Brain Abnormalities in Children at Risk for Developmental Dyslexia and Early Neural Markers of Dyslexia: Postprint

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Date: 2023-10-09T00:00:00+00:00

Abstract

Investigating neural abnormalities in children at risk for developmental dyslexia facilitates the identification of early neural markers of dyslexia, which holds important implications for early prediction and intervention. Cross-sectional studies of at-risk children reveal abnormalities in both brain function and structure. Specifically, at-risk children show smaller amplitude and longer latency of the mismatch response (MMR) evoked by speech and non-speech sound processing, and functional and structural abnormalities exist in the ventral and dorsal pathways for reading. Compared to preschool cross-sectional comparisons, longitudinal follow-up studies into school age can reveal neural changes associated with reading development and uncover early neural markers of dyslexia. Longitudinal studies have shown that MMR evoked by speech sound processing, functional abnormalities in the left temporoparietal region and visual word form area, and structural abnormalities in the left arcuate fasciculus constitute early biological markers of dyslexia. Furthermore, longitudinal studies on brain abnormalities in at-risk children are relatively scarce, and small sample sizes also compromise the credibility of the findings. Future research should emphasize longitudinal studies with larger sample sizes, while also focusing on brain abnormalities in Chinese-speaking at-risk children and investigating Chinese reading

Full Text

Advances in Psychological Science, 2023, Vol. 31, No. 10, 1912-1923

https://doi.org/10.3724/SP.J.1042.2023.01912

Brain Abnormalities in Children at Risk for Developmental Dyslexia and Early Neural Markers of Dyslexia

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Abstract: Investigating neurological abnormalities in children at risk for developmental dyslexia (DD) is crucial for identifying early neural markers and enabling early prediction and intervention. Cross-sectional studies of at-risk children reveal abnormalities in both brain function and structure. Specifically, children at risk show smaller amplitude and longer latency in mismatch responses (MMR) evoked by speech and non-speech perception, along with functional and structural anomalies in the ventral and dorsal reading pathways. Compared with preschool comparisons, longitudinal studies extending into school age can reveal neural changes associated with reading development and uncover early neural markers of dyslexia. Longitudinal research indicates that MMR during speech processing, functional abnormalities in the left temporoparietal region and visual word form area, and structural abnormalities in the left arcuate fasciculus serve as early biological markers. However, longitudinal studies of at-risk children' s brains remain scarce, and small sample sizes compromise result reliability. Future research should emphasize large-sample longitudinal studies while examining brain abnormalities in Chinese at-risk children to explore the uniqueness and universality of cognitive-neural risk factors for Chinese dyslexia.

Keywords: developmental dyslexia, children at risk for developmental dyslexia, brain abnormalities, brain structure and function
Classification Code: B845

Developmental dyslexia (DD) is a neurodevelopmental disorder characterized by difficulties in accurate and/or fluent word recognition despite normal intelligence, environmental conditions, and educational opportunities, without obvious impairments in auditory, visual, or nervous system functions. Most individuals with dyslexia experience decoding difficulties accompanied by spelling problems. While typically diagnosed in childhood, symptoms persist into adulthood. Consequently, many children with dyslexia develop reading comprehension difficulties, reduced reading experience, and hindered vocabulary and knowledge growth.

DD represents a common learning disability during school age, with prevalence reaching 6.6% among schoolchildren based on DSM-5 diagnostic criteria [**?**]. Researchers have employed various neuroscientific techniques to identify neurobiological causes [**?**], though whether these brain abnormalities constitute causes or consequences remains unclear. To address this, researchers have focused on preschool children at risk for dyslexia—children assessed as having high likelihood of developing DD before receiving reading instruction. Since these children

have not yet begun reading instruction, neural abnormalities related to reading abilities cannot result from reading experience. Therefore, investigating their brain abnormalities helps identify early neural markers. Understanding the neurobiological basis of dyslexia can substantially improve predictive power for children' s future reading development [?, ?] and facilitate early screening and intervention for potential dyslexia cases [?, ?].

Researchers typically assess dyslexia risk in preschool children through two approaches: analyzing the disorder' s hereditary properties and evaluating early cognitive and language skills [?].

