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John D. E. Gabrieli

Reading is essential in modern societies, but many children have dyslexia, a difficulty in learning to read. Dyslexia often arises from impaired phonological awareness, the auditory analysis of spoken language that relates the sounds of language to print. Behavioral remediation, especially at a young age, is effective for many, but not all, children. Neuroimaging in children with dyslexia has revealed reduced engagement of the left temporo-parietal cortex for phonological processing of print, altered white-matter connectivity, and functional plasticity associated with effective intervention. Behavioral and brain measures identify infants and young children at risk for dyslexia, and preventive intervention is often effective. A combination of evidence-based teaching practices and cognitive neuroscience measures could prevent dyslexia from occurring in the majority of children who would otherwise develop dyslexia.

The nexus of educational policies, evidence-based teaching practices, and cognitive neuroscience promises to use cutting-edge scientific methods and concepts to promote the growth and success of children. Reading is a focal point in this new synthesis because it is the most important portal to knowledge in our information age, from books to blackboards to the Internet. Learning to read is, however, perilous for the 5 to 17% of children who have developmental dyslexia, a persistent difficulty in learning to read that is not explained by sensory deficits, cognitive deficits, lack of motivation, or lack of adequate reading instruction (*1*).

Here, I provide an overview of research about the cognitive and brain bases of dyslexia, its treatment and brain plasticity associated with successful treatment, and how neuroscience may interact with education to help children with dyslexia. Particularly promising is the possibility that early identification of risk for dyslexia, through combined behavioral and neuroscience measures, may allow for preventive treatment such that many children with dyslexia who would otherwise fail to read would, instead, succeed at reading.

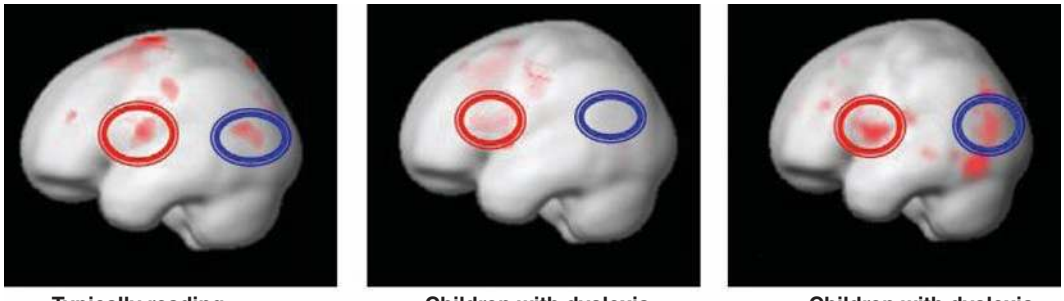

Fig. 1. Brain activation differences in dyslexia and its treatment [from (*36*)]. Functional magnetic resonance imaging activations shown on the left hemisphere for phonological processing in typically developing readers (**left**), age-matched dyslexic readers (**middle**), and the difference before and after remediation in the same dyslexic readers (**right**). Red circles identify the frontal region, and blue circles identify the temporo-parietal region of the brain. Both regions are hypoactivated in dyslexia and become more activated after remediation.

What Is Dyslexia and What Causes Dyslexia?

Definition of dyslexia. Most children have reading difficulties for three broad reasons: (i) dyslexia, which is characterized by a difficulty in understanding and using alphabetic or logographic principles to acquire accurate and fluent reading skills, (ii) reduced vocabulary and strategies needed for text comprehension, and (iii) reduced motivation to read. The latter reasons for reading failure often involve socioeconomic factors, at home and at school, that are beyond the scope of this review.

An initial difficulty in learning to read has wide and prolonged consequences. Difficulty in reading discourages children with dyslexia to practice their reading outside of the classroom, and lack of practice alone can impede the growth of reading skill and the acquisition of vocabulary and world knowledge (*2*). There are massive reading practice differences between good and poor readers: Outside of school in 5th grade, a good reader may read as many words in 2 days as a poor reader does in an entire year. Dyslexia is persistent: A student who fails to read adequately in 1st grade has a 90% probability of reading poorly in 4th grade and a 75% probability of reading poorly in high school. Thus, difficulty in early reading limits reading comprehension in the later years of education, as students shift from learning to read to reading to learn.

