Neuronal Asymmetries in Primary Visual Cortex of Dyslexic and Nondyslexic Brains

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Dyslexic brains exhibit histologic changes in the magnocellular (magno) cells of the lateral geniculate nucleus, and consistent with these changes, dyslexics demonstrate abnormal visually evoked potentials and brain activation to magnocellular stimuli. The current study was aimed at determining whether these findings were associated with changes in the primary visual cortex with the prediction that magnocellular components of this cortex would be affected. We measured cross-sectional neural areas in primary visual cortex (area 17) in dyslexic and nondyslexic autopsy specimens. There was a significant interaction between hemispheres and diagnostic category; ie, nondyslexic brains had larger neurons in the left hemisphere, whereas dyslexic brains had no asymmetry. On the other hand, cell layers associated with magnocellular input from the lateral geniculate nucleus did not show consistent changes in dyslexic brains. Thus, there is a neuronal size asymmetry in favor of the left primary visual cortex in nondyslexics that is absent in dyslexic brains. This is yet another example of anomalous expression of cerebral asymmetry in dyslexia similar to that of the planum temporale, which in our view reflects abnormality in circuits involved in reading.


Developmental dyslexia may be defined as the relatively selective impairment of reading despite normal intelligence, sensory acuity, motivation, and instruction. In the past 20 years much of the research on this subject has focused on language function. In recent years, however, there has been growing interest in exploring more fundamental sensory processing problems, including auditory and visual perception, which in turn may contribute to the well-documented linguistic deficits.1–6

Psychophysical studies have suggested that dyslexics process transient visual stimuli with low-contrast and high-repetition rates (ie, the domain of the magnocellular pathway) more slowly than nondyslexics. The pathway for processing sustained, high-contrast, slow stimuli, the parvocellular pathway, may be spared.2,7–13 An anatomical and visual evoked potential (VEP) study provided additional evidence that dyslexics showed a specific defect affecting the magnocellular pathway. Livingstone and colleagues3 found that, whereas the VEPs of dyslexics and nondyslexics were similar for high-contrast conditions, there was a delay in the early wave (from 0 to N100) in the dyslexic response to low-contrast stimuli. This delay is thought to implicate abnormal activity early in the magnocellular visual pathway from the retina up to visual area 1 (V1); also area 17 of Brodmann). Consistent with these results, quantitative analysis has shown that the neurons in the magnocellular layers of the lateral geniculate nucleus (LGN) in dyslexics were, on average, 27% smaller than those of the nondyslexics, but there were no measurable differences in the neurons of the parvocellular layers.

Additional physiological studies have explored the nature of the visual deficit in dyslexics. Several of these studies14–17 have measured VEPs using stimuli that are preferentially processed by the magnocellular and parvocellular pathways. Although the specific stimuli differ from that of Livingstone and colleagues,3 both Lehmkuhle and associates14 and Kubova and co-workers17 saw a delay in the early components of the VEP in dyslexic individuals. Victor and collaborators15 and Johannes and colleagues,16 on the other hand, did not measure any differences in the VEPs of dyslexics to stimuli processed by either the magnocellular or the parvocellular pathway. Victor and collaborators,15 however, did report an increase in the response variability in subjects with attention-deficit hyperactivity disorder.

Other studies have supported these findings. For instance, dyslexics had slower flicker fusion rates for stimuli that are specific for magnocellular function, but were not abnormal on tests of parvocellular function.18