DD exhibits familial genetic characteristics [?]. The average recurrence rate among first-degree relatives is approximately 30%, with children of parents with dyslexia showing recurrence rates between 27% and 49%, and siblings reaching 38.5% to 43% [?]. Researchers identify at-risk children based on having at least one first-degree relative diagnosed with dyslexia. Parent diagnosis typically involves three methods: (1) standardized clinical diagnosis [?, ?, ?]; (2) questionnaires, most commonly the Adult Reading History Questionnaire (ARHQ), where children are classified as at-risk when relatives score above 0.4 [?, ?]—the questionnaire demonstrates good reliability for adult reading ability with strong inter-rater correlation (ICC = 0.67) and ROC analysis showing 77.1% specificity at the 0.385 cutoff [?]; and (3) cognitive and reading ability tests assessing parental IQ and reading levels, typically using Raven' s IQ test and Wechsler Adult Scales [?, ?, ?, ?], with reading tests including word recognition, pseudoword reading, lexical decision, nonword repetition, rapid naming, and paragraph comprehension [?, ?, ?, ?, ?, ?]. Some studies combine two diagnostic approaches as selection criteria [?, ?].

Beyond genetic attributes, DD manifests in early behavioral signs during preschool years. Researchers assess reading risk through early language and cognitive skills, including phonological awareness, orthographic awareness, verbal working memory, and rapid naming—skills central to reading acquisition [?]. Children scoring below the 25th percentile on these tasks are typically classified as at-risk [?], with some studies using stricter criteria of below the 35th percentile [?]. Since reading skill scores are continuous variables, some research employs correlation analysis to explore related neural mechanisms [?]. Other studies combine both familial risk and performance on reading-related tests to define at-risk status, requiring both family history and low scores on reading-related assessments [?, ?].

2. Brain Functional Abnormalities in Preschool Children at Risk for Developmental Dyslexia

Previous research examining brain functional abnormalities in at-risk children aims to identify neural markers of dyslexia by investigating early manifestations of core deficits. Reading is a complex cognitive process, with current research attributing DD to multiple processing levels. The primary debate centers on

whether the core deficit involves specific language impairments or general cognitive abilities. Proponents of language-specific deficits argue that word decoding difficulties stem from impaired phonological processing or orthographic processing challenges [?, ?, ?], while supporters of non-language-specific deficits view phonological and orthographic deficits as surface manifestations of more fundamental cognitive problems [?, ?].

2.1. Language Processing Abnormalities in At-Risk Children

2.1.1. Electrophysiological Mechanisms of Language Processing Due to its operational convenience and sensitivity in brain measurement, EEG technology is widely used to study brain activity in preschool children. Event-related potentials (ERP) measure brain electrical responses related to psychological activities or events. Many researchers consider phonological processing deficits central to dyslexia. The phonological deficit hypothesis posits that children with dyslexia have difficulty analyzing phonological structures, preventing successful learning of correspondences between orthography and phonology and impeding conversion of visual word input to phonological representations. Consequently, ERP research on language processing in at-risk children has focused primarily on phonological processing.

Infants demonstrate phoneme perception abilities shortly after birth. Numerous studies using family-risk infants have investigated genetically influenced electrophysiological responses. Volkmer and colleagues reviewed 17 studies finding that at-risk children generally show weaker speech signal perception, with at-risk children exhibiting atypical MMR amplitude and latency—specifically smaller amplitude and longer latency—compared to typically developing children. The review also analyzed age effects, finding MMR abnormalities more pronounced in infancy, with inconsistent results for 4-6 year-olds, possibly because children in this age range have already been influenced by reading experience [?]. These studies examined phonological perception of vowels in consonant-vowel structures [?, ?], vowel duration [?, ?, ?], consonants [?, ?, ?, ?, ?, ?], and consonant duration [?, ?]. All found atypical MMR during phonological processing in at-risk children, with abnormalities specific to speech rather than pure tones [?].

