On Sensory Eye Dominance Revealed by Binocular Integrative and Binocular Competitive Stimuli

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Purpose. Two core processes underlie 3-D binocular vision. The first, a binocular combination/summation process, integrates similar feature signals from the two eye channels to form a binocular representation. The second, a binocular inhibitory process, suppresses interocular conflicting signals or falsely matched binocular representations to establish single vision. Having an intrinsic interocular imbalance within one or both processes can cause sensory eye dominance (SED), related to imbalances of combination (SED_{combo}) and/or inhibition ($SED_{inhibition}$). While much has recently been revealed about SED_{combo} and $SED_{inhibition}$, the relationship between them is still unknown.

Methods. We measured observers' foveal SED_{combo} and $SED_{inhibition}$, respectively, with a pair of dichoptic horizontal sine wave gratings with different phases and binocular rivalry stimulus with vertical and horizontal gratings. We then measured horizontal and vertical monocular contrast thresholds using sinusoidal grating stimuli, and stereo thresholds using random-dot stereograms.

Results. There exists a strong correlation between SED_{combo} and $SED_{inhibition}$. An observer's interocular difference in contrast threshold was not always consistent with his/her SED_{combo} and $SED_{inhihition}$, suggesting a partial binocular origin for the underlying imbalances. We also found stereo thresholds significantly increased with the magnitudes of SED_{combo}, as well as with the magnitude of $SED_{inhibition}$.

Conclusions. Our findings suggest a common origin for interocular imbalance in the two different binocular processes and that both types of sensory eye dominance are significant factors in impeding stereopsis.

Keywords: sensory eye dominance, interocular imbalance, interocular inhibition, binocular summation, stereopsis

Conceptually, the visual system relies on two functionally distinct binocular processes to achieve single three-dimensional (3D) vision from the two slightly disparate retinal images.[1] The binocular combination/summation process constructs a 3D binocular representation by integrating signals of similar visual features from the right and left eye's channels. In contrast, the interocular inhibitory process suppresses signals of the dissimilar features from one of the two eye channels to promote single binocular vision. These two binocular processes work in tandem to achieve optimal 3D binocular vision. It can further be argued that for these processes to work well, the two eyes need to be exposed to stimuli of roughly equal strengths within the binocular visual field. For example, it has been shown that 3D depth perception (stereopsis) of observers with clinically normal binocular vision is degraded when stimuli of unequal contrast values are presented to the two eyes.[2–7] This observation also predicts that stereopsis will be compromised when there exists an intrinsic imbalance between the two eyes. Indeed, observers with large sensory eye dominance (SED) have poor stereo acuity.[7–13]

The phenomenon of eye dominance has been documented in the early clinical literature[14–16] and have more recently been investigated with more quantitative psychophysical approaches.[8,9,13,17–20] For example, Ooi and He[13] investigated sensory eye dominance related to the interocular inhibitory process ($SED_{inhibition}$) by using a binocular rivalry display. The study found that several of their observers who were clinically considered to have normal binocular vision had significant $SED_{inhibition}$. Furthermore, for a subset of these observers, their weak eye monocular contrast sensitivity and monocular perceived brightness of suprathreshold gratings were no worse than the strong eye. This suggests that their $SED_{inhibition}$ were more likely caused by an imbalance in mutual inhibition between the two eyes rather than a difference between the two monocular pathways before binocular interaction.[8,13]

Figures 1A and 1B illustrate two pairs of dichoptic orthogonal gratings for measuring $SED_{inhibition}$ typically used in our laboratory.[8] During testing, stimulus A is displayed for a brief interval and the observer reports the predominant orientation of the perceived grating disc. For the next test trial, and depending on the observer's report in the preceding trial, the contrast of the vertical grating in the left eye (LE) is appropriately adjusted with an adaptive procedure[21] before stimulus A is presented again. This finetuning of the contrast is done after each trial until the observer experiences an equal percentage of seeing the two gratings (point of equality). Since the contrast of the horizontal grating in the right eye (RE) is kept constant, the contrast of the vertical grating obtained at