Dyslexia appears on a continuum with typical reading ability because specific psychological, neural, and genetic features of dyslexia also correlate with reading performance in a broad range of children. On one hand, this means that dyslexia may be understood in terms of normative psychological and computational models of reading and that discoveries about dyslexia may offer insights into mechanisms of normal reading acquisition (*3*, *4*). On the other hand, education and research findings depend on what behavioral boundary or criteria is selected to operationally define dyslexia. Dyslexia is often defined by a discrepancy between an average or above-average score on a test of general intelligence [intelligence quotient (IQ) test] and a low score on a standardized reading test. The core mechanism of dyslexia, however, appears to be similar in dyslexic readers, regardless of IQ over a broad range of IQ scores such that that children with low reading and IQ scores benefit from the same treatments as children with discrepant scores (*5*). These findings are consistent with the observation that dyslexia is independent of other talents that allow some children with dyslexia to grow into remarkably successful adults.

Dyslexia is strongly (54 to 75%) heritable, occurring in up to 68% of identical twins and 50% of individuals who have a parent or sibling with dyslexia (*6*). Environmental factors are also important in reading development, even in children at genetic risk for dyslexia. For example, heritability is greater among children whose parents have a higher educational level (*7*). This suggests that genetic risk factors account for more variance in highly supportive environments, but less so in environments that vary widely in support for reading. Identified candidate risk genes (*8*) are implicated in neural migration and brain development, which suggests that dyslexia may

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be a consequence of atypical neural migration in the developing brain.

Psychological bases of dyslexia. The causes of dyslexia can be considered at multiple levels of analysis and probably reflect multiple interacting mechanisms that vary across children. Historically, dyslexia was termed "word blindness"; however, the most common psychological cause of dyslexia for English speakers is a deficit in auditory processing of the sounds of language (phonological processing) (*9*). The diagnosis of dyslexia in the United States is commonly made in children ages 7 to 8 years old when reading difficulty is clearly measurable, although there is consensus that the roots of dyslexia begin before initial reading instruction, around 6 years of age (1st grade).

Beginning readers must decode print to access the identity and meaning of words. They already know the meanings of words in spoken language, but they have to learn to relate language to print through explicit phonological awareness that spoken words are composed of discrete sounds (phonemes) that can be mapped onto letters or syllables (graphemes). Children with dyslexia frequently exhibit poor phonological awareness, initially for spoken words and subsequently for printed words. These children have difficulty performing oral tasks that depend on phonological awareness, such as deciding which words start with the same sound as "hat"—"bat," "hot," or "sun," segmenting words into parts (knowing that "hat" is composed of "h," "a," and "t" sounds, or that those separate sounds can be blended into "hat"), and selectively deleting a sound within a word [what word remains if you take the "l" sound out of "clap" ("cap")]. For older children who can read, phonological impairment is most evident when asked to read aloud nonsense words ("twale") that are unknown and can only be pronounced or decoded on the basis of grapheme-to-phoneme mapping principles. These problems in phonological processing result in inaccurate recognition of words.

The expression of phonological difficulty in dyslexia varies as a consequence of differences in written languages (orthographies) (*10*). In alphabetic languages, such as English and Spanish, letters correspond to speech sounds, whereas in logographic languages, such as Chinese, characters correspond to meanings (morphemes). Alphabetic languages vary in their regularity (how consistently letters or letter clusters relate to one speech sound). Spanish and Italian are far more regular than English. Cross-cultural studies have shown that learning to read single words (grapheme-phoneme decoding) takes longer in less consistent orthographies. Current research suggests that across languages there are similar rates of dyslexia and that weakness in phonological processing is the most common etiology of dyslexia, but that the precise relation of phonological processing to reading and to the expression of dyslexia may vary across orthographies.

