What Factors Facilitate Resilience in Developmental Dyslexia? Examining Protective and Compensatory Mechanisms Across the Neurodevelopmental Trajectory

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ABSTRACT—***Developmental dyslexia is a specific learning disability characterized by deficits reading single words. Dyslexia is heritable and has been associated with neural alterations in regions of the left hemisphere in the brain. Cognitive and neural atypicalities have been observed before children with familial risk for dyslexia begin reading, yet children who are at risk subsequently develop reading abilities on a continuum from good to poor. Of those children who develop good reading skills, what factors are associated with more successful outcomes? In this article, we review findings describing genetic, cognitive, neurobiological, and environmental factors that facilitate reading development and propose a model of neural pathways to support successful reading development in at-risk children. This research can inform educational and clinical strategies to support at-risk children. Investigating factors that contribute to the variance in behavioral outcomes among at-risk children may help us understand developmental disorders and associated etiological, compensatory, and protective factors.***

KEYWORDS—*developmental dyslexia; brain development; resilience; reading; learning disabilities; neuroimaging; developmental disorders*

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DEVELOPMENTAL DYSLEXIA

Today, learning to read is critical for academic and professional achievement, yet 3–12% of individuals have difficulty due to developmental dyslexia (dyslexia; 1). Dyslexia is a specific learning disorder characterized by difficulties with decoding, speed, and accuracy of reading single words and poor spelling. These deficits cannot be explained by poor vision or hearing, lack of motivation, or disadvantageous educational opportunities. Moreover, the difficulties that characterize dyslexia can have severe psychosocial consequences such as anxiety, low self-esteem, and depression (2). Over time, these negative experiences often diminish motivation to read and lead to a profound gap in exposure to reading relative to typically developing peers, which can harm academic achievement and hinder vocational potential in the long term (3).

Dyslexia is heritable: In studies of twins and families, the prevalence of dyslexia among children with familial risk increased 30–60% (4, 5). While familial risk often increases the liability of dyslexia, children at risk develop reading abilities on a continuum ranging from good to poor (5, 6). What factors determine where along that continuum children's skills fall? In this article, we examine risk factors as well as protective and compensatory factors on genetic, cognitive, neurobiological, and environmental levels that contribute to the liability of dyslexia. In particular, we review findings on the putative compensatory neurobiological mechanisms that facilitate reading development, and we propose a model of the neural pathways that may protect and compensate for reading development in children with familial risk for dyslexia.

Child Development Perspectives

COGNITIVE FOUNDATIONS OF DYSLEXIA

Learning to read is a multifaceted process that relies on developing perceptual and cognitive skills that emerge even before birth. Early speech and language abilities are critical building blocks for developing phonological awareness, the ability to manipulate speech sounds within words (7). Subsequently, when children start to recognize letters, they map phonemes to graphemes to acquire knowledge of letter sounds, setting the foundation for subsequent decoding and reading acquisition.

In most studies, limited phonological awareness is one of the most reliable markers for dyslexia in school-age children (7). Other predictors of subsequent reading skills are letter-sound knowledge and rapid automatized naming, the ability to retrieve names for serially presented items quickly and accurately (8, 9). Several large-scale longitudinal studies have demonstrated that these skills predict long-term reading abilities (e.g., 6).

These causal factors have been reinforced in randomized control trials, in which interventions targeting phonological awareness and letter-sound mapping improved word-reading abilities in children at risk for dyslexia (10). Moreover, compared with preschoolers who are not at familial risk for dyslexia, children at familial risk perform less well on these key predictors (5). In addition, limited oral language and knowledge of vocabulary put children at risk for subsequent reading impairments (5, 6), although their effect on word decoding skills is likely mediated by the cognitive predictors of dyslexia (11).

GENETICS AND THE NEUROBIOLOGY OF DYSLEXIA

Scientists have identified several genes that are associated with dyslexia; most play an important role in brain development starting in utero (12, 13). Although several studies have questioned the impact of these genes and the methods used to detect them (14, 15), variant functions in these genes are associated with subtle cortical malformations in mice (e.g., 16). Furthermore, genes that suggest susceptibility for dyslexia have been hypothesized to affect neural circuits underlying perceptual and cognitive functions critical for reading acquisition (13). In line with this account, variants in some dyslexia susceptibility genes have been linked to alterations in the neural circuits in the brain that underlie reading (17). These genes interact with each other to contribute collectively to the genetic susceptibility to dyslexia (18).

