Disparity Tuning in Mechanisms of Human Stereopsis

SCOTT B. STEVENSON,*† LAWRENCE K. CORMACK,* CLIFTON M. SCHOR,* CHRISTOPHER W. TYLER†

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The change in sensitivity across some stimulus dimension which follows adaptation to a particular stimulus can reveal a great deal about the tuning characteristics of underlying sensory/perceptual mechanisms. In this study, a psychophysical adaptation paradigm was employed to characterize the disparity tuning of perceptual mechanisms involved in stereopsis. The stimulus was a dynamic random-dot stereogram (DRDS) portraying a surface which varied in interocular correlation (IOC) and retinal disparity. Adaptation to a fully correlated DRDS surface produced an elevation in IOC threshold over a relatively narrow range of disparities, with maximum effect at the disparity of the adapting stimulus. The width of these disparity tuning functions varied from 5 arc min for adaptation at the horopter to 20 arc min for adaptation at 20 arc min disparity. Frequently, IOC sensitivity was enhanced for disparities on either side of the adapted disparity, suggesting that an opponent center-surround organization operates at an early level of disparity processing. A model of underlying channel structure consistent with these data is presented.

INTRODUCTION

A common finding in visual psychophysics is that stimulus dimensions are processed through "channels" or "filters": perceptual mechanisms whose selectivity on a visual dimension is narrow relative to the overall sensitivity function for that dimension, but whose selectivity is often broad relative to the discrimination function for that dimension. The trichromacy of color vision provides the classic example of channeling in human vision. The photopic spectral sensitivity function reflects a weighted sum of somewhat narrower cone sensitivity functions, or wavelength channels. Comparison of the activity in these cone types in turn produces very precise wavelength discrimination.

Evidence for channels has been found for a number of visual dimensions including grating spatial frequency and orientation (Blakemore & Campbell, 1969), lateral motion (Levinson & Sekuler, 1980), motion in depth (Beverley & Regan, 1975) and static retinal disparity (Felton, Richards & Smith, 1972). In a review of such evidence, Regan (1982) offers a list of defining characteristics of a "visual channel" which restricts the term to mechanisms that are tuned for one, and only one, visual dimension. This paper concerns the tuning characteristics of mechanisms sensitive to static retinal disparity, and while we refer to "channels," it should be clear that we have not determined whether the mechanisms are tuned on other visual dimensions.

Early evidence for disparity tuned mechanisms in human vision was reported by Blakemore and Julesz (1971), who found that adaptation to a random-dot stereogram produced shifts in the apparent depth of subsequently viewed stereograms. They argued that these shifts indicated that relatively narrow disparity tuned mechanisms had been adapted. Further psycho-physical evidence for disparity tuning was provided by Felton et al. (1972) and by Blakemore and Hague (1972) in experiments that revealed disparity-specific elevation of contrast threshold after adaptation to a sine-wave grating. Thresholds were typically elevated the most at the disparity of adaptation, supporting the theory that the visual system includes a number of disparity-tuned mechanisms with peak sensitivities covering a range of disparities around the horopter.

The "many narrow channels" view of disparity processing can be contrasted with Richards (1971) three-pool hypothesis. He found, and Jones (1977) confirmed that certain "stereononalous" individuals were unable to make accurate disparity discriminations over a relatively broad range of disparities. Consistent with the pattern of deficits found, Richards proposed the existence of three pools of disparity detectors: a fine pool for...
small disparities, and near and far pools for larger crossed and uncrossed disparities, respectively.

Recent evidence from single cell recordings in monkey (Poggio, Gonzalez & Krause, 1988) provides some support for both of the above models of disparity channeling. Some cortical neurons ("tuned") exhibit narrow disparity tuning, with peaks distributed across a range of disparities. Others exhibit broad tuning functions ("Near/Far"), being excited only by either crossed or uncrossed disparity but not both. The difficulty in linking human stereopsis with the single cell results is that these disparity tuning profiles are found for both horizontal and vertical disparity. While sensory and motor fusion operates on both axes of disparity, stereoscopic depth occurs only for horizontal disparity. Therefore, it is difficult to know if all, or only a subset of these cell types might represent the neural basis of stereopsis.