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In a similar manner, Lehmkuhle and associates\textsuperscript{14} reported magnocellular dysfunction in dyslexics, although Victor and colleagues\textsuperscript{15} and Cornelissen and co-workers\textsuperscript{19} failed to do so. Similar psychophysical studies looking at metacontast masking,\textsuperscript{20,26} contrast sensitivity,\textsuperscript{19} and flicker sensitivity\textsuperscript{21} have reported specific delays and differences in processing “magnocellular stimuli” by dyslexics. In contrast, other studies do not support the magnocellular deficit hypothesis.\textsuperscript{22,23} These studies fail to show a difference in the dyslexic’s ability to process magnocellular stimuli. Besides studies looking at low-level visual processing, several studies have expanded the search for visual processing differences at the cortical level. By using psychophysical tasks, Cornelissen and associates\textsuperscript{19} found that dyslexics had a decreased ability to detect coherent motion, whereas Felmingham and Jakobson\textsuperscript{21} reported that dyslexics had more difficulty with completing tasks designed to test the function of the dorsal visual stream. Although these two studies only provide circumstantial evidence that the visual deficit extends to the cortical level, a functional magnetic resonance imaging study has provided more concrete evidence.\textsuperscript{24} Using a visual motion stimulus, Eden and collaborators\textsuperscript{24} measured the activation of the visual association area MT/V5 in both dyslexic and nondyslexic men, and found that the same stimulus, which produced robust activation of MT/V5 in nondyslexics, failed to activate this area in dyslexics. Demb and colleagues\textsuperscript{25} also showed reduced activation in MT in addition to primary visual cortex and several extrastriate areas in dyslexics compared with controls. By using whole-scalp neuromagnetic recordings, Vanni and associates\textsuperscript{26} failed to show any differences in the activation of MT to moving stimuli but did find slightly longer latencies within the dyslexic population. These recent studies provide preliminary evidence that the visual deficits seen in dyslexics may be the result of differences in the processing of visual stimuli at both the cortical and subcortical levels.

The present study was designed to follow up on the LGN findings in dyslexic brains by determining whether dyslexics’ neurons differed in size in the primary visual cortex (area 17), compared with nondyslexics, and if so, whether they affect magnocellular-linked layers differently from parvocellular-linked layers. Layer IV of area 17 receives input from the LGN. Neurons in layer IV\textsubscript{CB} receive direct input from the parvocellular layers, whereas neurons in layer IV\textsubscript{Ca} receive projections from the magnocellular LGN layers.\textsuperscript{27,28} We hypothesized in dyslexics that neurons receiving inputs from magnocellular LGN layers would show size differences, whereas those receiving projections from the parvocellular LGN layers would not.

Materials and Methods

We examined area 17 in autopsy specimens from 5 dyslexic subjects (4 men and 1 woman; mean age, 34.8 ± 13.6 years) and 5 nondyslexics (4 men and 1 woman; mean age, 39.4 ± 9.2 years). The dyslexics all had a clear diagnosis in life, and the brains had been used in previous anatomical studies of the LGN and medial geniculate nucleus (MGN).\textsuperscript{2,3,29} The nondyslexic subjects had sufficient educational histories to permit exclusion of the diagnosis of developmental dyslexia. Information about handedness was available on all dyslexic subjects and 4 of the 5 nondyslexic subjects, with 1 known left-handed subject in each group.

We used the method of Yakovlev\textsuperscript{30} for processing whole brains in serial histological sections. Brains were sectioned coronally at 35 μm, and every 20th section was stained for Nissl substance with cresyl violet. A section halfway between the caudal end of the splenium of the corpus callosum and the occipital pole was identified in each hemisphere, and a section rostral (five sections away) and one caudal (five sections away) to it were stained. Therefore, there were two stained sections from area 17 in each hemisphere in each specimen. The sections were coded so that the examiner was blind to both the diagnosis and hemisphere of the section during measuring. Within each section the boundaries of area 17 were marked based on the well-known tripartite appearance of layer IV under low-power light microscopy. Within area 17, fields selected for measurement were 77 μm wide and extended perpendicularly from the pial surface across all cortical layers. The demarcation of the layers was made based on the density and morphology of the neurons. These measuring boxes were oriented so that they would fall only on straight portions of the visual cortex, thus avoiding crests of gyri and bottoms of sulci. Under 1,250× magnification the outlines of all neuronal perikarya containing a nucleolus were traced using a camera lucida. The outlined cell areas were then measured by using a Macintosh Plus computer (Apple Computer, Cupertino, CA) coupled with a Zeiss MOP-3 electronic planimeter (Carl Zeiss, Inc, Thornwood, CA). Analyses were performed by using analysis of variance.