The second major problem for many children with dyslexia involves fluent reading of text. Even children who improve their accuracy for reading single words often continue to read text laboriously and slowly; the effort expended to read words in text often detracts from their ability to construct the meaning of what they are reading. This dysfluency may reflect a slowness evident even for naming a series of objects or colors. Children who have difficulties in both phonology and speed are described as having a double deficit (*11*). The dysfluency may also reflect difficulties in making up for the enormous amount of reading practice that these students miss out on when they remain poor readers in middle or late elementary grades (*12*). Much less is understood about the fluency deficit than the phonological deficit in dyslexia, but the fluency deficit is problematic for older children who must read increasingly sophisticated texts.

Scientists have been interested in discovering whether broader perceptual deficits precede reading deficits in dyslexia. Perhaps because these perceptual processes are less directly measurable in relation to reading and may exert their influences early in language development, there is debate about their precise role in dyslexia. The rapid temporal processing hypothesis derives from studies of children with "specific language impairment," a developmental language disorder estimated to occur in 7% of preschool children; these children have a difficulty in phonological awareness and/or morphosyntax, and they often progress to having dyslexia (*13*). Many of these children perform poorly at identifying the order of rapidly presented tones (*14*), and it is hypothesized that a broad auditory temporal processing deficit compromises accurate discrimination of language sounds that depends on very brief differences in auditory inputs (e.g., "b" and "d" differ by 50 msec or less of auditory information). The "magnocellular hypothesis" (*15*) is motivated by postmortem evidence in dyslexia for reduced area of the magnocellular layers of the lateral geniculate nucleus of the thalamus (*16*), which is part of the pathway mediating transient visual percepts such as motion. Individuals with dyslexia have exhibited subtle deficits in processing rapidly changing visual nonverbal information (e.g., gratings) and correlations between degrees of such visual impairment and reading difficulty (*17*). Other researchers report that children with dyslexia have, instead, a perceptual deficit in the exclusion of visual or auditory noise (*18*, *19*) or deficient stimulus-specific adaptation mechanisms (*20*). Conflicting reports on the presence or relation of these perceptual deficits to dyslexia raise the possibility that the relation between broader perceptual difficulties and reading difficulty may vary across children with dyslexia.

Brain basis of dyslexia. Functional neuroimaging studies have revealed differences in brain function and connectivity that are characteristic of dyslexia. Specific patterns of atypical brain activation in dyslexia relate to the specific reading or language processes examined in a neuroimaging study. When performing tasks that demand phonological awareness for print, such as deciding whether or not letters, words, or pseudoword letter strings rhyme, typically developing child and adult readers recruit several brain regions, including the left temporo-parietal cortex. In contrast, children and adults with dyslexia exhibit reduced or absent activation in this region (Fig. 1) (*21*–*23*). Hypoactivation of the left temporo-parietal cortex is evident when dyslexic children are compared with typically developing readers who are three years younger and reading at the same level as the dyslexic children (*24*). Therefore, left temporo-parietal hypoactivation appears to be related to the etiology of dyslexia per se, rather than delayed maturation or reading level. It is hypothesized that this left temporo-parietal region supports the cross-modal relation of auditory and visual processes during reading. Atypical activations in dyslexia are also found in the left prefrontal regions associated with verbal working memory [in some cases related to reading ability rather than dyslexia (*24*)], left middle and superior temporal gyri associated with receptive language, and left occipito-temporal regions associated with visual analysis of letters and words.

Functional neuroimaging studies have also examined cultural and perceptual influences on dyslexia. Adults with dyslexia in French, Italian, and English exhibit similar hypoactivation in the left temporal cortex (*25*). Chinese readers with dyslexia exhibit atypical activation in the left prefrontal cortex, but not in the left temporo-parietal regions that are commonly atypical in dyslexic individuals reading alphabetic languages (*26*). Dyslexic children do not show activation during the incidental auditory perception of rapidly (relative to slowly) changing non-speech stimuli that is shown in the left prefrontal cortex by typically developing children, but dyslexic children do show increased activation after remediation with a computer-based program focused on improving rapid auditory processing (*27*). There is also reduced or absent activation in individuals with dyslexia in response to gratings designed to preferentially stimulate the magnocellular pathway in visual cortices (*28*, *29*). Further, reading ability correlates with individual differences in activation in response to these nonverbal visual stimuli (*29*). Also, contrast responsivity to nonverbal stimuli in the motion-sensitive visual cortex correlates with behavioral measures of phonological awareness in children with a wide range of reading skills (*30*).