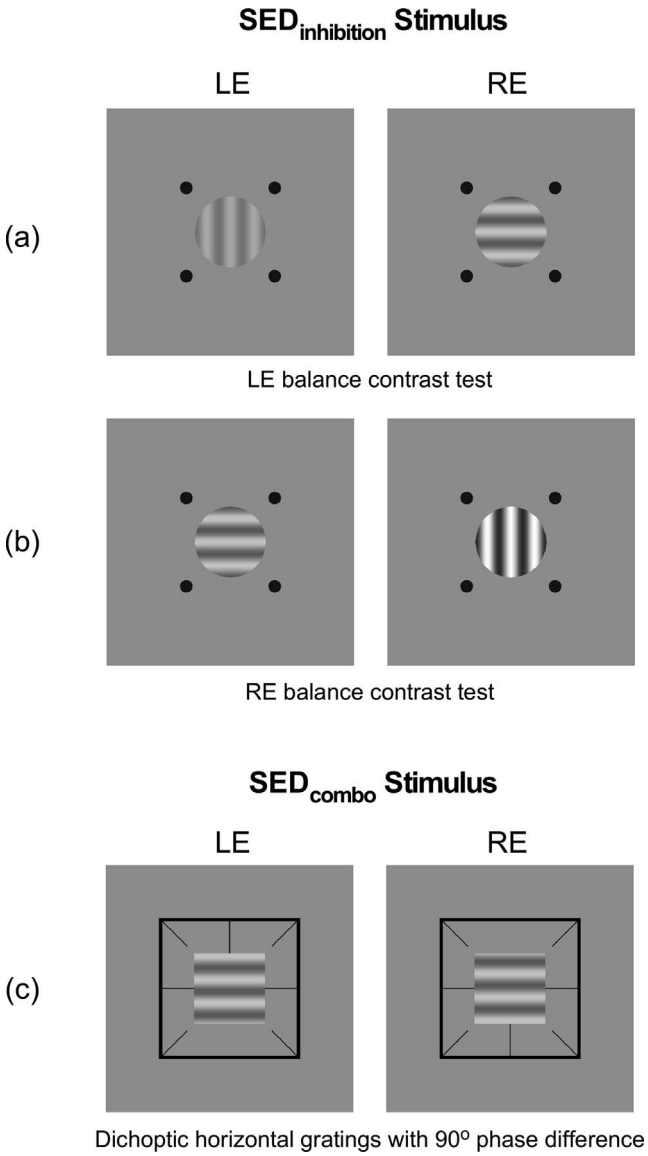

Figure 1. (**a**, **b**) Binocular rivalry stimulus used for measuring $SED_{inhibition}$. (**a**) Test 1: The LE balance contrast is obtained by varying the vertical grating contrast while keeping the contrast of the horizontal grating seen by the RE constant. The balance contrast is reached when the two eyes obtain an equal percentage of perceiving the two gratings (point of equality). (**b**) Test 2: Switching the grating orientation between the two eyes permits measurement of RE balance contrast. (**c**) Binocular combination stimulus for measuring SED_{combo}. The dichoptic horizontal gratings have a 90° interocular phase difference.

the point of equality is referred to as the LE balance contrast. But as the LE balance contrast could also be caused by a difference in sensitivity to the grating orientation (e.g., better sensitivity to vertical orientation), it is necessary to measure the RE balance contrast. This is obtained by switching the vertical and horizontal gratings between the two eyes as shown in stimulus B, and adjusting the contrast of the vertical grating now in the RE until the point of equality is obtained. The difference between the LE and RE balance contrast values defines the $SED_{inhibition}$.

To reveal SED associated with an imbalance in the binocular combination process (SED_{combo}), one can employ a pair of dichoptic stimuli with similar features that can be integrated as a single image representation.[22–25] Figure 1C illustrates the stimulus used in this study, which is adapted from Ding and Sperling.[25] The dichoptic display comprises two horizontal sinusoidal gratings with 90° spatial phase difference (–45° vs. 45°). A pair of reference horizontal lines is placed on each side of the grating. With fusion, the observer perceives an integrated (cyclopean) grating and judges its spatial phase relative to the horizontal reference lines. An observer with a balanced binocular combination process will report zero phase shift relative to the horizontal reference lines when the contrast values of the two dichoptic gratings are identical. Conversely, an observer with an unbalanced binocular combination (SED_{combo}) will perceive a phase shift toward that of the grating in the strong eye, indicating that the grating signal in the strong eye biases the spatial phase of the combined binocular grating. To measure SED_{combo}, one can add extra contrast to the grating seen by the weak eye until the observer perceives the integrated binocular grating to have zero phase shift. The extra contrast is a measure of SED_{combo}.

A number of studies have revealed that $SED_{inhibition}$ degrades binocular depth perception.[8,9,11,12,26] For example, Xu et al.[8] found stereo threshold increased as $SED_{inhibition}$ increased. However, $SED_{inhibition}$ can be reduced after visual training using the Push-Pull perceptual learning protocol that mainly targets the putative interocular inhibitory neural network.[9,10,12,26,27] During a training trial, an attention cue (monocular frame) was briefly presented to the weak eye, followed by a binocular rivalry stimulus (a pair of vertical and horizontal gratings). The brief cue attracts transient attention to the weak eye, resulting in the grating in the weak eye being perceived (push) while the grating with orthogonal orientation in the strong eye was suppressed (pull). The role of the cue was to deploy transient (involuntary, bottom-up) attention to the weak eye to cause its stimulus to be perceived in dominance during binocular rivalry. The push-pull training is based on the hypothesis that the suppression of the half-image in the strong eye during the push–pull training can effectively shift the balance of interocular inhibition between the two eyes.[9] This is because with the push–pull protocol, repetitive stimulation of the strong eye while preventing its signals from reaching the higher level (thus failing to induce conscious perception) could effectively degrade the efficiency of the excitatory synaptic transmission within the strong eye's channel and also depress the inhibition of the strong eye on the weak eye's channel.[9,28] Undergoing the protocol also resulted in a decrease in stereo threshold. Of significance, since the *Push-Pull* perceptual learning stimuli do not carry binocular disparity information, it suggests the improvement in binocular depth perception is likely a consequence of the reduced $SED_{inhibition}$. A similar learning effect was revealed in adult amblyopic patients who had much larger $SED_{inhibition}$ due to the stronger interocular suppression experienced by the amblyopic eye.[26]

In this paper, our first goal was to reveal whether $SED_{inhibition}$ and SED_{combo} are independent, given that they are associated with imbalances of the two functionally distinct binocular processes. To investigate this, we designed the stimuli and procedures for measuring both $SED_{inhibition}$ and SED_{combo} to be as similar as possible, the details of which are provided in the Supplementary Materials.

The second goal of this paper was to investigate whether SED_{combo} is similar to $SED_{inhibition}$ in influencing (increasing) stereo threshold. The answer might not be straightforward. First, previous psychophysical studies and modeling works suggest there exists a mutual inhibition between the two eyes' channels prior to binocular combination/summation.[29] This provides an explanation for the findings that a normal observer's stereo threshold increases with the interocular

contrast difference in the stereo stimuli.[3,4,6,29] Conceivably, having an interocular imbalance can further weaken the signals from the monocular channel that receives the lower contrast image, further resulting in poorer spatial resolution of the monocular representation as well as a poorer binocular representation. One could conceivably apply this explanation to SED_{combo}, wherein the larger suppression from the strong eye onto the weak eye can cause a poor spatial resolution of the binocular representation. Alternatively, one could argue that this explanation is not applicable to SED_{combo}. This is because SED_{combo} is often measured with dichoptic horizontal gratings (Fig. 1C) that stimulate neurons with horizontal orientation selectivity, which has little impact on the processing of horizontal binocular disparity for stereopsis. Accordingly, stereo thresholds may not be correlated with SED_{combo} measured with dichoptic horizontal gratings.

