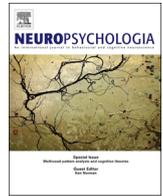




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Planum temporale morphology in children with developmental dyslexia



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ARTICLE INFO

Article history:

Received 18 September 2012

Received in revised form

23 April 2013

Accepted 7 May 2013

Available online 22 May 2013

Keywords:

Reading disability

Reading

Phonological processing

Auditory processing

Laterality

Neurobiology

Planum temporale

MRI

ABSTRACT

The planum temporale is a highly lateralized cortical region, located within Wernicke's area, which is thought to be involved in auditory processing, phonological processing, and language. Research has linked abnormal morphology of the planum temporale to developmental dyslexia, although results have varied in large part due to methodological inconsistencies in the literature. This study examined the asymmetry of the planum temporale in 29 children who met criteria for dyslexia and 26 children whose reading was unimpaired. Leftward asymmetry of the planum temporale was found in the total sample and this leftward asymmetry was significantly reduced in children with dyslexia. This reduced leftward asymmetry in children with dyslexia was due to a planum temporale that is larger in the right hemisphere. This study lends support to the idea that planum temporale asymmetry is altered in children with developmental dyslexia.

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1. Introduction

Developmental dyslexia, also referred to as a learning disability in reading or simply as reading disability, is defined as a dysfunction of reading that cannot be explained by intelligence, sensory impairment, or environment (American Psychiatric Association, 2000). This disorder is estimated to affect 3–6% of school-age children (Kibby & Hynd, 2001), and is characterized by particular cognitive deficits in addition to a deficiency in reading achievement. These cognitive deficits include phonological processing that is considered by many researchers to be the “core” deficit in developmental dyslexia (Blau et al., 2010; Lombardino, Riccio, Hynd, & Pinheiro, 1997; Ramus, 2003; Shaywitz & Shaywitz, 1999; Siegel, 1993). In fact, phonological processing skills in kindergarten are the most predictive factor of word reading achievement in elementary school, with correlations ranging from 0.4 to 0.6 (Torgesen, Wagner, & Rashotte, 1994). Some researchers, however, have theorized that phonological processing deficits alone are not sufficient to explain

dyslexia (Wolf & Bowers, 1999). Research has supported the presence of other cognitive deficits in individuals diagnosed with dyslexia, including difficulties in rapid naming, receptive and expressive language (Purvis & Tannock, 1997), verbal memory (Shaywitz et al., 1995), temporal processing (Klein, 2002), attention (Mayes, Calhoun, & Crowell, 2000), and orthographic processing (Eden, VanMeter, Rumsey, & Zeffiro, 1996).

1.1. The neurobiological basis of dyslexia

For more than a century, scientists have focused their attention on the neurobiological basis of language and reading (Kral, Nielson, & Hynd, 1998). Broca and Wernicke localized language to the left hemisphere of the brain in the late nineteenth century (Kral et al., 1998), leading Hinshelwood to suggest that damage to these cortical areas may be associated with reading problems (Hinshelwood, 1900). These brain areas are still the focus of the majority of research on the neurobiological basis of reading problems, and research linking abnormalities in left hemisphere cortical language areas to developmental dyslexia is copious (e.g., Filipek, 1995; Galaburda, 1993; Hynd & Semrud-Clikeman, 1989; Miller, Sanchez, & Hynd, 2003; Morgan & Hynd, 1998). Galaburda and colleagues have theorized that subtle cortical malformations,

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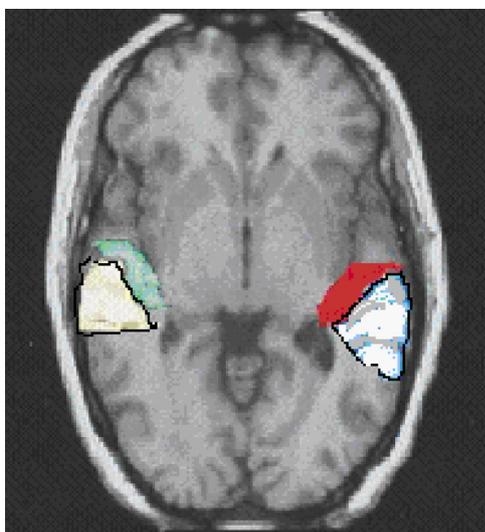


Fig. 1. An axial view of the planum temporal (the larger, triangular structure) and Heschl's gyrus of a normal adult. In this picture, the anterior of the brain is at the top and right and left are reversed. Thus, the fact that the left PT is larger is visible. Adapted from Hirayasu et al. (2000, p. 696).

which may be influenced by genes that increase susceptibility to dyslexia, lead to abnormal circuitry between and among cortical and thalamic areas, which in turn affects sensorimotor, perceptual, and cognitive processes important in learning (Galaburda, LoTurco, Ramus, Fitch, & Rosen, 2006). Specifically, dyslexia has been linked to neurobiological abnormalities in Broca's area, the angular gyrus, the planum temporale (PT) and the surrounding perisylvian cortex, the lateral geniculate nucleus of the thalamus, the occipital cortex, and the corpus callosum (Miller et al., 2003). However, the region of the planum temporale has received the most attention from researchers, as it is believed to play a pivotal role in the neurolinguistic deficits typically reported in dyslexia.

1.2. The planum temporale and developmental dyslexia

The planum temporale (PT) is a triangular-shaped area of cortex situated on the superior surface of the temporal lobe, adjacent to the Sylvian Fissure (SF) (see Fig. 1). It is bordered anteriorly by Heschl's gyrus and posteriorly by the termination of the horizontal aspect of the SF (Shapleske, Rossell, Woodruff, & David, 1999). The PT is a large structure within Wernicke's area, an area long known to play an important role in language comprehension (Barta et al., 1995; Nakada, Fujii, Yoneoka, & Kwee, 2001). It has long been associated with language lateralization, due to the fact that it is one of the most lateralized structures in the brain and typically shows pronounced leftward asymmetry (Geschwind & Levitsky, 1968). The function of the PT, as demonstrated by functional imaging, may be related to acoustic processing, phonological decoding, and language tasks (Blau et al., 2010; Dahaene et al., 2010). Research on subjects with lesions of the PT or near the PT has shown that those individuals are impaired in speech comprehension and auditory discrimination, suggesting the PT plays an important role in those cognitive tasks (Shapleske et al., 1999). Finally, some functional neuroimaging studies have suggested that the PT is activated during phonological decoding and language-related tasks (Nakada et al., 2001; Shapleske et al., 1999), which would connect the function of the PT to the major cognitive deficit in dyslexia.

In recent years, a theory regarding the importance of the left temporo-parietal area, which corresponds roughly to the PT, in the development of reading skills has been proposed by Shaywitz

and colleagues (Pugh et al., 2001; Shaywitz, 2003; Shaywitz & Shaywitz, 2007). In this theory, there are two left-hemisphere brain regions that are important in the development of fluent reading skills: the temporo-parietal area and the occipito-temporal area. The temporo-parietal system, which is thought to link orthographic and phonological processes, dominates as reading skills first develop. As word recognition becomes fluent and automatic, the occipito-temporal system becomes dominant over the temporo-parietal system. In individuals with dyslexia, functional activation is disrupted in both these systems, and increased activation is found in the corresponding right hemisphere posterior brain regions and inferior frontal regions (Pugh et al., 2001). The disruption of activation in the left temporo-parietal region, which corresponds roughly to the left PT, and increase in activation in the right temporo-parietal region, which corresponds roughly to the right PT, may reflect the functional consequences of atypical structural asymmetry of the PT. Interestingly, dyslexia readers who had made significant gains in reading fluency following a phonologically-based intervention demonstrated increased activation in left hemisphere regions, including the left superior temporal regions (Shaywitz et al., 2004).

