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A Theory of the Visual System Biology Underlying Development of Spatial Frequency Lateralization

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Abstract

The spatial frequency hypothesis contends that performance differences between the hemispheres on various visuospatial tasks are attributable to lateralized processing of the spatial frequency content of visual stimuli. Hellige has proposed that such lateralization could arise during infant development from the earlier maturation of the right hemisphere combined with the increasing sensitivity of the visual system to high spatial frequencies. This proposal is intuitively appealing but lacks an explicit theory with respect to the underlying visual system biology. In this paper, we develop such a theory based on knowledge of visual system processing and development. We then translate our theory into a computational model that serves as the basis for a series of development simulations. We find that the simulations produce spatial frequency lateralization effects consistent with those observed empirically. We relate the nature of the neural asymmetry implied by our theory to empirical findings on visual pathway bias and the relative spatial frequency lateralization effect.

Keywords

spatial frequency; hemispheric asymmetry; lateralization; visual development; computational model

1. Introduction

Numerous lesion studies and psychophysical experiments offer convincing evidence that the right and left hemispheres differ in their performance on various visuospatial tasks (for reviews see Hellige, 1993; Ivry & Robertson, 1998). The spatial frequency hypothesis (Sergent 1982, 1987) contends that many of the performance differences found on seemingly unrelated tasks are attributable to a common underlying cause: the lateralized processing of the spatial frequency content of visual stimuli. It further contends that the initial “sensory” processing of a visual stimulus is symmetric and that the lateralization, characterized as the more efficient processing of the low spatial frequency content of a stimulus by the right hemisphere and the high spatial frequency content by the left hemisphere, arises in the processing that takes place beyond that point. The existence of this hypothesized processing asymmetry is supported by the findings of extensive observational research (for reviews see Christman, 1997; Grabowska & Nowicka, 1996; see also Peyrin, Chauvin, Chokron, & Marende, 2003). In particular, psychophysical experiments that measured reaction time on identification tasks involving

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peripheral presentation of gratings of known spatial frequency to right-handed subjects produced the expected lateralization effects (Christman, Kitterle, & Hellige, 1991; Kitterle, Christman, & Hellige, 1990; Kitterle, Hellige, & Christman, 1992; Kitterle, Christman, & Conesa, 1993; Kitterle & Selig, 1991; Proverbio, Zani, & Avella, 1997). In these experiments, processing involving low frequencies (<2 cycles per visual degree) produced faster reaction times for right hemisphere/left visual field (RH/LVF) presentation, while processing involving high frequencies (>3 cycles per visual degree) produced faster reaction times for left hemisphere/right visual field (LH/RVF) presentation. The magnitude of lateralization observed was typically small – less than ± 0.05 when quantified using the asymmetry coefficient $\sigma = (t_R - t_L)/(t_R + t_L)$ where t_R and t_L are the reaction times for RH/LVF and LH/RVF presentation, respectively (Lezak, 1995) – but generally statistically significant.

An explanation of how such asymmetry in the processing of spatial frequencies could arise has been proposed by Hellige (1993, 1995, 1996, 2006). He hypothesized that the co-occurrence of two conditions in the neonate leads to the development of spatial frequency lateralization: the earlier maturation of the right hemisphere and the increasing sensitivity of the visual system to high spatial frequencies. Earlier maturation of the right hemisphere is supported by both anatomical and imaging evidence (Bracco, Tiezzi, Ginanneschi, Campanella, & Amaducci, 1984; Chi, Dooling, & Gilles, 1977; Chiron et al., 1997; Dooling, Chi, & Gilles, 1983; Scheibel, 1984; Gupta et al., 2005). Increasing sensitivity of the visual system to high spatial frequencies is supported by evidence from psychophysical and electrophysiological studies on infants (Banks & Dannemiller, 1987; Dobson & Teller, 1978; Gwiazda, Bauer, Thorn, & Held, 1997; Kelly, Borchert, & Teller, 1997; Norcia, Tyler, & Hamer, 1990) and is thought to be mainly attributable to retinal maturation (Youdelis & Hendrickson, 1986). Hellige argued that because the sensitivity of the neonatal visual system is limited to the low end of the spatial frequency range, the earlier developing right hemisphere acquires a specialization for processing low frequency input. Furthermore, because of improving visual system sensitivity to high spatial frequencies, the later developing left hemisphere acquires a specialization for the processing of high spatial frequency input. Hellige suggested that the developmental scenario may involve an interaction between the time course of visual development and the timing of “critical periods” of intense synaptic modification in the two hemispheres.

This proposal has intuitive appeal but lacks an explicit theory with respect to the underlying visual system biology. The goal of the work presented here is to develop such a theory based on current findings on visual system processing and development. In particular, we identify a set of visual system components and processes that could hypothetically work in combination with asynchronous hemispheric development to bring about the development of spatial frequency lateralization. We then provide a more definitive interpretation of our theory by specifying it as a computational model. Finally, we show that development simulations based on the model produce spatial frequency lateralization effects consistent with those observed empirically.

