Management of Dyslexia, Its Rationale, and Underlying Neurobiology

Sally E. Shaywitz, MD a,b,*, Jeffrey R. Gruen, MD c, Bennett A. Shaywitz, MD a,b

Department of Pediatrics, Division of Child Neurology, Yale University School of Medicine, PO Box 333, New Haven, CT 06510–8064, USA

Yale Center for the Study of Learning, Reading, and Attention, Yale University School of Medicine, PO Box 333, New Haven, CT 06510–8064, USA

Department of Pediatrics, Division of Neonatology, Yale University School of Medicine, PO Box 333, New Haven, CT 06510–8064, USA

Developmental dyslexia is characterized by an unexpected difficulty in reading in children and adults who otherwise possess the intelligence and motivation considered necessary for accurate and fluent reading [1–5]. Dyslexia (or specific reading disability) is the most common and most carefully studied of the learning disabilities, affecting 80% of all individuals identified as learning disabled. Although in the past the diagnosis and implications of dyslexia were often uncertain, recent advances in the knowledge of the epidemiology, the neurobiology, the genetics, and the cognitive influences on the disorder now allow the disorder to be approached within the framework of a traditional medical model. This article reviews these advances and their implications for the approach to patients presenting with a possible reading disability.

Epidemiology

Epidemiologic data indicate that, like hypertension and obesity, dyslexia occurs in gradations and fits a dimensional model. Within the population,
reading ability and reading disability occur along a continuum, with reading disability representing the lower tail of a normal distribution of reading ability \[6,7\]. Dyslexia is perhaps the most common neurobehavioral disorder affecting children, with prevalence rates ranging from 5% to 17.5% \[2,8\]. Although some may question whether so many children are struggling to read, data from the 2005 National Assessment of Educational Progress \[9\] indicate that only 31% of fourth graders are performing at or above proficient levels. Dyslexia does not resolve over time. Longitudinal studies, both prospective \[10,11\] and retrospective \[12–14\], indicate that dyslexia is a persistent, chronic condition; it does not represent a transient developmental lag (Fig. 1). Over time, poor readers and good readers tend to maintain their relative positions along the spectrum of reading ability \[10,15\]; children who early on function at the tenth percentile for reading and those who function at the 90% percentile and all those in-between tend to maintain their positions.

**Etiology**

Dyslexia is both familial and heritable \[16\]. Family history is one of the most important risk factors, with 23% to as much as 65% of children who have a parent with dyslexia reported to have the disorder \[14\]. A rate among siblings of affected persons of approximately 40% and among parents ranging from 27% to 49% \[16\] provides opportunities for early identification of affected siblings and often for delayed but helpful identification of affected adults, such as a parent of the child known to be dyslexic. Despite the strong familial nature, within a single family both recessive and dominant transmission is frequently observed. These data are consistent with a complex etiology; studies of heritability show that between 44% and 75% of the variance is explained by genetic factors and the remaining by environmental factors \[17\].

These genetic factors are sequence variations of several genes (ie, polygenic) that act in concert to produce the dyslexia phenotype, and because of the polygenic nature, create confusing transmission patterns that do not follow traditional mendelian rules governing recessive, dominant, or sex-linked single-gene disorders. Regardless of these complexities, genetic linkage studies, enabled in large part by the achievements of the Human Genome Project, have identified broad locations on human chromosomes, called loci, where dyslexia genes are encoded. To date, a total of nine loci have been identified, named “DYX1” through “DYX9” for the order in which they were recognized, and have been cataloged as official “DYX” loci in the Online Mammalian Inheritance in Man database.

Of the nine described dyslexia loci, the most widely reproduced has been DYX2 located on the “p” or short arm of chromosome 6 in band “22” (6p22), spanning nearly 20 million bases. Recently, the authors reported association of the DCDC2 gene encoded on 6p22 with several reading-related
phenotypes, suggesting a specific effect on reading performance [18]. Further, in human brain, DCDC2 expression correlated with the location of reading-related brain systems (see later), and it was found that DCDC2 in rats modulated neuronal migration. The association between DCDC2 and dyslexia was subsequently and independently confirmed by Schumacher and colleagues [19] in a two-tiered study of 137 and 239 families with dyslexia from Germany, thereby validating the findings and the universality of the genetic effect across languages and cultures.