Due to the association between the PT and language, the relationship between the PT and dyslexia has been the subject of considerable research. Abnormalities of the PT at the gross (Filipek, 1995; Hynd & Semrud-Clikeman, 1989; Leonard et al. 2001; Morgan & Hynd, 1998; Silani et al., 2005) and cellular levels (Galaburda & Kemper, 1979; Galaburda, Sherman, Rosen, Aboitiz, & Geschwind, 1990; Humphreys, Kaufman, & Galaburda, 1990) have been documented in dyslexia, although results have been inconsistent in that some document a larger right planum (reversed asymmetry; e.g., Galaburda & Kemper, 1979) whereas others reported smaller left planum (symmetry; e.g., Hynd, Semrud-Clikeman, Lorys, Novey, & Eliopoulos, 1990). This inconsistency is likely due to wide variety of methodological issues in measuring the PT, including a lack of consensus in the borders of the area and the limitations of early imaging technology (Barta et al., 1995; Honeycutt, Musick, Barta, & Pearlson, 2000). Furthermore, variables such as gender and handedness have not been well controlled in these studies, two variables that appear to have a relationship with PT asymmetry (Shapleske et al., 1999). Despite the inconsistent methods, a preponderance of the evidence suggests that individuals with dyslexia do not demonstrate the leftward asymmetry of the PT that is typically found in normal populations (Morgan & Hynd, 1998).

What might PT asymmetry have to do with developmental dyslexia? First of all, the PT is one of the most lateralized structures in the brain, which has led many researchers to conclude that it may be a structural representation of left-hemisphere dominance for language. Research has connected anomalous cerebral asymmetry to dyslexia, suggesting that language is not as strongly lateralized to the left hemisphere in dyslexia (Galaburda, 1995; Kertesz, Black, Polk, & Howell, 1986). Consistent with this theory, anomalous PT asymmetry would be expected in dyslexia. Furthermore, the mechanism for cerebral dominance may lie in the asymmetry of the homologous cortical regions, the larger of which may control its homologue via callosal connections, leading to left-hemisphere dominance for language (Galaburda, 1993). Galaburda and colleagues have found that when symmetry exists it is generally due to a larger than normal right PT rather than a smaller left PT (Galaburda, Corsiglia, Rosen, & Sherman, 1987; Humphreys et al., 1990). They suggest that the larger right PT in dyslexics as compared to controls may be interfering in the normal dominance of the left PT for language processing (Galaburda, 1995). In short, the structural asymmetry of the PT may reflect the functional dominance of the left hemisphere for language, which has been compromised in developmental dyslexia.

In fact, functional MRI research has demonstrated more activation of temporal cortex during reading in children who are typically developing readers than in children with dyslexia, suggesting that children with dyslexia fail to use brain areas specializing in language during reading tasks (Backes et al., 2002). Similarly, other researchers have found underactivation in the left temporo-parietal region, which corresponds roughly to the PT, in children with developmental dyslexia when compared to normally developing readers (Pugh et al., 2001; Shaywitz et al., 2002). Finally, and consistent with functional MRI findings, electrophysiological research has suggested a relationship between the abnormal PT hemispheric asymmetry often observed in dyslexia and poor performance in cortical auditory processing (Paul, Bott, Heim, Eulitz, & Elbert, 2005).

Clearly, the PT is considered an important region of interest when examining the neural correlates of dyslexia. Overall, several studies have identified abnormal patterns of PT morphology, represented either as reversed asymmetry or as lack of the typical leftward asymmetry. Variability across studies' findings may be largely associated with inconsistent methodological approaches, including measurement and definition of what constitutes the PT, and inclusion of variables such as handedness and gender in their analyses. With the re-emergence of sMRI research adding to DTI-based tractography and functional MRI, we believe that examination of PT morphology remains relevant as a source of reference for those conducting research in the neural markers of dyslexia. Furthermore, we do believe this study adds new information to the extant literature as efforts were made to: (a) obtain a large sample, (b) repeat classical measurement methodologies to avoid methodological inconsistencies that may add variability to findings, (c) control for handedness and gender, which have been largely disregarded in previous studies despite their relevance in evaluating between-groups PT differences, and (d) explore the brain-behavior relationships via examination of structural patterns and neuropsychological performance on testing. Given the range of literature we have tried to bridge, we hypothesized that (a) leftward-PT asymmetry will be positively correlated with right-handedness, male gender, verbal intelligence, and phonological processing, (b) that gradient of leftward PT asymmetry, phonological processing skills, and rapid naming skills will predict reading fluency, and (c) that children identified as having dyslexia will show less leftward asymmetry of the PT than children who do not meet criteria for dyslexia.

2. Method

2.1. Participants

Participants were children referred to a university-based developmental neuropsychology clinic in the southeastern United States, to participate in a research study. The study, funded by the National Institutes of Health (NIH), was designed to research variation in brain morphology and its relationship to neurolinguistic ability in developmental dyslexia. Families with at least one child who was experiencing serious reading problems or who had been previously diagnosed with developmental dyslexia were referred to the study through schools, local organizations, and advertisements. Both biological parents and school-aged siblings were strongly encouraged to participate in the testing process. The target child was required to be between 8 and 12 years of age and without a history of psychiatric disorders, neurological disorders, severe pre- or peri-natal complications, or traumatic brain injury. In exchange for their participation, parents received comprehensive neuropsychological reports for each of their participating children with results reported in a manner useful to school systems for making special education eligibility determinations. All children received a free t-shirt and a hard copy of their MRI scan.

There has been a great deal of controversy in recent years regarding the appropriate criteria for diagnosing reading disabilities, with many researchers arguing that the traditional IQ/achievement discrepancy model underrepresents children with lower IQ when, in fact, the cognitive deficits found in poor readers are the same regardless of IQ (Siegel, 1988; Stanovich & Siegel, 1994) as are the responses to intervention (Vellutino et al., 1996). A cut score model, which would

advocate the diagnosis of children who perform below average, typically defined as 85 standard score points or lower on a reading measure, has been proposed (Siegel, 1999). Despite its scientific support, this proposed criterion has not been widely accepted at the present time. In an effort to use the most stringent criteria for diagnosis, a diagnosis was made if the children met the following criteria: (1) reading achievement at or below 85 standard score points, as determined by a measure of oral reading fluency, (2) FSIQ above 85 standard score points, and (3) a discrepancy of at least 15 standard score points between FSIQ and reading achievement. These criteria satisfy both the cut score and discrepancy models of dyslexia diagnosis. Control subjects were children referred to the study that failed to meet these criteria but had FSIQs in the average range, i.e., greater than or equal to 85 standard score points. To insure that no children were included in the control group who might meet alternate criteria for dyslexia diagnosis, the six subjects who met the cut score criteria but not discrepancy criteria were excluded from the analyses.

Attention problems and previous diagnoses of attention-deficit/hyperactivity disorder (ADHD) were permitted. Prior research has demonstrated that the cognitive deficits found both in ADHD and RD are distinct (August & Garfinkel, 1990; Shaywitz et al., 1995) and thus the presence or absence of ADHD was unlikely to influence the results of the present study. Although symptoms of inattention are present in both groups, children with ADHD are characterized by having additional difficulties with behavioral disinhibition and/or hyperactivity, while children with RD show impairments in phonological processing (Sanchez, Miller, Garcia, & Hynd, 2005). Furthermore, children with solely ADHD do not show the atypical asymmetry of the left perisylvian cortex found in children with RD (Hynd et al., 1990). The decision was made to include children with ADHD because excluding them would have severely limited the available subjects and would have reduced the generalizability of the results, as these two disorders are frequently comorbid (Barkley, 1998).