The modeling simulations can be viewed as an extension of our earlier work that demonstrated the potential for asynchronous hemispheric development to produce functional lateralization (Howard & Reggia, 2004). In that modeling study, which examined the relationship between the timing of task learning and the state of hemispheric plasticity, we showed that differences in the timing of task learning periods, coupled with differences in the timing of plasticity for the two hemispheres, produced oppositely directed lateralization for two distinct tasks. The timing of the task learning periods was controlled by changing the stimuli presented to the network over time, thus changes external to the system played a critical role in eliciting the lateralized development. In this study, we show that change internal to the system that affects the input to asynchronously developing cortical areas can produce lateralized development in the absence of any change in the external stimuli. Conceivably, this set of circumstances could

arise not only during early visual system development, but also throughout the course of cortical development, contributing significantly to the lateralization of cognitive functions.

2. Theory Description and Rationale

Our theory is built on the finding that each retinotopic location is represented in layer 2/3 of the primary visual cortex (V1) by a set of cells that respond as selective filters in the spatial frequency domain (for in-depth discussion see De Valois & De Valois, 1988). Because the peak frequencies of these filters cover an approximately six-octave range (0.5 – 16.0 cycles per degree) and the mean bandwidth is approximately 1.4 octaves (De Valois, Albrecht, & Thorell, 1982), filters that respond best to low spatial frequencies are distinct from those that respond best to high frequencies. V1 filters form feed-forward connections to V2 cells in the visual system's ventral pathway, which mediates pattern identification and discrimination tasks (Livingstone & Hubel, 1988). Clearly, these feed-forward connections could produce frequency lateralization effects under discussion if connections from low frequency filters were relatively stronger in the right hemisphere, while connections from high frequency filters were relatively stronger in the left hemisphere. Motivated by this prospect, we next consider whether the postnatal increase in sensitivity to high spatial frequencies, in conjunction with the later development of the left hemisphere, can drive the development of this sort of hard-wired asymmetry in connection strengths.

Asymmetry in filter connectivity could emerge from an initially symmetric state if two conditions apply. First, V1 filters compete for connectivity to V2 cells such that connections from filters that contribute most to V2 cell activation strengthen at the expense of connections from filters that contribute least (competitive Hebbian development). Second, high frequency filters compete more effectively for connections to V2 cells during the later left hemisphere development than during the earlier right hemisphere development. The first condition is consistent with findings that indicate activity-driven development of a Hebbian nature occurs in the visual cortex (Kirkwood & Bear, 1994). The second condition could arise if the activity of high frequency filters increases relative to that of low frequency filters as the visual system matures.

The postnatal increase in sensitivity to high frequencies is consistent with a change in relative filter activity, but does not necessarily imply one. In fact, the change in sensitivity can be largely explained by retinal changes (Youdelis & Hendrickson, 1986) that researchers hypothesize have a uniform rather than a differential effect on the filters (Movshon & Kiorpes, 1988; Peterzell & Kelly, 1997; Peterzell & Teller, 1996; Peterzell, Werner, & Kaplan, 1993, 1995; Wilson, 1988). Specifically, they argue that cone migration shifts the sensitivity range of all filters toward higher frequencies while cone-lengthening increases the responsiveness of all filters, increasing visual system sensitivity to high frequencies without a change in relative filter activity. However, we contend that an additional factor contributes to increasing sensitivity to high spatial frequencies that does involve a change in relative filter activity, namely the maturation of the parvocellular visual pathway as explained below.

Both major retino-cortical pathways, the parvocellular and the magnocellular, contribute substantially to the spatial frequency filters of V1 layer 2/3 (Nealy & Maunsell, 1994). The relative pathway contributions to filter response as a function of filter peak frequency are unknown. However, the magnocellular pathway is more sensitive to low spatial and high temporal frequencies, while the parvocellular pathway is more sensitive to medium-high spatial frequencies and low temporal frequencies (Merigan, Byrne, & Maunsell, 1991; Merigan, Katz, & Maunsell, 1991). These sensitivities suggest that for the temporally-broadband stimuli typically used in lateralization investigations, the relative contribution of the magnocellular pathway is greatest for low frequency filters and decreases as peak frequency increases, while

the reverse holds for the parvocellular pathway. If so, then low frequency filters should be more affected by changes in the magnocellular pathway and high frequency filters by changes in the parvocellular pathway.

Evidence from psychophysical and electrophysiological studies suggests that, while both pathways mature during the first eight postnatal months, they do so at different times and rates (Burkhalter, 1993; Dobkins, Anderson, & Lia, 1999; Fiorentini, Pirchio, & Spinelli, 1980; Hammarrenger et al., 2003; Hartmann & Banks, 1992; LeVay Wiesel, & Hubel, 1980; Swanson & Birch, 1990; for review see Fiorentini, 1992). Specifically, the parvocellular pathway appears to mature later and more gradually than the magnocellular pathway. A physiological investigation by Hawken, Blakemore, and Morley (1997) found changes in the responsiveness of the two pathways that may account for the observed maturational differences. In particular, they found that the response rate (spikes per second) of magnocellular pathway cells in the thalamus of the macaque increases dramatically during the first two months after birth and then continues to increase slowly through the eighth month, while the response rate of parvocellular pathway cells increases steadily over the first eight months. Given the relative pathway contributions hypothesized above, this change in pathway responsiveness implies that the activity of high frequency filters should increase relative to that of low frequency filters between the second and the eighth postnatal months, satisfying the second condition for lateralized development mentioned earlier.

