Normal planum temporale asymmetry in dyslexics with a magnocellular pathway deficit

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Introduction

Developmental dyslexia is characterized by unexpectedly poor reading ability relative to IQ that cannot be explained by a lack of motivation, inadequate learning opportunity, or an acquired brain lesion. Two neurological abnormalities may help to explain this poor reading ability: abnormal hemispheric symmetry of the planum temporale, and a deficit in the magnocellular visual pathway. The exact relationship between these abnormalities remains unclear. However, confirming a relationship between these two abnormalities could help to better define a specific subtype of dyslexia.

The planum temporale (PT), an auditory association area of the temporal lobe, sits behind primary auditory cortex (Heschl’s gyrus) and is involved in early auditory processing [1]. The PT is larger in the left hemisphere in about 75% of human brains, and this asymmetry is present at birth [2–5]. The leftward asymmetry of the PT, observable in gross anatomical specimens, is correlated with leftward asymmetry of the auditory cytoarchitectonic area Tpt [6] and may be related to language lateralization [6,7]. However, several studies have shown symmetry or reversed asymmetry of the PT in the majority (~90%) of dyslexic brains [8–11].

The magnocellular (M) pathway is one of the major visual pathways between the retina and visual cortex and is primarily involved in processing lower spatial and higher temporal frequencies and visual motion [12]. There are several lines of evidence for an M pathway deficit in dyslexia. First, cell bodies in the M layers of the lateral geniculate nucleus (LGN) of the thalamus are abnormally small in dyslexia [13]. Second, visual performance is impaired when M pathway stimuli (lower spatial and higher temporal frequencies, low mean luminance) are presented [14–16]. Third, electrophysiological responses are reduced or delayed to M pathway stimuli [13]. Fourth, functional magnetic resonance imaging (fMRI) responses are reduced to M pathway stimuli [16,17].

Two studies examined both the laterality of the PT and the integrity of the M pathway within the same dyslexic subjects. The first study examined the brains of five dyslexic patients, post-mortem, and reported abnormally small M layer LGN cells [13]; all of these brains had symmetrical PT [8,9,13]. Livingstone et al. hypothesized that an underlying deficit in the heavily myelinated, rapid processing subdivisions of multiple sensory pathways could link these two abnormalities. However, a second study reported the opposite finding. Six dyslexics showed reduced M pathway-related brain activity and impaired M pathway-related psychophysical
performance [16]; these subjects showed normal leftward asymmetry of the PT [18]. The contradictory findings of these studies make it unclear whether the PT abnormality is associated with the M pathway deficit in dyslexia or is an independent neurological marker. The conflicting results may be explained by inconsistencies in the way the planum was measured. For example, in post-mortem studies such as the first study, it is traditional to measure the area of the planum that can be observed on the surface of the temporal lobe and exclude sulcal tissue. However, in MRI studies such as the second study, it is traditional to include sulcal tissue in the planum measurement. These differences in measurement technique may account for the discrepant findings.

To examine the relationship between these abnormalities, we measured PT asymmetry in dyslexics with a documented magnocellular deficit and controls [15,17]. We used three methods to measure the size of the planum temporale in sagittal MRI sections. The first was a method that includes all sulcal tissue. The other two were similar to methods used in the previous studies and differed to the extent that they included sulcal tissue.

Materials and Methods

Subjects: Five dyslexic subjects (two females) were solicited from the Stanford Disabilities Resource Center. All were Stanford students (mean age (± s.d.) 22.2 ± 2.9 years) and were assumed to be of above average intelligence. All had a childhood history of dyslexia, were diagnosed with dyslexia as adults, and received special services from the university (e.g. extra time on examinations). Five control subjects (two females) were solicited from the Stanford population (mean age 26.8 ± 6.1 years). None had a history of reading difficulty. Dyslexic subjects scored significantly lower than controls on measures of spelling, nonword pronunciation, reading speed and comprehension [15]. All subjects were right-handed, except one control who was left-handed. Two of the dyslexic subjects (1 female) were co-diagnosed with attention deficit disorder. None of the other subjects had a neurological or psychiatric illness that would interfere with the study. Subjects were paid or volunteered without pay, all subjects gave informed written consent, and all procedures were approved by the Internal Review Board at Stanford University.

