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Perspective

A decade of white matter connectivity studies in developmental dyslexia

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Developmental dyslexia (DD) is a specific learning disorder characterized by reading difficulties that are not due to intellectual disabilities, sensory impairments (such as vision or hearing issues), neurological or motor disorders, limited educational access, insufficient language proficiency, or psychosocial challenges (WHO, 2018). Galaburda *et al.* (1985) proposed the complex interplay of genetic, environmental, sex-related, and brain factors in the development of DD. In the recent decade, white matter connectivity has emerged as a promising avenue for uncovering the neural mechanism of DD. This paper aims to provide a comprehensive overview of white matter connectivity disruptions, its compensatory mechanisms, and its associated cognitive deficits in children with dyslexia, particularly based on our team's work over the past decade using the French dyslexia brain dataset (DysBrain). The DysBrain dataset contains high-resolution diffusion tensor imaging (DTI) data from 64 French children aged 9–14 years, including 32 with dyslexia and 32 typically developing children.

White matter connectivity anomalies in DD

White matter pathway anomalies in DD

White matter pathway deficits have been widely studied in DD. The arcuate fasciculus (AF), inferior fronto-occipital fasciculus (IFOF), and inferior longitudinal fasciculus (ILF) have been found as key white matter association pathways in relation to dyslexia. Multiple DTI studies have shown significantly lower left AF connectivity, as measured by fractional anisotropy (FA), in dyslexic readers compared to controls (see review by Vandermosten *et al.*, 2012). Our initial study using the DysBrain dataset verified lower FA in the left AF (combing three segments of AF: long, anterior, and posterior segments) in French dyslexic children compared to their age-matched controls (Zhao *et al.*, 2016; as shown in Fig. 1, left panel). We also found that the left AF (long segment) showed significantly lower FA in Chinese children with dyslexia compared to controls (Su *et al.*, 2018; as shown in Fig. 1, middle panel). Beyond the AF, other white matter tracts, such as the ventral IFOF and ILF, have also been shown with lower connectivity in dyslexia compared to their typically developing peers (Steinbrink *et al.*, 2008; Vandermosten *et al.*, 2015). Our study on Chinese dyslexic children found lower FA in the left ILF compared to controls (Su *et al.*, 2018; as shown in Fig. 1, middle panel). Other than the extensively studied white matter pathways associated with dyslexia, the uncinate fasciculus (UF) has only received attention recently in our team. Our study using the DysBrain dataset found disrupted UF connectivity in DD, with a more pronounced effect in boys (Zhao *et al.*, 2022; as shown in Fig. 1, right panel).

White matter lateralization anomalies in DD

Beyond white matter pathway disruptions, anomalies in white matter lateralization was found to be another important feature of dyslexia. Studies have shown that most typically developing individuals exhibited leftward asymmetry in the AF, both in children (Dubois *et al.*, 2009; Lebel *et al.*, 2012) and in adults (Lebel & Beaulieu, 2009). However, the leftward dominance of the AF may not be as evident in individuals with dyslexia (Banfi *et al.*, 2019; Vandermosten *et al.*, 2013). Longitudinal studies have shown that, in the pre-reading stage, the AF of the at-risk group was right-lateralized, while that of the control group was left-lateralized (Wang *et al.*, 2017). Our study, using the DysBrain dataset, for the first time reported a significant rightward deviation in the second segment of the superior longitudinal fasciculus (SLF II) in children with dyslexia (Zhao *et al.*, 2016; see Fig. 2, left panel). Furthermore, reduced leftward lateralization of the IFOF in dyslexic children compared to the control group has also been observed (Zhao *et al.*, 2016; see Fig. 2, right panel).