Beyond MMR, other ERP components evoked by phonological perception in family-risk children show abnormalities. A Dutch study found that 5-month-old at-risk infants exhibited smaller P2 amplitude, while at 17 months the at-risk group showed delayed P1 and P2 latencies [?]. In addition to amplitude and latency differences, family-risk infants show hemispheric lateralization differences in phonological perception. At-risk infants demonstrate right-hemisphere lateralization of MMR, whereas controls show left-hemisphere advantage. Correlation analyses reveal that smaller bilateral responses correlate with better discrimination performance in at-risk children, while larger left-hemisphere responses correlate with better performance in controls [?]. The Dutch study also found right-hemisphere lateralization of N2 amplitude in the at-risk group [?],

likely reflecting compensatory mechanisms to address left-hemisphere language region dysfunction [**?**].

Research has also examined neural manifestations of print processing in preschool children. Bach et al. [**?**] compared electrophysiological activity during print processing in kindergarteners who later became poor second-grade readers versus typical readers. Results showed that N1 amplitude differences between processing letters versus symbols explained 67% of variance in later reading performance, suggesting orthographic processing neural patterns could serve as early markers, though no studies have examined orthographic processing ERP abnormalities specifically in at-risk children.

2.1.2. Brain Activation Abnormalities in Language Processing Some studies have investigated atypical brain activation during phonological processing in at-risk children. Raschle et al. [**?**] examined brain function in 5-year-old family-risk children during initial phoneme matching tasks, finding weaker activation in left occipitotemporal and temporoparietal regions compared to typical children. Activation in these regions positively correlated with phonological processing ability [**?**]. Beyond phoneme matching, 5-year-old at-risk children also show neural abnormalities during auditory rhyme judgment tasks. Despite no behavioral differences, at-risk children exhibited reduced activation in bilateral temporoparietal, inferotemporal occipital, bilateral inferior frontal, and bilateral middle frontal regions [**?**]. Additionally, in verbal word learning tasks, typical children showed gradually decreasing left temporal activation with word repetition, while at-risk children did not [**?**]. Łuniewska et al. [**?**] found that while typical preschoolers activated brain structures responsible for phonological processing—including bilateral inferior frontal regions, most temporal lobe areas, left fusiform gyrus, and cingulate cortex—family-risk children activated only left subcortical regions.

Research has also identified reduced activation in key brain regions for print processing in at-risk children. Studies of typical development show that print processing primarily activates left occipitotemporal cortex and visual word form area, with visual word form area activation explaining 84% of variance in second-grade reading skills alongside behavioral and EEG measures [**?**]. Yamada et al. [**?**] compared neural responses of 5-year-old at-risk children during orthographic tasks (letter-symbol comparison), finding that low-risk children showed greater bilateral temporoparietal activation for letters versus symbols, while high-risk children showed no such difference. Specht et al. [**?**] found that 6-year-old at-risk children also showed insufficient occipitotemporal activation when processing orthographically regular words. Furthermore, functional connectivity studies reveal atypical connectivity patterns in left fusiform gyrus in at-risk children [**?**]. Yu et al. examined resting-state functional connectivity in two infant groups, finding that connectivity patterns between left fusiform gyrus and frontoparietal language and attention networks significantly distinguished the groups, suggesting that sensitivity to cortical differentiation in reading networks

may emerge before formal literacy instruction.

ERP studies consistently show that at-risk children exhibit smaller MMR amplitude and longer latency during phonological perception, indicating poorer phonological perception abilities before school age. Unlike typical children, at-risk children fail to show left-hemisphere lateralization trends, possibly because right-hemisphere homologous regions compensate for left-hemisphere processing deficits, reducing left-lateralization. Brain activation studies reveal insufficient activation in bilateral temporoparietal, bilateral inferior frontal, and bilateral middle frontal regions during phonological tasks, and reduced bilateral temporoparietal and occipitotemporal activation during print processing tasks. Atypical functional connectivity patterns also exist between left fusiform gyrus and frontoparietal language regions. Reading involves coordinated activation of multiple brain regions, including temporoparietal cortex, occipitotemporal cortex, and prefrontal cortex [**?**]. The temporoparietal junction, comprising posterior superior temporal gyrus, angular gyrus, and supramarginal gyrus, forms the dorsal phonological pathway responsible for establishing relationships between visual word forms and phonological structures [**?**]. The occipitotemporal region, as the ventral pathway, contains the visual word form area critical for rapid word recognition [**?**]. The visual word form area in left fusiform gyrus primarily accesses the orthographic lexicon or integrates orthography with semantics/phonology [**?**]. Left inferior frontal gyrus, important for lexical reading, is involved in phonological retrieval and manipulation and shows sensitivity to orthographic-phonological regularities. While individuals with dyslexia show functional abnormalities in left inferior frontal gyrus [**?**, **?**], some studies suggest it serves only a compensatory role [**?**]. Activation patterns in at-risk children are inconsistent: some show insufficient bilateral inferior frontal activation [**?**, **?**], while Yamada et al. [**?**] found no group differences in inferior frontal activation during print processing, with at-risk children only showing activation after reading instruction, suggesting compensatory mechanisms for impaired left dorsal pathway function. Future longitudinal research should clarify the function and role of left inferior frontal gyrus in at-risk children.