All subjects had participated in previous fMRI and psychophysical studies that measured the integrity of the M pathway. The dyslexic subjects showed reduced brain activity, relative to the controls, in several visual cortical areas including areas V1 and MT+ in conditions designed to evoke relatively strong activity in the M pathway (low mean luminance, moving gratings) [17]. However, the dyslexic subjects showed normal activity in conditions designed to evoke strong activity in multiple pathways (high mean luminance, contrast-reversing gratings). Using the same M pathway stimulus as in the fMRI experiment, the same dyslexic subjects had higher than normal psychophysical thresholds (i.e. worse performance) on a speed discrimination task [15]. This paradigm was modeled after a study that found speed discrimination deficits in monkeys with an M pathway lesion [12]. Across all 10 subjects, these fMRI and psychophysical measures of M pathway function were highly correlated (r = 0.79) [17].

Neuroimaging: MRI scans were performed using a 1.5 T General Electric Signa scanner. A high-resolution three-dimensional volume anatomy was performed using a standard SPGR sequence and a specially designed head coil (TR = 33 ms, TE = minimum full, flip angle = 40°, in-plane resolution = 0.94 × 0.94 mm). One hundred and twenty-four gapless 1 mm sagittal sections were acquired in each subject. The imaging protocol did not extend to the hemisphere edge in most cases. Using T2-weighted pseudo-coronal images collected as part of a companion study [17], hemisphere widths were measured as the distance between the midline and the edge of each hemisphere. Across all subjects, the mean widths of the two hemispheres were similar (left: 61.3 ± 2.6 mm; right: 62.4 ± 1.7 mm). After determining the midline slice and hemisphere widths, sagittal slice locations were normalized between 0 and 1, where 0 is the midline and 1 is the lateral edge of the hemisphere. The PT typically began around the standardized coordinate 0.50, and we measured the length in alternate slices beginning at the onset and ending at coordinate 0.85. Beyond this point, the PT length often became difficult to assess due to the disappearance of one of the landmarks (e.g. Heschl’s sulcus) or due to image artifacts near the very edge of the brain caused by the head coil. We measured this subregion of the PT and compared the area in the two hemispheres. Leonard et al. noted that measuring an even smaller portion of the PT (equivalent to 0.69–0.81 in our coordinate system) correlated well with overall area but could be measured more reliably because it avoids the often unreliable measurement of lateral slices [19]. We also measured PT asymmetry in this more restricted region (0.69–0.81), because it may better emphasize the hemispheric asymmetry in lateral slices.

The PT anatomical definition and the first measurement followed directly from methods described...
by Steinmetz and colleagues [20,21]. Specifically, we measured the length between the depth of Heschl’s sulcus (anterior border) and the terminal upswing of the posterior ascending ramus (PAR; posterior border). Heschl’s sulcus was identified at its onset in medial slices and followed out laterally. The PAR was identified in lateral slices and could be followed mediially. Figure 1 shows sections at a similar location in both hemispheres from two dyslexic subjects (D1 and D2). The first measurement followed the gray matter between the straight black arrow (depth of Heschl’s sulcus) and the curved black arrow (upswing of PAR). Two raters (MB and JD) measured the length of the PT on alternate sagittal slices by identifying points along the cortical surface extending into small sulci and including the posterior descending ramus (PDR) when present (controls PDR: 4/5 left, 3/5 right; dyslexics PDR: 4/5 left, 2/5 right). The total length of each slice was the total distance between consecutive points. Inter-rater reliability, with one rater (MB) blind to subject identity, was $r = 0.95$.

The second measurement method used the same boundaries as the first method. However, instead of including extensions into sulci, the raters identified the two end points of the PT (depth of Heschl’s sulcus and terminal upswing of PAR) and measured a straight line between them (Fig. 1, straight line between the two black arrows). This method is similar to that used by Rumsey et al. [18] and does not take into account the shape of the PT or small sulci on the surface. Inter-rater reliability was $r = 0.93$.