Seventy families, including 96 children, participated in the data collection, which included both neuropsychological assessment and Magnetic Resonance Imaging (MRI) scans. Although outside the scope of this current study, one of the objectives under the grant that supported this study was to investigate familial associations on the expression of dyslexia. Thus, siblings who met criteria for participation were included in the study, and may have been included as controls or as dyslexics, depending on how they scored. Only 18% of the subjects are under this category. For the purposes of this study, eleven children were eliminated from data analysis due to Full Scale Intelligence Quotient (FSIQ) scores that were below the average range (i.e., less than 85 standard score points). Six additional subjects who met the cut score criteria but not discrepancy criteria for dyslexia diagnosis were excluded from the study to insure that no children were included in the control group who might meet alternate criteria for dyslexia diagnosis. In addition, age of siblings admitted to the study was restricted to the range of 8 to 14 years, eliminating six subjects. In nine children, the MRI scans were unreadable due to excess movement. In an additional nine children, there was no scan due to technical difficulties or anxiety regarding the MRI scanner. Elimination of participants for the above reasons left 55 children (34 males).

Twenty-six children (47%) met the described criteria for dyslexia diagnosis (18 males, 8 females), while 29 children (16 males, 13 females) did not meet those criteria and were used as clinical control subjects. Thirty-six children in this sample did not meet criteria for ADHD, while nine were diagnosed with ADHD-Primarily Inattentive Subtype, one was diagnosed with ADHD-Hyperactive Impulsive Subtype, and nine were diagnosed with ADHD-Combined Subtype. Of children with a diagnosis of dyslexia, 16 did not meet criteria for ADHD, while four were diagnosed with ADHD-Primarily Inattentive Subtype, none were diagnosed with ADHD-Hyperactive Impulsive Subtype, and six were diagnosed with ADHD-Combined Subtype. Of the clinical control subjects, there were 20 subjects that did not meet criteria for ADHD, while five were diagnosed with ADHD-Primarily Inattentive Subtype, one was diagnosed with ADHD-Hyperactive Impulsive Subtype, and three were diagnosed with ADHD-Combined Subtype.

2.2. Procedure

The parents provided informed, written consent for their and their child's participation in the assessment and MRI scan. In addition, the child provided written assent witnessed by their parents. Neuropsychological assessment was completed during the day, with a 1-h lunch break and additional breaks as needed. Following the evaluation, each child underwent a structural MRI scan at a local imaging facility. The scan took approximately 20–25 min. All structural MRI scans were reviewed by a board-certified neurologist to screen for neurological conditions. All procedures were approved by the Institutional Review Board, Human Subjects Office at the university where the study was conducted.

2.3. Neuropsychological assessment

The target child and participating siblings underwent a comprehensive neuropsychological assessment. The Wechsler Abbreviated Scale of Intelligence (WASI; The Psychological Corporation, 1999), a brief, norm-referenced, individually administered test of intellectual ability. Participants were also administered the

Gray Oral Reading Test—Third Edition (GORT-3; Wiederholt & Bryant, 1992), a measure of oral reading fluency that combines both reading rate and reading accuracy into a composite score. A measure of reading fluency was chosen since many of the children in the study had received extensive intervention and thus had improved scores on measures of word identification and pseudoword reading. However, these children continued to show impairments in reading fluency, which is one of the last reading skills to develop and the most resistant to intervention (Wolf & Katzir-Cohen, 2001). Reading fluency measures have been used in other studies in individuals with developmental dyslexia due to their increased sensitivity to reading problems (Rumsey et al., 1997). Phonological processing was assessed using the Comprehensive Test of Phonological Processing (CTOPP; Wagner, Torgesen, & Rashotte, 1999). The subtests Elision and Phoneme Reversal were averaged to create a composite that was used as a measure of phonological awareness. These subtests were chosen to create the composite because previous research has demonstrated that they discriminate individuals with dyslexia from individuals without dyslexia (Lombardino et al., 1997). In addition, the Alternate Rapid Naming Composite Score, which is composed of Rapid Color Naming and Rapid Object Naming, was used as a measure of naming ability. The Edinburgh Handedness Inventory (Oldfield, 1971) was used to assess handedness. This task consists of 10 motor activities, such as writing, eating, and brushing teeth, which are either demonstrated or pantomimed by the participant. Scores are continuous and scored as a percentile of right-handedness, with a score of 100 indicating that the subject is completely right-handed and a score of 0 indicating that the subject is completely left-handed.

2.4. MRI acquisition and analysis

A 1.5 T GE Sigma scanner was used to obtain three-dimensional structural MRI scans. Slices were gapless, collected in the sagittal plane, and 1.5 mm thick (TE=Min Full; flip angle=30; Field of view=24; frequency & phase=256, frequency direction=S/I). Raw image (*.MERGE) data was compiled using Matlab, a Linux-supported software program. The images were then converted to individual sequential Tagged Image Format (TIF) pictures using MRlcro software (free-ware found at <http://www.psychology.nottingham.ac.uk/staff/cr1/linux.html>), and then compiled into a single TIF file using Scion Image (<http://www.scioncorp.com/>).

2.5. Measurement of the planum temporale

The measurement of the PT followed the technique proposed by Steinmetz et al. (1989) and subsequently used or adapted in many other investigations (Dos Santos Sequeira et al., 2006; Foundas, Leonard, & Hanna-Pladdy, 2002; Heiervang et al., 2000; Hugdahl et al., 1998, 2003; Preis, Jäncke, Schmitz-Hillebrechte, & Steinmetz, 1999; Steinmetz, Volkman, Jäncke, & Freund, 1991). In this technique, the folded cortical surface is traced on each sagittal slice in which the PT is visible. By multiplying each length measurement by the slice thickness, an area measurement of the complete folded cortical surface area in mm² can be obtained (Steinmetz et al., 1989). This measurement technique does not control for the possible differences in gyrification between hemispheres, but prior research has shown that the gyrification index is consistent across hemispheres (Zilles, Armstrong, Schleicher, & Kretschmann, 1988).

Borders of the PT were defined based on guidelines suggested by Shapleske et al. (1999) in their comprehensive review of the PT. The anterior border of the PT was defined as Heschl's sulcus, or the sulcus immediately behind the first Heschl's transverse gyrus. This definition included any additional Heschl's gyri into the measurement of the PT. In cases where Heschl's gyrus did not extend laterally to the surface of the temporal lobe, an imaginary line was drawn to complete the anterior border. The use of computer software that allowed measurement lines to

remain in place as different slices were shown aided in this process. The posterior border of the PT was defined as the transition from the horizontal ramus of the sylvian fissure (SF) into a posterior ascending ramus (PAR) and/or posterior descending ramus (PDR). All cortex buried in the PDR was included in the PT, in keeping with Steinmetz and colleagues. The lateral border was defined as the superolateral margin of the superior temporal gyrus, while the medial border was defined as the point at which the anterior and posterior borders met. The supertentorial area on the midsagittal slice was measured as an estimate of total brain size.

All measurements were done by the first author (J.S.B.) who was blind to group membership at the time of measurement. Inter-rater reliability was achieved in two phases. A training phase, aimed at achieving consensus, in which PT length was measured collaboratively with an experienced investigator (G.W.H.), who provided guidance as to boundaries and measurement. During the second phase, each investigator measured 10 scans (20 hemispheres) independently and an inter-rater reliability coefficient was determined. The training brain was not included in these measurements. Inter-rater reliability was calculated using the intra-class correlation (ICC), and with ICC > .90 as criterion. The reliability achieved was excellent, with ICC = .983, and a confidence interval (95%) ranging from .936 to .996.