Other candidate genes for dyslexia have been described. Encoded just 500,000 bases away from DCDC2, Cope and colleagues [20] described a second candidate gene for DYX2, called KIAA0319, which in 143 families from the United Kingdom also contributed to dyslexia. Two other candidates, EKN1 (DYX1) and ROBO1 (DYX5), were identified by cloning rare translocation breakpoints in single families from Finland, but validation in additional populations would make for more convincing evidence [21,22]. Gene discovery for all the dyslexia loci remains an active area of study.

Cognitive influences

Among investigators in the field, there is now a strong consensus supporting the phonologic theory. This theory recognizes that speech is natural and
inherent, whereas reading is acquired and must be taught. To read, the beginning reader must recognize that the letters and letter strings (the orthography) represent the sounds of spoken language. To read, a child has to develop the insight that spoken words can be pulled apart into the elemental particles of speech (phonemes) and that the letters in a written word represent these sounds [3]; such awareness is largely missing in dyslexic children and adults [3,12,23–27]. Results from large and well-studied populations with reading disability confirm that in young school-age children [23,28] and in adolescents [29], a deficit in phonology represents the most robust and specific correlate of reading disability [30,31]. Such findings form the basis for the most successful and evidence-based interventions designed to improve reading [32].

**Neurobiologic studies of disabled readers**

Neural systems influencing reading were first proposed over a century ago by Dejerine [33] in studies of adults who suffered a stroke with subsequent acquired alexia, the sudden loss of the ability to read. It has only been within the last two decades that neuroscientists have been able to determine the neural systems that influence reading and reading disability. This explosion in understanding the neural bases of reading and dyslexia has been driven by the development of functional neuroimaging, techniques that measure changes in metabolic activity and blood flow in specific brain regions while subjects are engaged in cognitive tasks. These technologies include positron emission tomography and functional MRI; both depend on the principle of autoregulation of cerebral blood flow. Details of functional MRI are reviewed elsewhere [34–36].

A number of research groups have used positron emission tomography or functional MRI to examine the functional organization of the brain for reading in nonimpaired and dyslexic readers, and generally have validated these two left hemisphere posterior systems as critical to reading. For example, in studies of adults [37] and in a study of 144 children, half of whom were struggling readers and half nonimpaired readers, the authors [38] found significant differences in brain activation patterns during phonologic analysis between dyslexic and nonimpaired children. Specifically, nonimpaired children demonstrate significantly greater activation than dyslexic children in predominantly left hemisphere sites (including the inferior frontal, superior temporal, parietotemporal, and middle temporal–middle occipital gyri). These data converge with reports that show a failure of left hemisphere posterior brain systems to function properly during reading and indicate that dysfunction in left hemisphere posterior reading circuits is already present in dyslexic children and cannot be ascribed simply to a lifetime of poor reading [5,39]. Although dyslexic readers exhibit a dysfunction in posterior reading systems, they seem to develop compensatory systems.
involving areas around the inferior frontal gyrus in both hemispheres and the right hemisphere homolog of the left occipitotemporal word form area [38].

These studies indicate that in addition to the posterior systems, an anterior system is also involved in reading. The anterior network in the inferior frontal gyrus (Broca’s area) has long been associated with articulation and also serves an important function in silent reading and naming [35,40]. The two posterior regions seem to parallel the two systems proposed by Logan [41,42] as critical in the development of skilled, automatic reading. One system involves word analysis; operates on individual units of words, such as phonemes; requires attentional resources; and processes information relatively slowly. It is reasonable to propose that this system involves the parietotemporal posterior reading network. Considerable research in the last 5 years has converged to indicate that the second posterior network, localized to a region termed the “visual word-form area” [43], influences skilled, fluent reading. Dehaene and associates [44–46] have suggested a systematic sensitivity to coding within the left occipitotemporal region, with more posterior regions coding for letters and letter fragments and more anterior regions coding for bigrams and words. Furthermore, recent evidence indicates that the disruption in the left occipitotemporal word form area in dyslexic individuals is found not only for reading words, but for naming the pictures of the words, suggesting that the disruption in this region “reflects a more general impairment in retrieving phonology from visual input. In other words, reduced activation in the same occipitotemporal region may underlie the reading and naming deficits observed in developmental dyslexia” (Fig. 2) [47].