Another consideration relates to the explanation that the measured SED_{combo} can be partially contributed by an imbalance in the binocular contrast gain control mechanism, which plays a major role in determining brightness/contrast perception of binocular surfaces. Several recent computational models of binocular contrast gain control have hypothesized that in addition to binocular summation, there exists mutual inhibitions between the two monocular channels.[22–25,30,31] Accordingly, the measured SED_{combo} could comprise of an imbalance of interocular summation and an imbalance of interocular inhibition. However, since SED_{combo} is typically measured with horizontal orientation gratings in the two eyes, the imbalance of interocular inhibition that contributes to SED_{combo} may not necessarily be the same as that which contributes to $SED_{inhibition}$. This is because that the latter is measured with a binocular rivalry stimulus that activates interocular inhibition between different feature (e.g., orientation) channels from the two eyes.

Methods

Observers

Eighteen observers (ages 18–25) who were naïve to the purpose of the study participated in the experiments. All had normal or corrected-to-normal visual acuity (at least 20/20), clinically acceptable fixation disparity ($\leq$8.6 arc min) and stereopsis ($\leq$40 arc sec). During the experiments, they viewed the computer monitor through a haploscopic mirror system attached to a head-and-chin rest from a distance of 100 cm.

We measured all observers' $SED_{inhibit}$, SED_{combo}, and interocular difference in contrast threshold (IDCT). Among them, seven observers also participated in the stereo disparity threshold experiment. All, except one observer, were also tested for motor eye dominance (MED).

The research conducted followed the tenets of the Declaration of Helsinki and was approved by the institutional review board (IRB). Informed consent was obtained from the observers after explanation of the nature and possible consequences of the study.

Apparatus

Gamma-corrected stimuli were generated on a Mac Pro computer running MatLab with PsychToolBox,[32,33] and presented on a 21-inch flat CRT monitor. The resolution of the monitor was set at 2048 × 1536 pixels @ 75 Hz refresh rate.

Stimuli and Procedures

$SED_{inhibition}$. The stimulus comprised a pair of dichoptic vertical and horizontal sinusoidal grating discs (diameter = 1°, 3 cycle/deg, 35 cd/m^2) on a gray background (8° × 8°, 35 cd/m^2; e.g., Fig. 1A). The contrast of the horizontal grating was held constant (1.5 log unit) while the contrast of the vertical grating was variable (0.376-1.976 log unit). (Note: log contrast is defined as $\log_{10}(C)$, where C is the Michelson contrast in percentage. Therefore, 1.5 log unit = 31.6% Michelson contrast). A trial began with the observer fixating on a white nonius target (0.45° × 0.45°, line width = 0.1°, 70 cd/m^2). When accurate fixation was achieved, the observer pressed the start button on the keyboard to remove the nonius target; 146 ms after the removal, a pair of dichoptic orthogonal gratings were presented for 400 ms. This was followed by a 200-ms mask (8° × 8° random dots [50% black and 50% white], 35 cd/m^2, dot size = 4.7 arcmin; contrast = 1.7 log unit) to end the trial. The observer responded to his/her percept by key presses. If a piecemeal pattern of vertical and horizontal orientation was seen, the observer would respond to the predominant orientation perceived. The vertical grating contrast was adjusted after each trial until equal predominance was achieved using the QUEST procedure (40 trials/block). When the vertical grating was presented to the LE (Fig. 1A) we refer to its contrast at equal predominance as the LE's balance contrast. To obtain the RE's balance contrast, the gratings were switched between the eyes (Fig. 1B). The difference between the LE and RE balance contrast values (i.e., $\log[C_{LE}] - \log[C_{RE}]$), is defined as $SED_{inhibition}$. The order of testing the two conditions was counterbalanced and each condition was repeated four times.

SED_{comb} With QUEST Procedure. The test stimulus was a pair of dichoptic horizontal grating squares (1° × 1°, 3 cycle/deg, 35 cd/m^2) with a 90° phase difference between them (Fig. 1C). The average phase of the two gratings was always held at 0° ($\theta_L = 45°$ and $\theta_R = -45°$, or $\theta_L = -45°$ and $\theta_R = 45°$). The contrast of the grating in one half-image was fixed at 1.5 log unit, while the contrast of the other grating in the tested eye varied from 0.376 to 1.976 log unit. The contrast in the tested eye was adjusted using the QUEST procedure over 40 trials in an experimental block, as the observer responded to either seeing the central dark band of the perceived grating as above or below the horizontal reference lines. When the variable contrast grating was presented to the LE, we refer to the contrast at which the grating was perceived to be aligned with the reference line as the LE's balance contrast. To measure the RE's balance contrast, we swapped the gratings between the two eyes so that the LE now received the grating with the fixed contrast and the RE the grating with the variable contrast. We refer to the difference between the LE and RE's balance contrast as SED_{combo}.

To control for the possible effect of contrast and grating phase in each half-image causing a positional bias, we tested SED_{combo} with two display types. In one display type, the variable contrast grating's phase in the tested eye was shifted upward relative to the fixed contrast grating's phase in the fellow eye. In the second display type, the variable grating's phase was shifted below the fellow eye's grating. For data analysis, SED_{combo} of the two types were averaged.