Two potential problems arise in the activity-driven development scenario posited so far that can be resolved by the inclusion of additional biological factors. First, the frequency-dependent differences in filter bandwidth and the non-uniform distribution of filters representing a retinotopic location (DeValois et al., 1982) result in frequency-dependent differences in overall V2 activation levels that could lead to a substantial loss in the frequency selectivity of V2 cells as connection strengths are modified. Second, the higher activity levels of the filters at the time of left hemisphere development could produce left lateralization of V2 activation for all frequencies. We resolve the first issue by assuming that V1-to-V2 connectivity modifications are based on the steady-state response of the V2 cell and that this response is subject to gain control via lateral inhibition generated by surrounding cells (Heeger, 1992). We resolve the second issue by assuming that homeostatic plasticity effectively normalizes total filter input to V2 as filter activity increases over time (Turrigiano & Nelson, 2004).

3. Materials and Methods

3.1 Model Design

The design of our computational model incorporates the underlying biology delineated above. The model's architecture, summarized schematically in Figure 1, is implemented (using Matlab) as a two-layer, feed-forward neural network with separate representations of the right and left hemispheres. The two layers, which are identical for the two hemispheres, comprise a layer of primary visual cortex (V1) filter units and a layer of secondary visual cortex (V2) cells, both associated with initial ventral stream processing. The V1 filter units are assumed to be cells that differ in response to a stimulus based strictly on its spatial frequency, therefore, model input consists purely of spatial frequency values given in cycles per second (cpd). Model output is defined as the total V2 activation produced in response to an input. Within each model hemisphere, V1 cells are fully connected to V2 cells via modifiable weighted connections. The retinotopic locations represented by the V1 cells in the two hemispheres correspond to mirror locations in peripheral visual field regions stimulated in psychophysical experiments investigating visual lateralization. These regions lack corpus callosum connections in areas V1 and V2, such that processing is maximally restricted to one hemisphere.

The V1 layer consists of 40 filter units drawn from a set of basic filters (F). The response (at full maturity) of filter $j \in F$ to input frequency ω given by the function

$$r_j(\omega) = [A_j \sigma_1 \exp[-(\pi \sigma_1 \omega)^2] - B_j \sigma_2 \exp[-(\pi \sigma_2 \omega)^2] + C_j \sigma_3 \exp[-(\pi \sigma_3 \omega)^2]]_+ \quad (1)$$

where $[\cdot]_+$ indicates the rectification operator defined as $[z]_+ = \max(z, 0)$, and $A, B, C, \sigma_1, \sigma_2$ and σ_3 determine the filter's shape, bandwidth, peak frequency and maximum response. This function is simply the Fourier transform of a difference-of-Gaussians function that can be used to describe a spatial filter's receptive field (in one spatial dimension) consisting of an excitatory center, an inhibitory surround and, in the case of smaller filters, a secondary excitatory region (Phillips & Wilson, 1984; Wilson, Levi, Maffei, Rovamo, & De Valois, 1990). The basic filter set (F), shown in Figure 2, consists of six filters with peak frequencies of 0.5, 1, 2, 4, 8 and 16 cpd that uniformly cover the range of peak frequencies, filter bandwidths that approximate mean bandwidths observed empirically in the macaque at these peak frequencies (De Valois et al., 1982), and equal peak frequency response (i.e., 1.0). The allocation of these filters across the 40 filter units of V1 roughly approximates the distribution of peak frequencies found in macaque parafoveal V1 cells (De Valois et al., 1982). The parameter values specifying these six filters together with the number of V1 units allocated to each are given in the Appendix.

The V2 layer in each hemisphere consists of 1000 cells fully connected to the filters in the V1 layer via weighted connections. The connection weights are initialized to random values between 0 and 1, then normalized such that the sum of the weights to a V2 cell from the V1 filters is 1. Identical initial weights are used for corresponding V1 filter-to-V2 cell connections in the right and left hemispheres so that the hemispheres start out in a symmetric state.

3.2 Simulation Design

During a simulation, the hemispheres undergo periods of development that are equal in length but different in their timing, with later left hemisphere development assumed in all cases. The timing of development in hemisphere $H \in \{R, L\}$ is determined by the value of t_H which represents the beginning (postnatal month) of a hemisphere's period of high plasticity. The length of the development period, t_{length} , is set to 6 months for both hemispheres. Hemispheric plasticity takes the form of a simple learning rate function of time t given by $\eta^H(t) = 0.6$ for $t_H < t < t_H + t_{\text{length}}$ and $\eta^H(t) = 0$ otherwise. Each simulation proceeds from the start of development in the right hemisphere (t_R) to the end of development in the left hemisphere ($t_L + t_{\text{length}}$) in equal time steps (epochs) of .005 months. At each epoch, the input set is randomly reordered and then presented to both hemispheres concurrently, eliminating the possibility of asymmetry arising from hemispheric differences in input sequences. In order to maximize activity-driven development over the course of a simulation, the six filter peak frequencies (0.5, 1, 2, 4, 8 and 16 cpd) are used as the input set. Weight changes that potentially allow the hemispheres to develop asymmetrically are made after the presentation of each input.

3.3 Simulation Dynamics

The activation of a V1 filter unit allocated to filter $j \in F$ in response to input of spatial frequency ω at time t , is computed as the product of the filter's relative maturity μ_j at time t and its response function at full maturity (Equation 1), that is,

$$a_j(t, \omega) = \mu_j(t) r_j(\omega) \quad (2)$$

The effect of the relative filter maturity factor, defined below, is the normalization of total V1 input over time as the overall V1 activity level rises due to visual pathway maturation.

