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The Development of Reading Impairment: A Cognitive Neuroscience Model

Bruce D. McCandliss[1] and Kimberly G. Noble[1, 2]

[1]Sackler Institute for Developmental Psychobiology, Weill Medical College of Cornell University, New York, New York

[2]University of Pennsylvania Center for Cognitive Neuroscience, Philadelphia, Pennsylvania

This review discusses recent cognitive neuroscience investigations into the biological bases of developmental dyslexia, a common disorder impacting approximately 5 to 17 percent of the population [Stanovich, 1986, Matthew effects in reading: Some consequences of individual differences in the acquisition of literacy. Reading Research Quarterly 21: 360–407]. Our aim is to summarize central findings from several lines of evidence that converge on pivotal aspects of the brain bases of developmental dyslexia. We highlight ways in which the approaches and methodologies of developmental cognitive neuroscience that are addressed in this special issue—including neuroimaging, human genetics, refinement of cognitive and biological phenotypes, neural plasticity and computational models—can be employed in uncovering the biological bases of this disorder. Taking a developmental perspective on the biological bases of dyslexia, we propose a simple *cascading model* for the developmental progression of this disorder, in which individual differences in brain areas associated with phonological processing influence the specialization of visual areas involved in the rapid processing of written words. We also discuss recent efforts to understand the impact of successful reading interventions in terms of changes within cortical circuits associated with reading ability.
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INTRODUCTION

Most diagnostic criteria invoke the term dyslexia "when accurate and fluent word reading and/or spelling develops very incompletely or with great difficulty. . ." [Reason, 2001]. As typically quantified in the US, an adult or child scoring at least 1.5 to 2 standard deviations below the norm for his or her age on standardized tests of reading ability would be considered to have skills in the dyslexic range. The diagnosis is considered to be one of "specific reading disorder" if reading scores are discrepant with overall IQ and cannot be directly attributable to sensory difficulties or educational deprivation. Several researchers, however, have challenged the validity and utility of using IQ discrepancies to define dyslexia, as well as the assumption that dyslexia represents a discrete syndrome rather than a continuum of disability [Fletcher et al., 1994; Shaywitz et al., 1992; Stanovich, 1994, see, however, Rutter, 1978]. It is possible that such debates may hinge upon a better understanding and quantification of the underlying disorder, which is being furthered by research seeking to create more refined, empirically-based cognitive and neurobiological phenotypes of reading disorder. Furthermore, given that reading is a cultural invention that is acquired only after years of extensive teaching and practice, research aimed at understanding the neurobiological basis of reading impairment is logically tightly coupled to investigations into the neurobiological basis of normal reading development.

In the last several years a great deal of developmental, cognitive, and neuroscience research has made progress in characterizing both typical reading development and dyslexia on several levels. Converging evidence has led to one candidate cognitive phenotype of developmental dyslexia, namely an underlying core deficit in phonological processing abilities [for a review see National Reading Panel, 2000]. A very specific pattern of linguistic deficits associated with phonological processing has been found to significantly predict the emergence of reading difficulties [Bradley and Bryant, 1983; Bishop and Adams, 1990; Gathercole and Baddeley, 1987]. This research suggests that developmental dyslexia is linked to a core cognitive deficit in phonological awareness tasks that require analysis and synthesis of the sounds within syllables [Wagner et al., 1997].

The newly emerging ability to characterize individual differences in the structural and functional properties of specific brain regions has the potential to aid in the formulation of a neurobiological phenotype of developmental dyslexia. Across several methodologies within cognitive neuroscience, evidence consistently points to two cortical areas that exhibit dysfunction in developmental dyslexia, and which, we propose, subserve the normal development of the cognitive achievement of reading. The first region—a left perisylvian area typically involving the superior temporal gyrus (STG)—is involved in phonological processing, and demonstrates significant structural and functional differences be-

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*Correspondence to: Bruce D. McCandliss, Sackler Institute for Developmental Psychobiology Weill Medical College of Cornell University, 1300 York Ave., Box 140, New York, NY 10021. E-mail: bdm2001@med.cornell.edu
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tween dyslexic and nonimpaired individuals. The second region—a portion of the left occipito-temporal extrastriate visual system typically centered on or near the middle portion of the fusiform gyrus—has been associated with the automatic process of visual word form perception in skilled adult readers. Responsiveness of this region reflects a form of perceptual expertise which normally develops over the course of learning to read, but which has also been shown to develop differently in dyslexic individuals. We suggest below that these two regions interact during the typical development of reading skills. Under this proposal, regions associated with phonology in the preliterate child impact the functional specialization of the left fusiform regions during the first several years of reading development, and the typical development of rapid and automatic word recognition ability is therefore disrupted in children with phonological processing deficits. Finally, it is possible that atypical patterns of activity in these regions might be altered via interventions that stress particular strategic approaches in word recognition processes and provide extensive practice. We shall discuss each of these issues in turn.