The observer prepared for a trial by maintaining accurate eye alignment on the fusion-lock (2° × 2°). Then to begin the trial, he/she would press a button on the keyboard. This was followed 146 ms later, with the presentation of the dichoptic grating stimulus (1° × 1°, 3 cycle/deg, 35 cd/m^2) for 400 ms. A 200-ms mask was then presented to end the trial (8° × 8° random dots patch, 35 cd/m^2, 1.7 log unit). The observer's task was to report by pressing one of two keys on the keyboard to indicate whether the grating band was perceived above or below the reference lines. The order of testing the balance contrast of the two eyes and the two display types was counterbalanced and each block was repeated four times.

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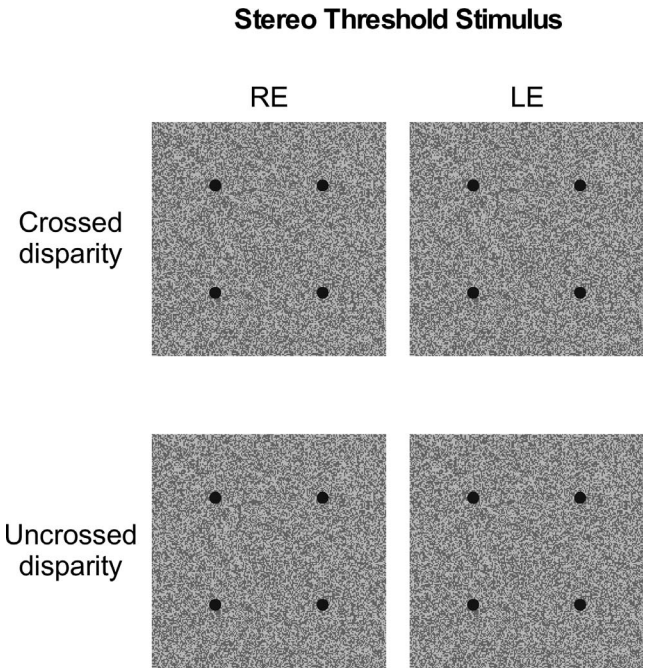

FIGURE 2. Random-dot stereogram for measuring stereopsis threshold. The disc target can be rendered in crossed (*top*) or uncrossed (*bottom*) binocular disparity.

Monocular Contrast Detection Threshold. The tested eye was presented with either a vertical or horizontal sinusoidal grating disc (35 cd/m^2, 3 cycle/deg, diameter = 1°), while a homogeneous gray (blank) field with the same mean luminance level was presented to the fellow eye. Each test trial was conducted using the 2IFC method, whose temporal sequence was: fixation, interval-1 (400 ms), blank (400 ms), interval-2 (400 ms), blank (200 ms), and mask (8° × 8° random dots, 35 cd/m^2, 1.7 log unit contrast, 200 ms). The grating disc was presented at only one interval while the other interval had a blank field. The observer responded whether he/she saw the grating in either interval-1 or -2 by a key press. The grating contrast was adjusted after each trial using the QUEST method to obtain the threshold. This contrast threshold test was repeated four times in each eye for each orientation. Throughout the experiment, four reference dots (size 0.18° × 0.18°) were displayed in a square formation (size 1.25° × 1.25°) surrounding the foveal location. The observer was instructed to maintain fixation at the center of the four dots where the stimulus was presented.

Stereopsis Threshold. An 8° × 8° random-dot stereogram (35 cd/m^2, 1.4 log unit contrast) with a crossed- or uncrossed-disparity 1° disc target was used (Fig. 2). The dot size of the random-dot was either 1.344 arc min (for four observers) or 2.016 arc min (for three observers), with the larger dot size only being used if the observer was unable to reliably perceive the smaller dot size. Five repeats of crossed disparity thresholds were measured before another five repeats of uncrossed disparity thresholds.

The standard 2IFC method in combination with the staircase procedure was employed to measure the stereo disparity threshold. The temporal sequence of the stimulus presentation was nonius fixation (0.45° × 0.45°, line width = 0.1°, 70 cd/m^2), blank (147 ms), interval-1 (53 ms), blank (400 ms), interval-2 (53 ms), and random-dot mask (200 ms, 8° × 8°, 1.7 log unit contrast, 35 cd/m^2). Both intervals comprised images with random-dot, but only the stimulus in one interval had some binocular disparity while the stimulus in the other interval had zero disparity. A block of trials comprised 10 reversals (step size = 0.672 arc min, total ~40–60 trials), and the average of the last six reversals were taken as the stereo threshold. During the experiment, the observer indicated whether the stimulus with the disk in depth was seen in interval-1 or -2 by pressing a key on the keyboard.

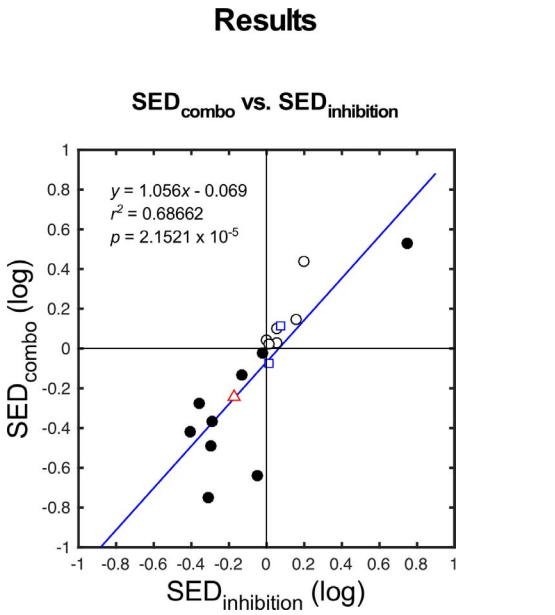

FIGURE 3. Relating the two types of sensory eye dominance. The graph plots SED$_{combo}$ as a function of SED$_{inhibition}$. Each *symbol* represents the data of an observer. Clearly, all symbols, except one, fall in the first and third quadrants, indicated that SED$_{combo}$ and SED$_{inhibition}$ are highly correlated. The data points are also plotted in different symbols, with all the *open symbols* regardless of their shapes representing the observers whose SED could not be explained by an interocular difference in contrast threshold (IDCT) for horizontal and/or vertical grating. The relationship between SED and IDCT will be further elaborated in Figure 5 where the differently shaped symbols will be defined.