Our purpose in these experiments was to examine the disparity tuning of human perceptual mechanisms processing interocular correlation (i.e. "solving the stereo-matching problem"). We used an adaptation paradigm to study the characteristics of visual mechanisms tuned to disparities in the range of 0.5 deg crossed to 0.5 deg uncrossed disparity. In order to target disparity processing mechanisms specifically, we have used dynamic random dot stereogram stimuli that varied in interocular correlation (IOC); that is, the proportion of left and right image dots which match each other at the target disparity (Julesz & Tyler, 1976). We began by measuring thresholds for detecting IOC over a range of disparities, and then determined the elevation in these thresholds which occurred after adaptation at a particular disparity. Finally, we generated a multichannel model to simulate a set of underlying mechanisms consistent with our results.

**GENERAL METHODS**

**Stimuli**

The dynamic random dot patterns were generated by special-purpose electronics that allowed for precise control of disparity and interocular correlation. Figure 1 is a schematic diagram of the stimulus generation system. Pseudo-random binary noise was generated by a 32 bit shift register with XOR feedback from bits 23 and 30. A second, uncorrelated noise source was produced with an XOR combination of bits 25 and 26. (Extensive computer simulation of the shift register system confirmed that these noise streams are uncorrelated.)
Zero interocular correlation was produced by directing one noise source to the left monitor and the other to the right monitor and a positive 1.0 interocular correlation was produced by directing the same noise source to both monitors. Various intermediate levels of interocular correlation were produced by switching rapidly between the two sources of noise and sending the result to the left monitor's image, while always directing the first source to the right monitor.* For example, at the lowest IOC level used (2%) one fiftieth of the dots were forced to be perfectly correlated in the two views (same noise source) while the remainder were randomly correlated or anti-correlated (independent noise sources). Although the percent correlation is small in this example, the actual number of matching dots is quite high since each frame of the display had over 18,000 dots visible to the subject.

Disparity was produced and controlled by directing the horizontal sync signal going to the left monitor through a programmable digital delay device. Delay of the sync resulted in a lateral shift of the noise pattern, producing a disparity of the noise with respect to the fixation dot. In our configuration, each unit of delay (nominally 5 nsec) resulted in a disparity change of about 0.27 arc min. The first 255 steps of delay allowed for a disparity range of about 70 arc min.

All stimuli in these experiments were viewed in a mirror haploscope at a distance of 57 cm. Convergence was adjusted for normal viewing at this distance. A chin rest and forehead rest stabilized the subject's head. The display luminance (measured through the haploscope mirrors) was 40 cd/m² and the contrast (measured on a static pattern) was 80%. A pair of 9 deg circular apertures concealed the edges of the display, producing a dark surround. The display dots were 5 arc min by 2 arc min tall. A black circle subtending 9 arc min served as a fixation dot. Vertical nonius lines subtending 3 by 40 arc min provided a reference for maintaining constant convergence. We estimate that convergence accuracy is kept to within 1 arc min by this method.†

The appearance of the display varied with IOC from a flat, opaque plane of dynamic dots at positive 1.0 IOC to a dynamic, space-filling swarm of dots at many depths at 0.0 IOC. Intermediate levels appeared as a surface embedded in the swarm, with the apparent density of the surface being monotonically, if not linearly, related to the level of IOC. Except for correlations right at threshold, subjects agreed that when the surface was visible its depth relative to the fixation mark was also evident.

Procedure

Thresholds for detection of interocular correlation were measured in three subjects using a method of constant stimuli and a temporal two alternative forced choice procedure. Subjects viewed a zero correlation pattern at all times except during the signal presentation. Each trial began with a tone to signal the first interval, a 500 msec pause, a 200 msec signal (or blank), and another 500 msec pause. The second interval followed the same sequence. Subjects then indicated which of the two intervals contained non-zero correlation by pressing a key on the keyboard. The next trial followed immediately after each key press. When fixation wandered or a blank or saccade interrupted a trial, it could be repeated, with a new random interval order, by pressing the space bar.

A set of 4–7 IOC levels presented in random order composed a block of trials and 30 blocks composed a single threshold determination. Threshold was defined as the level producing 75% correct performance, as interpolated from a linear regression between stimulus level and Z-transform of proportion correct.

Subjects

Subjects for these experiments were three of the authors of this paper. All had corrected to optimal visual resolution acuity and excellent stereoacuity for this stimulus, as reported previously (Stevenson, Cormack & Schor, 1989).

