The signal strength of a binocular stimulus affects stereopsis. For example, stereo threshold decreases as the contrast of the two half-images of a stereo grating increases. One reason is that high-contrast retinal images lead to their neural representations having better spatial resolution, which enables a finer three-dimensional binocular representation. Having a contrast difference between the two retinal images also affects stereo threshold. Stereo threshold decreases as the contrast difference between the half-images of the left (C_{LE}) and right (C_{RE}) eyes decreases, and it reaches a minimum when the contrast difference (C_{LE} - C_{RE}) is zero. There are two fundamental concepts explaining the effect of interocular contrast difference (ICD) on stereopsis. First, in the case of C_{LE} - C_{RE} = 0, correlated noise from the two eyes has similar magnitude that can be effectively canceled by the binocular visual system, assuming that disparity processing is based on the difference of the signals between the two eyes. Second, a mutual interocular inhibition between the left and right monocular channels before binocular convergence can affect stereopsis. Specifically, on receiving two monocular signals of different strengths, the interocular inhibitory mechanism exerts a stronger suppression on the monocular channel receiving the weaker stimulus, thereby effectively reducing its signal strength. Consequently, the spatial resolution of the subsequently integrated binocular representation becomes less optimal. Taking a modeling approach, Hou et al. used an extended multi-pathway contrast gain control model to account for the effect of binocular contrast on stereopsis. According to their model, the stereo signal strength depends on the product of the signal strengths in the two eyes (i.e., C_{LE} \cdot C_{RE}, where C_{LE} and C_{RE} represent the neural signal strengths in the left and right eyes, respectively) after the contrast gain control operation, which includes interocular gain control. Stereopsis is compromised when one eye's channel exerts a stronger gain control over the fellow eye's channel.

The hypotheses and models above can be investigated by testing observers with interocular imbalance, also commonly referred to as sensory eye dominance (SED). Previously, Ooi and He found observers with clinically normal binocular vision exhibited SED due to an imbalance of interocular inhibition (SED_{inhibition}). To measure SED_{inhibition}, they presented the observer with dichoptic orthogonal gratings to evoke a strong mutual inhibition between the two monocular channels (Fig. 1a). To achieve equal predominance between the two eyes, some observers needed a higher luminance or contrast.
A grating in one eye (the nondominant eye) to balance out the orthogonal grating in the fellow (dominant) eye. The extra amount of luminance or contrast reflects the magnitude of imbalance of interocular inhibition (i.e., $\text{SED}_{\text{inhibition}}$).

Further investigations revealed that the sensory dominant (strong) eye was not always the eye with the motor dominance. The sensory dominant eye did not always have the higher monocular contrast sensitivity or higher perceived monocular brightness than the nondominant (weak) eye, indicating that some, if not all, $\text{SED}_{\text{inhibition}}$ originated from an imbalance of interocular inhibition, rather than a mere monocular difference. Of significance, observers with larger $\text{SED}_{\text{inhibition}}$ had higher stereo thresholds and longer stereo reaction time (RT), even as the interocular inhibitory process and stereo process can operate concurrently. Although the reduced stereopsis is consistent with prevailing understanding, it is notable that the observed effects occurred while the observers were viewing stereo half-images with equal contrast ($C_{\text{LE}} = C_{\text{RE}}$). This suggests $\text{SED}_{\text{inhibition}}$ can cause a stronger suppression of the nondom-
iniant eye’s contrast signals, thus reducing its spatial resolution, leading to a compromised stereo resolution of the integrated binocular representation. Further supporting this hypothesis, we showed stereopsis improved after we reduced SEDinhibition through a push-pull perceptual learning protocol that did not directly train stereopsis with a stereo task.16,22–24 Rather, the protocol simply readjusted the excitatory-inhibitory balance through repetitive attentive binocular rivalry stimulation.