Motor Eye Dominance

A variation of the Ring sighting test was used.[8,13,34] To perform the test, the observer brought both hands simultaneously to the front of his/her face and formed a ring (2–3 inches in diameter) by bringing together the index finger and thumb from each hand. He/she then sighted a target with both eyes opened through this "ring", while carefully placing the sighted target in the center of the ring. After this, he/she closed each eye alternately to determine which eye saw the target as more centered in the ring. The eye that saw the target as more centered is defined as the motor-dominant eye.

RESULTS

Correlation Between SED$_{combo}$ vs. SED$_{inhibition}$ Using the QUEST Procedure

Figure 3 plots the SED$_{combo}$ versus SED$_{inhibition}$ data of all 18 observers. Clearly, all the data points (except for one observer who has very small SED$_{combo}$ and SED$_{inhibition}$), fall in quadrants I and III, indicating the sign of sensory eye dominance remains the same for both inhibitory and combination measures. This observation is corroborated by a correlation analysis revealing a large r^2 ($n = 18$, $P < 0.0001$, $r^2 = 0.687$) and a slope of the

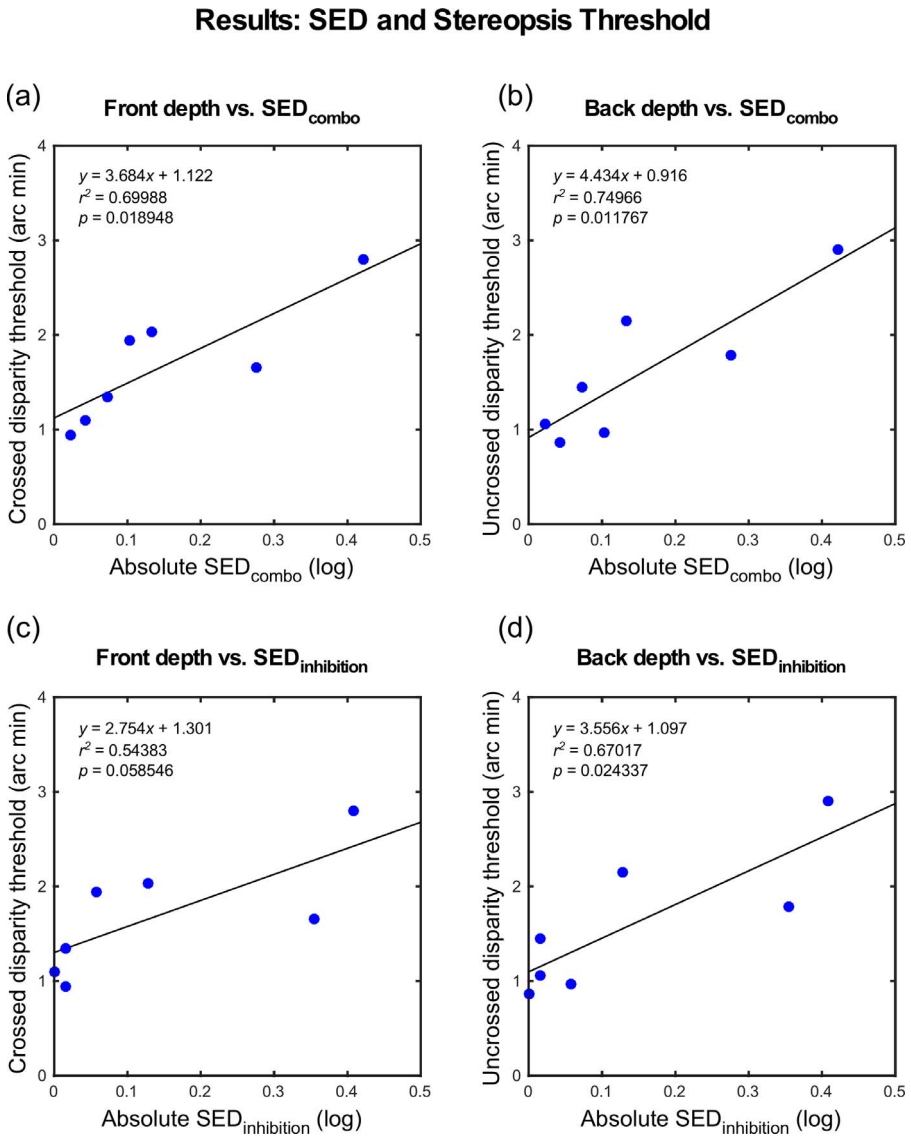

Figure 4. Relating sensory eye dominance to stereo threshold. (**a**) SED_{combo} versus crossed/front depth threshold; (**b**) SED_{combo} versus uncrossed/back depth threshold. (**c**) $SED_{inhibition}$ versus crossed/front depth threshold; (**d**) $SED_{inhibition}$ versus uncrossed/back depth threshold. Generally, a larger interocular imbalance results in a higher stereo threshold for both types of SED.

regression line being close to 1 (slope = 1.056). This means that despite the SED_{combo} and $SED_{inhibition}$ being measured with different stimuli and tasks, they result in the same eye being dominant by about the same magnitude. (Note: The observers' data in Figure 3 are plotted in different symbols to represent how their SED relates to luminance contrast thresholds, which will be elaborated in Fig. 5).