EXPERIMENT I: BASELINE CORRELATION SENSITIVITY

We began our experiments by establishing baseline performance in the correlation detection task, for purposes of comparison to adapted performance.

Methods

Five threshold measures for each of 17 disparity levels (0, 2.5, 5, 10, 15, 20, 25, 30 and 35 arc min for both crossed and uncrossed directions, and additionally 40 and 45 arc min uncrossed for SBS) composed a complete baseline correlation sensitivity function. Prior to each run, subjects were shown a fully correlated surface at the disparity to be tested in order to minimize uncertainty about where test stimuli would appear.

Results

The results from the baseline measures of correlation sensitivity are plotted in Fig. 2. Disparity of the stimulus relative to fixation is plotted on the horizontal axis...
against interocular correlation at threshold (log scale). Correlation detection thresholds vary from about 0.10 at the horopter to 1.0 at about 1 deg of crossed or uncrossed disparity, the upper disparity limit (see below). In general, all three subjects show a logarithmic increase in correlation threshold with linear increases in disparity away from zero. However, each subject’s function shows localized irregularities and extensive baseline measurements made in the context of a follow-up study (Cormack, Stevenson & Schor, 1991a; in preparation) reveal that the coarser features are stable over time (e.g. the plateau from +10 to +20 arc min the data for LKC). Of course, some of these features are due to normal statistical variation and were not replicated exactly in the follow-up (e.g. the bump at 0 arc min in the data for SBS).

Figure 3 shows the baseline data replotted to compare sensitivity for near and far disparities. Data for the three subjects have been displaced vertically for clarity. Error bars represent ±1 SE. Subject CMS shows a small but consistent difference in sensitivity between near and far disparity values, being more sensitive to near (crossed) disparity over the range tested. The other two subjects show no overall difference between near and far sensitivity, although LKC tends to be more sensitive to far disparities greater than 15 arc min.

EXPERIMENT II: UPPER DISPARITY LIMIT

The upper disparity limit (UDL) for detecting correlation under our conditions is the disparity at which correlation threshold reaches 100% and beyond which even fully correlated surfaces are indistinguishable from 0 correlation. We measured the UDL for both crossed and uncrossed disparity in all three subjects.

Methods

Upper disparity limits for correlation detection were determined with a variant on the baseline procedure in which the correlation was kept at 100% and the disparity was varied according to the method of constant stimuli. Otherwise, the trial sequence was the same as described above for baseline measures. The resulting psychometric functions were used to determine a threshold value reflecting the largest disparity for which a 1.0 correlated surface is discriminable from 0.0 correlation.

Results

The upper disparity limits are plotted in Fig. 2 as the intersection between the sensitivity curves and the horizontal line at 0.0 on the vertical axis. They are between 45 and 65 arc min for all three subjects and both directions of disparity, and are consistent with the exponential trend in the baseline data for each subject.

EXPERIMENT III: ADAPTATION

The effect of adaptation on correlation sensitivity was determined for all three subjects and compared to the baseline measures in order to reveal the extent to which adaptation at a particular disparity affects sensitivity over the visible disparity range revealed in the data of Fig. 2.

Methods

Adaptation trials followed the same sequence as baseline trials except that a 1 min adaptation interval preceded each threshold determination and a 500 msec "refresher" adaptation interval preceded each interval of each trial. The adaptation stimulus was a 1.0 correlated surface at some fixed disparity. Three threshold

![Figure 2](https://example.com/figure2.png)

**FIGURE 2.** Baseline correlation thresholds for three subjects. The log of interocular correlation at threshold is plotted against horizontal disparity of the test surface relative to fixation. Negative values of disparity indicate near (crossed) disparity. A value of 0.0 on the vertical axis represents a correlation of 1.0, the maximum possible. The intersection of each subject’s curve with the line at 0.0 indicates the upper disparity limits for correlation detection in our task. Correlation thresholds were measured for each subject out to ±35 arc min disparity, with each point representing the mean of 5 runs. Upper disparity limits were measured in a separate experiment. The general trend can be characterized as being symmetric about 0 disparity, with an exponential relationship between correlation threshold and disparity. Each subject showed idiosyncratic departures from this exponential trend, such as the bumps at 0 disparity for SBS, at +5 arc min for CMS and at +10 for LKC.