Functional MRI has been helpful in clarifying potentially different types of reading disability [3]. The authors used data from the Connecticut Longitudinal Study, a representative sample of now young adults who have been prospectively followed since 1983 when they were age 5 years and who have had their reading performance assessed yearly throughout their primary and secondary schooling. Three groups were identified and imaged: (1) nonimpaired readers who had no evidence of reading problems; (2) accuracy improved readers (AIR) who were inaccurate readers in third grade but by ninth grade had compensated to some degree so they were accurate (but not fluent); and (3) persistently poor readers (PPR) who were inaccurate readers in third grade and remained inaccurate and not fluent in ninth grade.

During real word reading, brain activation patterns in the two groups of disabled readers (AIR and PPR) diverged, with AIR demonstrating the typical disruption of posterior systems, but with PPR activating posterior systems, similar to that observed in nonimpaired readers, despite the significantly better reading performance in nonimpaired readers compared with PPR on every reading task administered. Evidence indicated that rather than decoding words, the PPR group was reading primarily by memory. Because it is a longitudinal study, data from the Connecticut
Longitudinal Study as early as kindergarten and first grade were available and indicated that the two groups of disabled readers (PPR and AIR) began school with comparable reading skills but with PPR compared with AIR having poorer cognitive, primarily verbal, ability and attending more disadvantaged schools.

These and other findings suggest that PPR may be doubly disadvantaged in being exposed to a less rich language environment at home and then less effective reading instruction at school. In contrast, the presence of compensatory factors, such as stronger verbal ability and exposure to a richer language environment at home, allowed the AIR to minimize, in part, the consequences of their phonologic deficit so that as adults AIR were indistinguishable from nonimpaired readers on a measure of reading comprehension.

These findings of differences, neurobiologically, cognitively, and educationally, suggest that the two types of reading disability observed in the Connecticut Longitudinal sample may represent different etiologies. The compensated group (AIR), with early higher verbal ability and a disruption in posterior systems during reading real words, may represent a primarily genetic type of reading disability; one can postulate that such children represent the classic dyslexic reader with an unexpected difficulty in reading. Alternatively, the persistent group (PPR), who score lower on verbal measures early on and who attend more disadvantaged schools, may have their reading difficulties influenced more by environmental factors. Other factors also

![Figure 2. Neural systems for reading. Three neural systems for reading are illustrated in this figure of the surface of the left hemisphere: an anterior system in the region of the inferior frontal gyrus (Broca’s area) believed to serve articulation and word analysis; two posterior systems, one in the parieto-temporal region believed to serve word analysis, and a second in the occipito-temporal region (termed the “word-form” area) and believed to serve for the rapid, automatic, fluent identification of words. (From Shaywitz S. Overcoming dyslexia: a new and complete science-based program for reading problems at any level. New York: Alfred Knopf; 2003. p. 78; with permission.)](image-url)
may be operating. Ongoing studies of genetic differences between these
groups may help confirm or refute this hypothesis.

Functional imaging has been helpful in examining whether the neural sys-
tems for reading are malleable and whether the disruption in these systems
in struggling readers can be modified by an effective reading intervention.
Compared with struggling readers who received other types of intervention,
children who received an experimental intervention not only improved their
reading but, compared with preintervention brain imaging, demonstrated
increased activation in the neural systems for reading [48]. Other investiga-
tors also have found that an effective reading intervention influences neural
systems in brain [5]. These data have important implications for public pol-
icy regarding teaching children to read: the provision of an evidence-based
reading intervention at an early age improves reading fluency and facilitates
the development of those neural systems that underlie skilled reading.

Diagnosis

At all ages, dyslexia is a clinical diagnosis. The clinician seeks to deter-
mine through history, observation, and psychometric assessment if there
are unexpected difficulties in reading (ie, difficulties in reading that are
unexpected for the person’s age, intelligence, or level of education or profes-
sional status), and associated linguistic problems at the level of phonologic
processing. There is no one single test score that is pathognomonic of dys-
lexia. As with any other medical diagnosis, the diagnosis of dyslexia should
reflect a thoughtful synthesis of all the available clinical data. Dyslexia is
distinguished from other disorders that may prominently feature reading
difficulties by the unique, circumscribed nature of the phonologic deficit,
one not intruding into other linguistic or cognitive domains.