Relative filter maturity, μ_j , for filter $j \in F$ at time t is defined as the weighted sum of the maturity (m) of the magnocellular (M) and parvocellular (P) pathways at time t , normalized across all filters as given by

$$\mu_j(t) = (c_j^M m^M(t) + c_j^P m^P(t)) / \sum_{\ell} (c_{\ell}^M m^M(t) + c_{\ell}^P m^P(t)) \quad (3)$$

where the weights c_x^M and c_x^P represent the relative contributions of the two pathways to the activity of filter $x \in F$ at full maturity as defined below. Although empirical evidence indicates that a filter's response is not the simple sum of the responses elicited under separate pathway activation, it does suggest that it is greater than or equal to the response elicited by single pathway activation (Allison, Melzer, Ding, Bonds, & Casagrande, 2000; Nealy & Maunsell, 1994) – an attribute captured by the simple summation used in the model.

The relative contributions of the parvocellular and magnocellular pathways to the activity of filter $x \in F$ at full maturity are defined as linear functions of filter index x , designed to be consistent with the assumption that the contribution of the parvocellular pathway increases, while that of the magnocellular pathway decreases, with increasing filter peak frequency (Merigan et al., 1991a,b). Specifically,

$$c_x^P = 1.0 - (c_{\max}^P - c_{\min}^P) * (6 - x) / 5 \quad (4)$$

$$c_x^M = 1 - c_x^P \quad (5)$$

where $c_{\max}^P$ and $c_{\min}^P$ are the maximum and minimum relative contributions of the parvocellular pathway, respectively. The values $c_{\max}^P = 1.0$ and $c_{\min}^P = 0.33$, used in the simulations, effectively set the total parvocellular contribution to layer V2 to roughly twice the magnocellular contribution consistent with the dominance of the parvocellular contribution to the ventral pathway, and provide the constraint $0 \leq c_x^X \leq 1$, $X \in \{M, P\}$.

Maturity levels for the magnocellular and parvocellular pathways (m^M and m^P in Eq. 3) are estimated as indices ranging from 0 and 1 (adult-like maturity) given by

$$m^M(t) = .5 + \frac{1}{\sqrt{\pi}} \int_0^{.55(t-2)} e^{-x^2} dx, \quad 0 \leq t \leq 8 \quad (6)$$

$$m^P(t) = .5 + \frac{1}{\sqrt{\pi}} \int_0^{.4(t-4)} e^{-x^2} dx, \quad 0 \leq t \leq 8 \quad (7)$$

Indices are set to 1 for $t > 8$. As evident in Figure 3, the indices capture the differences in pathway maturation suggested by empirical data (Fiorentini, 1992; Hawken et al., 1997).

The activation of cell k in layer V2 of hemisphere H is given by the weighted sum of its V1 filter input less an activation threshold (0.03) chosen to maximize the frequency sensitivity of V2 cells while ensuring the each cell responds to at least one member of the stimulus set. Specifically,

$$a_k^H = \sum_j^F w_{kj}^H a_j - 0.03 \quad (8)$$

where a_j is the activation of filter $j \in F$ and w_{kj}^H is the sum of the weights to unit k from the N_j units allocated to filter j in hemisphere H , that is, $w_{kj}^H = \sum_i^{N_j} w_{kji}^H$.

Weight adjustments are based on a Hebbian learning rule with the adjusted weight total to V2 cell k from all units allocated to filter j in hemisphere H given by

$$w_{kj}^H = w_{kj}^H + \eta^H a_j a_k^H / \sum_{\ell}^{V2^H} a_{\ell}^H \quad (9)$$

where η^H is the current learning rate for hemisphere H , a_j is the activity of filter $j \in F$ in response to the input, a_k^H is the activity of V2 cell k in hemisphere H , and $\sum_{\ell}^{V2^H} a_{\ell}^H$ is the sum of activity for all V2 cells of hemisphere H . This learning rule implies the existence of lateral connections between V2 cells that normalize the steady-state response of a V2 cell based on the overall response of the cells in the surrounding region (Dayan & Abbott, 2001). The rule implements a weak competition for filter connections among V2 cells whereby the changes in weights on connections to a particular cell depend on its activity relative to the total activity in the region. The weight change is followed by weight normalization such that

$$w_{kj}^H = w_{kj}^H / \sum_j^F w_{kj}^H \quad (10)$$

The weight normalization gives rise to competition among the filters converging on a V2 cell resulting in a strengthening of connections from filters with relatively higher activity and a weakening of connections from filters with relatively lower activity.