CONTRIBUTION OF LEFT PERISYLVIAN REGIONS

Functional Neuroimaging

Given the cognitive evidence that core deficits in phonological processing form a causal pathway to reading impairment, the use of functional neuroimaging to investigate the biological bases of phonological processing in vivo has proven to be a fruitful step in elucidating an associated neurobiological phenotype. The predominant strategy has been to develop cognitive tasks that engage phonological processing, and to then image the brain structures that are selectively activated during these tasks. Such tasks as rhyming and phoneme monitoring require the subject to manipulate the sound structure within syllables or words. Several investigators have used neuroimaging to link phonological processing with posterior perisylvian structures in normal readers [Price et al., 1997; Rumsey et al., 1997a, but see Pugh et al., 1996]. Extending this strategy to investigations of dyslexia, investigators have attempted to link cognitive impairment of phonological processes with dysfunction of left perisylvian structures, typically implicating the posterior superior temporal gyrus (STG) (see Fig. 1), and sometimes the angular gyrus and the insula.

This line of reasoning has resulted in a wide number of studies converging on the same conclusion: there is a reduced tendency of dyslexic individuals to recruit left perisylvian regions when faced with a phonologically challenging task. This common finding has been proposed as a candidate for a hallmark neurophysiological phenotype of developmental dyslexia [Shaywitz et al., 1998]. As demonstrated in Table 1 (entries marked with PS for perisylvian), many neuroimaging paradigms employing a wide variety of tasks provide evidence to support this central conclusion.

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For example, in a series of positron emission tomography (PET) studies, Rumsey and colleagues demonstrated that a reading pronunciation task led to increased activity in left STG in nonimpaired subjects [Rumsey et al., 1997a], but that dyslexic subjects showed no such increase [Rumsey et al., 1997b]. Shaywitz et al. [1998] further investigated this pattern with a hierarchical set of tasks that systematically increased phonological demands. As phonological demands increased, dyslexics exhibited a relative underactivation of posterior regions, including the left posterior superior temporal and angular gyri, as well as marginally reduced activity in the inferior lateral extrastriate area. Even the subtle phonological demands inherent in deciding whether the names of two letters rhyme produce differential recruitment of the STG region in dyslexics and controls [Paulesu et al., 1996].

Considerations for Interpreting Neuroimaging Results

Although the results summarized above produce strong converging evidence that posterior STG dysfunction underlies reading difficulties, interpretation of these neuroimaging findings should proceed with caution. One limitation commonly raised is that it is unclear whether brain activation patterns observed in adult dyslexics are the result of the original difficulties that were manifested during the early years of literacy acquisition, or rather reflect one of many potential long-term sequelae of years of reading difficulties. The use of functional magnetic resonance imaging (fMRI) with younger populations has recently permitted the investigation of these phenomena in children, and such studies have generally found results consistent with the adult literature (see Table 1, entries marked with Dev, for Development). For a description of this technique and its use in typically developing children, readers are referred to Davidson et al. [2003, this issue].

Another complication arises when considering the fact that in the majority of the above studies, dyslexic subjects generally performed more poorly on tasks than did controls, as evidenced by accuracy and reaction time measures. Perhaps the different degrees of activation reported in dyslexics and control subjects were not due to an underlying neurophysiological impairment per se, but rather, to a tendency for dyslexic subjects to fail to fully engage in tasks that are known to be challenging for them. Such concerns are tempered, however, by the fact that dyslexic individuals often produce greater activation in other brain regions, as compared to control subjects. Furthermore, the neurobiological phenotype described above has been demonstrated even in a group of dyslexics performing a letter rhyming task at levels of accuracy equal to those of control subjects [Paulesu et al., 1996].

An alternative possibility is that abnormal functional activity does not reflect fundamental processing limitations, but rather a strategic failure to engage this otherwise functioning region in the face of phonological challenges. In support of this, Paulesu et al. [1996] demonstrated equivalent recruitment of posterior STG in both dyslexics and controls performing a memory task, thus suggesting that both