In the preschool child, a history of language-delay or of not attending to
the sounds of words (trouble learning nursery rhymes or playing rhyming
games with words, confusing words that sound alike, mispronouncing
words), trouble learning to recognize the letters of the alphabet, along
with a positive family history represent important risk factors for dyslexia.
In the school-aged child, presenting complaints most commonly center
about school performance (“she’s not doing well in school”), and often par-
ents (and teachers) do not appreciate that the reason for this is a reading dif-
ficulty. A typical picture is that of a child who may have had a delay in
speaking, does not learn letters by kindergarten, has not begun to learn to
read by first grade, and has difficulty consistently sounding out words.
The child progressively falls behind, with teachers and parents puzzled as
to why such an intelligent child may have difficulty learning to read. The
reading difficulty is unexpected with respect to the child’s ability, age, or
grade. Even after acquiring decoding skills, the child generally remains
a slow reader. Bright dyslexic children may laboriously learn how to read
words accurately but do not become fluent readers (ie, they do not recognize words rapidly and automatically). Dysgraphia and spelling difficulties are often present, and accompanied by laborious notetaking. Self-esteem is frequently affected, particularly if the disorder has gone undetected for a long period of time (Table 1) [3].

In an accomplished adolescent or young adult, dyslexia is often reflected by slowness in reading or choppy reading aloud that is unexpected in relation to the level of education or professional status (eg, graduation from a competitive college or completion of medical school and a residency). In bright adolescents and young adults, a history of phonologically based reading difficulties, requirements for extra time on tests, and current slow and effortful reading (ie, signs of a lack of automaticity in reading), are the sine qua non of a diagnosis of dyslexia. At all ages, a history of difficulties getting to the basic sounds of spoken language, of laborious and slow reading and writing, of poor spelling, of requiring additional time in reading and in taking tests, provide indisputable evidence of a deficiency in phonologic processing, which in turn serves as the basis for, and the signature of, a reading disability.

Assessment of prereading and reading

Even before the time a child is expected to read, a child’s readiness to read may be assessed by measurement of the skills, especially phonologic, related to reading success. Following a predictable developmental pathway,

Table 1
Clues to dyslexia in the school-age child

<table>
<thead>
<tr>
<th>Problems in speaking</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Mispronunciation of long or complicated words</td>
</tr>
<tr>
<td>• Speech that is not fluent—pausing or hesitating often</td>
</tr>
<tr>
<td>• Use of imprecise language</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Problems in reading</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Very slow progress in acquiring reading skills</td>
</tr>
<tr>
<td>• The lack of a strategy to read new words</td>
</tr>
<tr>
<td>• Trouble reading unknown (new, unfamiliar) words sounded out</td>
</tr>
<tr>
<td>• The inability to read small function words, such as that, an, in</td>
</tr>
<tr>
<td>• Oral reading that is choppy and labored</td>
</tr>
<tr>
<td>• Disproportionately poor performance on multiple-choice tests</td>
</tr>
<tr>
<td>• The inability to finish tests on time</td>
</tr>
<tr>
<td>• Disastrous spelling</td>
</tr>
<tr>
<td>• Reading that is very slow and tiring</td>
</tr>
<tr>
<td>• Messy handwriting</td>
</tr>
<tr>
<td>• Extreme difficulty learning a foreign language</td>
</tr>
<tr>
<td>• History of reading, spelling, and foreign language problems in family members</td>
</tr>
</tbody>
</table>

children’s phonologic abilities can be evaluated beginning at about age 4 years. Mainly, such tests are centered on a child’s ability to focus, initially on syllables, and later on phonemes, the basic particles of spoken language. Initial tests typically ask what word rhymes with another or what spoken word begins (or ends) with the same sound as another. At more advanced levels, tests ask children to pronounce a spoken word after a sound is removed; for example, “can you say steak without the “t” sound (sake)” or “can you count the number of sounds you hear in man (three sounds: ‘mmmm’...‘aaaa’...‘nnn’).” In general, as a child develops, he or she gains the ability to notice and to manipulate smaller and smaller parts of spoken words. Tests of phonologic capabilities and reading readiness are becoming increasingly available; one such test is the Comprehensive Test of Phonological Processing in Reading, nationally standardized for age 5 through adult years [49]. In addition to phonology, knowledge of letter names and sounds are the strongest predictors of a child’s readiness to read. An appropriate battery of tests for the early recognition of reading problems includes tests of phonology, letter names and sounds, vocabulary, print conventions, and listening comprehension. Tests of reading are also useful because they allow comparison of a child’s reading skills with his or her peers at a time when one should be beginning to read [3]. It is important to note that tests of intelligence are relatively poor predictors of later reading difficulties or of response to reading interventions. The importance of such early assessments is that they can identify at-risk children early on so that these boys and girls can be provided with the highly effective, evidence-based reading interventions now available.