Impact of SED_{combo} and $SED_{inhibition}$ on Stereo Threshold

We measured stereo thresholds for detecting front and back depth of a random dot stereogram display and plotted the data as a function of the observer's SED_{combo} and $SED_{inhibition}$ in Figures 4A through 4D ($n = 7$). Figures 4A and 4B, respectively, show the stereo thresholds increased with SED_{combo} for the front ($P = 0.019$, $r^2 = 0.700$) and back ($P = 0.012$, $r^2 = 0.750$) depth threshold conditions. To the best of our knowledge, this finding is the first to reveal the relationship between SED_{combo} and stereo threshold in clinically normal observers. This relationship is similar to those for $SED_{inhibition}$, as found in the current study (Figs. 4C, 4D; front: $P = 0.059$, $r^2 = 0.544$; back: $P = 0.024$, $r^2 = 0.670$) and in our earlier studies.[8,9,13]

Monocular Contrast Thresholds Versus SED_{combo} and $SED_{inhibition}$. Given that the test stimuli for SED_{combo} are dichoptic horizontal gratings, we also explored how horizontal grating contrast threshold affected SED_{combo}. Figure 5A shows the relationship between SED_{combo} and interocular difference in contrast threshold (IDCT) of the horizontal grating. If an observer's SED_{combo} could be accounted for by IDCT, the data point should fall in quadrants I and III (i.e., the dominant eye based on SED_{combo} measurement is also the strong eye based on monocular contrast threshold measurement). Instead, we found 7 out of 18 data points (39%, the six open circles and the triangle) fall in quadrants II and IV, although SED_{combo} and IDCT are significantly correlated ($P = 0.031$, $r^2 = 0.260$). A similar trend is found in the $SED_{inhibition}$ and IDCT plot in Figure 5B (the average contrast thresholds of horizontal and vertical grating stimuli since both orientations make up the $SED_{inhibition}$ test stimulus). Here too, we found that 8 out of 18

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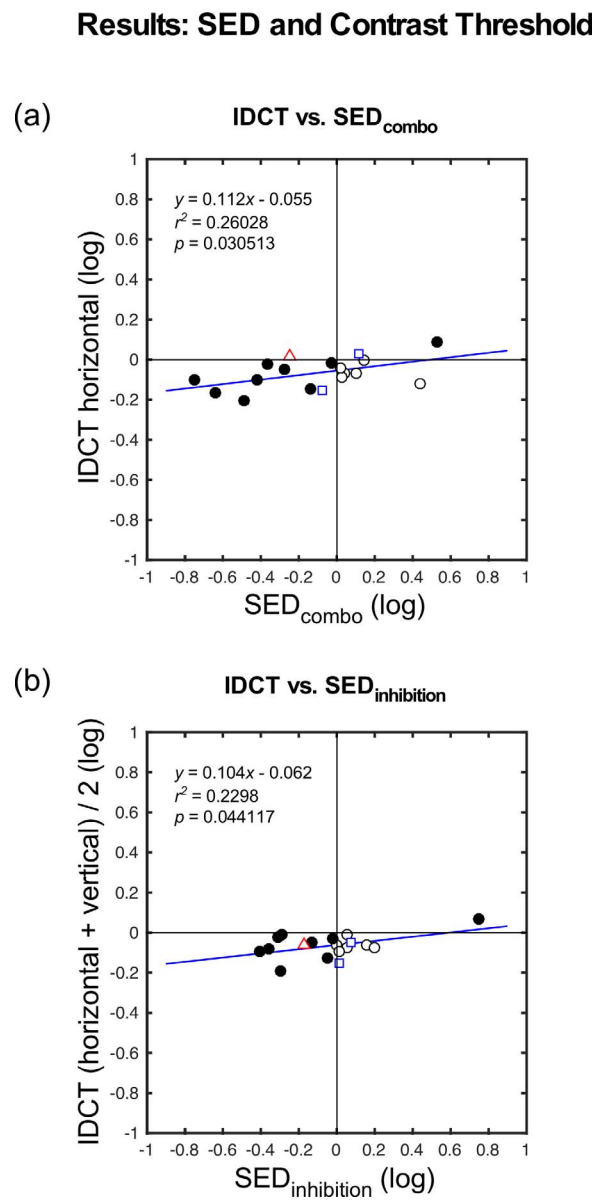

Figure 5. Relating sensory eye dominance to the interocular difference in contrast threshold (IDCT). (**a**) SED_{combo} versus IDCT for horizontal grating. (**b**) $SED_{inhibition}$ versus IDCT for vertical and horizontal gratings. *Filled circles* represent observers whose SED_{combo} and $SED_{inhibition}$ were consistent with IDCT. *Open circles* represent observers whose SED_{combo} and $SED_{inhibition}$ were both opposite from the prediction made based on IDCT. The *triangle* represents the observer whose $SED_{inhibition}$, but not SED_{combo}, was consistent with IDCT. The *squares* represent observers whose SED_{combo}, but not $SED_{inhibition}$, was consistent with IDCT. Taken together, the data suggest that both monocular and binocular channels can cause interocular imbalance.

observers' data points (44%, the six open circles and the two squares) fall in quadrant IV. There is also a weak but significant correlation ($P = 0.044$, $r^2 = 0.230$) between $SED_{inhibition}$ and IDCT, which is also consistent with the previous $SED_{inhibition}$ results from our lab.[8,13] In one of these previous studies, we also measured observers' monocular suprathreshold brightness perception and found a similar trend as in the monocular contrast threshold test.[13] Namely, we found that there were a number of observers with dominant eyes that had a relatively weaker brightness perception than the nondominant eyes. Furthermore, there were also observers whose dominant eyes had a stronger brightness perception. However, the difference in brightness perception between the two eyes was much smaller than the magnitude of $SED_{inhibition}$.[13] It will be interesting to learn if future investigations of other types of suprathreshold measurements, such as interocular contrast matching and contrast increment threshold discrimination measurements, would reveal a similar trend.