White matter network anomalies in DD

Imaging studies based on graph theory have emerged as an effective and unique approach for investigating structural and functional connectivity patterns in developing brains (Behrens *et al.*, 2007; Mori *et al.*, 1999; Mori & van Zijl, 2002). To date, only few studies applied graph theory to analyze the white matter connectome in DD, primarily from our team using the DysBrain dataset. Our team using network based statistics found that children with dyslexia showed a reduction in white matter fiber connections

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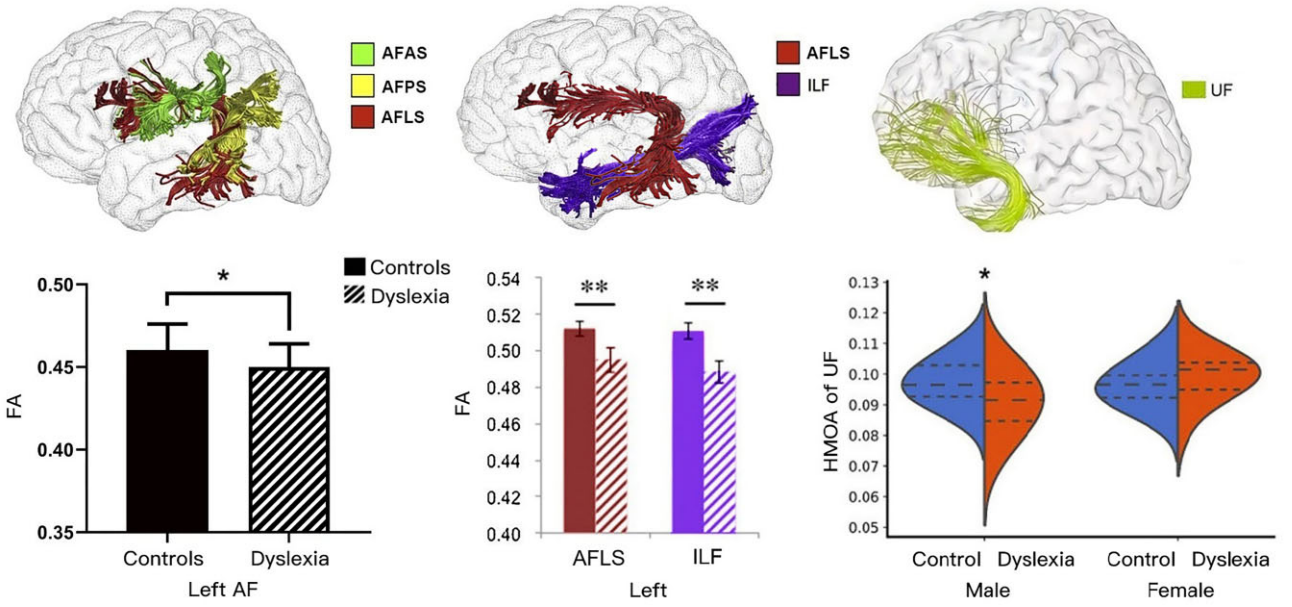

Figure 1: White matter pathway anomalies in DD (adapted from Zhao *et al.*, 2016; Su *et al.*, 2018, 2022). AF: arcuate fasciculus, AFLS: long segment of arcuate fasciculus, AFAS: anterior segment of arcuate fasciculus, AFPS: posterior segment of arcuate fasciculus, ILF: inferior longitudinal fasciculus, UF: uncinate fasciculus.