It might appear puzzling that there would be a significant correlation between stereo threshold and SEDinhibition, given that SEDinhibition is measured with a pair of orthogonal gratings (typical binocular rivalry stimulus) that does not induce binocular depth perception. One possible explanation lies in the fact that during binocular rivalry, the entire stimulus image in the suppressed eye at the corresponding area is not perceived (i.e., suppressed), indicating interocular suppression of all visual channels. This suggests that binocular rivalry stimuli trigger a broad-based interocular inhibitory process that could be related to the suppression of false matches. Thus, it is reasonable to assume that SEDinhibition can, at least in part, characterize the interocular inhibitory mechanism that contributes to horizontal disparity processing.6,8,25,26

Observers also can exhibit sensory eye dominance when binocularly combining similar monocular images, in what we refer to as SEDcombo.27–29 SEDcombo is measured with a pair of dichoptic horizontal gratings having the same spatial frequency but different phases (Fig. 1b).27,28,30–32 These studies assume that the gain control mechanism plays a crucial role in binocular combination and determines the contrast/ brightness perception of the combined binocular representa- tion.5,8,28,30–34 It has been hypothesized that a difference between the channels of the two eyes and an imbalance of mutual inhibition between the channels of the two eyes contribute to SEDcombo. SEDcombo differs from SEDinhibition in that the latter specifies an imbalance between the channels of the two eyes carrying vastly different feature signals (e.g., orthogonal orientation). Despite this important difference, we recently found SEDcombo and SEDinhibition are strongly correlated, and stereo threshold increases with the magnitude of SEDinhibition as well as SEDcombo.19

Existing evidence indicates that optimal stereopsis is achieved when the two eyes receive half-images with equal external contrast (Cle = CRE). But it is unknown how having an SED would affect stereopsis when the half-images have different interocular contrast. Here, extending the previous approaches,6,8 we hypothesized that for observers with SED but otherwise clinically normal binocular vision, stereopsis is optimal not when cle = CRE, but when the nondominant eye receives the stronger half-image that balances the signal strengths in the two eyes. We investigated this by measuring how observers’ SED affected their stereo disparity thresholds and stereo RT as we varied the interocular contrast difference (ICD = log10[C1] − log10[C2]) of random-dot-stereograms (RDS) (Fig. 1c). To do so, we kept the product of the external contrast in the two eyes, cle·cre or log10(C1) + log10(C2), constant while varying the ICD. This allowed us to obtain a quantitative relationship to reveal whether there was an interocular-stimulus-compensating effect, that is, for observers with SED, both stereo disparity thresholds and RT would be lower when the nondominant eye received the stronger half-image (compensated), rather than when the dominant eye received it (anticompensated). The quantitative relationship also allowed us to estimate the ICD at which stereo performance would be optimal. If indeed, observers with SED could be estimated to exhibit optimal stereopsis when ICD is not zero, but at a compensated level, it would open new avenues to enhance stereopsis using devices that compensate for the interocular imbalance.

**Methods**

The study used stereo stimuli with five levels of ICD (ICD = log10[C1] − log10[C2] = −0.4, −0.2, 0.0, 0.2 or 0.4 log unit) (note: log contrast is defined as log10[C], where C is the Michelson contrast in percentage; for example, 1.4 log unit is equivalent 25.1% contrast). The average contrast received by the two eyes, (log10[C1] + log10[C2])/2, was constant at 1.2 log unit or 15.8% contrast, so that the five contrast combinations were (1.0, 1.4), (1.1, 1.3), (1.2, 1.2), (1.3, 1.1), (1.4, 1.0). The order of testing the contrast combinations was pseudo-randomized.

Using the above-mentioned contrast settings, stereo disparity threshold and stereo RT of both crossed (front depth) and uncrossed (back depth) binocular disparity stimuli were tested. Observers were tested on the threshold task before the RT task. In addition, SEDinhibition and SEDcombo were also tested during each stereo testing session. For each type of SED, we took the average of 12 pairs of data for analysis. Below, we describe the detailed stimuli and procedures for the stereo threshold, stereo RT, SEDinhibition, and SEDcombo measurements.