White-matter pathways of the brain may be characterized by diffusion tensor imaging (DTI), which provides a quantitative index of the organization of large myelinated axons constituting the long-range connections of brain networks. White-matter organization appears to be weaker in the left posterior brain region of people with dyslexia than is typical (*31*), and this measure of organization correlates positively with reading scores among both typical and

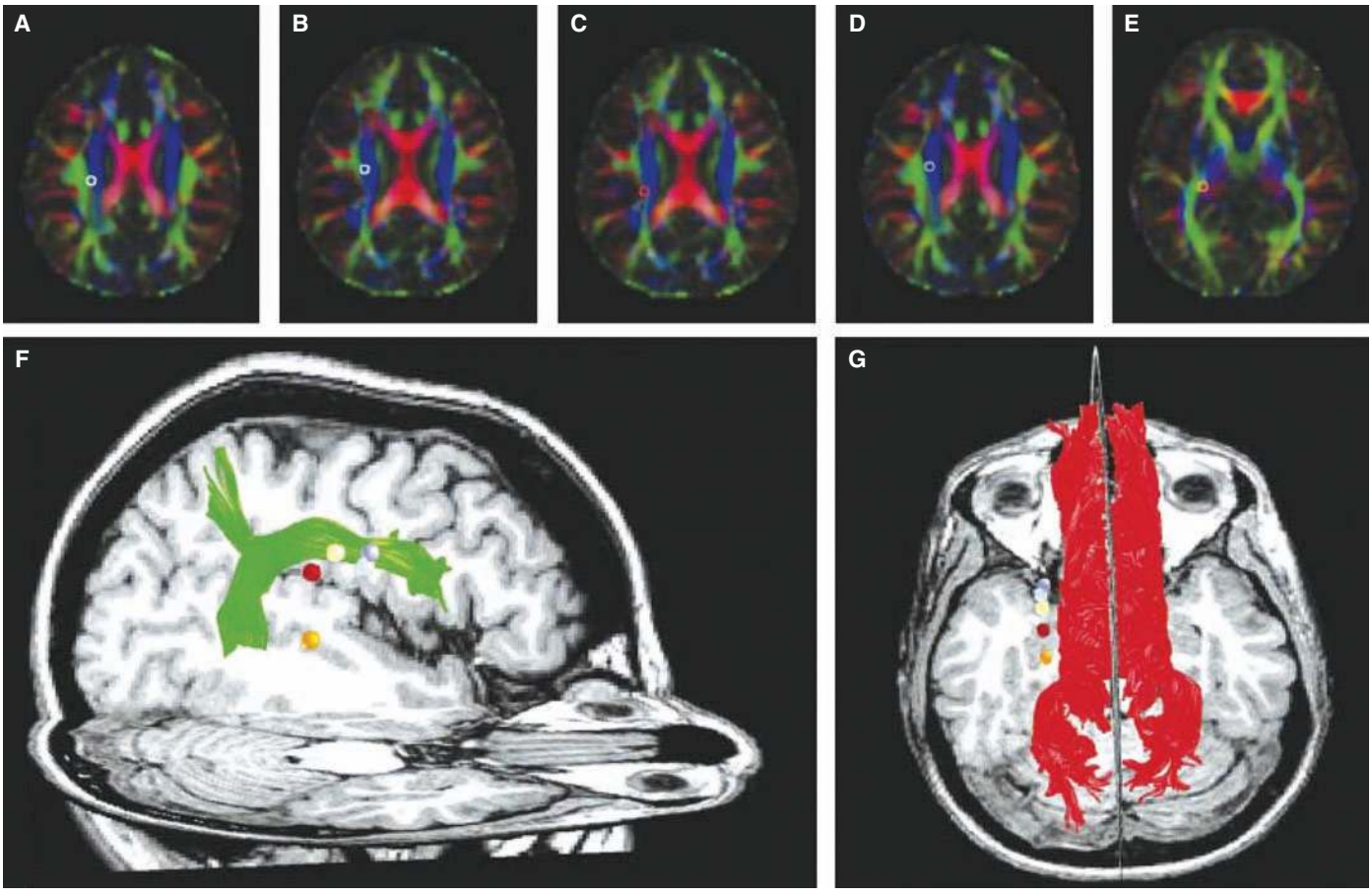

Fig. 2. Reading-related group differences in white matter as measured by DTI [from (*48*)]. Top row (**A** to **E**) shows reading-related differences in five independent studies; same locations of group differences are viewed sagitally (**F**) and axially (**G**). Colors correspond to estimated directions of white-matter pathways: left-right, red; anterior-posterior, green; inferior-superior, blue.

dyslexic readers (Fig. 2) (*31*–*33*). DTI studies of dyslexia also report greater-than-normal white-matter connectivity in the corpus callosum, the large white matter tract connecting homotopic regions of the left and right hemispheres (*34*). These findings suggest that, in dyslexia, white-matter pathways supporting reading project too weakly within the primary reading pathways of the linguistic left hemisphere, but they project too strongly between hemispheres (which may reflect an atypical reliance on right-hemisphere regions for reading that is observed in a number of functional neuroimaging studies). DTI is suitable for young children because its measurement does not require task performance. Studies with children conducted before reading instruction may determine whether the differential organization of white matter is predictive of developing dyslexia or is a consequence of reading practice.

Can Dyslexia Be Treated?

Remediation of dyslexia. Once children are diagnosed with dyslexia because of reading failure, treatments are instructional. Typical public school and special education interventions often stabilize the degree of reading failure rather than remediate (normalize) reading skill (*35*). Well-controlled studies involving random assignment to treatment and control groups consistently show that instruction yields substantial improvement in reading accuracy for many, but not all, children if instruction is more intensive (for instance, 100 min per day for 8 weeks), occurs in small groups (1 or 2 students per teacher), and includes explicit and systematic instruction in phonological awareness and decoding strategies (although