-responsive neurons) has so far only been studied in

perceptual experiments (Alpern, 1964). Alpern found that simultaneous brightness contrast was reduced by color contrast. We think it is reasonable to hypothesize that Alpern’s results could be explained by the mutual inhibition of brightness-responsive neurons by color-responsive neurons, also occurring early in the visual system, in V1. This topic should be the target of future research.

The suppressive effect on color appearance of brightness contrast near the edge of a color target has been shown to cause other perceptual effects, such as the gamut expansion effect (Brown and MacLeod, 1997; Faul et al., 2008). Our results show that these perceptual effects are mediated by corticocortical inhibition early in the visual cortex. Corticocortical inhibition has been found to have an important function in visual cortex (Shapley and Xing, 2013) and in other sensory cortices (Wu et al., 2008; Franks et al., 2011; Isaacson and Scanziani, 2011). Throughout the cortex, neurons engage in a recurrent inhibitory computation to determine what is the most likely sensory information in the environment. Figure 1 illustrates that cortical computation in action in the effect of brightness context on color.

Proposed neural mechanism of luminance–color suppression

Our results strongly suggest that luminance–color suppression mainly takes place at the border of the stimulus. Here we propose a simple explanation (Fig. 7). V1 neurons can be categorized into three groups (Johnson et al., 2001). One group of neurons are only interested in luminance contrast (Lum-Neuron); another group of neurons are only interested in pure color difference (Color-Neuron); the third group of neurons are interested in both color and luminance contrast (Color-Lum-Neuron) (Johnson et al., 2001; Li et al., 2014). Generally, Color neurons are the minority in the V1 population, and they respond best at low spatial frequency (Johnson et al., 2001; Schluppeck and Engel, 2002; Conway and Livingstone, 2006). Based on the spatial frequency tuning of the cVEP (Rabin et al., 1994), we infer that Color neurons do not contribute very much to the population activity (cVEP). Also, because it is not clear whether the responses of “Color-Neuron” are suppressed by brightness, we think that the color–luminance (double-opponent) cells are the most likely candidates for brightness–color suppression. Let us only consider Lum neurons and Color-Lum neurons. In our experiment, when a square alternates its color but keeps its luminance constant, V1 neurons whose receptive fields are on the square’s surface will not generate strong responses (Fig. 7) because V1 neurons (both Lum neurons and Color-Lum neurons) are tuned to medium spatial frequency and respond poorly to very low spatial frequency, such as a uniform surface (Johnson et al., 2001; Schluppeck and

Engel, 2002). Cells that mainly respond to the alternation of a square's color are those at the border of the square (Friedman et al., 2003). At the border, Color-Lum neurons' responses strongly synchronize to the alternation of the square color, but Lum neurons respond poorly to the color alternation due to a lack of luminance change. However, Lum neurons can be driven by the stimulus due to asynchronous modulation, such as brain state fluctuations or small eye movements, when spatial luminance contrast exists at the square border (Fig. 7*B,C*). Because the fluctuations are not synchronized with color alternation of the square, Lum neurons' responses are invisible in trial-averaged cVEP. But the asynchronous Lum neurons' responses play an important role in color–luminance interaction: they provide the source of a constant suppression onto Color-Lum neurons. When there is no spatial luminance contrast (Fig. 7*A*), Lum neurons are inactive. Therefore, Color-Lum neurons simply respond to color alternation without any suppression originating from Lum neurons. But when there is a spatial contrast (Fig. 7*B,C*), the color-locking responses of Color-Lum neurons are modulated by a constant suppression (Johnson et al., 1998) that originates from Lum neurons' asynchronous responses to luminance contrast.

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