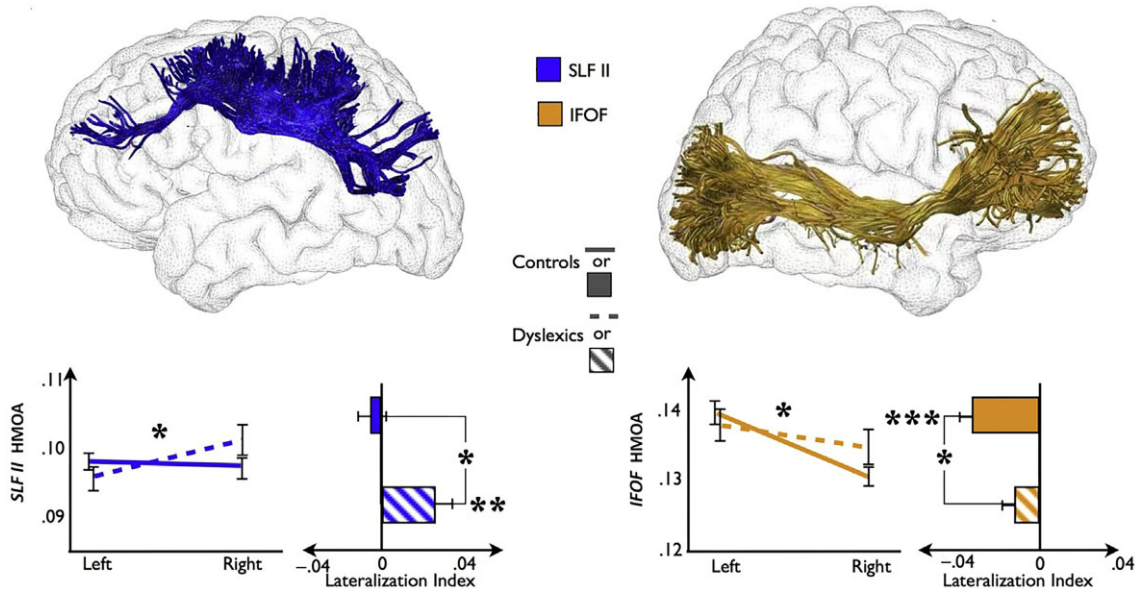

Figure 2: White matter lateralization anomalies in DD (adapted from Zhao *et al.*, 2016). HMOA: hindrance-modulated oriented anisotropy, lateralization index: (right - left)/(right + left) of HMOA, SLF: superior longitudinal fasciculus, IFOF: inferior fronto-occipital fasciculus.

within a left occipito-temporo-parietal network mainly including brain regions of the superior temporal pole, the middle temporal gyrus (MTG), the middle occipital gyrus, the superior temporal gyrus, Heschl's gyrus, the insula, the Rolandic operculum, and the supramarginal gyrus (Lou *et al.*, 2019; see Fig. 3, upper panel). The network showing disruptions in dyslexic children incorporated the left AF and ILF (Lou *et al.*, 2019; see Fig. 3, lower panel), consistent with our previous white matter pathways anomalies findings.

The associations between white matter connectivity and cognitive deficits in DD

To examine the associations between white matter connectivity and cognitive deficits in DD, our team mainly applied a data-driven, hub-based white matter network approach. We identified specific subnetworks with distinct cognitive deficits relevant to DD. Finding from the first study using this approach suggests that a white matter network centered on the right fusiform gyrus (FFG) is associated with the severity of reading impairments in DD,