**Observers**

Ten observers (ages 18–30; three males and seven females) who were naïve to the purpose of the study participated. All 10 observers participated in the stereo disparity threshold experiments and 7 in the stereo RT experiments. All had normal or corrected-to-normal visual acuity (at least 20/20), clinically acceptable fixation disparity (<8.6 arc min) and stereopsis (<40 arc sec). The research conducted followed the tenets of the Declaration of Helsinki and was approved by the institutional review board. Informed consent was obtained from the observers before the experiments.

**Design**

Gamma-corrected stimuli were generated on either a PC (Linux) or Mac Pro computer running MatLab (MathWorks, Natick, MA, USA) with PsychToolBox35,36 and presented on a 21-inch flat CRT monitor. The resolution of the monitor was set at 2048 × 1536 @ 75 Hz refresh rate. During the experiments, the observers viewed the computer monitor through a haploscopic mirror system attached to a head-and-chin rest from a distance of 100 cm.

**Stimuli and Procedures**

**Stereo Disparity Threshold.** An 8° × 8° random-dot-stereogram (35 cd/m²) with a variable crossed or uncrossed disparity disc target (1° diameter) was used in separate experimental blocks (example in Fig. 1c). The dot size of the random-dot was 1.34, 2.01, or 3.36 arc min, with the larger dot size being used only if the observer was not able to reliably perceive the smaller dot size. The contrast levels of the stereo half-images were set at various predetermined levels. The mean contrast of the two half-images, (log10[C1] + log10[C2])/2, was 1.2 log unit, so that the five contrast combinations were (1.0, 1.4), (1.1, 1.3), (1.2, 1.2), (1.3, 1.1), (1.4, 1.0).

The 2IFC method in combination with the staircase procedure was used 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/m²), blank (147 ms), interval-1, blank (400 ms), interval-2, and random-dot mask (200 ms, 8° × 8°, 1.7 log unit contrast, mean luminance of 35 cd/m²). Both intervals comprised images with random-dot, but only the images in one interval carried the binocular disparity signal, whereas the images in the other...
interval had zero disparity. The interval duration was individually set between 27 and 93 ms to avoid the floor or ceiling effect. During the experiment, the observer indicated by key presses whether the stimulus with the disk target was seen in interval 1 or −2. A block of trials comprised 10 reversals (step size = 0.2 arc min, total ~40–60 trials), and the average of the last 6 reversals was taken as the disparity threshold.

Each observer’s stereo disparity threshold was tested with five combinations of ICD (ICD = log10[C1]Log – log10[C2]Log) = −0.4, −0.2, 0, 0.2, or 0.4 log unit) and two binocular disparity types (crossed and uncrossed). Threshold testing for each contrast combination was repeated five times (i.e., five 2IFC blocks). Observers were tested for crossed disparity thresholds before uncrossed disparity thresholds. All, except observer S10 (who discontinued from the study due to her busy schedule), were tested for both crossed and uncrossed disparity thresholds.

Stereoscopic RT. The random-dot stereograms used to measure stereoscopic RT were similar to those used for measuring stereo thresholds in that the disparity disk was 1°, and the mean luminance was 35 cd/m². However, the binocular disparity of the disk was fixed at either ±0.67°, ±1.34°, or ±2.04° arc min. The particular disparity value chosen was scaled according to each observer’s stereo disparity threshold to avoid the floor or ceiling effect. Similar to the stereo disparity threshold testing, each observer’s stereo RT was tested with the contrast levels of the stimulus set similar to the stereo disparity threshold testing. The order of testing the contrast combinations was pseudo-randomized.

An experimental block comprised 50 trials, with 20 front-trials, 20 back-trials, and 10 catch trials in which the random-dot stereogram carried zero binocular disparity. All trials were semi-randomly interleaved, with the provision that no four consecutive trials had the same depth sign. The observer began a trial by aligning his or her eyes on the nonius fixation before pressing the start button. The target was then presented for a predetermined fixed duration and followed by a random-dot mask (200 ms, 8° × 8°, 1.7 log unit contrast, 35 cd/m²). The fixed duration (within a range of 160–1000 ms) was individually determined to avoid the floor or ceiling effect. The observer’s task was to press one of two keys on the keyboard immediately on detecting the disc target, to indicate whether the disc was seen in front or in back. If no depth was perceived, either because the depth was not perceived or there was no depth stimulus due to it being a catch trial, the observer did not need to respond and simply waited for the next trial to begin 2000 ms later. If the observer made a false alarm by pressing either response key in a catch trial, he or she would be given an audio feedback. No more than two false alarms were permitted in a block of trial. If a third false alarm was made, the computer would terminate the program and the observer had to repeat the same block of trials. Each observer was tested over five blocks of trials for each interocular contrast combination.