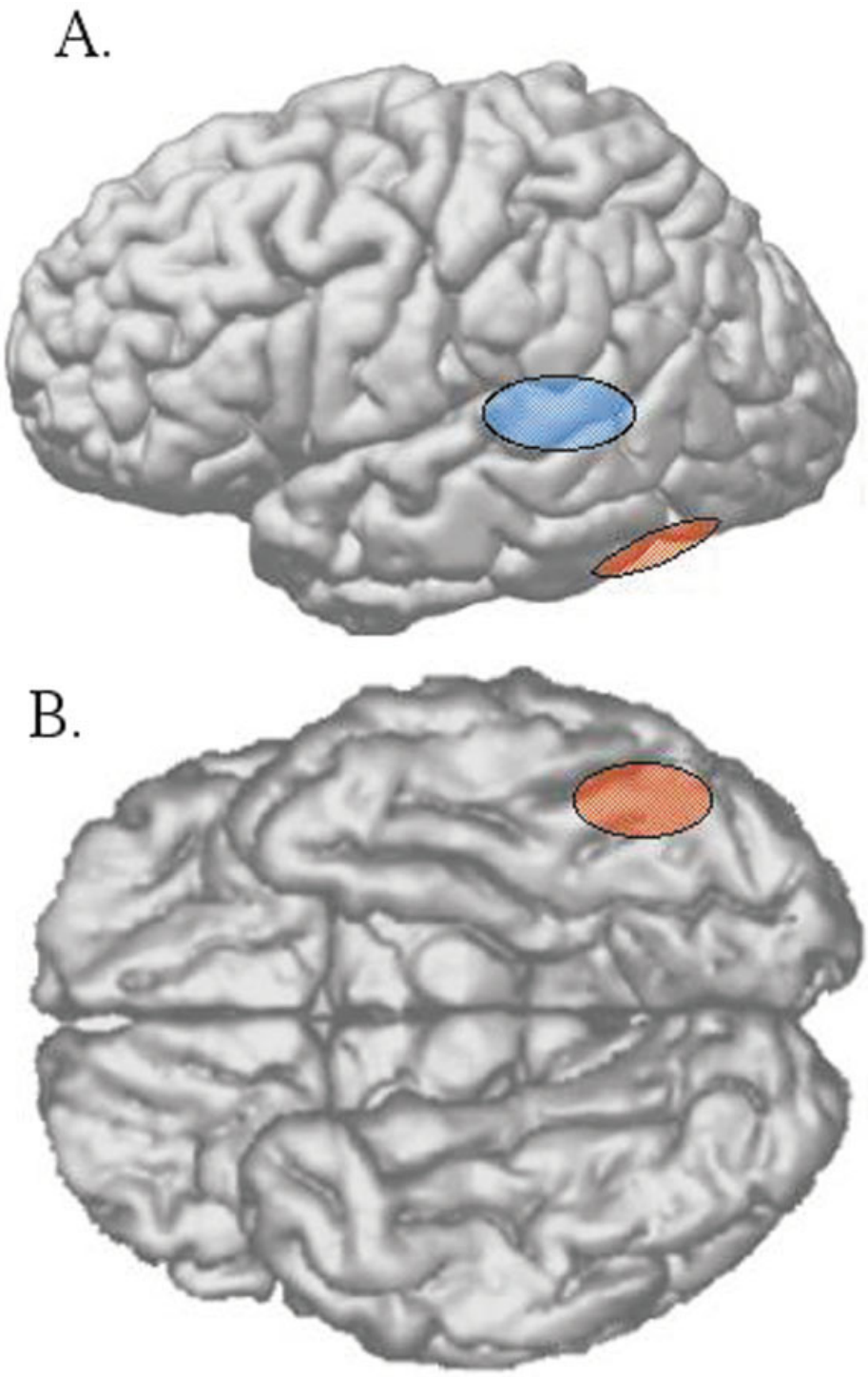

Fig. 1. (a) Lateral cortical surface of the left cerebral hemisphere. A region of the superior temporal gyrus (highlighted in blue) has been associated with phonological processes, and is implicated in dyslexia. (b) Ventral surface of the cerebral hemispheres. A region of the mid-fusiform gyrus (highlighted in red) has been associated with visual word form processing, and has also been implicated in dyslexia.

groups were able to equivalently activate this region when the demands were nonphonological.

Anatomical Contributions

Populations with reading difficulties have been found to exhibit anomalous anatomy of left perisylvian structures. Differences in white-matter tracts as well as gross morphological differences in the volume and symmetry of particular brain regions might be functionally linked to reading impairment. As reviewed by Watts [2003] in this issue Diffusion Tensor Imaging (DTI) has been shown to be sensitive to individual differences in white-matter tract properties. DTI is sensitive to properties of white matter microstructure by virtue of measuring the direction of the diffusion of water, which follows an anisotropic pattern that is greater in healthy, densely myelinated white matter tracts. DTI has recently been used to examine subtle white matter tract anomalies in dyslexia [Klingberg et al., 2000]. Reading impairments were associated with decreased an-

Table 1. Summary of Neuroimaging Studies Involved in Contrasts Between Dyslexic and Non-Impaired Control Subjects.*