Interhemispheric and intrahemispheric asymmetry coefficients were determined for the PT in accordance with Steinmetz et al. (1990). The coefficient (R-L)/[(R+L)(0.5)] was used to determine interhemispheric asymmetry, with negative scores indicating leftward asymmetry and positive scores indicating rightward asymmetry.

2.6. Analyses

Statistical analyses for this study were conducted for each hypothesis. The first hypothesis was that, regardless of diagnostic group, leftward-PT asymmetry will be positively correlated with right-handedness, male gender, verbal intelligence, and phonological processing. Correlations were conducted to test these hypotheses, with the exception of gender, for which a directional t-test was conducted. To investigate the second hypothesis, that leftward PT asymmetry, phonological processing skills, and rapid naming skills will predict reading fluency, a multiple regression analysis was performed separately for boys and girls to assess for gender differences in the models, as gender is potentially a confounding variable in studies of PT morphology. The third hypothesis is that children identified as having dyslexia will show less leftward asymmetry of the PT than children who do not meet criteria for dyslexia. Analysis of covariance (ANCOVA) was used to test this hypothesis, with handedness, verbal intelligence, and supertentorial area used as covariates. Significance level was determined at the one-tailed level.

3. Results

3.1. Descriptive statistics

Group means and standard deviations for age, handedness quotient, phonological processing, rapid naming, FSIQ, VIQ, PIQ, and reading fluency are presented in Table 1. Table 2 contains means and standard deviations for neuroanatomical variables, including the left and right PT, supertentorial area, and PT asymmetry. As means and standard deviations were calculated on the entire sample as well as sub-groups categorized by the

Table 1
Demographic and psychometric variables in dyslexics, non-dyslexics, and the total sample.

	Total sample (N=55)		Dyslexics (N=26)		Non-dyslexics (N=29)		t-test	
	Mean	SD	Mean	SD	Mean	SD	t	p
Age (in months)	126.04	17.26	129.65	18.376	122.79	15.808	-.448	.656
Laterality quotient	87.74	23.77	90.77	17.011	84.81	28.872	-.950	.346
WASI FSIQ	102.45	12.66	102.65	11.85	101.76	13.463	.488	.629
WASI PIQ	104.25	15.74	105.85	13.77	102.45	17.541	.523	.604
WASI VIQ	100.11	12.35	99.19	12.869	100.93	12.035	.984	.329
GORT-3 Passage	83.52	16.30	70.19	8.658	95.89	11.060	9.015	.000*
CTOPP Phoneme reversal and elision	89.34	11.61	85.00	10.478	92.931	11.438	-.628	.536
Alt. Rapid naming	87.38	16.93	80.59	17.440	92.71	14.707	3.584	.001**

Abbreviations: WASI=Wechsler abbreviated scale of intelligence; FSIQ=Full scale intelligence quotient; PIQ=Performance intelligence quotient; VIQ=Verbal intelligence quotient; GORT-3=Gray Oral Reading Test—third edition; CTOPP=Comprehensive test of phonological processing.

* p < .05.
** p < .01.

Table 2
Neuroanatomical variables in dyslexics, non-dyslexics, and the total sample.

	Total sample (N=55)		Dyslexics (N=26)		Non-dyslexics (N=29)		t-test	
	Mean	SD	Mean	SD	Mean	SD	t	p
Left PT area (in mm ²)	811.68	167.98	815.12	128.154	808.59	199.33	.390	.698
Right PT area (in mm ²)	761.53	187.47	811.99	159.104	716.29	201.73	-2.42	.019*
Supertentorial area (in mm ²)	10392.95	846.12	10480.3	872.758	10314.63	828.95	-.266	.791
PT Asymmetry	-0.075	0.2376	-.0094	.20542	-.1341	.25208	-2.27	.027*

Abbreviations: PT=Planum temporal.

* $p < .05$.

presence or absence of dyslexia diagnosis, these descriptive statistics are presented in both manners.

In the total sample, leftward asymmetry was found for the PT asymmetry. It should be noted that the ratio variables measure asymmetry of the PT, with negative values indicating leftward asymmetry and positive values indicating rightward asymmetry.

Regarding handedness, quotients for the total sample and subgroups were calculated, excluding two members of the sample who did not complete the Edinburgh Handedness Inventory and, therefore, handedness quotient data were not available for those subjects. It appears that handedness quotient in this sample ($n=53$) is distributed in a similar manner to the normal population distribution of handedness, demonstrating a majority of right-handedness (96.22%, handedness quotients ranging from 80–100). However, we obtained a lower representation of left-handedness, than that typically observed in the normal population (1.8%, handedness quotient=20). One subject presented mixed handedness (1.8%, handedness quotient=65). A non-directional t -test indicated that handedness quotient does not differ significantly depending on the presence or absence of dyslexia diagnosis, $t(51)=-.810$, $p=.422$.

3.2. Correlational analyses

Pearson correlations were calculated on selected variables as a test of the first hypothesis. A summary of the results of these correlations is presented in Table 3. PT asymmetry was correlated with handedness quotient but not verbal intelligence or phonological processing. Verbal intelligence and phonological processing were also positively correlated in this sample.

3.3. Independent samples t -test

Directional t -tests were conducted to examine systematic differences in PT asymmetry associated with gender, under the assumption that a larger leftward PT asymmetry would be identified in males. In addition, total plana ratio was included in this analysis, but as this secondary analysis was exploratory a non-directional t -test was used. None of these analyses yielded statistically significant results.

Regression analyses

A multiple regression analysis was conducted to explore the second hypothesis. The model tested used PT asymmetry, rapid naming skills, and phonological processing skills to predict reading achievement in the total sample. The model was significant overall, with 46.5% of the variance in reading achievement explained by these three independent variables. PT asymmetry did not contribute significantly to the model ($p=.691$), while phonological processing ($p=.000$) and rapid naming skills ($p=.019$) did contribute significantly to reading achievement.

Table 3

Pearson correlations between PT asymmetry, phonological processing, verbal intelligence, and handedness.

	PT Asymmetry ^a	Phonological processing	WASI VIQ	Handedness quotient ^b
PT Asymmetry	1	-.077	.088	-.270*
Phonological processing		1	.362**	.171
WASI VIQ			1	.204
Handedness quotient				1

Abbreviations: PT=Planum temporale; WASI VIQ=Wechsler abbreviated scale of intelligence -verbal intelligence quotient.

* $p < .05$.

** $p < .01$.

^a PT asymmetry is a measure of the asymmetry of the PT, which is leftward in this sample.

^b Handedness quotient is an indicator of handedness, with higher numbers indicating right-handedness.

Previous research has suggested that gender may influence the asymmetry of the PT. In order to assess for gender differences that may be present in the multiple regression model described above, a new multiple regression model was created that included all the previously included variables plus gender and the cross-products of gender and phonological processing, rapid naming, and PT asymmetry. Including gender and the cross-products in the model did not significantly change the model R squared. Neither the cross-products nor gender contributed significantly to the model, indicating that gender differences in the coefficients are not statistically significant. In addition, the sample was divided by gender and the regression analysis was run again for each gender for descriptive purposes. The pattern of findings was similar for males and females.