the proportion of such instruction relative to reading meaningful text can vary widely with similar success). Gains are maintained for at least a year or two by ~50% of children after they return to the school's standard curriculum. Those children who retain their benefits improve from year to year, but they do not further catch up to typical readers. Such improvements are much more likely to occur in children who are beginning to read (ages 6 to 8) than in older children and are much more difficult to achieve for fluency than for accuracy. Thus, these resource-demanding interventions are effective for many children, but there are still challenges in developing interventions that are effective for all children.

How remediation of dyslexia alters the brain. Functional neuroimaging studies have revealed brain plasticity associated with effective intervention for dyslexia. In general, effective remediation is associated with increased activation, or normalization, in the left temporo-parietal and frontal regions that typically show reduced or absent activation in dyslexia for phonological processing of visually presented letters, words, or sentences (*36*–*40*). Immediately after intervention, increased right-hemisphere activations are also observed (*36*–*39*). Typical reading development is characterized by decreased right-hemisphere engagement and increased left-hemisphere engagement (*41*), which may reflect a shift in interpreting visual inputs like letters and words from specific percepts to categorical linguistic representations. Thus, individuals with dyslexia receiving intervention may engage, in a contracted period, both right- and left-hemisphere mechanisms underlying reading development. These changes in brain function can be maintained for at least a year after remediation is completed and students have returned to their standard curriculum (*37*, *40*).

Neuroimaging studies have not yet revealed what is different in the brains of children who do or do not respond to an intervention or sustain the benefits of intervention. It would be especially useful if neuroimaging markers were identified that could predict, before a specific intervention is provided, which children would benefit from a treatment, so that a given child could be offered an intervention most likely to help that child. To be informative, such neuroimaging studies would need to be longitudinal and involve many participants so that variation

among children with dyslexia could be characterized rigorously.

Can Dyslexia Be Predicted and Prevented?