3.4 Metrics

Three metrics are used in the analysis of asymmetry that develops over the course of a simulation. The first measures the strength of the projection from filters of a particular peak frequency to the V2 cells of a hemisphere. This metric, defined as the total of weights from V1 units allocated to filter $j \in F$ to the V2 cells in hemisphere H , is given by

$$W_j^H = \sum_{\ell}^{V2^H} w_{\ell j}^H \quad (11)$$

The second metric measures the activation that is produced in a hemisphere in response to a particular stimulus frequency. This metric, defined as the total activation of the V2 cells in hemisphere H in response to input $i \in I$, is given by

$$A_i^H = \sum_{\ell}^{V2^H} a_{\ell}^H(\omega_i) \quad (12)$$

where $a_{\ell}^H(\omega_i)$ is the activation of V2 cell ℓ in hemisphere H in response to input frequency ω_i . The third metric measures the total strength of the visual pathway projections to V2 in each

hemisphere. This metric, defined as the contribution of pathway $X \in \{M, P\}$ to the V1-to-V2 connection weights in hemisphere H is given by

$$W^{XH} = \sum_j^F c_j^X W_j^H \quad (13)$$

where W_j^H is given by Equation 11. The degree of asymmetry that develops for each of these metrics is then computed as a coefficient of asymmetry (Lezak, 1995) given by

$$\rho_Y = (Y^R - Y^L) / (Y^R + Y^L) \quad (14)$$

where Y^R and Y^L are the values of metric $Y \in \{W, A, X\}$ for the right and left hemispheres, respectively. The value of ρ_Y can range from -1 to 1 with negative values indicating left lateralization and positive values indicating right lateralization.

4. Results

This section reports the results of several model simulations in which we examined the asymmetry that developed under a variety of hemispheric development timing conditions, as measured by the coefficients of asymmetry for the metrics described above. For each timing condition, we performed twenty simulations that differed only in the initial random values assigned to the V1-to-V2 weights. At the start of a simulation, the hemispheres were set to a symmetric state (via symmetric initial weights) such that the coefficients of asymmetry for all metrics were equal to zero. At the conclusion of a simulation, the coefficients of asymmetry for the metrics were recomputed with non-zero values signifying the occurrence of lateralized development. Results from the twenty simulations were averaged to obtain mean values for the post-development coefficients of asymmetry. These are shown, together with the 95% confidence limits (error bars), in the figures that follow.

4.1 The Basic Simulation

For the basic simulation, we used timing conditions consistent with empirical observations of a period of intense synaptogenesis occurring in the visual cortex between 2 and 8 months (Huttenlocher, de Courten, Garey, & Van der Loos, 1982) and a gap between right and left hemisphere development of 1 to 2 weeks (Chi et al., 1977; Dooling et al., 1983). Specifically, for the right hemisphere, the period of intense development was set to start at postnatal month 2 and end at month 8, while for the left hemisphere, it was set to start a half-month later at month 2.5 and end at month 8.5. We found that, under these conditions, low frequency filters develop relatively stronger connections to V2 in the right hemisphere, while high frequency filters develop relatively stronger connections to V2 in the left hemisphere, as measured by ρ_W (Figure 4). This asymmetry in connection strength produces greater V2 activation in the right hemisphere in response to low spatial frequency input and greater V2 activation in the left hemisphere in response to high frequency input, as measured by ρ_A (Figure 5). The weight asymmetry and activation lateralization effects for the low frequencies of .5 and 1 cpd and the high frequencies of 4, 8 and 16 cpd are small ($< \pm 0.03$) but statistically significant, as evidenced by the 95% confidence limits on the mean values for ρ_W and ρ_A . A linear regression analysis of asymmetry as a function of $\log(\text{frequency})$ indicates that the negative slope characteristic of low/right-high/left asymmetry is highly significant in the case of both filter-to-V2 weights [$F(1,5) = 78.35, p < .0009$] and V2 activation [$F(1,4) = 649.6, p < .0001$].

The typical course of development for filter weight asymmetry (Figure 6) and V2 activation lateralization (Figure 7) shows that the earlier start of development in the right hemisphere causes an initial jump, particularly for low frequencies, that tends to diminish as development

proceeds in both hemispheres. Similarly, the later termination of development in the left hemisphere causes the more pronounced changes, generally in the direction of reduced asymmetry, that appear at the end of hemispheric development in both asymmetry functions. This pattern suggests that co-termination of development in the two hemispheres would produce greater lateralization than the later termination of left hemisphere development for most spatial frequencies. It should be noted that the time course of the general diminution of lateralization that occurs during development depends on the change in plasticity as a function of time that is assumed in the model. If plasticity is actually more concentrated in time, then the decrease in asymmetry will be abridged correspondingly.

Our results also indicate that hemispheric biases with respect to the visual pathways are present in the V1-to-V2 connection weights (Figure 8). However, they are small in comparison to the weight asymmetries associated with the low and high frequency filters (Figure 4). This is understandable considering that both the magnocellular and the parvocellular pathways contribute to both low and high frequency filters, albeit unequally. Thus, although differences in the maturation of the two visual pathways drive the asymmetric development, the resulting asymmetry exhibits more of a hemispheric bias toward the outputs of the low and the high frequency filters than toward the outputs of a particular visual pathway.

As a check on the validity of the model's basic design, we examined the post-development mean distribution of peak frequencies for V2 cells by determining which of the six input frequencies produced the greatest response in each of the 2000 (combined right and left hemisphere) V2 cells. We found that, although no V2 cell exhibited peak response at the lowest input frequency (0.5 cpd), the V2 distribution was, on average, shifted lower in comparison with the peak frequency distribution assumed for V1 filters (Figure 9). This shift is agreement with findings (in the macaque) of a mean peak frequency of 1.4 cpd for V2 cells (Levitt, Kiper, & Movshon, 1994) and 3.5 cpd for V1 cells (De Valois et al., 1982). It is also consistent with the evidence of a shift in peak frequency sensitivity to lower frequencies at the V1/V2 border obtained in fMRI imaging experiments (Sasaki et al., 2001).