In individuals with dyslexia, brain regions primarily in the left hemisphere that are important for language and reading are altered structurally and functionally (19). Specifically, the neural signature of dyslexia has been characterized by reduced volume of gray matter and decreased functional activation during literacy tasks in left-hemispheric anterior inferior frontal, posterior temporoparietal junction (dorsal), and occipitotemporal (ventral) regions (19, 20; Figure 1A). The temporoparietal junction is implicated in mapping speech sounds within words to corresponding written code, whereas the occipitotemporal region is involved in fast automated orthographic processing and in retrieving associated meanings (21). The left inferior frontal region is an important hub in reading, integrating the dorsal and ventral reading pathways and serving as an important region for speech planning and lexical access, as well as semantics and comprehension (22). Dyslexia has also been associated with weaker functional connectivity within the reading network (23) and altered pathways in left-hemispheric white matter that are linked with language and reading (24; Figure 1B). Moreover, in a few studies, dyslexia has been associated with a more bilateral neural network for reading. Compared with individuals without dyslexia, individuals with dyslexia have increased right-hemispheric activation and white-matter connectivity of the corpus callosum, a pathway that connects both hemispheres (22, 25), suggesting that individuals with dyslexia may compensate by relying on bilateral brain circuits.

Recent advances in pediatric neuroimaging have allowed researchers to investigate brain structure and function in children before they begin formal reading instruction (26). In preschoolers and infants with familial risk as well as preschoolers who subsequently develop poor reading skills, neural responses to basic auditory properties and speech are atypical and the left-hemispheric reading network is altered functionally and structurally (e.g., 27–29). In addition, in longitudinal studies, neuroimaging in early childhood predicts long-term reading outcomes (e.g., 28, 30), although replication studies with larger samples are needed to evaluate the generalizability of these findings.

PROTECTIVE FACTORS THAT FACILITATE SUCCESSFUL READING DEVELOPMENT IN CHILDREN WITH FAMILIAL RISK FOR DYSLEXIA

Despite the behavioral and neural deficits associated with familial risk for dyslexia, children at risk subsequently read on a continuum from good to poor (5). Nevertheless, 30–60% of at-risk children develop poor reading skills, which indicates that reading skills in these children are distributed atypically (31). Emerging evidence supports the notion that the liability distribution is determined by the interaction among many genetic, cognitive, and environmental factors, which in turn spurs reading outcomes along a continuum rather than a dichotomous categorization for dyslexia (32). Accordingly, children at risk who develop typical reading abilities show higher values on reading-related tasks than children who develop dyslexia, but show lower values on reading-related tasks than typical readers with no risk (32). This variance in reading outcomes among at-risk children suggests that we should investigate the protective factors that facilitate reading development for those at-risk children who subsequently read well.

Several cognitive-linguistic factors have been linked positively with reading outcomes in at-risk children. A recent meta-analysis (5) revealed that early language abilities play a