Code	Authors	Subjects/Tasks	Methods	Findings
PS	Paulesu et al. (1996)	Adults/Letter-rhyming, phonological short-term memory	PET	Nonimpaired readers activated the insula and posterior STG during letter rhyming; dyslexics failed to activate these areas. However, both dyslexics and controls activated left STG during the phonological short term memory task, suggesting that dysfunction of this region is specific to phonological tasks.
PS, FG	Rumsey et al. (1997a, b)	Adult males/Psuedoword vs. real-word pronunciation	PET	Dyslexics showed decreased activations in left STG, bilateral middle and inferior temporal gyri and inferior parietal lobes, and the left fusiform. Also showed significantly increased activation of left insula relative to control subjects.
PS, FG	Horwitz et al. (1998)	Adults/Pseudoword reading	PET	In normal readers, large positive correlations found between angular gyrus and lingual and fusiform gyri, as well as left STG and left inferior frontal areas. In dyslexics, no significant correlation was seen between angular gyrus activity and that of any of the above regions
PS	Shaywitz et al. (1998)	Adults/case judgement, single letter rhyme, nonword rhyme	fMRI	Nonimpaired readers demonstrate significantly increased activation in left STG, angular gyrus, striate cortex, as phonological demands increase; dyslexics fail to show this pattern, and also demonstrate relative overactivation in inferior frontal gyrus
PS, FG	Paulesu et al. (2001)	Italian, French and English-speaking adults/reading words and nonwords	PET	Dyslexics from all countries showed significantly reduced activity in left STG, left middle and inferior temporal gyri, left and middle occipital gyrus in response to reading aloud.
PS, Dev	Simos et al. (2001)	7–17-year-old children/pseudoword rhyme-matching	MEG	Initially, dyslexic children showed decreased left STG activity relative to controls. Following a phonologically-based intervention, all dyslexic children showed a neural pattern of activity more similar to that of controls.
PS, Dev	Temple et al. (2001)	8–12-year-old children/Letter-rhyming; letter-identity matching.	fMRI	During letter rhyming, both normal and dyslexic children had activity in left frontal regions, whereas only nonimpaired children had activity in left temporoparietal cortex. During letter-matching, normal readers showed activity throughout extrastriate cortex, whereas dyslexic children showed little extrastriate activity.
PS, FG, Dev	Shaywitz et al. (2002)	7–18-year-old children/Same as above	fMRI	During nonword reading, nonimpaired readers demonstrated significantly greater activation compared to dyslexics in left STG, middle temporal gyrus, superior temporal sulcus, and inferior frontal gyrus. Performance on pseudoword rhyming was positively correlated with activation in left VWFA during nonword reading, across all readers.
FG	Brunswick et al. (1999)	Adults (childhood hist. of Dys. vs. controls)/explicit word & pseudoword reading; implicit feature detection task (words and pseudowords vs. false fonts)	fMRI	Explicit: Both groups showed increased left STG, frontal, and occipital areas. Dys. showed reduced VWFA, medial extrastriate, lingual areas. Implicit: Both groups showed increased left STG, and frontal, Dys. showed reduced activity in VWFA, mid temporal gyrus, and inferior parietal regions.
PS, FG	Salmelin et al. (1996)	Adults/passive viewing of words	MEG	Nonimpaired demonstrated a left occipito-temporal source within 200 msec; the dyslexics did not.
PS, FG	Helenius et al. (1999)	Same as above	MEG	Same as above; except refined analysis demonstrated both produced equivalent responses in early visual processing areas, but that dyslexics produced reduced or not in left occipito-temporal cortex during the first 200 msec.

PS marks converging patterns of evidence concerning perisylvian brain areas. FG marks converging patterns of evidence concerning the role of fusiform gyrus in visual word recognition. Dev indicates that subjects were children.

isotropy bilaterally in the white matter of temporoparietal regions. Further, the degree of anisotropy in a left temporoparietal region was highly correlated with reading skill, across both dyslexic and control subjects. Importantly, this finding has recently been replicated in a group of children and adolescents, demonstrating the same pattern of correlation between reading and anisotropy [Nagy et al. 2002].

Other studies have investigated gross anatomical structural differences in brain morphology as a potential biological phenotype for dyslexia (see Kennedy et al., [2003] in this issue for overview of MRI and its use in morphometry studies of development). Several early publications reported that dyslexia is marked by a lack of the normal asymmetry in planum temporale, which might serve as such a phenotypic marker [Galaburda et al., 1985; Rumsey et al., 1986]. However, a recent review [Eckert and Leonard, 2000] of over 20 such investigations argued that this general finding has not been consistently replicated, and that this literature is complicated by differences in measurement techniques, classification criteria, and potential confounds such as IQ and handedness.

Despite these complications, studies of brain morphology have produced

some useful conclusions concerning individual differences in reading ability. Studies that have opted to quantify phonological abilities on a continuum rather than to focus on categorical distinctions between dyslexics and nonimpaired readers have demonstrated robust correlations between such cognitive skills and asymmetries in perisylvian brain regions. For example, phonological ability and degree of asymmetry in the planum temporale are correlated both in normal and reading-impaired children, even when handedness, IQ, and socioeconomic status (SES) are taken into account [Eckert et al., 2001]. A similar correlation was reported by Habib and Robichon [1996] in a nearby perisylvian region closer to parietal areas. Taken together, these two studies support the notion that individual differences in perisylvian structures systematically translate into individual differences in function, a finding which has a close parallel in the DTI literature [Klingberg et al., 2000].