3.4. Analysis of covariance

An analysis of covariance was used to test hypothesis three. As previous research has shown that handedness, verbal intelligence, and an estimate of total brain size are correlated with the PT, these variables were included as covariates in all three analyses. Although previous correlations did not support a significant relationship between verbal intelligence and PT asymmetry in this sample, it was decided that, because the literature suggests a relationship between those variables, it would be prudent to continue to use verbal intelligence as a covariate. Directional hypotheses used one-tailed analyses, while non-directional hypotheses used two-tailed analyses. Neuroanatomical variables were used as the dependent variables.

Hypothesis three stated that children classified as having dyslexia would show less leftward asymmetry of the PT than

Table 4

Results from the analysis of covariance examining the relationship between PT asymmetry and dyslexia diagnosis. Dependent variable: PT asymmetry.

	Degrees of freedom	F	p
Covariates			
Verbal intelligence	1	2.194	.073
Laterality quotient	1	7.358	.005**
Supertentorial area	1	2.466	.062
Independent variable			
Dyslexia diagnosis	1	5.485	.012*

Note: $R^2 = .214$.

Abbreviation: PT=Planum temporale.

* $p < .05$.

** $p < .01$ (1-tailed).

Table 5

Estimated marginal means for PT asymmetry based on dyslexia diagnosis in the total sample.

	PT Asymmetry	
	Mean	Standard error
Dyslexics ($N=26$)	.008	.041
Non-dyslexics ($N=29$)	-.128	.040

children who did not meet criteria for dyslexia. Table 4 summarizes the results. The relationship between PT asymmetry and diagnostic group was significant, as was the relationship between handedness quotient and PT asymmetry. The relationships between verbal intelligence and PT asymmetry and supertentorial area and PT asymmetry were not significant. The R squared suggests that 21.4% of the variance in dyslexia diagnosis can be explained by PT asymmetry, handedness quotient, supertentorial area, and verbal intelligence. Examination of the means of left and right PT area as well as PT asymmetry across diagnostic groups reveals that PT symmetry was found in children with dyslexia while leftward asymmetry of the PT was found in children who did not meet criteria for dyslexia.

The estimated marginal means and standard error of PT asymmetry divided by diagnostic group are presented in Table 5. The estimated marginal means are generated during analysis of covariance and give an estimate of what the ratio means might be if verbal intelligence, handedness quotient, and supertentorial area were the same in each group. It is important to note that negative numbers indicate leftward asymmetry and positive numbers indicate rightward asymmetry. Consistent with our previous observation, the estimated marginal means for PT asymmetry show that children with dyslexia have symmetrical PTs while individuals without dyslexia show leftward asymmetry. An examination of the left and right PT area in Table 2 shows that this difference is due to a larger right PT in individuals with dyslexia. An independent t -test reveals that this difference is statistically significant, $t(53) = -1.938$, $p < .05$.

4. Discussion

The current study contributes to the literature on the relationship between plana morphology, reading ability, and dyslexia diagnosis in several ways. Compared to the sample sizes ranging from 9 per group to 25 per group found in the literature on plana morphology and dyslexia diagnosis, we were able to obtain sample sizes of 26 and 29 per group. Sample sizes tend to be relatively

small in this literature due to the time and expense of full neuropsychological evaluations, MRI scans, and measurement of brain areas.

Furthermore, the use of widely accepted methodologies in the measurement of the PT and the calculation of the neuroanatomical ratios that derive from those measurements, adds value to this study. The use of borders of the PT has not been consistent in the literature, but recent reviews of the literature on the PT have suggested the use of specific borders so that a standard for PT measurements is emerging in the literature (Barta et al., 1995; Beaton, 1997; Shapleske et al., 1999). These suggested borders were followed in this study. These reviews of PT measurement procedures also suggested that MRI scans should have gapless slices with 1.5 mm slice thickness or smaller. The MRI scans used in this study met this criterion. In addition, the use of the Steinmetz methodology (Steinmetz et al., 1989) in the measurement of the PT and the calculation of plana ratios is a strength of the current study. This method is popular in the literature currently being published on the PT. While criticized by some, this methodology is a great improvement over length measurements as it is a measure of area that takes cortical folding into account.

Perhaps the most significant contributions of this study are the facts that we controlled for handedness and gender, and that we were able to examine brain and behavior relationships, a component often ignored in the neuroimaging studies. The use of reading fluency as the outcome measure in the multiple regression analysis and as a part of the diagnostic criteria for dyslexia diagnosis is both a contribution to the literature and a limitation of this study as it limits its ability to generalize to other studies. Reading fluency has not been used extensively in this literature as a means of diagnosing dyslexia, although some studies have employed it due to its increased sensitivity in detecting reading problems (Rumsey et al., 1997). However, due to the number of children in this study who had received extensive phonologically-based intervention, the use of the most sensitive measure to reading problems was deemed necessary and appropriate.

4.1. PT morphology and ratios

Results of this study suggest that children with dyslexia differ from a clinical control sample with regards to asymmetry of the PT. This study supports the findings of Heiervang et al. (2000), Hugdahl et al. (1998), Hynd et al. (1990), and Larsen, Høien, Lundberg, and Ødegaard (1990) who found atypical asymmetry of the PT at statistically significant levels in children with dyslexia using structural MRI. This study also corroborates postmortem studies of adults with dyslexia in which symmetrical PT were found (Galaburda & Kemper, 1979; Galaburda, Sherman, Rosen, Aboitiz, & Geschwind, 1985; Humphreys et al., 1990). Specifically, this study found symmetry of the PT in children with dyslexia while controlling for handedness, verbal intelligence, and total brain size. This symmetry was due to a larger right PT in the brains of children with dyslexia. This result is consistent with the postmortem studies of Galaburda et al. (1987), Humphreys et al. (1990), who have suggested that the right PT is larger in individuals with dyslexia due to insufficient pruning during corticogenesis. Galaburda and colleagues have theorized that excess cortex in the PT on the right side of the brain may interfere with language processing in the PT on the left side of the brain, putting individuals at risk for developmental dyslexia (Galaburda, 1993). In fact, one study found that larger right PTs are associated with weaker linguistic skills (Foster, Hynd, Morgan, & Hugdahl, 2002). Findings from this study lend further support to Galaburda's theory.

4.2. The relationship between PT asymmetry, neuropsychological test performance, and demographic variables

In the current study, leftward PT asymmetry was significantly correlated with right-handedness, but not with verbal intelligence or phonological processing. As is typically reported in the literature, phonological processing and verbal intelligence were significantly correlated. It is possible that sufficient power was not present in this study to find relationships between the neuroanatomical variables and verbal intelligence and phonological processing. Another possibility is that previous findings linking verbal intelligence and phonological processing to atypical PT asymmetry used samples in which the children with dyslexia had lower IQs than the control subjects. The lack of a relationship between PT asymmetry and phonological processing may also be explained by the fact that many children in our sample had received reading interventions that were phonologically-oriented. Some of the children who had received intervention had improved phonological processing skills, which in some had led to improvements in reading, while some were able to read accurately but not fluently. In fact, post-intervention hemispheric reorganization of reading and language brain areas in dyslexia has been demonstrated (Spironelli, Penolazzi, Vio, & Angrilli, 2010). Furthermore, a recent study conducted by Welcome, Leonard, and Chiarello (2010) reported increased variability in PT asymmetry in a sample of readers with adequate reading comprehension but poor phonological and orthographic processing. These resilient readers appear to compensate their deficits by relying on semantic associations between words while reading, a common alternative strategy used to improve reading in dyslexia. Future studies should attempt to control for reading interventions, particularly as many children are receiving phonologically-oriented interventions at young ages, sometimes before diagnoses are made.