Results

At low magnification, area 17 showed no obvious qualitative differences in cytoarchitectonic appearance between the dyslexic and the nondyslexic cortex. As expected, neuronal sizes showed significant laminar differences (F\textsubscript{6,68} = 93.0, p < 0.0001), both in the dyslexic and nondyslexic specimens. Mean cross-sectional neuronal areas combined over all layers of bilateral visual cortex showed no significant difference between dyslexics and nondyslexics (Tables 1 and 2). However, when the means were computed for the hemispheres separately there was a significant hemisphere-by-diagnosis interaction for all cortical layers combined (F\textsubscript{1,8} = 5.70, p < 0.05); Nondyslexics had larger neurons in the left hemisphere (left = 80.48 ± 3.17 μm\textsuperscript{2}, right = 70.44 ± 2.32 μm\textsuperscript{2}), whereas dyslexics had neurons of similar size in both the right and
the left hemisphere (left = 77.42 ± 2.86 μm², right = 79.77 ± 2.89 μm²; Fig 1).

To ascertain whether this hemisphere × diagnosis interaction was confined to specific layers of the cortex, each cortical layer was analyzed independently. This analysis showed that the hemisphere × diagnosis interaction was significant in layers II/III (F_{1,8} = 6.2, p < 0.05; dyslexic left = 82.28 μm², right = 87.76 μm²; nondyslexic left = 84.04 μm², right = 75.29 μm²), IV (F_{1,8} = 8.2, p < 0.05; dyslexic left =
Table 2. Mean Cell Area (μm²) and Number of Neurons in Dyslexic and Nondyslexic Brains in Layer IV of the Visual Cortex

<table>
<thead>
<tr>
<th>Subject</th>
<th>Layer</th>
<th>Mean</th>
<th>No.</th>
<th>Mean</th>
<th>No.</th>
</tr>
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<tr>
<td>ORT-01</td>
<td>IVCB</td>
<td>75.97 ± 3.72</td>
<td>90</td>
<td>64.00 ± 1.82</td>
<td>142</td>
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<td></td>
<td>IVCα</td>
<td>70.55 ± 2.39</td>
<td>107</td>
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<td></td>
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<td>57.83 ± 1.20</td>
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<tr>
<td>ORT-02</td>
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<td>86</td>
<td>73.47 ± 2.53</td>
<td>74</td>
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<tr>
<td></td>
<td>IVCB</td>
<td>58.6 ± 1.74</td>
<td>121</td>
<td>62.63 ± 1.89</td>
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<td></td>
<td>IVCα</td>
<td>54.51 ± 1.05</td>
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<td>116</td>
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<tr>
<td></td>
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<td>120</td>
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<tr>
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<td>55.09 ± 1.28</td>
<td>170</td>
</tr>
<tr>
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<td>135</td>
<td>73.23 ± 1.91</td>
<td>129</td>
</tr>
<tr>
<td></td>
<td>IVCα</td>
<td>57.58 ± 1.36</td>
<td>168</td>
<td>73.90 ± 1.72</td>
<td>137</td>
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<td>Nondyslexics</td>
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<tr>
<td>ORT-07</td>
<td>IVB</td>
<td>78.11 ± 3.11</td>
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<td>71.09 ± 4.10</td>
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<tr>
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<td>125</td>
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<td>199</td>
<td>52.06 ± 1.04</td>
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<tr>
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<td>60.00 ± 1.51</td>
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<tr>
<td></td>
<td>IVCB</td>
<td>57.05 ± 1.53</td>
<td>134</td>
<td>54.63 ± 1.09</td>
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<tr>
<td></td>
<td>IVCα</td>
<td>54.09 ± 1.24</td>
<td>151</td>
<td>54.26 ± 1.88</td>
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<td>IVB</td>
<td>58.15 ± 1.68</td>
<td>164</td>
<td>55.30 ± 1.89</td>
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<td>53.38 ± 1.10</td>
<td>200</td>
<td>47.39 ± 1.05</td>
<td>159</td>
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</table>