Sensory Eye Dominance of Inhibition (SEDInhibition). The stimulus comprised a pair of dichoptic vertical and horizontal sinusoidal grating discs (diameter = 1°, 3 cycle/deg, 35 cd/m²) on a gray background (8° × 8°, 35 cd/m²) (Fig. 1a). The contrast of the horizontal grating was held constant (1.5 log unit), whereas the contrast of the vertical grating was variable (0.376–1.976 log unit). A trial began with central fixation on the nonius target (0.45° × 0.45°, line width = 0.1°, 70 cd/m²), which was removed after the observer was ready and pressed the start button. The dichoptic orthogonal gratings were presented 147 ms after fixation removal and lasted for 400 ms, followed by a 200-ms mask (8° × 8° random dots, 35 cd/m², 1.7 log unit). The observer responded to his or 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, we refer to its contrast at equal predominance as the balance contrast of the LE. To obtain the balance contrast of the RE, the gratings were switched between the eyes. The difference between the LE and RE balance contrast values is defined as SEDInhibition.

Sensory Eye Dominance of Combination (SEDcombo). The test stimulus comprised a pair of dichoptic horizontal grating squares (1° × 1°, 3 cycle/deg, 35 cd/m²) with a 90° phase difference between them (Fig. 1b). The average phase of the two gratings was always held at 0 degree (θL = −45 and θR = −45, or θL = −45 and θR = 45). A pair of horizontal reference lines was located adjacent to each side of the dichoptic grating. The contrast of the grating in one half-image was fixed at 1.5 log unit, whereas the contrast of the other grating in the tested eye varied from 0.376 to 1.976 log unit. The observer prepared for a trial by maintaining eye alignment on the fusion-lock (2° × 2°) before pressing a button on the keyboard. This was followed 147 ms later, with the dichoptic grating stimulus for 400 ms. A 200-ms mask followed to terminate the trial (8° × 8° random dots patch, 35 cd/m², 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 perceived location of the grating band depended on the variable contrast in the tested eye, which was adjusted using the QUEST procedure.

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 (LE). To measure the RE’s balance contrast (RE), 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 SEDcombo.

To control for the possible effect of contrast and grating phase in each half-image causing a positional bias, we tested SEDcombo 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. In our data analysis below, SEDcombo of the two display types were averaged in order to cancel the possible positional bias (slight).

**RESULTS**

**SEDInhibition and SEDcombo.**

Figure 2 plots all observers’ SEDcombo as a function of SEDinhibition (n = 10). There is a significant correlation between the two measures of SED (r² = 0.653, P = 0.005). Further examination of individual observers’ data reveals that all observers had the same sign of dominant eye for SEDinhibition and SEDcombo, even though the absolute magnitudes of the SED differed.

**Effects of ICD on Stereo Threshold**

Figures 3a and 3b, respectively, show one observer’s (S3) crossed and uncrossed disparity thresholds as a function of ICD (the remaining nine observers’ data are provided in the Supplementary Material). Overall, stereo threshold was lowest at approximately ICD = 0, and increased as the magnitude of ICD increased. This trend is consistent with previous reports in
The graph plots the correlation between SED_{combo} against SED_{inhibition}. Each data point represents the results of an observer. The solid line represents the regression function, whereas the dashed curves reveal the 95% confidence interval.

The results showed a positive correlation between SED_{combo} and SED_{inhibition}. The correlation coefficient was calculated to be 0.972 with a p-value of 0.031.