![Figure 3](https://example.com/figure3.png)

**FIGURE 3.** Baseline correlation thresholds replotted for purposes of near/far comparison. Absolute value of the disparity of the test stimulus is plotted horizontally against the log of the correlation at threshold, with data from each subject displaced vertically for clarity (0.5 log units for LKC, 0.8 for CMS). Open symbols plot data for far (uncrossed) disparity, solid symbols for near (crossed) disparity. Error bars indicate ±1 SE.
measures for disparities at and surrounding the site of adaptation composed a single adapted sensitivity function. Functions were measured for adaptation at four values of disparity for each subject (0, +10, -10, +20 arc min for SBS and LKC, and 0, +10, -10, -20 arc min for CMS). Disparity tuning functions were calculated by taking the log of the ratio of adapted to baseline threshold for each test disparity.

Results

Sample psychometric functions for correlation detection at 20 arc min far are shown in Fig. 4 for one subject before (open symbols) and after (solid symbols) adaptation to a fully correlated surface at the same disparity. The effect of adaptation on correlation sensitivity across the range of disparities used is shown in Fig. 5 for two adaptation conditions for subject SBS. The baseline data (open symbols) are replotted from Fig. 2, the solid symbols show correlation thresholds measured after adaptation to a zero disparity (panel A) and a 10 arc min near (panel B) surface. Error bars represent ±1 SE. In both panels of Fig. 5, threshold is elevated at the disparity of adaptation by about a factor of two, and the range of disparities for which threshold is elevated is relatively narrow. The adaptation curve crosses the baseline curve 5-10 arc min on either side of the adaptation locus and is below baseline for relative disparities greater than 10-20 arc min in both directions. The principal difference between the results presented in panels A and B of Fig. 5 is that adaptation at 10 arc min near produces a somewhat broader and asymmetric threshold elevation.

Disparity tuning functions were obtained from these data by plotting the log of the ratio of adapted to baseline threshold, and the results are shown in Fig. 6 for all three subjects and all values of adapting disparity. The horizontal axis plots disparity in arc min, the vertical axis plots threshold ratio on a log scale. The arrow on the horizontal axis indicates the disparity of adaptation, and the horizontal line at 0 on each vertical axis indicates a ratio of one (no effect of adaptation). Values above the line reflect a loss of sensitivity due to adaptation, and values below it reflect an enhanced sensitivity due to adaptation. In the bottom panel, results are shown for two subjects adapting at 20 arc min far and for one subject adapting at 20 arc min near disparity.

In most cases, the tuning functions obtained show the greatest threshold elevation at the disparity of adaptation. An exception to this general rule occurred for subject LKC (dashed line): the tuning function for adaptation to 10 arc min far disparity showed maximum threshold elevation at 20 arc min. In the same plot, subject CMS (dotted line) shows a smaller shift, with threshold elevation peaking at 15 arc min. In most cases, the tuning functions show facilitation for disparities on either side of the adaptation locus. In some cases, where the data set is sufficiently extensive, there is an indication that performance returns to baseline for more distant disparity values.

The full width at half height of these disparity tuning functions was measured directly from these plots and the results are shown in Fig. 7. The horizontal axis plots the absolute value of the disparity at which threshold elevation is maximum (which was not always the locus of adaptation, as noted above). The vertical axis plots the full width at half height of each tuning function obtained. The best fitting line, its equation and $R^2$ value.
FIGURE 6. Disparity tuning functions for three subjects after adaptation at (A) 0, (B) -10, (C) +10, (D) +20 (two subjects) and -20 (one subject) arc min disparity. The log of threshold ratio (adapt/baseline) is plotted against the disparity of the test surface in each panel. Arrows on disparity axis indicate the disparity of the adapting surface. The solid curve in panel A plots the difference between the two functions shown in Fig. 5(a) for SBS, the solid curve in panel B plots the difference between the two functions shown in Fig. 5(b). In general, the disparity tuning functions show the greatest elevation at the locus of adaptation, with facilitation for more distal disparities. Note, however, the peak at +20 for LKC after adaptation to +10 arc min (dashed curve in panel C).

FIGURE 7. Width of disparity tuning functions plotted against location of peak elevation. Each point represents the full width at half height of a tuning function, measured directly from the curves plotted in Fig. 6. Tuning width is approximately a linear function of peak disparity, with an intercept of about 5 arc min.