4.2 Variation in the Timing of Hemispheric Development Relative to Pathway Maturation

We next examined the effect of variation in the timing of hemispheric development relative to visual pathway maturation on the development of lateralization in our model. Four sets of simulations were performed with the starts of right hemisphere development given by $t_R = 1.5, 3.5, 5.5$ and 7.5 months. As in the previous simulation, the difference in the timing of development between the hemispheres was 0.5 months ($t_L = t_R + 0.5$). The results show a consistent pattern of right lateralization for low frequencies and left lateralization for high frequencies for all timing conditions (Figure 10). However, the lateralization effects tend to decline as the timing of hemispheric development moves later relative to pathway maturation until they are virtually extinguished for development that starts at 7.5 months. An analysis of covariance based on the mean coefficient of asymmetry values for the four timing conditions found the reduction in V2 activation lateralization as a function of the timing of hemispheric development to be significant [$F(3,19) = 7.14, p < .0052$].

4.3 Variation in the Difference in the Timing of Development for the Two Hemispheres

Finally, we examined the effect of variation in the magnitude of the difference in the timing of development between the two hemispheres. We performed three simulations with differences in the timing of development between the hemispheres of .25, .5, and 1 month. All three simulations had a common midpoint of development – 5 months – determined by setting t_R to 1.875, 1.75, and 1.5 and t_L to 2.125, 2.25, and 2.5, respectively ($t_{\text{length}} = 6.0$). As shown in Figure 11, lateralization of V2 activation increased as the offset between right and left hemisphere development increased. An analysis of covariance based on the mean coefficient

of asymmetry values for the three timing conditions found the increase to be highly significant [$F(2,14) = 99.64, p < 10^{-7}$].

5.0 Discussion

Our theory of the development of spatial frequency lateralization, which we propose as a biological specification of Hellige's (1993) hypothesis on this topic, contends that certain visual system components and processes work in conjunction with asynchronous hemispheric development to bring about the development of spatial frequency lateralization. The key visual system elements include the increasing contribution of the parvocellular (relative to the magnocellular) pathway to V1 filter response as filter peak frequency increases, the later and more gradual development of parvocellular (relative to magnocellular) pathway responsiveness, and the modification of the connections from V1 filters to V2 cells via a competitive Hebbian process during postnatal development. The second of these three elements contributes to the increasing sensitivity of the visual system to higher spatial frequencies during the first several postnatal months, proposed as a causal factor of lateralized development in the Hellige (1993) hypothesis. The visual system elements incorporated in our simulation model represent the necessary conditions for the development of spatial frequency lateralization in the context of our theory. Consequently, research that supports or refutes the occurrence of these conditions addresses our theory's validity as well. Consistent with Hellige's hypothesis, our theory assumes that right hemisphere development occurs earlier than left hemisphere development in most humans. This assumption implies that, in the absence of other lateralizing factors, reversed spatial frequency lateralization would develop if and only if this pattern of asynchrony was reversed in an individual.

Our simulation results support the potential validity of our theory, including the completeness of its biological assumptions. In the simulations, which assumed the earlier development of the right hemisphere as prescribed by our theory, right lateralization of low frequency processing and left lateralization of high frequency processing developed under a variety of hemispheric development timing conditions. The results were consistent with lateralization effects observed in psychophysical experiments (as noted in the Introduction) in terms of magnitude as well as the direction, that is, the effects were small but statistically significant. In simulations that examined the effect of the overall timing of hemispheric development, we found that development concurrent with rapid changes in relative pathway maturities produced greater lateralization than development concurrent with slow (or no) change. In simulations that examined the effect of the difference in the timing of right and left hemisphere development, we found that greater differences in the state of relative visual pathway maturity at the time of right versus left hemisphere development produced greater lateralization. Thus, consistent with the implications of our theory, our simulation results showed that the magnitude of lateralization increased with either an increasing difference in the timing of hemispheric development or the earlier timing of hemispheric development within the period of visual pathway maturation. The sensitivity of the results to either sort of variation in the timing of hemispheric development, especially in comparison with sensitivity to variations in initial V1–V2 connectivity, suggests that individual differences in the timing of hemispheric development could account for intersubject variability in spatial frequency lateralization.

The finding that variation in the timing of hemispheric development relative to pathway maturation can affect the magnitude of the lateralization that arises from hemispheric development asynchrony implies that spatial frequency lateralization and other functional lateralization effects that also arise from asynchronous hemispheric development in the neonate may appear to be only weakly correlated. One possible source of such variation is the length of the gestational period. For example, visual cortex development may be triggered or stimulated by postnatal experience while visual pathway maturation is unaffected. Although

the state of most nervous system development is thought to depend on time since conception, activity-driven cortical development may be subject to environmental influences, and, indeed, visual cortex development correlated with time since birth has been observed in an EEG study of the alpha rhythm in infants (Stroganova, Orekhova, & Posikera, 1999). This suggests that an empirical finding of a relationship between gestational period and spatial frequency lateralization would provide support for our theory.