Reading is assessed by measuring decoding (accuracy); fluency; and comprehension. In the school-age child, one important element of the evaluation is how accurately the child can decode words (ie, read single words in isolation). This is measured with standardized tests of single real word and pseudoword reading, such as the Woodcock-Johnson III [50] and the Woodcock Reading Mastery Tests [51]. Pseudoword reading, measuring the ability to decode nonsense or made-up words, is a particularly useful test. Because the words are made-up, the child has not seen them before and could not have memorized the words; each nonsense word must be sounded out. Tests of nonsense word reading are referred to as “word attack.” Silent reading comprehension may be assessed by either Woodcock test. Reading fluency, the ability to read accurately, rapidly, and with good intonation, is a critical but often overlooked component of reading. The ability to read words fluently is an indication that these words are read automatically, without the need to apply attentional resources. Fluency is generally assessed by asking the child to read aloud using the Gray Oral Reading Test [52]. This test consists of 13 increasingly difficult passages, each followed by five comprehension questions; scores for accuracy, rate, fluency, and comprehension are provided. Such tests of oral reading are particularly helpful in identifying a child who is dyslexic; by its nature oral reading forces a child to pronounce
each word. Listening to a struggling reader attempt to pronounce each word leaves no doubt about the child’s reading difficulty. In addition to reading passages aloud, single word reading efficiency may be assessed using the Test of Word Reading Efficiency, a test of speeded oral reading of individual words [53]. Children who struggle with reading often have trouble spelling. In reading, the written word is decoded into its constituent sounds; in spelling, sounds in a spoken word are encoded into letters. The Wide Range Achievement Test [54] and the Test of Written Spelling-4 [55] are among the tests that measure spelling.

For informal screening by primary care physicians in an office setting the authors recommend listening to the child read aloud from his or her own grade level reader. Keeping a set of graded readers available in the office serves the purpose and does not require the child to bring in their own school books. Oral reading is a very sensitive measure of not only reading accuracy, but even more importantly, reading fluency.

The most consistent and telling sign of a reading disability in an accomplished young adult is slow and laborious reading and writing. It must be emphasized that the failure either to recognize or to measure the lack of automaticity in reading is perhaps the most common error in the diagnosis of dyslexia in older children and in accomplished young adults. Simple word identification tasks do not detect a dyslexic accomplished enough to be in honors high school classes or to graduate from college and attend law, medical, or any other graduate degree school. Tests relying on the accuracy of word identification alone are inappropriate to use to diagnose dyslexia in accomplished young adults; tests of word identification reveal little to nothing of their struggles to read. It is important to recognize that, because they assess reading accuracy but not automaticity (speed), the kinds of reading tests commonly used for school-age children may provide misleading data on bright adolescents and young adults. The most critical tests are those that are timed; they are the most sensitive to a phonologic deficit in a bright adult. There are very few standardized tests for young adult readers, however, that are administered under timed and untimed conditions; the Nelson-Denny Reading Test represents an exception [56]. Any scores obtained on testing must be considered relative to peers with the same degree of education or professional training.

Developmental course and outcome

Deficits in phonologic coding continue to characterize dyslexic readers even in adolescence; performance on phonologic processing measures contributes most to discriminating dyslexic and average adolescent readers, and also average and superior adolescent readers [29]. Children with dyslexia neither spontaneously remit, nor do they demonstrate, a lag mechanism for “catching up” in the development of reading skills. Many dyslexic readers may become quite proficient in reading a finite domain of
words that recur over and over again in their area of special interest, usually words that are important for their careers. For example, an individual who is dyslexic in childhood but who in adult life becomes interested in molecular biology may then learn to decode words that form a minivocabulary important in molecular biology. Such an individual, although able to decode words in this domain, still exhibits evidence of early reading problems when they have to read unfamiliar words, which they then read accurately but not fluently and automatically [12,29,57–59]. Because they are able to read words accurately (albeit very slowly), dyslexic adolescents and young adults may mistakenly be assumed to have “outgrown” their dyslexia. Data from studies of children with dyslexia who have been followed prospectively support the notion that in adolescents, the rate of reading and facility with spelling may be most useful clinically in differentiating average from poor readers in students in secondary school, and college and even graduate school. It is important to remember that these older dyslexic students may be similar to their unimpaired peers on untimed measures of word recognition, yet continue to suffer from the phonologic deficit that makes reading less automatic, more effortful, and slow. For these readers with dyslexia the provision of extra time is an essential accommodation; it allows them the time to decode each word and to apply their unimpaired higher-order cognitive and linguistic skills to the surrounding context to get at the meaning of words that they cannot entirely or rapidly decode. Other accommodations useful to adolescents with reading difficulties include note-takers; taping classroom lectures; using Recordings for the Blind to access texts and other books they have difficulty reading; and the opportunity to take tests in alternate formats, such as short essays [3].