DISCUSSION

Disparity range of interocular correlation detection

The results of the baseline and upper disparity limit experiments indicate that, in general, correlation sensitivity follows an exponential relationship to disparity. This is reminiscent of the finding by Blakemore (1970) that relative depth judgments on a disparity pedestal follow an exponential relationship to the magnitude of the pedestal (but see also Badeck & Schor, 1985; Regan & Beverley, 1973). Since the fixation reference was a pair of nonius lines, a disparity value of zero on the horizontal axis of Fig. 2 indicates the nonius horopter and sensitivity to correlation was greatest at or near this point. One might alternatively measure a correlation horopter, i.e. the locus of disparities for which correlation threshold is at a minimum. For our subjects, this appears to be within 2-3 arc min of the nonius horopter.

The humps and dips in the baseline data of Fig. 3 produce some local asymmetries between crossed and uncrossed disparities of the same value: for example, a comparison of 20 Near vs 20 Far for subject LKC in Fig. 3 shows about a 0.2 log unit difference in threshold. Subject CMS shows an asymmetry in the opposite direction over most of the disparity range tested, but the magnitude of the difference varies considerably. Considering all three subjects, these data indicate no overall difference in sensitivity between near and far mechanisms, particularly if the correlation horopter is used as the reference. This localized, mild form of "stereoanomaly" is more consistent with a model of many narrow band disparity channels, some of which may be relatively insensitive, than with the Richards (1971) pooling hypothesis. The effects are not robust enough in these subjects to be conclusive, but it suggests that further study may turn up individuals with pronounced deficits in correlation sensitivity at a particular disparity—a kind of correlation scotoma.

For each subject, the triangular area in Fig. 2 bounded by the baseline sensitivity curve and the horizontal line at 0.0 on the vertical axis (top of Fig. 2) represents those combinations of disparity and correlation values which are distinguishable from 0.0 correlation. Everything outside that area has the appearance of 0.0 correlation: a dynamic swarm of dots filling a volume in depth. Values inside the triangle produce the percept of a stable surface in depth, floating amidst a less dense swarm.

The baseline values of Fig. 2 are specific to the conditions we tested under and should not be generalized beyond them. In particular, correlation threshold at

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zero disparity varies with stimulus area, duration (Tyler & Julesz, 1978) and contrast (Cormack, Stevenson & Schor, 1991b). One would also expect the upper disparity limit to change with these variables, so that the figure of 1 deg might be exceeded under different conditions. In general, we chose values that optimize performance in the tasks so most alterations in the configuration of our stimuli would result in lowered correlation sensitivity.

**Disparity-specific adaptation**

The results of the adaptation experiments indicate that those visual mechanisms which process interocular correlation are sharply tuned for retinal disparity, both at and away from the horopter. While our data set is not sufficiently extensive to make a definitive statement, it appears that a relatively large number of "channels" exist based on the fact that threshold was usually most elevated at the locus of adaptation. Had there been only a few (e.g. 1 or 4) channels mediating correlation detection, adaptation would have produced the same elevation profile for a range of adapted disparities. Our data indicate that there are a large number, if not a continuum of channels, but a precise estimate of how many and where they peak would require an order of magnitude more data than we have presented here.

The finding of facilitation for disparity values distant from the locus of adaptation suggests that adaptation weakened an inhibitory mechanism that affects sensitivity under baseline conditions. One explanation, which has been used to explain facilitory effects in spatial frequency channels (DeValois, 1977), is that channels are mutually inhibitory and that adaptation of a channel releases neighboring channels from inhibition. This assumes that surrounding channels which are not excited by test stimuli are significantly inhibiting those which are, thus implying a relatively high, constant level of activity in all channels. However, in the current context, the spurious matches which occur at random at all disparities in a DRDS stimulus might excite surrounding channels.

A somewhat different explanation holds that each channel has an opponent, center-surround organization due to its inputs from earlier levels of processing rather than from inhibitory interactions at the same level. In this case, adaptation in the surround region produces enhanced sensitivity of the channel more directly, perhaps by adaptation of subunits in the inhibitory surround. This puts the inhibitory mechanism at an earlier stage in visual processing and requires no spontaneous activity in surround channels, but retains the intuitive notion that facilitation comes from a release of inhibition. Both models yield the same opponent-center-surround characteristics and may be equivalent at the computational level despite differences in implementation.