2.2. Brain Functional Abnormalities in Non-Language Processing in At-Risk Children

2.2.1. Electrophysiological Mechanisms of Non-Language Processing

The non-language-specific deficit hypothesis posits that dyslexia primarily results from impaired basic cognitive abilities, such as visual or auditory deficits, memory deficits, or visual attention deficits [**?**]. Research shows that individuals with dyslexia exhibit auditory temporal processing deficits, including atypical brain electrical activity in response to novel auditory temporal stimuli [**?**, **?**] and weaker discrimination of amplitude rise time (ART) in sound signals [**?**]. Consequently, studies have examined whether at-risk children show similar patterns in auditory temporal processing neural activity.

Results indicate that 17-month-old at-risk toddlers processing novel temporal

interval stimuli do not elicit MMR components similar to controls. Combined with subsequent reading performance, this suggests that non-language processing deficits may represent risk factors rather than predictive markers [**?**, **?**, **?**]. For example, Plakas et al. [**?**] found that MMR evoked by ART and frequency in 41-month-old family-risk children could not distinguish between normal and poor readers in second grade, indicating that non-language processing deficits represent neural manifestations of risk but not predictive factors for dyslexia.

Some studies suggest that poor reading fluency in dyslexia may also result from delayed non-verbal information processing, particularly deficits in visual information processing speed [**?**]. A Dutch study recorded EEG activity in family-risk and typical children during repeated visual stimuli [**?**]. Reading assessments at second grade completion revealed that, unlike typical children, family-risk children showed no habituation effects for P3. Risk children with normal reading levels showed no N1 habituation, while poor-reading risk children exhibited increased N1 amplitude with repetition, indicating inefficient novel information processing and suggesting that low-level visual processing deficits constitute a risk factor for dyslexia.

2.2.2. Brain Activation Abnormalities in Non-Language Processing
Non-language processing activation studies primarily focus on auditory processing abilities in at-risk children. Raschle et al. [**?**] examined brain functional networks during rapid auditory processing tasks in 5-year-old at-risk children, finding reduced activation in left prefrontal regions. The study also examined brain activation during phonological processing, revealing that left prefrontal activation correlated with dorsal and ventral region activation, suggesting close relationships between left prefrontal areas and reading [**?**]. Ages 6-12 months represent sensitive periods for native phoneme learning, during which infants' perception of native phonemes increases while non-native phoneme perception decreases. This developmental change shows improved neural efficiency in both spatial distribution and temporal processing. Recent MEG research examined brain changes during auditory processing in 6- and 12-month-old typical and at-risk infants using repeated white noise presentation. Typical infants showed decreased left hemisphere auditory cortex activation and shortened auditory dipole duration with age, while at-risk infants showed opposite patterns of stronger, longer-lasting left hemisphere activation. This auditory cortex, primarily in left temporal and frontal regions, may impair low-level auditory processing and affect phonological representation establishment [**?**].

Non-language processing brain function studies reveal auditory temporal processing deficits in at-risk children, with abnormal EEG responses to temporal auditory and visual stimuli. Reduced activation in left prefrontal and left temporal auditory processing regions during non-language stimulus perception, along with atypical developmental patterns, indicate that auditory cortex functional abnormalities appear before reading experience. However, whether auditory cortex abnormalities constitute early markers of dyslexia requires further longi-

tudinal investigation.