CONTRIBUTIONS OF THE LEFT FUSIFORM REGION

We next consider the contribution of the left fusiform gyrus region in the neural basis of dyslexia (see Fig. 1). In this case, however, understanding this region's role in the disorder is first contingent upon our understanding its role in typically developing reading function. A number of neuroimaging, electrophysiological and anatomical studies provide evidence that the later stages of skilled reading development are linked to a specialization of response properties of this portion of the extrastriate visual system. Skilled readers develop a form of visual expertise that allows them to automatically combine the letters of a word form into an integrated visual percept within approximately 200 milliseconds [for review see Rayner and Pollatsek, 1995]. This rapid visual word recognition ability is supported by a specialization of a region in the extrastriate visual system that slowly emerges over the first several years of reading experience [Posner and McCandliss, 1999]. This region is often referred to as the Visual Word Form Area, and is typically functionally defined as the extrastriate area that demonstrates an increase in response to word-like stimuli as compared to other control stimuli [McCandliss et al., 2003a].

Below, we consider several neurobiological lines of evidence that demonstrate a link between the functional specialization of this region and the cognitive ability of skilled adult readers to rapidly perceive visual word forms. In addition, we present evidence that this region responds to word stimuli differently in dyslexic readers. Finally, we consider the significance of this finding in terms of a developmental model of normal reading progression in which individual differences in phonological skill may impact the development of automatic word reading ability.

Functional Neuroimaging

Recent studies have produced strongly converging data supporting the claim that the perception of visual word forms reliably activates the left fusiform gyrus in a way that other stimuli that control for visual stimulation do not [Price et al., 1997; Puce et al., 1996; Beauregard et al., 1997; Wagner et al., 1998; Brunswick et al., 1999; Fiez et al., 1999; Kiehl et al., 1999; Paulesu et al., 2000; Xu et al., 2001]. Perhaps the most compelling evidence comes from studies that localize the functional properties of the Visual Word Form Area within individual subjects. Across four independent samples, the Visual Word Form Area has been discretely localized in approximately 90% of subjects via contrasts between words and other visual stimuli such as checkerboards, pseudo-letters, and random letter strings [Cohen et al., 2000, Polk and Farah, 2002; Dehaene et al., 2001; Cohen et al., 2002]. Across these studies, activation of the Visual Word Form Area tends to reliably cluster around similar Talairach coordinates (i.e., x = -43,y = -54, z = -12) with a standard deviation of approximately 5 mm.

In addition to precisely localizing a functional region, neuroimaging studies have helped to further characterize the response properties of the Visual Word Form Area, both in terms of the specific low-level characteristics of stimuli that drive the system, and the representations that characterize the level of processing associated with this region. Responsiveness of the Visual Word Form Area has been shown to be highly specific to visually presented alphabetic characters, which produce a greater response than either auditory words [Dehaene et al., 2002] or visually presented false font characters that control for low-level visual qualities [Price et al., 1997; Garrett et al., 2000]. The Visual Word Form Area is also sensitive to the organization of letters within a word form. Both familiar words and novel letter strings that follow the patterns of the writing system (i.e., pseudowords such as 'blard') typically produce greater responses than randomly ordered strings, or strings of consonants [McCandliss et al., 2003a]. Furthermore, responses in the Visual Word Form Area have also been demonstrated to be sensitive to the presentation of words even under subliminal presentation conditions which produce chance levels of stimulus detection, suggesting that this region can be activated even in the absence of conscious awareness [Dehaene et al., 2001].

One challenge in localizing the neural correlates of rapid visual word recognition involves localizing functionally specific neural activity in time as well as space. As mentioned above, skilled readers typically require less than a quarter of a second to form a stable representation of a visual word. Event related potentials (ERPs) recorded over posterior visual regions demonstrate sensitivity within 150–200 msec to differences between presentations of visual words and other stimuli that control for visual features [Bentin et al., 1999] including consonant strings [Compton et al., 1991; McCandliss et al., 1997]. Similar functional contrasts and time frames have been found using Magnetic Source Imaging [Tarkiainen et al., 1999; 2002; Salmelin et al., 1996; Helenius et al., 1999] and intracranial electrical recordings [Nobre, 1994], in regions consistent with the fMRI coordinates for the Visual Word Form Area. These studies provide further evidence that specialization within left extrastriate regions of the visual system supports rapid word recognition abilities in skilled readers.