57.56 μm², right = 60.96 μm²; nondyslexic left = 62.45 μm², right = 54.59 μm²), and V (F1,8 = 7.32, p < 0.05; dyslexic left = 86.74 μm², right = 90.06 μm²; nondyslexic left = 94.51 μm², right = 78.69 μm²), but not for layer VI. The layer IV analysis was further extended to sublayers IVB, IVCα, and IVCβ. The results showed that the interaction held for IVCβ (F1,8 = 12.13, p < 0.01; dyslexic left = 54.53 μm², right = 60.05 μm²; nondyslexic left = 59.72 μm², right = 53.01 μm²; Figs 2 and 3). The above interaction effect shows that the dyslexics and nondyslexics differ with respect to each other in cross-sectional neuronal area between the two hemispheres. Further analyses were performed to determine whether there were significant asymmetries within both populations. Whereas nondyslexic brains showed significant interhemispheric differences in neuronal sizes over all layers combined (left larger than right; F1,24 = 8.381, p < 0.05), there were no such differences in the dyslexic brains, either over all layers combined or layer by layer. Interhemispheric differences in nondyslexics were significant in layers IV and V (F1,4 = 11.523, p < 0.05; F1,4 = 8.598, p < 0.05, respectively; see Fig 2). Further analysis for hemispheric asymmetries in sublayers IVB, IVCα, and IVCβ in nondyslexic and dyslexic brains showed significant asymmetries in all sublayers in the nondyslexics (F1,4 = 11.119, F1,4 = 12.737, F1,4 = 9.185; p < 0.05, respectively); but not in the dyslexics (see Fig 3).

To establish whether the lack of asymmetry in the dyslexics was caused by changes in the left or the right hemisphere, direct comparisons of the hemispheres were made across diagnosis. Neuronal sizes in the left hemisphere of the dyslexics were not significantly different from those of nondyslexics (F1,8 = 0.339, NS), nor were there significant differences between the right hemispheres (F1,8 = 3.73, NS) despite a sizable mean difference (dyslexic = 79.8 μm², nondyslexic = 70.4 μm²).

A mean asymmetry coefficient was calculated for cell sizes for each individual brain by using the following formula: Right − Left/[0.5(Right + Left)]. This coefficient was then used in an analysis of variance to compare the dyslexic and nondyslexic groups for extent of
asymmetry. As predicted from the mean cell area comparisons, nondyslexics were significantly more biased to the left hemisphere than dyslexics ($F_{1,8} = 5.6, p < 0.05$) over all cortical layers combined. Layer-by-layer comparisons of the coefficients of asymmetry revealed that nondyslexics were biased to the left hemisphere in layer IV ($F_{1,8} = 10.2, p < 0.05$) and V ($F_{1,8} = 8.5, p < 0.05$) and within layer IV, they were asymmetric in IVcα ($F_{1,8} = 14.9, p < 0.005$). They were not asymmetric in layers II/III and VI.

**Discussion**

Anatomical studies have revealed that the magnocellular layers of the LGN project to layer IVcα of the primary visual cortex whereas the parvocellular layers project to layer IVcβ. Assuming a developmental interaction between magnocellular neurons in the LGN and those of the primary visual cortex, we postulated that neurons in cortical layer IVcα would be smaller too, perhaps as a result of diminished input from these thalamic neurons. In fact, we did not find this to be the case. The fair degree of blending of the two visual pathways in the cortex, including the primary visual cortex, may explain the lack of specific effects in the magnocellular thalamic projection layers. Furthermore, the primary visual cortex is under the effect of projection from areas both upstream (the thalamus) and downstream (temporal, parietal, preoccipital, and frontal cortical areas). The further blending of magnocellular and parvocellular systems downstream from the primary visual cortex could obviate the developmental influences from the thalamus. The cross-sectional area of neurons in area 17 did differ between the dyslexics and nondyslexics. Hemispheric asymmetries in the size of neurons over all layers combined, in layers IV and V and in sublayers IVB, IVcα, and IVcβ were seen in nondyslexic brains, but not dyslexic brains. A lack of asymmetry has been noted in previous studies of dyslexics. The symmetry of the size of neurons in area 17 is yet another example of symmetry in cortical regions in the dyslexic population.