Gender differences in neuroanatomical ratios were not found in the current study. However, it is important to continue to assess for gender differences when studying the PT due to previous findings showing differences in interhemispheric asymmetry of the PT in males and females (Honeycutt et al., 2000). Furthermore, when using non-ratio neuroanatomical variables, it is important to control for the effect of total brain size, as males typically have larger brains than females, even as children (Schultz et al., 1994).

4.3. The relationship between PT asymmetry, phonological processing, rapid naming, and reading achievement

Results from this study suggest that PT asymmetry does not predict reading achievement, while phonological processing and rapid naming skills do predict reading achievement. These results are consistent across males and females. There has not been another study that has attempted to use PT asymmetry to predict reading achievement in a regression model, likely due to sample size limitations (e.g., Hynd et al., 1990; Leonard et al., 1993; Robichon, Levrier, Farnarier, & Habib, 2000; Rumsey et al., 1997; Schultz et al., 1994; Semrud-Clikeman, Hynd, Novy, & Eliopoulos, 1991). Consistent with the double-deficit hypothesis (Wolf & Bower, 1999), this study found that phonological processing and rapid naming skills are good predictors of reading achievement.

4.4. Limitations of the study

One limitation of this study is its reliance on a clinical sample, which is more likely to show comorbidity and severity of a disorder due to the process of self-referral to the study. Parents with more severely impaired children or children whose behavior was more externalizing were more likely to bring their children in for the assessment. Furthermore, head tilt can impact volumetric

analysis particularly when the boundaries of the region of interest are poorly defined. Our measurements were conducted with the same software program and protocol used in other studies (Heiervang et al., 2000; Hugdahl et al., 2003, 1998), which employed a well-calibrated PT measurement methodology outlined in Steinmetz et al. (1989). The measurement protocol relies on precise morphological landmarks that facilitate the identification of the PT. It was noted that Steinmetz et al.'s protocol does not include head tilt correction, and therefore we did not include this procedure in our protocol. The lack of alignment can introduce small sources of variability not corrected by the easily identifiable measurement landmarks. Finally, given the potential genetic component of dyslexia (Scerri, & Schulte-Körne, 2010), the fact that there are a few siblings in the control group (< 18%) may have obscured some of our findings.

5. Conclusions and future directions

This study demonstrates that there is a relationship between PT asymmetry and dyslexia diagnosis that exists even when controlling for handedness, verbal intelligence, and total brain size. The question remains as to exactly how the PT affects neural processing in a way as to put an individual at risk for dyslexia. This study does not support a relationship between PT asymmetry and phonological processing, considered by many researchers to be the core deficit in dyslexia. This may be due to the amount of intervention the children in this sample have received, particularly phonologically-oriented reading interventions. Previous research has shown that PT function is involved in language-related tasks other than phonological decoding (Shapleske et al., 1999) and other studies have found a relationship between the PT and both receptive and expressive language (Morgan, 1996). It is possible, therefore, that a relationship exists between PT morphology and other types of language skills, such as receptive language, expressive language, and confrontation naming. As many children with severe and specific language impairments have difficulty with reading, it may be the relationship between PT asymmetry and language impairments that explains the association between PT asymmetry and dyslexia diagnosis. Some research has suggested that larger right PTs are associated with weaker linguistic skills (Foster et al., 2002), while other studies have posited that plana morphology is associated with global language skills (Morgan, 1996). Further research is needed to target this question specifically by employing detailed language assessments as part of the diagnostic process.

Another important area for future study is to relate structural MRI research on the PT with functional MRI research. Researchers have found underactivation of the left temporo-parietal region, which corresponds roughly to the left PT, and overactivation in the right temporo-parietal region, which corresponds roughly to the right PT, in children with dyslexia during reading tasks in functional MRI studies (Pugh et al., 2001; Shaywitz et al., 2002). Based on findings from structural MRI studies that the right PT is larger in children with dyslexia, it is possible that the overactivation of the right temporo-parietal area and corresponding underactivation of the left temporo-parietal area found in children with dyslexia in functional MRI studies is due to the interference of a larger right PT in the processing of language in these children. This mechanism would be consistent with the theory of interference of the right PT in language processing as postulated by Galaburda (1993). This larger right PT may be interfering with language processing in the left PT, causing underactivation of that region in children with dyslexia. Linking these two areas of research would help elucidate the function of the PT as well as

the role this structure may play in developmental dyslexia in children.

Financial support

Preparation of this manuscript was supported in part by a grant to the final author (GWH; NIH/NICHD-1-R01-26890-07). This study was conducted at the former Center for Clinical and Developmental Neuropsychology, University of Georgia.

Acknowledgements

We gratefully acknowledge the contributions of William Rylie Moore (BSc [Hon]) to editing and proofreading this manuscript. Your input was very helpful to us.