Anatomical Contributions

Critical evidence establishing that the Visual Word Form Area is *necessary* for the perception of word forms rather than simply *associated* with reading comes from anatomical lesion analyses of a specific syndrome of functional loss known as *word form dyslexia, pure alexia*, or *letter-by-letter reading* [Warrington and Shallice, 1980]. This syndrome preserves some ability to spell, write, and recognize letters, but drastically impairs the ability to perceive the letters of a word in an integrated fashion, resulting in laborious attempts at reading, with reaction time proportional to the number of letters in the word. Binder and Mohr [1992] demonstrated that across a number of studies, damage restricted to the left ventro-temporal area leads to word form dyslexia. This result is consistent with the majority of cases of this syndrome in which damage is reported either to this region or to fibers that provide input to this region

[Beversdorf et al., 1997], although additional regions associated with this disorder have also been described [Dejerine, 1892].

LINKING PERISYLVIAN AND FUSIFORM CONTRIBUTIONS TO DYSLEXIA

Taking these lines of evidence together, activity in the Visual Word Form Area appears to reflect a form of perceptual expertise, akin to that seen in certain domains of object recognition [see Gauthier and Tarr, 2002, for review], in which extensive experience with a class of visual objects leads to specialized, domain-specific response properties of a fusiform area. This specialization in turn serves to enhance perception within that domain. In the case of visual words, this perceptual expertise appears to support the rapid integration of letters into a perceptual word-form. This raises questions about how visual experience with words leads to a reorganization of the functional properties of this region, as well as questions about how individual differences might impact the specialization of this region.

Since evidence links visual word form processing to left-mid fusiform areas, it stands to reason that dyslexic individuals, who typically demonstrate great difficulty in rapidly identifying visual words, may demonstrate an atypical response pattern in the Visual Word Form Area. A number of neuroimaging studies have demonstrated that, in contrast to nonimpaired readers, dyslexic adults typically under-activate this region during word reading tasks (See Table 1, items marked FG). For example, when a word is presented visually, dyslexics activate the Visual Word Form Area less than nonimpaired readers, both within tasks that explicitly require reading words, and also for tasks that do not require reading, but allow for implicit contrasts between presentation of words and false fonts [Brunswick et al., 1999]. Paulesu et al. [2001] demonstrated this same Visual Word Form Area contrast between dyslexics and nonimpaired readers across several writing systems, including both English, in which the letter-sound mapping is complex, and Italian, in which the relationship is much more direct. This cross-cultural study highlights an additional advantage of pursuing a biological phenotype rather than a cognitive phenotype. The authors point out that while the surface symptoms of dyslexia may take on very different forms in different writing systems, it is possible that groups of different symptoms share one underlying biologically-defined core deficit that can be studied in a similar way across cultures. Furthermore, both of the above studies demonstrated that the difference in activity between dyslexics and controls was more pronounced in the Visual Word Form Area than in the left STG regions discussed above (see Table 1). This suggests that, for reading tasks, the Visual Word Form Area might account for more variance in performance between dyslexic and nonimpaired groups.

Magnetoencephalography (MEG) studies have specifically contrasted the time-course of Visual Word Form Area activity in dyslexics and nonimpaired readers. While both groups demonstrate equivalent responses in early visual areas, only nonimpaired readers demonstrate an additional response approximately 150–200 msec after the onset of a visual word; this response is localized to left inferior temporal regions [Helenius et al., 1999]. Although both dyslexic and nonimpaired subjects demonstrated later sources associated with left superior temporal activity that peaked around 300 msec, these signals showed functional disruption in dyslexics. MEG studies thus suggest that during word recognition, the left fusiform region is actively involved in computations concerning the visual word form within the first 150 to 200 msec of processing, and that activity in left superior temporal regions occurs much later, perhaps associated with processing of phonological information.

Finally, a relatively new approach has examined how cortical areas interact differently in nonimpaired and dyslexic readers. Horwitz et al. [1998] reasoned that functional connectivity between two or more regions during performance of a given task should produce within-task, across-subject correlations in activity. They examined correlated areas of neural activity during the reading of exception words and pseudowords, in both dyslexics and control subjects. In nonimpaired subjects, left angular gyrus activity was strongly correlated with activity in the left posterior STG, the left inferior frontal gyrus (IFG), and extrastriate visual areas in the occipital and temporal cortices of the left hemisphere. Angular gyrus activity was also correlated with areas in the lingual and fusiform gyri. In dyslexics, however, there were no significant positive correlations between activation in the angular gyrus and STG, IFG, or the fusiform or lingual gyri. The authors thus conclude that dyslexia involves a functional disconnection between the angular gyrus and perisylvian and extrastriate temporo-occipital regions.

DEVELOPMENTAL CONSIDERATIONS

Several questions to be addressed by future research will involve understanding how the two functional-neurobiological hallmarks of dyslexia reviewed above—dysfunction of the perisylvian and fusiform regions—are linked during reading development. The development of perceptual expertise associated with the VWFA appears to progress quite gradually over several years of reading experience, and is still not at adult levels even after five years of reading experience [Gibson and Levin, 1975; Aghabanian and Nazir, 2000]. McCandliss and colleagues investigated the development of the VWFA by tracking the development of an N200 ERP component that, in adults, demonstrates a focus over posterior visual areas that differentiated familiar words from consonant strings [McCandliss et al., 1997]. In investigating the potential emergence of similar effects in four, seven, and ten-year-old children, it was revealed that only at age ten did a small and delayed N200 begin to demonstrate sensitivity to words over consonant strings [McCandliss et al., 1997; Posner and McCandliss, 1999].