The figures also reveal that the stereo thresholds were generally lower on the negative side of the x-axis where \( \log_{10}(C_{LE}) - \log_{10}(C_{RE}) < 0 \) (high contrast in RE), than on the positive side of the x-axis where \( \log_{10}(C_{LE}) - \log_{10}(C_{RE}) > 0 \) (high contrast in the LE). This asymmetric pattern is consistent with the observer's SED, which revealed LE dominant for (high contrast in the LE). This asymmetric pattern is consistent with the observer's SED, which revealed LE dominant for (high contrast in the LE).

We next analyzed the ratio of stereo thresholds of each anticompenating to compensating stimulus pair for [ICD] = 0.4 log unit and [ICD] = 0.2 log unit. For example, in Figure 3a (as alluded to earlier in the section "Effects of ICD on Stereo Threshold"), because the observer had a dominant LE SED_{inhibition} and SED_{combo}, the stereo threshold with the +0.4 log unit stimulus is defined as the anticompenating pair (strong stimulus in the dominant LE), whereas the stereo threshold with the –0.4 log unit stimulus is defined as the compensating pair (strong stimulus in the nondominant eye). If there exists an interocular-stimulus-compensating effect, the ratio is expected to be larger than 1. Accordingly, we pooled all observers' data according to their dominant eye and plotted the geometric average of crossed and uncrossed disparity threshold ratios in Figure 3e. (Note: All observers had the same dominant eye for SED_{inhibition} and SED_{combo} [Fig. 2].)

The notched box plot in Figure 3e shows that for both [ICD] = 0.2 and 0.4 log unit contrast pairs, the median ratios are larger than 1, demonstrating the interocular-stimulus-compensating effect. Further statistical analysis reveals a significant interocular-stimulus-compensating effect for [ICD] = 0.4 log unit test \( \left( t[9] = 5.916, P < 0.001, \text{one-tailed } t\text{-test} \right) \), but not for [ICD] = 0.2 log unit \( \left( t[9] = 1.752, P = 0.117, \text{one-tailed } t\text{-test} \right) \). In addition, the ratio is significantly larger for [ICD] = 0.4 than 0.2 log unit \( \left( t[9] = 3.207, P = 0.011, \text{one-tailed } t\text{-test} \right) \), suggesting that the interocular-stimulus-compensating effect reliably increased with [ICD].