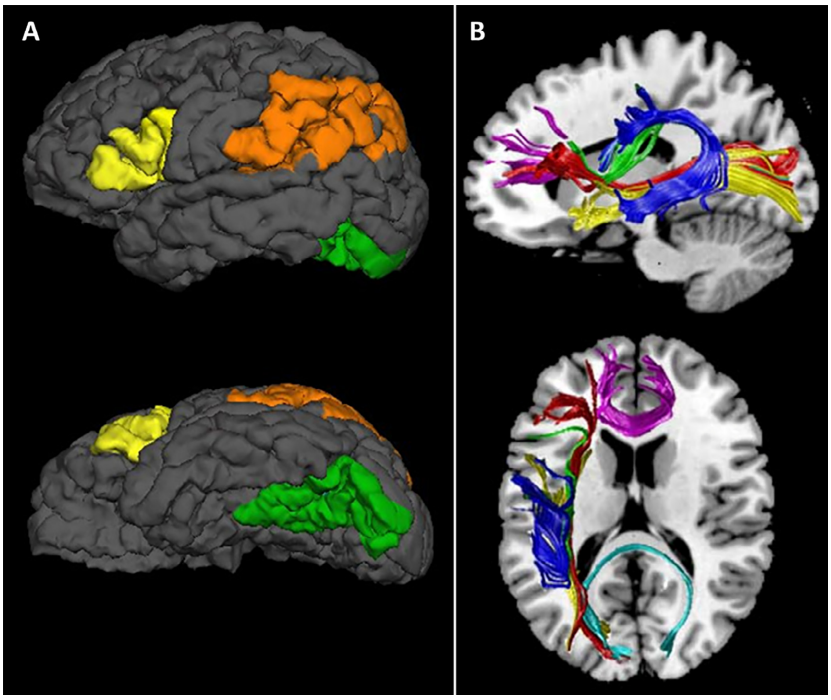

Figure 1. The neurobiology of dyslexia. (A) A left-hemispheric reading network that is commonly observed to exhibit structural and functional alterations in individuals with dyslexia. Brain images are presented on the lateral (upper) and ventral (lower) views. Orange: the temporoparietal junction; green: the occipitotemporal cortex; yellow: the inferior frontal cortex. (B) Atypical white-matter tracts implicated in dyslexia (upper: the sagittal view; lower: the transverse view). Blue: left arcuate fasciculus; green: left superior longitudinal fasciculus; red: left inferior fronto-occipital fasciculus; yellow: left inferior longitudinal fasciculus; light green: corpus callosum splenium; magenta: corpus callosum genu.

critical role in literacy acquisition for children with familial risk of dyslexia (5). Specifically, preschoolers with a family history of dyslexia who developed typical reading skills had more advanced language skills, particularly in vocabulary knowledge, than preschoolers who developed reading difficulties. Moreover, early language abilities predict reading outcomes among children with and without risk. These advantages in language may protect at-risk children from developing dyslexia by facilitating their phonological development or providing rich contextual information to compensate for difficulties in learning to read (33). Finally, typical reading outcomes in at-risk children have also been linked positively with increased attention skills, memory, and general intelligence, though further research is warranted to verify the protective nature of these factors (34).

Several environmental factors also affect reading development positively. For instance, in several studies, socioeconomic status is linked strongly with language and reading abilities (35), likely because socioeconomic status reflects access to resources for education as well as for general health and nutrition, which are critical for early childhood development and general brain development, oral language, and subsequent reading acquisition (36). Another environmental factor that facilitates reading development is an enriched home literacy environment, characterized by many children's books in the home, shared reading in early infancy, and frequent shared-reading experiences with parents and caregivers. The home literacy environment contributes to reading outcomes even after controlling for socioeconomic status (37). However, few studies have investigated the impact of socioeconomic and home literacy factors on the reading outcomes of children with familial risk for dyslexia. In those that have done so, parents of good readers at risk for dyslexia tended to be more educated and read to their offspring more often than parents of poor readers at risk for dyslexia, although these differences were not significant in small-scale meta-analyses (5). These environmental factors may build resilience to the liability of dyslexia, which may protect children with familial risk for dyslexia.

PUTATIVE NEURAL MECHANISMS UNDERLYING SUCCESSFUL READING DEVELOPMENT IN CHILDREN WITH FAMILIAL RISK FOR DYSLEXIA

Besides cognitive and environmental factors, other possible protective factors in struggling readers are right-hemispheric pathways in the brain (22, 38). Recruitment of these regions may underlie successful reading compensation in struggling readers because increased right-hemispheric activation in children with dyslexia during a reading task has been associated with more successful reading outcomes over time (38). Moreover, individuals with dyslexia who received intervention and improved

significantly in reading had increased activation in the right-hemispheric regions (39). These changes resulting from experience were specific to children who responded to the treatment and were not seen in those who did not improve, suggesting that increased right-hemispheric activation may be a compensatory neural mechanism that facilitates reading development in these children (40).

Given the role of experience-induced neuroplasticity in shaping brain development (41), we should also consider environmental experiences that may facilitate the development of a potential right-hemispheric compensatory reading network. For instance, in several studies, socioeconomic status and home literacy affected brain development significantly, including bilateral and right-hemispheric networks important for reading (42, 43). Furthermore, in a recent neuroimaging study, distinct approaches to teaching adults how to read words in an artificial language differentially shaped hemispheric preference for reading these words (44). Based on these findings, environmental variables may assist in facilitating the development of compensatory reading network components in the right hemisphere in children with atypical brain development in left-hemispheric regions.

These right-hemispheric pathways already seem to exist prior to the onset of reading, presumably supporting literacy acquisition in children with familial risk. In at-risk children, researchers have identified increased right-hemispheric neural responses for speech perception as early as infancy (for a review, see 26). Moreover, preschoolers with familial risk for dyslexia had right lateralization of white-matter tracts important for reading development; preschoolers without familial risk had primarily a left-hemispheric dominance. Furthermore, an increased rate of development of white matter in the right hemisphere has been associated with more successful reading in at-risk children (28). Thus, converging evidence suggests that right-hemispheric neural pathways play a role in compensating for atypical brain development in at-risk children.