How may we use neurobiology to link the gradual emergence of automatic reading to the well-established association with phonological processing? We propose that functional and structural abnormalities in the perisylvian regions that subserve phonological processing may have a cascading effect on the development of rapid word recognition processes during the years when the VWFA is becoming increasingly specialized to respond to regularities within the writing system. Little is presently known, however, about the mechanism by which functional properties of the perisylvian regions associated with phonology influence the development of visual expertise effects supported by the fusiform gyrus. One possibility, suggested by cognitive research on reading development, is that a child's *decoding skill*—which involves simultaneously attending to both letters and the sounds those letters make within a word—plays a fundamental role in establishing rapid word recognition ability [Perfetti, 1985; Share and Stanovich, 1995]. Share and Stanovich argue that

effortful decoding processes provide a way for automatic word reading skills to be progressively strengthened through a form of *self-teaching*. Decoding letters into sounds provides a child with a means to both generate plausible pronunciations for unfamiliar visual words, and to generate error-signals that allow successive approximations from decoding attempts to be stored as phonological patterns associated with known words. Interestingly, this form of error correction in self-teaching is conceptually parallel to the error-correction neural circuitry active in the self-practice phase of songbird learning [Wilbrecht and Nottebaum, 2003, this issue], perhaps suggesting that this research could provide a potential model for understanding self-teaching in reading.

The notion that decoding ability plays a causal role in the specialization of the VWFA provides a pathway through which disrupted function of perisylvian regions could have a potentially cascading influence on the specialization processes that occur in left fusiform areas during literacy acquisition. Phonological deficits have been directly linked to difficulties in grapheme-phoneme decoding [for review, see National Reading Panel, 2000]. This suggests a developmental sequence in which functional anomalies in left perisylvian regions may lead to childhood deficits in phonological processing skills that are crucial for grapheme-phoneme decoding. In normally developing children, successful application of decoding skill may then serve to focus attention on the regularities of grapheme-phoneme mapping in the writing system, making those regularities more salient; this attentional factor may serve a critical role in the gradual specialization of the response properties of the VWFA. Throughout the early years of reading, subtle differences in decoding ability may impact the gradual specialization of the VWFA that allows it to respond automatically to regularities with the writing system.

Shaywitz et al. [2002] provide support for this developmental relationship between decoding ability and the development of the VWFA. In a recent study involving over 140 dyslexic and nonimpaired children aged 7–18, standardized scores for decoding ability were significantly and positively correlated with the degree of VWFA activation in response to pseudowords. This brain-behavior relationship was present across the full range of scores including both dyslexic and nonimpaired children, and remained significant even when the effects of age were controlled. This correlation suggests a significant relationship between the degree to which a child might successfully engage in decoding and the degree to which his or her left fusiform gyrus becomes tuned via experience to become responsive to the orthographic structure of the writing system.

Finally, one implication raised by this cascading model of developmental reading disabilities is that multiple pathways leading to poor phonological decoding ability might have the same negative impact on the specialization of the VWFA. We have discussed how impairment in phonological skills may be rooted in neurobiological anomalies of the left posterior perisylvian region.

Several studies have provided strong support for the claim that children with mild to severe reading impairments can benefit significantly from intervention techniques that involve explicit training and support in phonological awareness training and alphabetic decoding skills.

However, environmental factors that lead to poor phonological awareness skills might have a similar cascading impact on the development of the VWFA. Although children from socioeconomically disadvantaged homes perform significantly worse on many measures of academic ability and achievement [Smith et al., 1997; Duncan et al., 1998; Haveman and Wolfe, 1995], measures of reading, language, and phonological skills are particularly influenced by differences in socioeconomic status (SES) [see Whitehurst, 1997; Lonigan et al., 1998]. These differences have been empirically tied to environmental factors, such as characteristics of home literacy environments [Payne et al., 1994]. Furthermore, differences in phonological skill associated with SES have been demonstrated to be orthogonal to at least one neurobiological influence on phonology, degree of asymmetry in planum temporale [Eckert et al., 2001]. Since these two factors demonstrate additive (i.e., noninteracting) effects on phonological skill, an intriguing possibility is raised. Perhaps a child's neural profile and environmental background may each account for unique variance in responsiveness to different phonologically-based interventions.