**Effects of ICD on RT**

Figures 4a and 4b show the same observer's mean RT, respectively, for detecting crossed and uncrossed disparity stereo stimuli. Generally, the graphs reveal that stereo RTs were lower when the dominant eye received the lower contrast stereo half-image (i.e., exhibiting the interocular-stimulus-compensating effect). To quantify this, we used the same analyses as those used for the stereo disparity threshold data above.
FIGURE 3. Stereo disparity thresholds obtained with RDS of different interocular contrast. The symbols in graphs (a) and (b) represent the disparity thresholds of an observer as a function of ICD, respectively, for crossed and uncrossed disparity RDS. The quadratic function was used to define the curve that fit the data in each graph. The minimum of each curve is indicated by the blue vertical line in each graph, and its intercept on the x-axis provides a measure of SEDstereo-threshold. The red and green vertical lines intercepting the curve indicate, respectively, SEDinhibition and SEDcombo of the observer. Overall, notice that the minimum of each curve is shifted leftward, which is consistent with all three types of SEDs. Graphs (c) and (d) provide an alternative way to demonstrate the consistency among the three types of SEDs by plotting, respectively, each observer’s SEDstereo-threshold as a function of SEDinhibition and SEDstereo-threshold as a function of SEDcombo. The solid lines represent the regression functions and the dashed curves represent the 95% confidence interval. The notched box plot in graph (e) shows the geometric average of crossed and uncrossed disparity stereo threshold ratios for both |ICD| = 0.2 and 0.4 log unit contrast pairs.
**FIGURE 4.** Stereo RT obtained with RDS of different interocular contrast. The symbols in graphs (a) and (b) represent the stereo RT of an observer as a function of ICD, respectively, for crossed and uncrossed disparity RDS. The quadratic function was used to define the curve that fit the data in each graph. The minimum of each curve is indicated by the blue vertical line in each graph, and its intercept on the x-axis provides a measure of $S_{\text{ED,RT}}$. The red and green vertical lines intercepting the curve indicate, respectively, the $S_{\text{ED,inhibition}}$ and $S_{\text{ED,combo}}$ of the observer. Overall, notice that the minimum of each curve is shifted leftward, which is consistent with all three types of SEDs. Graphs (c) and (d) provide an alternative way to demonstrate the consistency among the three types of SEDs by plotting, respectively, each observer’s $S_{\text{ED,RT}}$ as a function of $S_{\text{ED,inhibition}}$ and $S_{\text{ED,combo}}$ as a function of $S_{\text{ED,combo}}$. The solid lines represent the regression functions and the dashed curves represent the 95% confidence interval. The notched box plot in graph (e) shows the geometric average of crossed and uncrossed disparity stereo RT ratios for both $|\text{ICD}| = 0.2$ and 0.4 log unit contrast pairs.
Curve Fitting for SEDstereo-RT. We fit all observers’ stereo RT data with the quadratic function (see Fig. 4a for crossed disparity and 4b for uncrossed disparity; the remaining six observers’ data are provided in the Supplementary Material). Overall, the results exhibit an interocular-stimulus-compensating effect, wherein RTs were shorter when the nondominant eye (ICD < 0) rather than the dominant eye (ICD > 0) viewed the higher contrast stereo half-image. We next obtained the ICD where RT was minimum from the fitted curves and defined it as the SED due to stereo RT, SEDstereo-RT. From this, we then plotted each observer’s average crossed and uncrossed SEDstereo-RT versus SEDinhibition and SEDstereo-RT versus SEDcombo in Figures 4c and 4d, respectively. (There is no significant difference between the crossed and uncrossed data (t(6) = 0.216, P = 0.836, two-tailed paired t-test).) We found the slopes of both regression lines are positive, indicating that the sign of eye dominance from all three SED definitions are the same (SEDstereo-RT versus SEDinhibition: \[ r = 0.655, P = 0.027 \] or \[ r = 0.797, P = 0.007 \] with Spearman’s rank correlation coefficient); SEDstereo-RT versus SEDcombo: \[ r = 0.360, P = 0.155 \] or \[ r = 0.460, P = 0.094 \] with Spearman’s rank correlation coefficient). Also note the average SEDstereo-RT (0.107 ± 0.027 log unit) was much smaller than SEDinhibition (0.212 ± 0.036 log unit, t(6) = 3.075, P = 0.011, one-tailed paired t-test) and SEDcombo (0.365 ± 0.107 log unit, t(6) = 2.202, P = 0.035, one-tailed paired t-test).

RTs for Compensating Versus Anticompensating Stimuli. As with the stereo disparity threshold data, we sorted the observers’ mean RTs according to their dominant eye based on their SED (SEDinhibition and SEDcombo had the same sign for all observers, as shown in Fig. 2).

The notched box plot in Figure 4e depicts the geometric average ratios from crossed and uncrossed disparity conditions. For both ICD = 0.2 and 0.4 log unit contrast pairs, the median ratios are larger than 1, demonstrating the interocular-stimulus-compensating effect. Further statistical analysis reveals a significant interocular-stimulus-compensating effect for [ICD] = 0.4 log unit (\[ t(6) = 3.131, P = 0.010 \] one-tailed t-test), but not for [ICD] = 0.2 log unit (\[ t(6) = 0.747, P = 0.424 \] one-tailed t-test). The ratio is significantly larger for \[ |ICD| = 0.4 \] than 0.2 log unit (\[ t(6) = 2.141, P = 0.038 \] one-tailed paired t-test), suggesting that the interocular-stimulus-compensating effect reliably increased with [ICD]. This finding, along with the stereo disparity threshold results, demonstrates the interocular-stimulus-compensating effect in observers with SED.