Although anatomical evidence for laterality in the visual system has not been shown before, many have suggested that there exists hemispheric specialization for some complex visual perceptual tasks. For instance, in a review, Christman described a left hemisphere bias for high spatial frequencies in contrast to a right bias for low spatial frequencies. Others have suggested that the visuospatial capacity of the left hemisphere is

![Fig 1. Mean neuronal cell area differs in nondysexic and dyslexic autopsy brains in area 17. The histograms depict the interaction between diagnosis and hemisphere, revealing an asymmetry in nondyslexics but not in dyslexics ($p < 0.05$).](image)

![Fig 2. These histograms show the asymmetry in the nondysexic and the lack of asymmetry in dyslexic for each of the cortical layers ($p < 0.05$).](image)
The asymmetry of neuronal size in the visual cortex, or whether these neuronal differences impact the reading difficulties, or perhaps some as yet unknown interactive effect between the neuronal size asymmetry and reading.

The absence of propagation of size effects from the LGN to the primary visual cortex in dyslexics is at first puzzling. We have already suggested that this may be in part the result of blending of both pathways in visual cortical areas, including area 17, or developmental or functional effects acting top-down from downstream connected cortical areas. The top-down hypothesis may be cited to explain the findings regarding asymmetry and lack thereof. We know that other cortical areas are more symmetric in dyslexic brains, and symmetry in these other areas of the cortex may propagate top-down to the neurons of the primary visual cortex. On the other hand, there is no reason to exclude the possibility that downstream areas are symmetric because of bottom-up developmental effects from the primary cortex. Furthermore, rather than LGN neurons causing changes in area 17 neurons, the opposite may be true. As stated above, we have shown that symmetry of cortical areas is associated with increased numbers and distribution of interhemispheric callosal projections. Because there is a normally occurring competition for cortical targets between thalamic neurons and transcallosal neurons, it is possible that with increased symmetry, as seen in the dyslexic brain, and greater numbers of transcallosal neurons, thalamic neurons are deprived of targets resulting in smaller neurons. In all of these cases, it appears likely that the visual system of dyslexics is built or functions differently by virtue of the reported anomalies.

The cause of symmetric development of cortical areas in dyslexic brains is not known. Genetic factors may play a role, as they appear to do for other manifestations of brain laterality. However, we have also shown that the induction of abnormal neuronal migration leading to minor cortical malformations in rats is associated with anomalies of anatomical brain lateralization and callosal and thalamic connectivity. In addition, studies of the New Zealand Black mouse, which spontaneously develops ectopias that resemble those seen in dyslexics, have revealed ectopias as early as embryonic day 14 to 15, suggesting that they occur in the middle of the neurons’ migrational period. Tracer studies have shown that these ectopias make anomalous connections to both thalamic nuclei and cortical regions. It is possible, therefore, that the first developmental event in dyslexia is the appearance of such cortical anomalies, which alter the development of cortical asymmetry and of cell sizes in connectionally related cortical and subcortical regions via anomalous connections.

Lack of asymmetry in the cross-sectional neuronal areas of the primary visual cortex of dyslexic brains is...
interesting in and of itself, and so is the presence of such asymmetries in nondyslexic brains. Psychophysical and neuropsychological experiments can be used to test whether these anatomical asymmetries may underlie sensory-perception ability and whether the abnormal lateralization in dyslexics may result in abnormal visual perception.

Finally, although this report concentrates on findings in the visual system of dyslexics, related work from our laboratory has shown that the auditory system, too, is anomalous in this population, and that similar fast systems may be preferentially affected. By focusing on the visual system of dyslexics, we do not mean to imply that the anomalies reported here and elsewhere in this system are causally related to the reading problem. It is well documented that dyslexics fail in metalinguistic (metaphonological) tasks that require them to break words down into their component phonemes and other similar tasks. This clearly indicates some problem between language and other cognitive systems, implicating particularly auditory language. We anticipate that neuronal analysis in the primary auditory cortex will also show anomalies in dyslexic brains.

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