References

- American Psychiatric Association (2000). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author, text rev.
- August, G. J., & Garfinkel, B. D. (1990). Comorbidity of ADHD and reading disability among clinic-referred children. *Journal of Abnormal Child Psychology*, *18*, 29–45.
- Backes, W., Vuurman, E., Wennekes, R., Spronk, P., Wuisman, M., van Engelshoven, J., et al. (2002). Atypical brain activation of reading processes in children with developmental dyslexia. *Journal of Child Neurology*, *17*, 867–871.
- Barkley, R. A. (1998). *Attention-deficit hyperactivity disorder: a handbook for diagnosis and treatment* (2nd ed.). New York, NY: Guilford Press.
- Barta, P. E., Petty, R. G., McGilchrist, I., Lewis, R. W., Jerram, M., Casanova, M., et al. (1995). Asymmetry of the planum temporale: methodological considerations and clinical associations. *Neuroimaging*, *61*, 137–150.
- Beaton, A. A. (1997). The relation of planum temporale asymmetry and morphology of the corpus callosum to handedness, gender and dyslexia: a review of the evidence. *Brain and Language*, *60*, 255–322.
- Blau, V., Reithler, J., van Atteveldt, N., Seitz, J., Gerretsen, P., Goebel, R., et al. (2010). Deviant processing of letters and speech sounds as proximate cause of reading failure: an fMRI study of dyslexic children. *Brain*, *133*, 868–879.
- Dahaene, S., Pegado, F., Braga, L. W., Ventura, P., Filho, G. N., Jobert, A., et al. (2010). How learning to read changes the cortical networks for vision and language. *Science*, *330*, 1359–1364.
- Dos Santos Sequeira, S., Woerner, W., Walter, C., Kreuder, F., Lueken, U., Westerhausen, R., et al. (2006). Handedness, dichotic-listening ear advantage, and gender effects on planum temporale asymmetry: a volumetric investigation using structural magnetic resonance imaging. *Neuropsychologia*, *44*, 622–636.
- Eden, G. F., VanMeter, J. W., Rumsey, J. M., & Zeffiro, T. A. (1996). The visual deficit theory of developmental dyslexia. *Neuroimage*, *4*, S108–S117.
- Filipek, P. A. (1995). Neurobiologic correlates of developmental dyslexia: how do dyslexics' brains differ from those of normal readers. *Journal of Child Neurology*, *10*(Suppl. 1), 62–69.
- Foster, L. M., Hynd, G. W., Morgan, A. E., & Hugdahl, K. (2002). Planum temporale asymmetry and ear advantage in dichotic listening in developmental dyslexia and attention-deficit/hyperactivity disorder (ADHD). *Journal of the International Neuropsychological Society*, *8*, 22–36.
- Foundas, A. L., Leonard, C. M., & Hanna-Pladdy, B. (2002). Variability in the anatomy of the planum temporale and posterior ascending ramus: do right- and left-handers differ? *Brain and Language*, *83*, 403–424.
- Galaburda, A. M. (1993). Neuroanatomical basis of developmental dyslexia. *Behavioral Neurology*, *11*, 161–173.
- Galaburda, A. M. (1995). Anatomic basis of cerebral dominance. In: R. J. Davidson, & K. Hugdahl (Eds.), *Brain asymmetry* (pp. 51–74). Cambridge, MA: MIT Press.
- Galaburda, A. M., Corsiglia, J., Rosen, G. D., & Sherman, G. F. (1987). Planum temporale asymmetry, reappraisal since Geschwind and Levitsky. *Neuropsychologia*, *25*, 853–868.
- Galaburda, A. M., & Kemper, T. L. (1979). Cytoarchitectonic abnormalities in developmental dyslexia: a case study. *Annals of Neurology*, *6*, 94–100.
- Galaburda, A. M., LoTurco, J., Ramus, F., Fitch, R. H., & Rosen, G. D. (2006). From genes to behavior in developmental dyslexia. *Nature Neuroscience*, *9*, 1213–1217.
- Galaburda, A. M., Sherman, G. F., Rosen, G. D., Aboitiz, F., & Geschwind, N. (1985). Developmental dyslexia: four consecutive patients with cortical anomalies. *Annals of Neurology*, *18*, 222–233.
- Galaburda, A. M., Sherman, G. F., Rosen, G. D., Aboitiz, F., & Geschwind, N. (1990). Developmental dyslexia: four consecutive patients with cortical abnormalities. *Annals of Neurology*, *18*, 222–233.
- Geschwind, N., & Levitsky, W. (1968). Human brain: left-right asymmetries in temporal speech region. *Science*, *161*, 186–187.
- Heiervang, E., Hugdahl, K., Steinmetz, H., Smievoll, A. I., Stevenson, J., Lund, A., et al. (2000). Planum temporale, planum parietale and dichotic listening in dyslexia. *Neuropsychologia*, *38*, 1704–1714.
- Hinshelwood, J. (1900). Congenital word-blindness. *The Lancet*, *1*, 1506–1508.
- Hirayasu, Y., McCarley, R. W., Salisbury, D. F., Tanaka, S., Kwon, J. S., Frumin, M., et al. (2000). Planum temporale and Heschl gyrus volume reduction in Schizophrenia. A magnetic resonance imaging study of first-episode patients. *Archives of General Psychiatry*, *57*, 692–699.
- Honeycutt, N. A., Musick, A., Barta, P. E., & Pearlson, G. D. (2000). Measurement of the planum temporale (PT) on magnetic resonance imaging scans: temporal PT alone and with parietal extension. *Psychiatry Research: Neuroimaging Section*, *98*, 103–116.
- Hugdahl, K., Heiervang, E., Erslund, L., Lundervold, A., Steinmetz, H., & Smievoll, A. I. (2003). Significant relationship between MR measures of planum temporale area and dichotic processing of syllables in dyslexic children. *Neuropsychologia*, *41*, 666–675.
- Hugdahl, K., Heiervang, E., Nordby, H., Smievoll, H., Stevenson, J., & Lund, A. (1998). Central auditory processing, MRI morphometry and brain laterality: applications to dyslexia. *Scandinavian Audiology*, *27*, 26–34.
- Humphreys, P., Kaufman, W. E., & Galaburda, A. (1990). Developmental dyslexia in women: evidence for a subgroup with a reversal of cerebral asymmetry. *Annals of Neurology*, *28*, 727–738.
- Hynd, G. W., & Semrud-Clikeman, M. (1989). Dyslexia and brain morphology. *Psychological Bulletin*, *106*, 447–482.
- Hynd, G. W., Semrud-Clikeman, M., Lorys, A. R., Novey, E. S., & Eliopoulos, D. (1990). Brain morphology in developmental dyslexia and attention deficit/hyperactivity disorder. *Archives of Neurology*, *47*, 919–926.
- Kertesz, A., Black, S. E., Polk, M., & Howell, J. (1986). Cerebral asymmetries on magnetic resonance imaging. *Cortex*, *22*, 117–127.
- Kibby, M. Y., & Hynd, G. W. (2001). Neurobiological basis of learning disabilities. In: D. P. Hallahan, & B. K. Keogh (Eds.), *Research and global perspectives in learning disabilities: essays in Honor of William M. Cruickshank* (pp. 25–42). Mahwah, NJ: Lawrence Erlbaum.
- Klein, R. M. (2002). Observations on the temporal correlates of reading failure. *Reading and Writing: An Interdisciplinary Journal*, *15*, 207–232.
- Kral, M., Nielson, K., & Hynd, G. W. (1998). Historical conceptualization of developmental dyslexia: neurolinguistic contributions from the 19th and early 20th centuries. In: R. Licht, A. Bouma, W. Slot, & W. Koops (Eds.), *Child Neuropsychology* (pp. 1–16). Delft, Netherlands: Eburon Publishing.
- Larsen, J. P., Høien, T., Lundberg, I., & Ødegaard, H. (1990). MRI evaluation of the size and symmetry of the planum temporale in adolescents with developmental dyslexia. *Brain and Language*, *39*, 289–301.
- Leonard, C. M., Eckert, M. A., Oakland, L. T., Kranzler, J., Mohr, C. M., King, W. M., et al. (2001). Anatomical risk factors for phonological dyslexia. *Cerebral Cortex*, *11*, 148–157.
- Leonard, C. M., Voeller, K. K. S., Lombardino, L. J., Morris, M. K., Hynd, G. W., Alexander, A. W., et al. (1993). Anomalous cerebral structure in dyslexia revealed with magnetic resonance imaging. *Archives of Neurology*, *50*, 461–469.
- Lombardino, L. J., Riccio, C., Hynd, G. W., & Pinheiro, S. B. (1997). Linguistic deficits in children with reading disabilities. *American Journal of Speech-Language Pathology*, *6*, 71–78.
- Mayes, S. D., Calhoun, S. L., & Crowell, E. W. (2000). Learning disabilities and ADHD: overlapping spectrum disorders. *Journal of Learning Disabilities*, *33*, 417–424.
- Miller, C. J., Sanchez, J., & Hynd, G. W. (2003). Neurological correlates of reading disabilities. In: L. Swanson, & S. Graham (Eds.), *Handbook of research on learning disabilities* (pp. 242–255). New York, NY: Guilford Press.
- Morgan, A. E. (1996). *Anatomical variation of the planum temporale: implications for dyslexia and linguistic ability*. Athens, Georgia: Unpublished Dissertation, University of Georgia.
- Morgan, A. E., & Hynd, G. W. (1998). Dyslexia, neurolinguistic ability, and anatomical variation of the planum temporale. *Neuropsychology Review*, *8*, 79–93.
- Nakada, T., Fujii, Y., Yoneoka, Y., & Kwee, I. L. (2001). Planum temporale: where spoken and written language meet. *European Neurology*, *46*, 121–125.
- Oldfield, R. C. (1971). The assessment and analysis of handedness: the Edinburgh Inventory. *Neuropsychologia*, *9*, 97–113.
- Paul, I., Bott, C., Heim, S., Eulitz, C., & Elbert, T. (2005). Reduced hemispheric asymmetry of the auditory N260m in dyslexia. *Neuropsychologia*, *44*, 785–794.
- Preis, S., Jäncke, L., Schmitz-Hillebrecht, J., & Steinmetz, H. (1999). Child age and planum temporale asymmetry. *Brain and Cognition*, *40*, 441–452.
- Pugh, K. R., Mencl, W. E., Jenner, A. R., Katz, L., Frost, S. J., Lee, J. R., et al. (2001). Neurobiological studies of reading and reading disability. *Journal of Communication Disorders*, *34*(6), 479–492.
- Pugh, K. R., Mencl, W. E., Jenner, A. R., Lee, J. R., Katz, L., Frost, S. J., et al. (2001). Neuroimaging studies of reading development and reading disability. *Learning Disabilities Research and Practice*, *16*(4), 240–249.
- Purvis, K. L., & Tannock, R. (1997). Language abilities in children with attention-deficit hyperactivity disorder, reading disabilities, and normal controls. *Journal of Abnormal Child Psychology*, *25*, 133–144.
- Ramus, F. (2003). Developmental dyslexia: specific phonological deficit or general sensorimotor dysfunction? *Current Opinion in Neurobiology*, *13*, 212–218.
- Robichon, F., Levrier, O., Farnarier, P., & Habib, M. (2000). Developmental dyslexia: atypical cortical asymmetries and functional significance. *European Journal of Neurology*, *7*, 35–46.
- Rumsey, J. M., Donohue, B. C., Brady, D. R., Nace, K., Giedd, J. N., & Andreason, P. (1997). A magnetic resonance imaging study of planum temporale asymmetry in men with developmental dyslexia. *Archives of Neurology*, *54*, 1481–1489.
- Silani, G., Frith, U., Demonet, J. F., Fazio, F., Perani, D., Price, C., et al. (2005). Brain abnormalities underlying altered activation in dyslexia: a voxel based morphometry study. *Brain*, *128*, 2453–2461.