To summarize, these observations are graphed in Figures 5A and 5B with different symbols. Filled circles represent observers whose SED_{combo} and $SED_{inhibition}$ were both consistent with IDCT. Open circles represent observers whose SED_{combo} and $SED_{inhibition}$ were both opposite from the prediction made based on IDCT. The triangle represents the observer whose $SED_{inhibition}$, but not SED_{combo}, was consistent with IDCT. The squares represent observers whose SED_{combo}, but not $SED_{inhibition}$, was consistent with IDCT. These same symbols are also used to plot Figure 3 above.

Additionally, we notice there exists an overlap between the two groups of "inconsistent" observers: six observers (open circles in Fig. 5) from the SED_{combo}-IDCT inconsistent group (six out of seven) also exhibited inconsistent $SED_{inhibition}$-IDCT (six out of eight). Overall, half of our observers (nine open symbols) had SEDs that are not accountable for by the monocular contrast threshold difference between the two eyes. The remaining half (filled circles in Fig. 5) had SED and IDCT that are consistent, suggesting that an imbalance in the binocular visual processes could be due in part to a difference in the monocular channels' sensitivity. In other words, both binocular and monocular channels together can cause SED in our observers with clinically normal binocular vision. We recognize that although the correlations between the SEDs and IDCT in Figure 5 are statistically significant, they are nevertheless small. Thus, they do not suggest a strong causal relationship between SED and IDCT, and in general, the contribution of monocular channels to SED in our sample of clinically normal observers was not substantial. One possible reason is that most observers with clinically normal binocular vision have small differences between their monocular channels. However, whether this conclusion could be generalized to the amblyopic population who often has a larger IDCT, requires further psychophysical and physiological investigations of both $SED_{inhibition}$ and SED_{combo}. Pertaining to $SED_{inhibition}$, a study with a small sample size[26] as well as from other unreported observations in our laboratory suggest the weakness of the amblyopic eye can be attributed to poor monocular contrast sensitivity and strong interocular suppression onto the weak eye.

Separately, modeling studies from other laboratories suggest that both monocular deficits and asymmetric interocular inhibition contribute to SED_{combo} in amblyopic observers.[23,24,30,35] Neurophysiological studies on macaque monkeys have also shown that deficits in a monocular channel (e.g., ocular dominant neurons in V1) are correlated with the amblyopic eyes' poor visual functions.[36–38] However, little is known about the neurophysiological substrates of interocular suppression in amblyopia.[39]

Motor Eye Dominance Versus SED_{combo} and $SED_{inhibition}$

Figures 6A and 6B, respectively, plot the $SED_{inhibition}$ and SED_{combo} of each observer and his/her motor eye dominance (MED; $n = 17$). Negative values along the *y*-axis of each graph represent the amount of LE sensory eye dominance. Filled bars represent observers with LE motor dominance. We performed analysis by averaging the SEDs of all observers with the same sign of eye dominance as shown in Figures 6C and 6D,

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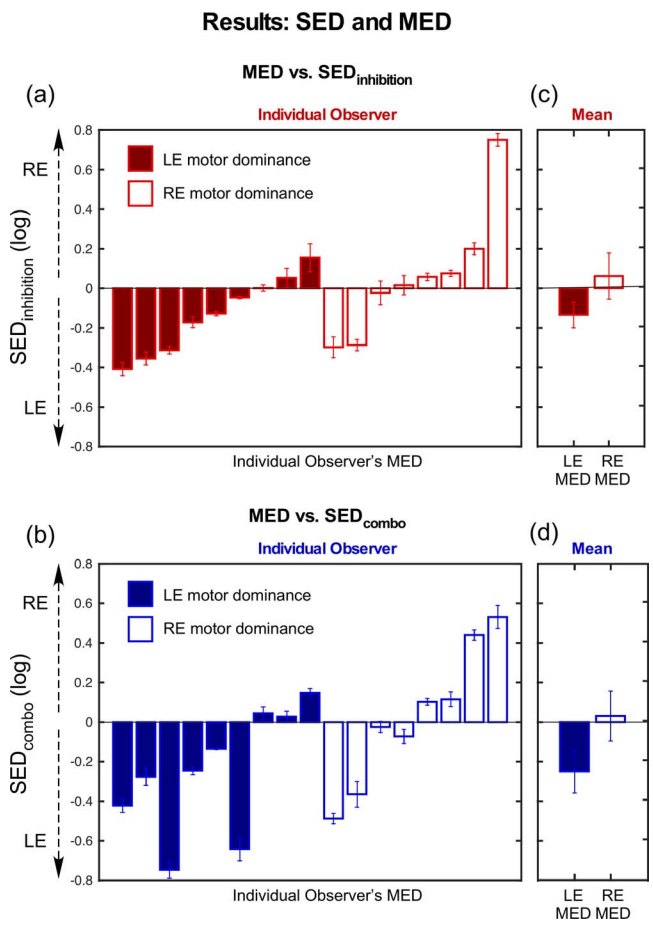

Figure 6. Relating sensory eye dominance (SED) to motor eye dominance (MED). (**a**) $SED_{inhibition}$ versus MED. (**b**) SED_{combo} versus MED. Each bar along the *x*-axis represents the MED data of an individual observer. The data for observers with RE MED are plotted with *open bars* while the data for observers with LE MED are plotted with *filled bars*. The length of the bar is determined by his/her SED, whose magnitude is represented along the *y*-axis, with data for the observers with RE dominance being plotted as positive values. Therefore, should SED and MED be consistent, all *filled bars* should have negative values. Our data show that this is not the case. Graphs (**c**) and (**d**), respectively, plot the average $SED_{inhibition}$ versus MED and average SED_{combo} versus MED. Although each graph shows the mean SED was consistent with the mean MED, statistical analyses fail to show a significant relationship.