The simulation results also support the existence of a right hemisphere–magnocellular pathway bias and a left hemisphere–parvocellular pathway bias. However, the results suggest that pathway-based asymmetry is very small in comparison to the filter-based asymmetry. This implies that for tasks that depend primarily on the processing of low or high frequency information, hemispheric differences in task performance may be evident, while hemispheric differences in the change in task performance resulting from manipulations affecting a particular visual pathway, may be too small to detect. Such a situation could account for the results obtained in the Roth and Hellige (1998) study that investigated the impact of visual pathway manipulations on the performance of two oppositely-lateralized visuospatial tasks. That study found performance on the right-lateralized task to be more affected by manipulation of the magnocellular pathway, while performance on the left-lateralized task was more affected by manipulation of the parvocellular pathway. However, the study also found that the impact of the pathway manipulations on task performance for the two hemispheres did not differ significantly, consistent with an absence of hemispheric asymmetry specific to the visual pathways. With respect to spatial frequency identification and discrimination experiments, our theory predicts that the use of gratings that differentially affect the performance of the magnocellular and parvocellular systems will produce very similar changes in task performance in the two hemispheres. This prediction distinguishes our theory of spatial frequency lateralization from those that hypothesize asymmetric contributions of either the magnocellular and parvocellular systems or the dorsal (magnocellular-dominated) and ventral (parvocellular-dominated) pathways as the primary underlying cause.

A potentially controversial aspect of our theory is its implication that hard-wired asymmetry in the feedforward connections of the perceptual system underlies spatial frequency lateralization. The hypothesized asymmetry clearly seems adequate to explain lateralization effects based on absolute spatial frequency. However, it is not so obvious that it can account for the relative spatial frequency lateralization effect observed in the Christman et al. (1991) study. In that study, subjects distinguished between two compound grating stimuli, one consisting of a base pair of sine-wave components and the other of three sine-wave components including the base pair. Two experiments were conducted, the first using low spatial frequencies (.5 and 1 cpd) and the second using high spatial frequencies (4 and 8 cpd) for the base pair, with an identical third component (2cpd) in both experiments. The results showed that identification of the low frequency base pair was faster for LVF/RH presentation while identification of the high frequency base pair was faster for RVF/LH presentation, consistent with lateralization based on absolute frequency. The results also showed that adding the third component slowed identification for both experimental conditions, but with a lateralized impact. Specifically, the addition of the 2 cpd component produced a shift in performance in favor of the right hemisphere when its frequency was low relative to the base pair components, but in favor of the left hemisphere when its frequency was high relative to the base pair components. Thus, the direction of lateralization for the 2 cpd component appears to change as a function of its relative placement in the stimulus frequency range rather than being fixed as would be expected if it were determined solely by hard-wired asymmetry.

In seeking to explain both absolute and relative spatial frequency lateralization effects, researchers have hypothesized the involvement of mechanisms that go beyond a hard-wired asymmetry affecting feedforward processing. Ivry and Robertson (1998) have proposed that

attentional processes effectively shift the lateralization crossover point based on the range of frequencies contained in the stimulus frequency (the Double Filtering by Frequency theory). Christman (1997) has suggested that lateralized frequency interference effects, that is, greater low frequency interference with high frequency processing in the right hemisphere and greater high frequency interference with low frequency processing in the left hemisphere, may change the lateralization of mid-spectrum frequencies as a function of stimulus frequency range.

Although the hard-wired asymmetry in feedforward connections implied by our theory is compatible with and readily integrated into these hypotheses, we claim that it alone is sufficient to explain both relative and absolute frequency lateralization effects. Our argument is as follows. An input of a particular frequency activates not only filters of the corresponding peak frequency but also, to some degree, filters with peak frequencies falling an octave above and below it (see Figure 2). Consequently, both a low frequency stimulus composed of 0.5 and 1 cpd sinusoidal components and a high frequency stimulus composed of 4 and 8 cpd sinusoidal components produce some activation in 2 cpd filters. If a 2 cpd component is added to either the low or the high frequency stimulus it will increase the activation of the 2 cpd filters but, perhaps more saliently, will newly activate either 1 cpd filters or 4 cpd filters depending on the base stimulus. As a result, the most effective information on which to base the identification of the three-component stimulus may be provided by 1 cpd filters in the high frequency condition and 4 cpd filters in the low frequency condition. If indeed this information is used in the task, then the hard-wired right-lateralization of 1 cpd processing and left-lateralization of 4 cpd processing could produce an impact on performance that is oppositely lateralized for the low and high frequency conditions, that is, the relative frequency lateralization effect.

While we believe that the proposed asymmetry in bottom-up, perceptual system connections is sufficient to account for frequency lateralization effects, it is also possible that asymmetries in lateral connections and in top-down, attentional system connections that are not incorporated in our model could contribute as well. With respect to the origin of such asymmetries, we speculate that established asymmetry in V1-to-V2 connections could provide the basis for further asymmetric development in later developing connections that could include intra-areal long-range connections and both interareal feed-forward and feed-back connections. In fact, it seems quite conceivable that small asymmetries arising early in the visual processing stream, coupled with continued asynchronous development of homologous visual areas (Thatcher, 1992; Thatcher, Walker, & Giudice, 1987) could result in functionality that grows more divergent with increasing level in the visual hierarchy. A particularly interesting possibility is that frequency-based, hard-wired asymmetry in V1-V2 connections influences the establishment of intercolumnar connections in a way that substantially enhances global-level processing in the right hemisphere and local-level processing in the left hemisphere, by creating, in effect, hemispheric asymmetry in receptive field size. In turn, asymmetric receptive fields might interact with the attentional system to produce observed lateralization in the allocation of attention to either the global (right hemisphere) or the local (left hemisphere) level (Fink et al., 1996; Robertson, Lamb, & Knight, 1988; Weissman & Woldorff, 2005; Yamaguchi, Yamagata, & Kobayashi, 2000). While outside the scope of our current study, both the development of asymmetries in cortical connections beyond those between the V1 filters and V2 cells on the ventral pathway and the interplay of perceptual and attentional systems in producing spatial frequency lateralization are important areas to address in future extensions of this research.