Environmental factors also facilitate the emergence of right-hemispheric reading circuits in at-risk children. Specifically, home literacy was associated more strongly with right inferior frontal regions during phonological processing in young children with familial risk for dyslexia than in young children without such risk (43), suggesting an interaction effect between familial risk and environment. This leads us to question whether these networks are truly compensatory by emerging in response to difficulties in learning to read, or whether these children are predisposed to process linguistic stimuli with a less left-lateralized network. If these right-hemispheric networks exist when children start learning to read, this could enable them to develop typical reading skills despite left-hemispheric brain alterations associated with a risk of dyslexia.

In addition, not all children with a familial risk have a genetic or neurobiological susceptibility for dyslexia. Although nonaffected siblings typically have reduced reading abilities and mild phonological deficits compared with children without siblings with dyslexia, some of these children have no risk or are at minimal genetic risk and therefore learn to read typically. However, identifying protective factors that facilitate typical reading development in children with a genetic risk has important implications for developing educational and clinical strategies to support these children as they learn to read.

A MODEL FOR PROTECTIVE AND COMPENSATORY PATHWAYS TO SUPPORT SUCCESSFUL READING DEVELOPMENT

Based on evidence, we propose a model for putative protective and compensatory right-hemispheric pathways that emerge through an interaction between genetic and environmental factors, which then results in a more bilateral, less-lateralized reading network supporting typical reading development in at-risk children (Figure 2). Researchers have hypothesized that variant function in critical genes (those related to susceptibility for dyslexia) involved in cortical development cause subtle left-hemispheric cortical malformations that, in turn, lead to the atypical left-hemispheric network in individuals with dyslexia (13); this has been outlined in the schematic hypothesis of the neural underpinnings associated with poor reading outcomes (Figure 2).

Moreover, variant function in some susceptibility genes for dyslexia may also cause atypical brain development in the corpus callosum, which may then set a foundation for greater connectivity between the left and right hemispheres. This may facilitate increased engagement of the right hemisphere for language and literacy skills during early development. Consistent with this hypothesis, right-lateralized specialization in the reading network has been observed in children with familial risk for dyslexia even before they start reading (for a review, see 26). Subsequently, as they learn to read, effective right-hemispheric compensatory mechanisms emerge through an interaction with positive environmental impacts in at-risk children (Figure 2). However, these atypical brain asymmetries and alterations in the corpus callosum may not always lead to the development of successful detour pathways, as the same patterns have also been observed in some children with dyslexia compared with typically developing children (24).

Accordingly, collective evidence suggests that environmental factors such as home literacy and specific instructional approaches may help shape these putative right-hemispheric and colossal neural specializations for reading in at-risk children who subsequently develop good reading skills, particularly during the period of heightened plasticity in early childhood (see the dashed effect pathways in Figure 2). As a consequence, a compensatory reading network develops, promoting resilience and facilitating the emergence of typical reading skills. This developmental model may be one way some children with a genetic predisposition for dyslexia develop typical reading skills despite deficits in early literacy skills or neural alterations in the left-hemispheric reading network.