respectively, for $SED_{inhibition}$ and SED_{combo}. For $SED_{inhibition}$, the mean $SED_{inhibition}$ for observers with LE motor dominance was −0.1346 ± 0.0648 log unit (negative value denotes LE dominance), and the mean $SED_{inhibition}$ was 0.0617 ± 0.1161 log unit (positive value denotes RE dominance) for observers with RE motor dominance. Although the mean $SED_{inhibition}$ for each eye was consistent with the motor eye dominance, statistical analysis fails to show a significant effect (point-biserial correlation coefficient: $r_{pb} = 0.3658$; 2-tailed unpaired *t*-test with equal variances for correlation coefficient: $t[15] = -1.522$, $P = 0.1487$]). Similarly for SED_{combo}, the means of SED_{combo} were −0.2501 ± 0.1031 log unit and 0.0299 ± 0.1246 log unit, respectively, for observers with LE and RE motor eye dominance, which is not significantly different (point-biserial correlation coefficient: $r_{pb} = 0.4109$; 2-tailed unpaired *t*-test with equal variances for correlation coefficient: $t[15] = -1.746$, $P = 0.1013$). This observation is consistent with our previous findings using a binocular rivalry stimulus, that $SED_{inhibition}$ and motor eye dominance do not necessarily reside in the same eye of an individual.[8,12,13] Our current finding is also in agreement with the view that motor eye dominance may not always correspond with the asymmetry in visual functions.[40]

Other laboratories[11,41,42] have measured normal observers' $SED_{inhibition}$ using binocular rivalry stimuli that were presented for a longer duration (1 minute). They defined the eye having the higher predominance of seeing its half-image as the $SED_{inhibition}$ dominant eye. These studies also measured motor eye dominance. One study by Dieter et al.[11] with a large sample size[17] failed to show significant relationship between $SED_{inhibition}$ and MED. Conversely, two of other studies reported a statistically significant relationship between sensory and motor eye dominance.[41,42] However, we do not think the difference between these reports and our current findings can be attributed to the shorter stimulus duration used in our current study (400 ms). This is because a previous study from our laboratory revealed a significant correlation between $SED_{inhibition}$ with a short duration presentation (500 ms) and $SED_{inhibition}$ based on predominance over longer duration presentation (30 seconds).[12]

Discussion

The current paper reveals a significant correlation between SED_{combo} and $SED_{inhibition}$ for observers with clinically normal vision. Our study also shows that stereo threshold increases with SED_{combo}, a trend that is similar to that with $SED_{inhibition}$. Furthermore, by comparing monocular contrast thresholds between the two eyes, we found half of our observers' SED_{combo} and $SED_{inhibition}$ cannot be accounted for by an interocular difference in contrast threshold (IDCT). For these observers, their SED are likely caused by an imbalance within the binocular visual processes. For the remaining half of our observers, both difference between the monocular channels and imbalances within the binocular visual processes are likely to contribute to their SED.

Presumably, the SED_{combo} and $SED_{inhibition}$ psychophysically measured here with two different types of binocular stimuli, respectively, reflect imbalances in the binocular combination and interocular inhibitory processes. If these two binocular processes were independent, a poor correlation would be found between an observer's SED_{combo} and $SED_{inhibition}$. However, this prediction is not borne out by our current study. One possible explanation is that while the two processes are implemented by distinct distributed neural networks along the visual pathway from LGN to primary visual cortex (V1) and extrastriate visual cortical areas, they do interact with each other along the different stages of the visual pathway. For example, one could speculate that the LGN is one location where the interaction takes place. Neurophysiologic studies have shown that binocular suppression begins as early as dLGN.[43–46] From modeling work, Ding and Levi[47] suggested that the interocular inhibition in the LGN, which lacks of orientation selectivity, could provide the neurophysiologic substrate for the interocular gain-control in binocular combination. This would predict that an imbalance of interocular inhibition at dLGN can affect SED_{combo}. On the other hand, $SED_{inhibition}$ is revealed with binocular rivalry stimuli (e.g., orthogonal orientation gratings) that activate the interocular inhibitory network at V1.[48–52] The interocular inhibitory activities at V1 during binocular rivalry can further affect dLGN neurons via feedback networks.[53,54]

It is also reasonable to hypothesize that both binocular combination (compatible binocular stimulus) and interocular inhibitory (incompatible/rivalrous stimulus) processes belong to a larger integrated network that represents binocular

surfaces. Consistent with this explanation, there are empirical findings showing the binocular combination and interocular inhibition can operate concurrently.[5,55,56] Of course, this hypothesis needs to be further developed and tested. For example, it would be interesting to learn how modeling works that focuses on binocular combination (e.g., the gain-control theory of binocular vision by Ding and Sperling,[25] Ding et al.,[22,30] and Ding and Levi[47,57]) could account for binocular rivalry perception, which in turn could provide insights on our findings of significant correlation between SED_{combo} and $SED_{inhibition}$.

It is also possible that even if the two binocular visual processes have little interaction, the interocular imbalance is caused by the same factors during development. For example, when one eye receives weaker signals during development, it will affect both binocular visual processes in a similar manner, which can result in a similar ocular dominance. In particular, if the monocular deficits are located in the early visual pathway before binocular interaction occurs, the deficits can further affect the development of the binocular processes, causing permanent interocular imbalances. Further studies are needed to investigate these possibilities.

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