- Sanchez, J., Miller, C. J., Garcia, M., & Hynd, G. W. (2005). Reading disabilities in children with ADHD. In: D. Gozal, & D. Molfese (Eds.), *Attention deficit hyperactivity disorder: from genes to animal models to patients* (pp. 337–358). Totowa, NJ: Humana Press.
- Scerif, T. S., & Schulte-Körne, G. (2010). Genetics of developmental dyslexia. *European Child & Adolescent Psychiatry*, 19(3), 179–197.
- Schultz, R. T., Cho, N. K., Staib, L. H., Kier, L. E., Fletcher, J. M., Shaywitz, S. E., et al. (1994). Brain morphology of normal and dyslexic children: the influence of sex and age. *Annals of Neurology*, 35, 732–742.
- Semrud-Clikeman, M., Hynd, G. W., Novey, E. S., & Eliopoulos, D. (1991). Dyslexia and brain morphology: relationships between neuroanatomical variation and neurolinguistic tasks. *Learning & Individual Differences*, 3, 225–242.
- Shapleske, J., Rossell, S. L., Woodruff, P. W. R., & David, A. S. (1999). The planum temporale: a systematic, quantitative review of its structural, functional, and clinical significance. *Brain Research Reviews*, 29, 26–49.
- Shaywitz, B. A., Fletcher, J. M., Holahan, J. M., Shneider, A. E., Marchione, K. E., Stuebing, K. K., et al. (1995). Interrelationships between reading disability and attention-deficit/hyperactivity disorder. *Child Neuropsychology*, 1, 170–186.
- Shaywitz, B. A., Shaywitz, S. E., Blachman, B., Pugh, K. R., Fulbright, R. K., Skudlarski, P., et al. (2004). Development of left occipitotemporal systems for skilled reading in children after a phonologically-based intervention. *Biological Psychiatry*, 55, 926–933.
- Shaywitz, B. A., Shaywitz, S. E., Pugh, K. R., Mencl, W. E., Fulbright, R. K., Skudlarski, P., et al. (2002). Disruption of posterior brain systems for reading in children with developmental dyslexia. *Biological Psychiatry*, 52(2), 101–110.
- Shaywitz, S. E. (2003). *Overcoming dyslexia: a new and complete science-based program for reading problems at any level*. New York, NY: Alfred A. Knopf.
- Shaywitz, S. E., & Shaywitz, B. A. (1999). Cognitive and neurobiologic influences in reading and in dyslexia. *Developmental Neuropsychology*, 16, 383–384.
- Shaywitz, S. E., & Shaywitz, B. A. (2007). The neurobiology of reading and dyslexia. *ASHA Leader*, 12(12), 20–21.
- Siegel, L. S. (1988). Evidence that IQ scores are irrelevant to the definition and analysis of reading disability. *Canadian Journal of Psychology*, 42, 201–215.
- Siegel, L. S. (1993). Phonological processing deficits as the basis of a reading disability. *Developmental Review*, 13, 246–257.
- Siegel, L. S. (1999). Issues in the definition and diagnosis of learning disabilities: a perspective on Guckenberger v. Boston University. *Journal of Learning Disabilities*, 32, 304–320.
- Spironelli, C., Penolazzi, B., Vio, C., & Angrilli, A. (2010). Cortical reorganization in dyslexic children after phonological training: evidence from early evoked potentials. *Brain*, 133, 3385–3395.
- Stanovich, K. E., & Siegel, L. S. (1994). Phenotypic performance profile of children with reading disabilities: a regression-based test of the phonological core variable-difference model. *Journal of Educational Psychology*, 86, 24–53.
- Steinmetz, H., Rademacher, J., Huang, Y., Hefter, H., Zilles, K., Thron, A., et al. (1989). Cerebral asymmetry: MR planimetry of the human planum temporale. *Journal of Computer Assisted Tomography*, 13, 996–1005.
- Steinmetz, H., Rademacher, J., Jäncke, L., Huang, Y., Thron, A., & Zilles, K. (1990). Total surface of temporoparietal cortex: diverging left–right asymmetries. *Brain and Language*, 39, 1–16.
- Steinmetz, H., Volkman, J., Jäncke, L., & Freund, H. (1991). Anatomical left–right asymmetry of language-related temporal cortex is different in left- and right-handers. *Annals of Neurology*, 29, 315–319.
- The Psychological Corporation (1999). *The Wechsler Abbreviated Scale of Intelligence*. San Antonio, Texas: Author.
- Torgesen, J. K., Wagner, R. K., & Rashotte, C. A. (1994). Longitudinal studies of phonological processing and reading. *Journal of Learning Disabilities*, 27, 276–287.
- Vellutino, F. R., Scanlon, D. M., Sipay, E. R., Small, S. G., Pratt, A., Chen, R., et al. (1996). Cognitive profiles of difficult-to-remediate and easily remediated poor readers: early intervention as a vehicle for distinguishing between cognitive and experiential deficits as basic causes of specific reading disability. *Journal of Educational Psychology*, 88, 601–638.
- Wagner, R. K., Torgesen, J. K., & Rashotte, C. A. (1999). *The comprehensive test of phonological processing*. Austin, Texas: Pro-Ed.
- Welcome, S. E., Leonard, C. M., & Chiarello, C. (2010). Alternate reading strategies and variable asymmetry of the planum temporale in adult resilient readers. *Brain and Language*, 113, 73–83.
- Wiederholt, L., & Bryant, B. (1992). *Gray Oral Reading Tests—3*. Austin, TX: PRO-ED.
- Wolf, M., & Bowers, P. G. (1999). The double-deficit hypothesis for developmental dyslexia. *Journal of Educational Psychology*, 91, 415–438.
- Wolf, M., & Katzir-Cohen, T. (2001). Reading fluency and its intervention. *Scientific Studies of Reading*, 5, 211–239.
- Zilles, K., Armstrong, E., Schleicher, A., & Kretschmann, H. (1988). The human pattern of gyrification in the cerebral cortex. *Anatomy and Embryology*, 179, 173–179.