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**Disparity channels model: assumptions**

It is important to note that the tuning functions shown in Fig. 6 are not necessarily plots of disparity-tuned channels, but may be the result of adapting many overlapping channels, each with sensitivity to the disparity of the adapting surface. To understand better the implications of our results, we have developed a model of underlying channel structure which produces similar results and have explored some boundary conditions to limit the possibilities. Our search is by no means exhaustive and our model is by no means definitive, but we have been able to rule out some general schemes that have been proposed elsewhere.

We constructed the model according to the general form of the results presented above, with a number of tuned mechanisms whose sensitivity decreases with disparity and whose tuning width increases with disparity such that the area under the channel profile is constant for all channels. We chose a difference of Gaussians as the response profile of each mechanism, with an excitatory central region and a broader, inhibitory surround that exactly balances the overall function. The response of the visual system to a pulse of interocular correlation was modeled as the vector sum of the individual responses.* The effect of adaptation on an individual mechanism was assumed to be proportional to its sensitivity to the adapting stimulus. Figure 8 shows the channel profiles used in the modeling, after several channels have been desensitized by adaptation to a stimulus at \( \pm 10 \) arc min. One channel has been plotted more darkly to illustrate the form of the profile.

The stimulus is represented as being at a single disparity value in the modeling. In actuality, the energy of the stimulus is distributed across disparity by some combination of optical, neural and oculomotor factors (Stevenson, Cormack & Schor, 1989; 1991). To simplify the model, we consider the channel profiles to be the response across disparity of each channel to the stimulus, without specifying the stimulus profile exactly.

Quantitative agreement between the human data and the modeling results was achieved by adjusting the number of channels and eight constants in relatively simple formulas which determined the location, sensitivity, breadth and inhibitory sidelobe extent of each channel. In the configuration presented here, there are 21 channels, peak sensitivity falls off as the square root of disparity from the horopter, the space constant is inversely proportional to sensitivity, the inhibitory surround has six times the spatial extent of the excitatory center and the peak separation increases exponentially with disparity. The use of formulas to specify these variables served to reduce the number of free parameters considerably, given the large number of channels used.

**Disparity channels model: results**

The results of our simulations are shown in Figs 9, 10 and 11. Figure 9 shows simulated pre-adaptation performance for this set of mechanisms (solid curve), with

*Wiebull fits to our psychometric functions show that Beta is almost exactly 2.0, so a vector sum is equivalent to probability summation across channels.
log threshold plotted vertically against disparity. For comparison, the baseline data from two subjects have been replotted from Fig. 2. The model baseline captures the basic form of the human data, absent the idiosyncratic bumps and dips. These variations can be reproduced in the model by adjustment of individual channel sensitivities. Figure 10 shows the same model baseline data (solid curve) along with the post-adaptation model baseline (dashed curve), obtained after adaptation to a stimulus at +10 arc min. Note that the adapted baseline drops below the unadapted baseline for disparities that are distant from the locus of adaptation. Figure 11 shows the difference between these curves (dotted curve in Fig. 11), plotted in the same way as the tuning functions of Fig. 6, with the log of the threshold ratio plotted vertically against the disparity of the test stimulus. Also plotted in Fig. 11 are model threshold elevation curves for adaptation at 0 and 20 arc min disparity. While the profiles do not match exactly to those found experimentally, they capture four important characteristics: they peak at the disparity of adaptation, are narrowly tuned, show facilitation for surrounding disparities and the off-horopter functions are slightly asymmetric in form. Note that the asymmetry is not a property of the individual channels, but of the channel distribution overall. Note also that the facilitation observed at large disparities after adaptation to zero disparity (solid curve in Fig. 11) is produced by the sidelobes of channels peaking at large disparities not by the sidelobe of the channel peaking at zero disparity. Thus, the disparity tuning profile produced by adaptation to a particular disparity does not accurately reflect the channel profile of a single mechanism which peaks at that disparity.
FIGURE 11. Model tuning functions, obtained by taking the threshold ratio from the model baseline and post adaptation model sensitivity. Log of the threshold ratio is plotted vertically against the disparity of the test pulse. Functions are shown for adaptation at 0 (solid curve), +10 arc min (dotted curve) and +20 arc min (dashed curve). Note the similarity of these functions to the tuning functions plotted in Fig. 6(b) and 6(c).

Rather, it is a reflection of the aggregate activity of many channels.