INTERVENTION STUDIES

Several studies have provided strong support for the claim that children with mild to severe reading impairments can benefit significantly from intervention techniques that involve explicit training and support in phonological awareness training and alphabetic decoding skills [Foorman et al., 1998; Vellutino et al., 1996; Torgesson et al., 2001]. Recently, the availability of noninvasive neuroimaging techniques have afforded the possibility of investigating the impact of such intervention techniques on functional neural activity, through measures collected over the course of the intervention. Such studies can address questions about the malleability of observed functional patterns of activity associated with dyslexia, and can also provide information concerning the nature of how an intervention achieves its effect. While it is possible that some intervention approaches may reverse the core deficits in abnormal patterns of functional activity, others may achieve their effects by recruiting compensatory mechanisms, or may have no measurable effect on underlying brain mechanisms.

One study used magnetic source imaging to demonstrate the typical neural profile described earlier, in which dyslexic children showed relatively decreased activation of the left posterior superior temporal gyrus during a phonologically challenging task, relative to nonimpaired children [Simos et al., 2002]. Children then received an intervention in which they were given 80 hours of one-on-one instruction in one of two commercial programs designed to increase awareness of phonological structure and provide extensive practice related to the alphabetic principle. Following intervention, all dyslexic children showed significant increases in reading skill, as well as increased activation in the left posterior STG, with additional in-

creases in activation noted in the left supramarginal and angular gyri. The neural profiles of nonimpaired children did not change over the course of the study.

In a similar intervention study, children with demonstrated reading impairments participated in an fMRI scanning session before and after 20 sessions of a decoding-skills intervention protocol [McCandliss et al., 2001]. The intervention was a highly scripted procedure designed to focus a child's attention on grapheme-phoneme relationships within written words by engaging the child in activities such as changing a single grapheme within a word to make a new word. This intervention produced gains in decoding skills equivalent to 1.2 grade levels of improvement [McCandliss et al., 2003b]. The activation task involved a simple one-back repetition monitoring task with familiar words and pseudowords. Similar to the results of Simos et al. [2002] reported above, regions of left superior temporal gyrus demonstrated significantly greater recruitment after the intervention. A similar within-subject control task involving nondecodable consonant strings demonstrated no such change over the course of the study, suggesting that the observed changes were specific to decodable letter strings.

These studies suggest that the phonological awareness and decoding intervention techniques employed had a direct impact on reversing one of the primary neurobiological hallmarks of developmental dyslexia-limited recruitment of left superior temporal gyrus in the face of a phonological challenge. This evidence represents the first important step in demonstrating the possibility for eliciting functional reorganization associated with targeted interventions.

Additional research is needed, however, to identify the cognitive and neurobiological principles through which such interventions can produce rapid change in reading-related skills and associated patterns of cortical activity. For example, McCandliss et al. [2003b] suggested that one mechanism involved in the intervention's success rests on the use of single grapheme changes to focus a child's attention on individual graphemes within a word form. This leads to testable predictions, both in the form of research designed to isolate the 'critical ingredients' that serve as effective components of the intervention, as well as in the form of neurobiological research to investigate whether focusing attention on individual graphemes can drive changes in cortical reorganization.

Furthermore, empirical and theoretical developments are needed to create neurobiological models that can account for both the nature of the underlying disorder and the mechanisms through which intervention-based changes occur. One promising example of new research initiatives in this direction is offered by Harm and Seidenberg [1999], who study the development of reading ability in computational models created with different gradations of phonological impairment. Such models have demonstrated that subtle impairments in phonology can lead to reading development problems that parallel most of the symptoms of developmental dyslexia, and can also be used to investigate principles by which particular intervention protocols can lead to generalizable changes in performance [Harm et al., 2003].

Another promising line of research capitalizes on the significant heritability of reading impairment [DeFries et al., 1996]. As reviewed in this issue by Fossella et al. [2003], human genetics research has also made significant contributions toward understanding the biological bases of developmental disorders, by associating well specified cognitive phenotypes with genotypic information. The development of specific cognitive phenotypes has allowed research to isolate separable heritability patterns and genetic linkages for phonological awareness and decoding and orthographic skill [see Olson 2002, for a review]. Future work is needed to elucidate whether the genetic profiles underlying these separable cognitive phenotypes are associated with functional or structural differences in distinct brain regions.

In sum, the various methods of developmental cognitive neuroscience reviewed in this special issue have produced converging results that can be synthesized into an account of the biological bases of developmental dyslexia. In this selective review, we have focused on the two cortical regions that have produced the most promising pattern of evidence linking cortical activation and cognitive function in typically developing and dyslexic readers. Despite a large degree of convergence among methods, we argue that in order to fully understand the functional organization of these regions and the nature of their interactions in dyslexia, we must adopt a developmental perspective. We propose a simple descriptive model of one possible interaction across development. We believe, however, that a more complete theoretical treatment of the neurobiological basis of reading ability and disorder will require an account of the mechanisms by which the functional properties of these brain areas change with learning, development, and intervention.

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