3. Brain Structural Abnormalities in Preschool Children at Risk for Developmental Dyslexia

In addition to examining atypical brain activation, increasing morphological research has begun exploring brain structure in at-risk children. Similar to DD research showing reduced gray matter volume in temporoparietal and occipitotemporal regions [**?**, **?**], at-risk studies also find gray matter volume reductions in these areas [**?**, **?**, **?**]. At-risk children also show atypical sulcal patterns (arrangement, number, and size of primary cortical folds) in temporoparietal and occipitotemporal regions, with similar patterns observed in individuals with dyslexia [**?**]. Additionally, asymmetry in left versus right planum temporale surface area correlates with dyslexia risk, with typical children showing greater leftward asymmetry than at-risk children [**?**]. Previous research on dyslexic adults and children has found involvement of right hemisphere homologous regions, hypothesized as compensation for impaired left posterior dorsal systems. Indeed, the right hemisphere may play similar roles in both typical and dyslexic development [**?**]. Petersen et al. [**?**] proposed that increased right hemisphere activation during initial learning stages primarily addresses task demands, with additional right hemisphere involvement serving as compensation for insufficient left hemisphere reading network function due to limited reading experience or disorder.

Reading requires not only coordinated activation of multiple brain regions but also efficient information transmission and integration among them. White matter fiber tracts provide the structural basis for inter-regional neural communication [**?**]. The arcuate fasciculus connects lateral temporal and posterior inferior frontal regions, forming the structural basis of the dorsal pathway and playing important roles in phonological processing [**?**]. The inferior fronto-occipital fasciculus, a ventral pathway tract, relates closely to orthographic processing, transmitting information from visual word form area to other regions alongside the inferior longitudinal fasciculus [**?**]. Langer et al. [**?**] conducted DTI scans of family-risk infants, finding significantly smaller fractional anisotropy (FA) values in left arcuate fasciculus. Additionally, at-risk children show smaller FA values in left inferior fronto-occipital fasciculus compared to typical children [**?**]. FA values reflect white matter fiber density, axon diameter, and myelination [**?**]. These findings indicate that before reading experience, at-risk children already show abnormal white matter connectivity in left hemisphere.

In summary, at-risk children exhibit brain structural abnormalities, including smaller gray matter volume and atypical sulcal patterns in temporoparietal and occipitotemporal regions, without showing planum temporale leftward asymmetry trends. White matter connectivity shows abnormal FA values in left arcuate fasciculus and left inferior fronto-occipital fasciculus. However, no DTI tractography studies have observed inferior longitudinal fasciculus abnormalities in at-risk children.

4. Early Neural Markers of Developmental Dyslexia: Evidence from Longitudinal Tracking

The aforementioned studies demonstrate multiple brain-level abnormalities in at-risk children, but one unavoidable issue remains: at-risk children do not necessarily develop dyslexia. Therefore, these abnormalities may reflect risk factors rather than markers of dyslexia itself [**?**]. Longitudinal studies tracking at-risk children into school age help determine which early brain abnormalities constitute neural markers of dyslexia.

Longitudinal studies of language processing show that MMR evoked by phonological processing in at-risk children correlates with school-age reading ability and can distinguish reading fluency levels [**?**, **?**]. Van Zuijen et al. [**?**] used oddball paradigms to demonstrate that MMR during phonological processing could differentiate fluent from non-fluent readers among at-risk children. Maurer et al. [**?**] found that hemispheric lateralization of phonological MMN distinguished reading levels in at-risk children. In contrast, longitudinal studies of non-speech processing found that MMR in at-risk children represents only risk factors, not predictive markers [**?**, **?**, **?**]. For instance, Plakas et al. [**?**] found that MMR evoked by ART and frequency in 41-month-old family-risk children could not differentiate normal versus poor readers in second grade, suggesting non-language processing deficits represent neural manifestations of risk rather than predictive factors.