The third measurement was similar to measurements reported in post-mortem studies by Galaburda and colleagues but adapted for MRI sagittal sections [8,9]. The two raters identified the anterior border of the PT that would be observed from above if the frontal lobes were removed (Fig. 1, white arrow). A straight line was measured between this point and the posterior border used in the second method (Fig. 1, straight line between white arrow and curved black arrow). This measurement excludes gray matter extending into Heschl’s sulcus, small sulci on the surface of the temporal lobe, and the PDR. Inter-rater reliability for this method, after correcting three initial measurements from one rater, was $r = 0.93$.

To determine interhemispheric asymmetry, a laterality index (LI) was computed for each subject and for all methods. First, we performed linear interpolation between the length measurements of alternate slices starting at the onset and ending at 0.85 in standardized coordinates in each hemisphere. Next, we summed the area of the planum for each hemisphere (L and R) by adding the lengths at every 0.01 coordinate step. Finally, we used the formula $LI = (L - R)/(0.5[L + R])$ to measure hemispheric asymmetry. Positive LIs indicate leftward asymmetry and negative LIs indicate rightward asymmetry. In previous studies, an LI of between $-0.05$ and $0.05$ has been used to indicate symmetry (e.g. [9]).

**Results**

Using Method 1, similar to Steinmetz and colleagues [20,21], all five dyslexic subjects showed leftward asymmetry of the PT. Figure 1 shows the planum...
measurement as a function of distance from the midline for two dyslexic subjects (Method 1 panels). The LI for dyslexic subjects ranged from 0.11 to 0.47 (mean LI 0.26 ± 0.14; see Table 1). Three of five control subjects showed leftward PT asymmetry, and two controls (both females) showed rightward asymmetry (mean LI 0.17 ± 0.29). These results from the control group are consistent with 75% leftward lateralization in non-dyslexic subjects previously demonstrated using the Steinmetz method on a large sample [20,21]. It is also consistent with the finding that females are less likely than males to show leftward asymmetry of the PT [4].

Using Method 2, similar to Rumsey and colleagues [18], three of five dyslexic subjects showed leftward asymmetry of the PT, one showed symmetrical PT, and one showed rightward asymmetry (mean LI 0.17 ± 0.26). Planum measurements as a function of distance from the midline for two dyslexic subjects are shown in Fig. 1 (Method 2 panels). The control group showed almost identical results (mean LI 0.14 ± 0.31; Table 1).

Using Method 3, similar to Galaburda and colleagues [8,9], three of five subjects in both groups again showed leftward PT asymmetry (mean LI: dyslexic 0.10 ± 0.31; control 0.10 ± 0.30). The planum measurement as a function of distance from the midline for two dyslexic subjects is shown in Fig. 1 (Method 3 panels).

In most cases, the LI was similar across the three methods. However, in one dyslexic subject (D2) there was a large difference in the LI across techniques. Figure 1 shows sections from the left (LH) and right (RH) hemispheres for this subject. The black arrows show the beginning and end of the PT in slices at a standardized coordinate of about 0.79 in each hemisphere. The left PT was slightly larger using Method 1 (following the cortical surface between the black arrows). However, much of the PT cortex dips into Heschl’s sulcus in the left hemisphere. Thus, based on Method 3, the right hemisphere measurement was larger. This was the case in several lateral slices, and so the LI switched from leftward to rightward in this subject. This was not the case for three dyslexic subjects who showed leftward asymmetry by all three techniques. For example, two slices for a second dyslexic subject (D1) are shown in Fig. 1 (at standardized coordinate 0.73). The left hemisphere has a larger PT length than the right by all methods.

We calculated the LIs for all subjects using a more restricted lateral range of the PT (0.69–0.81 in normalized coordinates) that better emphasizes the asymmetry that largely occurs in lateral slices (see Fig. 1), and is directly comparable to a previous study [19]. In this more restricted range, both groups were more left-lateralized (Methods 1–3), and both groups again became less left-lateralized using Methods 2 and 3 that exclude sulcal tissue (Table 1).

We calculated the probability of our results under the hypothesis that the majority of dyslexics (90%) have abnormal PT asymmetry (symmetrical or rightward) [8–11]. Under this hypothesis, the probability that three of the subjects would show left lateralization (Methods 2 and 3) is very small (P = 0.01). The probability that all five would show left lateralization (Method 1) is even smaller (P < 0.0001).