The considerations in this paper naturally raise several questions: First, what factors might cause the right hemisphere to develop earlier than the left? Second, are there other potential sources of lateralized development? Lastly, what purpose does lateralized development serve? Although there is no definitive answer to the first question, a recent study suggests that the disparity in the timing of hemispheric development could be linked to the earlier expression

of a particular gene in the right hemisphere of human embryonic brains (Sun et al., 2006). With regard to the second question, there are a number of relevant hypotheses about the sources of asymmetric development and their relationship to various functional lateralizations (for review see Hellige 1993,2006). For example, fetal positioning in-utero could play a role in right-handedness, and fetal cranio-facial asymmetries in speech lateralization (Previc, 1991). As for the question of purpose, several hypotheses relate lateralization to increased reproductive fitness during evolution due to associated benefits such as the avoidance of interference between complementary processes (Hellige, 2006) and the minimization of energy/connectivity costs (Grushin & Reggia, 2005; Shkuro & Reggia, 2003).

Finally, considering our theory as it might relate to the evolution of hemispheric specialization in humans (for review see Hellige, 2006), we note that the functionality mediated by the parvocellular system appears to have evolved more recently than that mediated by the magnocellular system as it is found only in the primates (Shapley & Perry, 1986). If ontogeny recapitulates phylogeny in human development, then a possible hypothesis suggested by our work is that the right hemisphere develops a specialization for cognitive functions of a more ancient origin and the left a specialization for functions of more modern origin. An examination of this hypothesis in light of current knowledge about human nervous system phylogeny and ontogeny could lead to new theories on the sources of lateralized functional development.

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Figure 1.

Model architecture. Input, in the form of a value representing the spatial frequency of a stimulus presented at mirror locations in the right (RVF) of left (LVF) visual fields, is passed to filters in V1 of the left and right hemispheres, respectively. Within each hemisphere, filter response feeds forward to V2 cells via modifiable weighted connections.

Figure 2.
Response functions for the six filters in filter set F .

Figure 3. Maturity indices for the magnocellular and parvocellular pathways, as given by $m^M(t)$ and $m^P(t)$, respectively, for postnatal months 0 to 8.

Figure 4. Post-development asymmetry in the total weight of the connections to V2 cells from filters of specified peak frequencies, computed as the coefficient of asymmetry ρ_W . Depicted are mean values together with the (very small) 95% confidence limits (error bars) based on 20 simulations, with positive (negative) values indicating rightward (leftward) asymmetry.

Figure 5.

Post-development lateralization in V2 activation in response to inputs of the specified spatial frequencies, computed as the coefficient of asymmetry ρ_A . Lateralization is not shown for input frequency 16 cpd as it produces little or no activation. Depicted are mean values for the simulation set together with the 95% confidence limits (error bars), with positive (negative) values indicating rightward (leftward) asymmetry.

Figure 6. The development of asymmetry in filter-to-V2 connection weights, as measured by the coefficient of asymmetry, ρ_W , for filters designated by peak frequency as shown in legend.

Figure 7.
The development of functional lateralization as measured by the coefficient of asymmetry, ρ_A , computed for the input frequencies shown in legend.

Figure 8. Hemispheric bias toward output of the magnocellular or the parvocellular pathway computed as ρ_M and ρ_P , respectively.

Figure 9.
The distribution of peak frequencies assumed for V1 filters and found post-development for V2 cells (combined mean values for right and left hemispheres).

Figure 10.

The effect on V2 activation lateralization, ρ_A , of varying the overall timing of hemispheric development while keeping the timing of visual pathway maturation constant.

Figure 11.

The effect of varying the difference in the timing of hemispheric development on V2 activation lateralization, ρ_A .

Table 1

Filter Parameters

filter index j	V1 units N_j	peak (cpd)	bandwidth (octaves)	A	B	C	σ_f	σ_2	σ_3
1	2	0.5	2.17	4.4440	0.3330	0.0	0.3330	1.0000	0.0
2	6	1.0	1.83	8.8570	0.5417	0.111	0.1950	0.5080	0.7200
3	12	2.0	1.63	18.050	0.7217	0.222	0.1075	0.2590	0.3570
4	12	4.0	1.49	37.120	0.8940	0.333	0.0590	0.1320	0.1770
5	6	8.0	1.34	88.766	1.1055	0.426	0.0344	0.0637	0.0844
6	2	16.0	1.19	241.70	1.2800	0.480	0.0200	0.0312	0.0412

Parameters specifying the 4 cpd filter are taken from Wilson & Gelb, 1984.