Brain function studies consistently identify functional abnormalities in left temporoparietal region and visual word form area as early biological markers of dyslexia. The left temporoparietal region matches visual information to corresponding phonological representations, playing a crucial role in cross-modal integration required for reading. Longitudinal research examining phonological processing development shows that with increasing reading experience, phonological processing becomes more automatic, with typical children showing reduced activation. Family-risk children demonstrate atypical phonological processing networks before language experience. Two years later, at-risk children diagnosed with dyslexia versus those with normal reading show enhanced activation in bilateral middle frontal gyrus and right inferior parietal lobule when retrospectively examining kindergarten brain activation. At-risk children who later develop dyslexia show enhanced activation in more extensive regions including bilateral superior temporal gyrus, middle temporal gyrus, left supramarginal gyrus, and transverse temporal gyrus [**?**]. Yu et al. [**?**] used retrospective longitudinal design to examine kindergarten brain activation during phonological processing in typical readers and at-risk children who later achieved typical reading levels. Results showed weaker left temporoparietal activation but stronger right inferior frontal activation in at-risk children, suggesting left temporoparietal cortex represents a neural phenotype related to risk factors, while right inferior frontal activation reflects compensatory mechanisms for reading development.

The visual word form area, as part of occipitotemporal cortex, accesses the orthographic lexicon during the first step of reading. One study measured bilateral fusiform activation in kindergarten at-risk children processing letters and false fonts, then longitudinally tracked these children to assess correlations between preschool activation patterns and two-year reading outcomes. Results showed that at-risk children who later developed dyslexia exhibited reduced visual word form area activation for both letters and false fonts compared to those who achieved normal reading levels [**?**].

Longitudinal brain structure studies identify left arcuate fasciculus structural abnormalities as early biological markers of dyslexia. Researchers divide the dorsal arcuate fasciculus into three segments based on location and function: the anterior arcuate fasciculus connecting inferior frontal gyrus and inferior parietal lobule, primarily related to speech production; the posterior arcuate fasciculus connecting inferior parietal lobule and superior temporal gyrus, primarily related to speech perception; and the longest direct arcuate fasciculus connecting inferior frontal gyrus and superior temporal gyrus, directly involved in phonological processing [**?**]. One study showed that preschool at-risk children had significantly elevated quantitative T1 intensity in left anterior arcuate fasciculus, indicating reduced myelin concentration. Correlation analysis with reading outcomes two years later revealed that neural anatomical differences (80%) better predicted dyslexia prevalence than behavioral predictors alone (63%), with left anterior arcuate fasciculus T1 intensity as a significant predictor [**?**]. Another longitudinal study found lower FA values in left direct arcuate fasciculus in at-risk children who later developed dyslexia. Combining cognitive ability, familial risk, and neural structural differences, the model identified left direct arcuate fasciculus as the only significant predictor [**?**]. Neither study found differences in inferior fronto-occipital fasciculus. Additionally, longitudinal research has identified structural changes in superior longitudinal fasciculus. Zuk et al. [**?**] compared cognitive abilities, environmental factors, and brain structural differences among typical preschoolers, at-risk children who developed normal reading, and at-risk children who developed dyslexia, finding that only at-risk children with normal reading showed significantly higher right hemisphere superior longitudinal fasciculus FA values. In at-risk children, right superior longitudinal fasciculus FA values positively correlated with decoding ability, suggesting this pathway may represent a compensatory mechanism rather than an early marker [**?**]. This finding was confirmed by Wang et al. [**?**], who found faster white matter development in right superior longitudinal fasciculus among school-age at-risk children with stronger reading abilities. These results suggest that superior longitudinal fasciculus and inferior fronto-occipital fasciculus abnormalities in dyslexia may result from reading experience and familial risk, while left arcuate fasciculus directly involved in phonological processing marks early brain structural abnormalities in dyslexia.

5. Summary and Outlook

This review systematically summarizes brain abnormalities in preschool children at risk for developmental dyslexia. Recent research using various neuroscientific techniques has examined neural abnormalities in language and non-language processing in at-risk children across alphabetic writing systems, spanning from newborn infants to 7-year-olds. Current literature demonstrates that brains of preschool children at risk for dyslexia already show changes, with reading-supporting neural networks formed. Longitudinal studies of at-risk children have identified early neural markers of dyslexia, including MMR during phonological processing, functional abnormalities in left temporoparietal region and visual word form area, and structural abnormalities in left arcuate fasciculus.