We directly compared the LIs from the first and third methods that yielded results that were most different (see Table 1). The correlation of the LIs across all subjects was high (r = 0.83, n = 10), and became much higher after removing subject D2 who showed an unusually large switch in the LI across the techniques (r = 0.95, n = 9). Eight of ten subjects were more right-lateralized (lower LI scores) with the third method, consistent with the change in the mean values. Thus, the measurements were

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correlated across methods, even though the average laterality changed across methods.

Discussion

We found normal, leftward asymmetry of the planum temporale in a group of dyslexic subjects with a documented magnocellular pathway deficit [15,17]. Using a conventional method for analyzing MRI sections [20,21], all five subjects showed leftward asymmetry. Using methods similar to previous studies on dyslexia, three or four of five subjects showed leftward asymmetry depending on the method and the range of the PT measured.

The reason that measurements in some subjects switched laterality can be explained by the differences between the measurement techniques. Method 1 includes cortical tissue in Heschl’s sulcus, small sulci on the planum surface, and tissue in the posterior descending ramus. Method 2 also includes Heschl’s sulcus but not the other small sulci. Method 3 excludes all sulcal tissue. Therefore, subjects with a deep Heschl’s sulcus or several small sulci on the PT surface will show varying degrees of laterality depending on the measurement technique. The relative validity of these three techniques will require further investigation. However, at present we see no reason to exclude sulcal tissue from the planum measurement.

One series of studies reported that all five cases showed both abnormally small M layer LGN cells and abnormally symmetrical PT [8,9,13]. This suggests a possible link between an M pathway deficit and abnormal PT symmetry in dyslexia. However, normal leftward PT asymmetry has been reported in recent studies of dyslexia in cases where the integrity of the M pathway was not investigated [18,19], and in cases where it was shown to be impaired [16,18]. Our results provide further support that abnormal PT symmetry may be a marker of a subtype of dyslexia that is independent from the M pathway deficit.

PT measurements that excluded sulcal tissue showed less leftward laterality in both subject groups. This indicates that the measurement technique can influence PT laterality, but that it does not affect the subject groups differently. Therefore, the different measurement techniques used in previous studies cannot explain the different relationships described between the PT and the M pathway [13,18].

It is possible that a different subtype of dyslexia is specifically related to the planum abnormality. For example, it could be that dyslexic subjects with phonological deficits have abnormal PT symmetry. However, subjects in the current study were impaired on a test of phonological processing (non-word pronunciation) as were subjects in two previous studies that also found normal PT asymmetry [18,19]. Alternatively, it could be that specifically those dyslexic subjects without an M deficit have abnormal PT symmetry. Some research estimates that only about three quarters of dyslexic subjects exhibit the M pathway deficit [22]. If this were so, studies of PT asymmetry that used large samples but did not explicitly test visual function should have found significantly lower rates of leftward asymmetry (e.g. 50–60%) given that about one-quarter of their sample would not have the visual deficit. However, recent studies reported normal or slightly exaggerated rates of leftward asymmetry [18,19]. Finally, it could be that dyslexic subjects with accompanying developmental language delay have abnormal PT symmetry. However, there has been only mixed support for this relationship [23–25]. Thus, future research is required to delineate the relationship between subtypes of dyslexia and a planum temporale abnormality.

Conclusion

We measured the planum temporale in dyslexics with a documented magnocellular pathway deficit to examine the relationship between abnormally symmetrical plana and a deficit in the magnocellular pathway. Sagittal brain images were acquired using magnetic resonance imaging (MRI). The planum temporale was measured using three methods that varied in the extent that they included sulcal tissue. The first two methods were similar to previous MRI studies, and the third was similar to previous post-mortem studies. All five dyslexic subjects showed normal, leftward asymmetry of the planum by the first method that included all sulcal tissue. Depending on the region of the planum measured, three or four of the five subjects showed leftward asymmetry using the second or third methods. Within subjects, differences in laterality can arise from the exclusion of tissue in Heschl’s sulcus and small sulci on the planum surface by the third method. Some previous studies suggested a possible relationship between abnormal planum symmetry and a magnocellular pathway deficit in dyslexia, but others have not found this to be the case. Our results support the hypothesis that these two neurological markers for the disorder are independent.

References

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