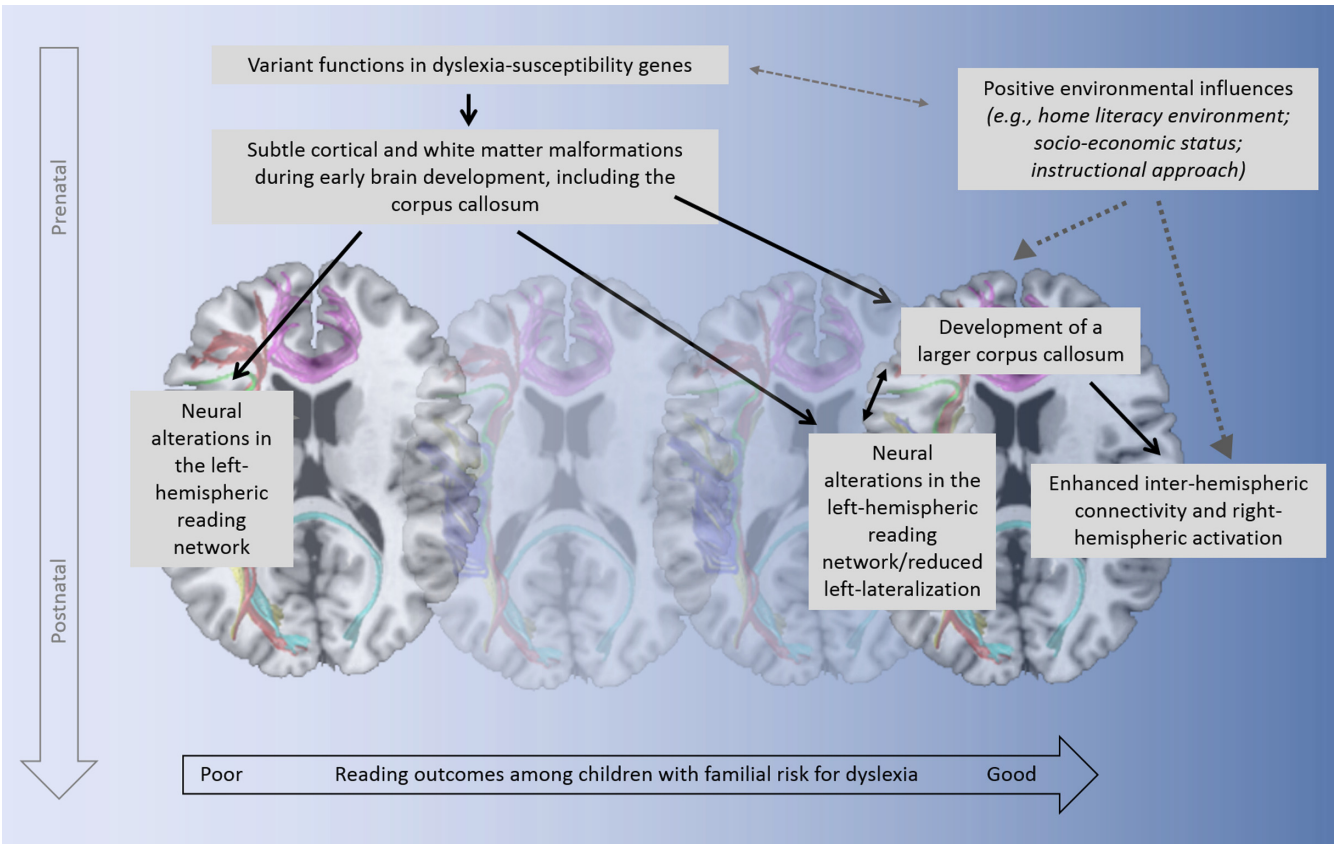

Figure 2. **Hypothesized neurodevelopmental trajectories underlying reading development in children with a familial risk for dyslexia.**

LOOKING AHEAD

Researchers are just beginning to investigate protective and compensatory mechanisms in children at risk for dyslexia who become successful readers. The proposed model brings forth an initial hypothesis due to the limited empirical research available. We need more research with a developmental focus to empirically evaluate the viability of this model and determine the developmental trajectory of putative protective and compensatory factors, as well as their specificity and interactions. In particular, we need longitudinal investigations from infancy to school age to determine whether the altered corpus callosum and right-hemispheric pathways are in place from birth as protective factors that support reading acquisition from the start, or whether these experience-driven changes develop in the brain during reading acquisition. Furthermore, it is unclear if only children with familial risk exhibit these alterations or if these compensatory networks also appear in remediated learners with no familial risk. In addition, we still need to determine whether putative compensatory networks are formed only for reading in these children or whether they could be observed for language or other cognitive processes. These longitudinal studies should account for familial risk for dyslexia from the beginning, consider possible contributing environmental factors, and measure the behavioral and neural correlates of the cognitive-linguistic effects reported. Finally, we should consider the continuous nature of reading outcomes and the associated multifactorial influences to minimize potential inconsistencies due to variation in the definition of dyslexia.

Overall, this work can inform best practices to support children at risk for dyslexia. Identifying protective factors that facilitate reading development may allow clinicians and educators to approach reading instruction and intervention comprehensively, recognizing children's relative strengths and promoting alternate strategies in addition to traditional interventions to maximize the potential for success in every child. Furthermore, investigating children with familial risk for dyslexia who develop typical reading skills may also be a way to understand other developmental disorders and their compensatory and protective factors.

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