This configuration of the model we present was chosen so as to capture both the baseline and the tuning behavior of the human data and some parameters are more critical than others. For example, as few as 10 channels could be used to match the overall baseline and tuning characteristics, but then the baseline data showed considerable bumpiness due to gaps between channels. Broadening these 10 channels removed the bumps but then made the tuning functions broader than the human data. Using more than 21 channels does not significantly alter the behavior of the model, so our configuration is close to the minimum needed to get the idealized baselines and tuning functions shown.

The baseline irregularities and the peak shift from the human data (seen for LKC adapting at 10 arc min) suggest that the mechanisms tapped by our experiments depart from these ideals. These were successfully simulated by simply adjusting the sensitivity of individual channels to make localized regions of higher or lower sensitivity, resulting in an idiosyncratic set of channels. Thus the model could be easily tuned to capture the individual differences seen in the data.

The results of our modeling serve to confirm what was implied by our data concerning the narrow tuning of multiple underlying mechanisms subserving correlation and disparity processing. However, much is also revealed by the models we constructed that didn't work. Unfortunately, there is always the possibility that a more creative design might have made these alternatives work. Our conclusions from these negative results are therefore tentative.

That being said, we conclude that narrowly tuned threshold elevation profiles cannot arise from a scheme based solely on broadly tuned underlying mechanisms such as the Near and Far neural mechanisms of Poggio et al. (1988) or the Near and Far pools of Richards (1971). The models which produce the kind of functions we used were successful in simulating the overall baseline and post adaptation model, but then the baseline data showed considerable bumpiness due to gaps between channels. Broadening these 10 channels removed the bumps but then made the tuning functions broader than the human data. Using more than 21 channels does not significantly alter the behavior of the model, so our configuration is close to the minimum needed to get the idealized baselines and tuning functions shown.

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That being said, we conclude that narrowly tuned threshold elevation profiles cannot arise from a scheme based solely on broadly tuned underlying mechanisms such as the Near and Far neural mechanisms of Poggio et al. (1988) or the Near and Far pools of Richards (1971). The models which produce the kind of functions we obtained require narrowly tuned mechanisms off the horopter, more like the Tuned Near and Tuned Far neural mechanisms of Poggio et al. (1988), the continuum of disparity tuned mechanisms proposed by LeVay and Voigt (1988) or the disparity tuned mechanisms proposed by Lehky and Sejnowski (1990). In particular, one cannot account for the fact that tuning functions return to baseline on both sides of their peak without invoking the existence of a tuned mechanism, off the horopter, with a relatively narrow profile. We found that models based on channels with broad sensitivity profiles extending out to large disparity values (Near/Far channels) always produced very broad threshold elevation functions. It is still possible that very broadly tuned channels are operative in processing correlation but their contribution is not apparent in our data due to the overwhelming influence of more narrowly tuned channels.

The magnitude of side-lobe facilitation in Fig. 11 is somewhat less than in the data. It is likely that the data of Fig. 6 could be more faithfully reproduced by varying the profiles of the individual mechanisms. Additionally, as discussed above, it is possible that the opponent center/surround behavior could be captured by using mechanisms without side lobes and then invoking mutual inhibition at a later stage. However it is implemented, we conclude from our simulations that inhibitory interactions between test and surrounding disparities are required to account for the facilitation effects.

Finally, while the model we present has accounted for the data by assuming many channels along a line of sight, it is possible that these data resulted from the aggregate response of many channels distributed across the visual field, with only a small number representing any given visual direction. The data we present show that there are a number of channels available to process correlation over the 9 deg field size we used, but the distribution of these channels within that field is unknown. The luminance spatial frequency tuning of these channels is likewise open to speculation, since the stimulus we used was spatially broad band.

SUMMARY AND CONCLUSIONS

Thresholds for detecting pulses of positive interocular correlation against a background of zero correlation were found to increase logarithmically with linear increases in both crossed and uncrossed retinal disparity. While significant irregularities occurred in the functions for our individual subjects, only one subject showed an overall difference between the two directions of disparity.

Adaptation to 100% correlation at several different disparity levels revealed threshold elevation tuning functions that were relatively narrow and usually peaked at the locus of adaptation. More distant disparities showed a facilitation effect after adaptation.

Efforts to model these results suggest that they are most likely the result of a large number of disparity channels, with relatively narrow tuning, broad inhibitory side lobes and peak sensitivities that are distributed about the horopter.
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