Discussion

We investigated the possibility that for observers with SED but otherwise clinically normal vision, stereo perception was optimal not when \( C_{LE} = C_{RE} \), but when the non-sensory-dominant eye received the stronger half-image. Supporting this hypothesis, we found a small but significant tendency for the observers’ SEDstereo-threshold and stereo-thresholdmin to deviate from ICD = 0 in the direction consistent with their SED. Furthermore, we found that observers with SEDinhibition and SEDcombo had significantly lower stereo thresholds and shorter stereo RT on stimuli with unequal interocular contrast when the non-sensory-dominant eye viewed the higher contrast half-image. This is because stereo stimuli with large \( |ICD| \) tend to induce a larger imbalance of interocular inhibition. By linking the observer’s SEDinhibition and SEDcombo, with the effect of ICD on stereopsis, we provided further support for the notion that the effect is mediated by the interocular inhibitory and interocular gain control mechanisms.\(^2,6,8\)

We also estimated from individual observer’s data, the ICD that would lead to the minimum disparity threshold (SEDstereo-threshold) and stereo RT (SEDstereo-RT). If we assume that the stereo process achieves optimal stereopsis when the signals within the binocular channel are balanced, then SEDstereo-threshold and SEDstereo-RT both reflect the sensory eye dominance associated with the stereo process. Interestingly, when estimated this way, our observers’ SEDstereo-threshold and SEDstereo-RT were much smaller than their empirical interocular imbalance from SEDinhibition and SEDcombo measurements. However, we believe this observation is not surprising because these SEDs were measured with different binocular stimuli that very likely activated various binocular subprocesses differently. The two half-images of the stereo stimulus used for measuring SEDstereo-threshold and SEDstereo-RT were quite similar and are not likely to evoke a large interocular inhibitory activity. Conversely, the binocular stimuli for measuring SEDinhibition (orthogonal gratings) and SEDcombo (horizontal gratings with large relative phase shift) had larger interocular image differences, which induced stronger interocular inhibitory activities. It is also noteworthy to point out that the random dots stereograms used to obtain the SEDstereo-threshold and SEDstereo-RT had spectrally broad spatial frequency content in contrast to the grating stimuli (3 cpd) used for measuring SEDinhibition and SEDcombo. It is possible that the extent of interocular imbalance differs among the various spatial frequency channels, with channels having small SEDs contributing more to the stereo perception. Future studies are needed to reveal whether SEDs are significantly affected by spatial frequency.

The current study is the first to investigate the effect of ICD on stereopsis of observers with clinically normal binocular vision who exhibited SEDinhibition and SEDcombo. Of significance, our study differs from previous studies that varied ICD as well as the average contrast of the stereo half-images. Here, by keeping the latter constant, we were able to reveal the sole effect of ICD on stereopsis. Our findings could perhaps be understood in the context of the model by Hou et al.,\(^9\) which posits that the stereo signal strength depends on \( C_{LE}C_{RE} \), where \( C_{LE} \) and \( C_{RE} \) represent the neural signal strengths of the left and right eyes, respectively. According to their model, if the visual system has equal interocular gain control, test conditions that vary \( C_{LE} \) and \( C_{RE} \) while keeping \( C_{LE}C_{RE} \) or \( \log_{10}(C_{LE}) \) and \( \log_{10}(C_{RE}) \) constant lead to a symmetrical U-shaped function of stereo threshold versus ICD. Although Hou et al.\(^9\) did not explicitly address a scenario with significant imbalance of interocular gain control, and based on their model, one can predict an asymmetrical U-shape function of stereo threshold versus SED. This prediction would be consistent with our data (e.g., Figs. 3a, 3b).

The finding of an interocular-stimulus-compensating effect in observers with SED has a potential application. It suggests that using a stereo device that could augment contrast of the images presented to the nondominant eye could lead to the observer experiencing improved stereopsis of three-dimensional visual scenes. But because our current study tested observers only in the laboratory setting with stimuli having large ICDs, further research is needed to realize this potential application.

Acknowledgments

Supported by National Institutes of Health grants EY023561 and EY023374.

Disclosure: C. Han, None; Z.J. He, None; T.L. Ooi, None

References