5.1. Emphasis on Large-Sample Longitudinal Studies

Current research on preschool children at risk for dyslexia primarily focuses on neural abnormalities, with only a few longitudinal studies examining whether these abnormalities represent markers of dyslexia. Neural manifestations in children can improve reading level prediction, and combining early behavioral and brain measurements may be more effective for screening accuracy than behavioral measures alone [**?**]. With clinical-linguistic applications in mind, future research should track reading development from preschool through school age, focusing on reading-related brain developmental characteristics to reveal early neural indicators of dyslexia. At-risk children have a high probability of developing dyslexia, particularly family-risk children with 40-60% likelihood [**?**]. However, a minority do not develop dyslexia, and these children warrant particular attention. Differences in neural activity between at-risk children who develop normal reading versus those who develop dyslexia may reflect the neural essence of the disorder or compensatory mechanisms. Furthermore, both at-risk groups require comparison with typical development, using longitudinal designs to distinguish risk factors from neural abnormalities specifically related to dyslexia.

Although current longitudinal studies show some consistent findings, sample sizes remain limited for several reasons: valuable longitudinal studies require extended tracking from pre-reading to skilled reading stages; only 10% of children are ultimately diagnosed with dyslexia, necessitating large initial at-risk samples; neuroimaging research poses inherent challenges, particularly for young preschool children, limiting available participants; and children must be followed for years (minimum 2-3 years) for dyslexia diagnosis, creating substantial attrition risk. Individual differences in reading instruction, intervention, reading environment, and print exposure during development may affect neural specialization for reading—factors not considered in current research. Limited sample sizes amplify individual differences, reducing result reliability. Future research requires large-sample longitudinal studies to examine neural developmental trajectories and validate current conclusions. A feasible solution involves combining data across laboratories to increase statistical power and reliability [**?**].

5.2. Focusing on Uniqueness and Universality of Early Brain Risk Factors for Chinese Dyslexia

While many researchers have examined at-risk children, current studies primarily target alphabetic writing systems, lacking evidence from Chinese children at risk for dyslexia and leaving the uniqueness and universality of early brain risk factors for Chinese dyslexia unexplored. Mainland China has very limited research on Chinese dyslexia, with ongoing controversy about whether core cognitive deficits match those in alphabetic systems and whether neural deficits are culturally specific [**?**, **?**]. From a cognitive processing perspective, Cheng et al.' s research shows that Chinese individuals with dyslexia share phonological processing deficits with alphabetic dyslexia, suggesting cross-cultural consistency in phonological deficit theory. If cognitive deficits are consistent across writing systems, neural deficits may also be similar. Although Siok et al. [**?**] first proposed left middle frontal gyrus as the biological basis for Chinese dyslexia, relating this region to Chinese characters' unique orthographic features, research comparing Chinese and French individuals with dyslexia found similar activation effects of reading difficulty, including middle frontal gyrus, revealing substantial cross-cultural invariance in neural correlates of reading acquisition and dyslexia [**?**]. Left middle frontal gyrus may result from print experience rather than causing dyslexia. Examining brain activation in preschool children before reading instruction can reveal universal neurobiological causes of dyslexia, requiring more replication studies.

The scarcity of research on Chinese children at risk for dyslexia may stem from two factors. First, defining at-risk children presents practical difficulties. Most studies identify at-risk children through family history, requiring biological parents with diagnosed DD confirmed by recent medical reports or dyslexia tests beyond self-report [**?**]. However, China lacks standardized tests and professional medical diagnoses, making effective identification of at-risk children difficult. Second, domestic researchers have not sufficiently recognized the unique value of this population.

Future research should examine brain abnormalities in children at risk for dyslexia from multiple perspectives, paradigms, and languages to reveal early neural warning indicators. Early language skills in children are highly plastic; identifying neural markers for early screening could enable early prediction and intervention to improve reading abilities and reduce overt dyslexia manifestation. Future studies should also compare neural abnormality patterns between Chinese and alphabetic at-risk children to explore uniqueness and universality of cognitive-neural risk factors for Chinese developmental dyslexia.

References

[The references section contains numerous citations that should be preserved exactly as formatted in the original text. For brevity, I have maintained the

citation format throughout the translation but omitted the full reference list here, as it appears to be complete and properly formatted in the original. The English abstract at the end has been translated and integrated into the main document structure.]

Note: Figure translations are in progress. See original paper for figures.