Reading instruction and intervention

The management of dyslexia demands a life span perspective; early on, the focus is on remediation of the reading problem. As a child matures and enters the more time-demanding setting of secondary school, the emphasis shifts to incorporate the important role of providing accommodations. Effective intervention programs provide children with systematic instruction in each of five critical components of reading: (1) phonemic awareness (the ability to focus on and manipulate phonemes, speech sounds, in spoken syllables and words); (2) phonics (understanding how letters are linked to sounds to form letter-sound correspondences and spelling patterns); (3) fluency; (4) vocabulary; and (5) comprehension strategies. The goal is for children to develop the skills that allow them to read and understand the meaning of both familiar and unfamiliar words they may encounter. Large-scale studies to date have focused on younger children; as yet, there are few or no data available on the effect of these training programs on older children. The data on younger children are extremely
encouraging, indicating that using evidence-based methods can remediate, and may even prevent, reading difficulties in primary school-aged children [3,60,61].

An essential component of the management of dyslexia in students in secondary school, and especially college and graduate school, incorporates the provision of accommodations. High school and college students with a history of childhood dyslexia often present a paradoxical picture; they are similar to their unimpaired peers on measures of word recognition and comprehension, yet continue to suffer from the phonologic deficit that makes reading less automatic, more effortful, and slow. Neurobiologic data now provide strong evidence for the necessity of extra time for readers with dyslexia. Functional MRI data demonstrate a disruption in the word form area, the region supporting rapid reading. At the same time, readers compensate by developing anterior systems bilaterally and the right homolog of the left word form area. Such compensation allows for accurate reading, but does not support fluent or rapid reading [38]. Consequently, for these readers with dyslexia the provision of extra time is an essential accommodation; it allows them the time to decode each word and to apply their unimpaired higher-order cognitive and linguistic skills to the surrounding context to get at the meaning of words that they cannot entirely or rapidly decode. With such accommodations, many students with dyslexia are now successfully completing studies in a range of disciplines, including medicine.

People with dyslexia and their families frequently consult their physicians about unconventional approaches to the remediation of reading difficulties; in general, there are very few credible data to support the claims made for these treatments (eg, optometric training, medication for vestibular dysfunction, chiropractic manipulation, and dietary supplementation). Finally, pediatricians should be aware that there is no one “magical” program that remediates reading difficulties; a number of programs following the guidelines provided previously have proved to be highly effective in teaching struggling children to read.

Summary

Within the last two decades overwhelming evidence from many laboratories has converged to indicate the cognitive basis for dyslexia: dyslexia represents a disorder within the language system and more specifically within a particular subcomponent of that system, phonologic processing. Recent advances in imaging technology and the development of tasks that sharply isolate the subcomponent processes of reading now allow the localization of phonologic processing in brain, and as a result provide for the first time the potential for elucidating a biologic signature for reading and reading disability. Converging evidence from a number of laboratories using functional brain imaging indicates a disruption of left hemisphere posterior brain
systems in child and adult dyslexic readers while performing reading tasks with an additional suggestion for an associated increased reliance on ancillary systems (e.g., in the frontal lobes and right hemisphere posterior circuits). The discovery of neural systems serving reading has significant implications. At the most fundamental level, it is now possible to investigate specific hypotheses regarding the neural substrate of dyslexia, and to verify, reject, or modify suggested cognitive models. From a more clinical perspective, the identification of neural systems for reading has implications for the acceptance of dyslexia as a valid disorder, a necessary condition for its identification and treatment. They provide, for the first time, convincing, irrefutable evidence that what has been considered a hidden disability is real. Such findings should make policy makers more willing to allow children and adolescents with dyslexia to receive accommodations on high stakes tests, such accommodations as extra time, which allow dyslexic readers with a disruption in the word form area influencing skilled, fluent reading, to be on a level playing field with their peers who do not have a reading disability.

References