A major goal for all behavioral disorders is their prevention. Dyslexia is currently identified by reading failure that is difficult for the child and that discourages reading practice. If children at risk for dyslexia could be identified before reading instruction or early during this process (between infancy and 1st grade), there is opportunity to intervene therapeutically and minimize or eliminate reading failure.

There is good evidence that dyslexia can be predicted and prevented in many children. Individually administered screening assessments for children in kindergarten and 1st grade have been developed that are brief and easy to give and yield strong predictions about future reading ability; these assessments focus on knowledge of letter names and sounds, phonological awareness, and speed of naming. Further, when beginning readers identified as "at risk" are provided with the sort of intensive instruction described above, 56 to 92% of at-risk children across six studies were brought within the range of average reading ability (*42*). Further, early intervention reduces the risk of the difficult-to-remediate fluency deficit that emerges in 4th grade.

One challenge regards the specificity of screening measures. It is estimated that to identify all of the weakest 10% of beginning readers, current measures would identify 20% of children as being at high risk. Because effective prevention is resource-demanding, more accurate identification of at-risk children would be valuable.

Brain measures predict risk for language and reading difficulty. Longitudinal studies have shown that brain measures can predict future language and reading problems in infants and young children before reading instruction. These studies measured event-related potentials (ERPs), which are time-locked changes in electrical activity in response to stimuli measured with scalp electrodes that have excellent temporal (millisecond) resolution, although the brain locations of the sources of the electrical activity are uncertain. ERPs can be performed readily with infants and children, so that brain mechanisms relevant for ultimate language and reading achievement can be measured before overt manifestations of language or reading. Most of these studies examined infants and children with familial risk for reading disorders to have a reasonably large percentage of participants go on to exhibit reading difficulties.

Newborns from families with versus without familial risk for dyslexia exhibit differences in ERP responses to language sounds within hours or days of birth, a finding all the more impressive because only about half the newborns with familial risk are expected to become dyslexic years later (*43*). Longitudinal ERP studies have shown impressive relations between brain responses at infancy and later language and reading success or failure. ERP responses to speech sounds within 36 hours of birth discriminated with over 81% accuracy those infants who would go on to become dyslexic readers at age 8 (*44*). Newborns, tested within a week of birth, had ERPs in response to speech sounds that correlated with language scores at ages 2.5, 3.5, and 5 years of age (*45*). These studies indicate that brain differences are present near the time of birth that greatly enhance the risk for and underscore the developmental nature of dyslexia. The findings also suggest that a deep understanding of the developmental pathways that lead to dyslexia demand prospective, longitudinal studies, from birth to early reading experience around ages 6 to 8.

Perhaps the most practical, near-term synergy between education and cognitive neuroscience arises from an integration of behavioral and brain measures in the service of predicting reading difficulty and then offering intervention to avoid reading failure. One example of this synergy comes from a study focused on decoding, the ability to determine the sound of a letter string from its constituent letters and syllables (*46*). Children identified by teachers as being at risk for reading difficulty at the start of a school year received a standardized test of decoding and 12 additional behavioral measures of language and reading, and they also underwent brain imaging. The behavioral and brain measures taken at the beginning of the school year were then related to the children's decoding ability at the end of the same school year, which improved on average after a year of education. The behavioral test scores and the brain imaging values in the fall accounted for 65 and 57%, respectively, of the variance in end-of-year decoding performance, but the combination of behavioral and brain measures accounted for significantly more of the variance (81%). Another longitudinal study related ERP measures in kindergarten to reading performance 5 years later and found that the addition of the ERP measures not only improved the prediction of reading ability over behavioral measures alone, but that only the ERP measures significantly predicted reading success in 5th grade (*47*). In both studies, brain measures significantly enhanced accuracy, beyond that possible with behavioral measures alone, in predicting long-term reading outcomes in children.

These findings suggest that the combination of behavioral and brain measures, perhaps together with genetic and familial information, may enhance the certainty with which dyslexia can be predicted for a child and promote the possibility of preventive intervention that allows many more children to succeed at learning to read.

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