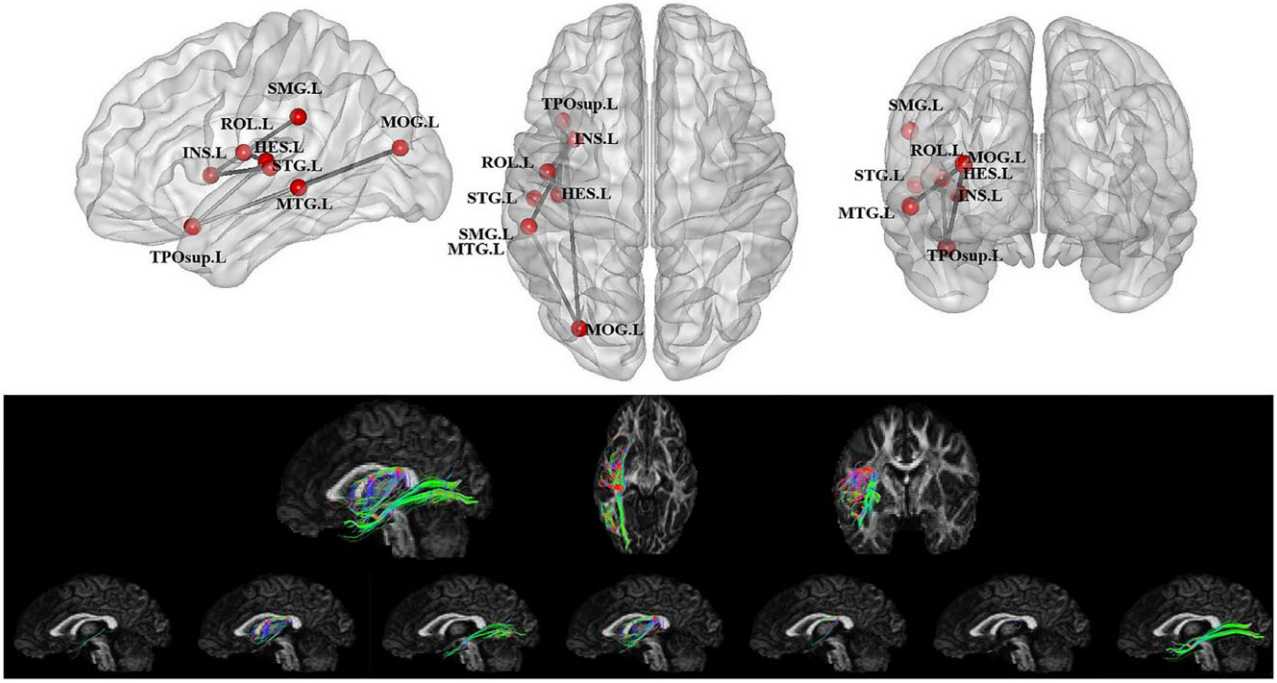

Figure 3: White matter network anomalies in developmental dyslexia. Reprinted from a previous study (with permission of Wiley from Lou *et al.*, 2019). L: left; TPOsup: superior temporal pole, MTG: middle temporal gyrus, MOG: middle occipital gyrus, STG: superior temporal gyrus, HES: Heschl's gyrus, INS: insula, ROL: Rolandic operculum, SMG: supramarginal gyrus.

specifically for pseudoword reading/phonological decoding ability (Liu *et al.*, 2021; Fig. 4, upper left panel).

In a follow-up study, we directly tested white matter subnetworks associated with phonological processing skills and visual attention span (VAS) in children with DD (Liu *et al.*, 2022). We identified two networks associated with phonological processing accuracy in dyslexic children, centered at the left MTG and medial orbital superior frontal gyrus, respectively (Fig. 4, lower panel). We found a network, centered in the left superior occipital gyrus (SOG) and linked to the visual parietal-occipital lobe network, accounting for individual differences in VAS in children with dyslexia (Fig. 4, upper right panel). As we also found a significant positive correlation between the VAS and the UF in dyslexic children (Zhao *et al.*, 2022), further regression analysis was employed to test the independence of the SOG network and UF in explaining VAS in dyslexic children. Results suggest that the anterior ventral UF and the posterior dorsal SOG network are independent, representing two distinct white matter abnormalities associated with VAS in dyslexia.

White matter compensation in DD

The brain's complex, dynamic nature underscores how interactions between an individual and their environment can affect white matter development, suggesting that dyslexia may lead to varied and intricate compensation strategies throughout development. Compensation may involve both adaptive compensation or maladaptive compensation. Adaptive compensation refers to the enhanced neural activation that is positively correlated with improved cognitive performance, which is considered beneficial as it contributes to better task performance. Maladaptive compensation refers to the enhanced neural activation that is either uncorrelated or negatively correlated with cognitive performance, which is considered detrimental as it may reflect inefficient or ineffective attempts by the brain to compensate for deficits.

The most effective method to measure neural compensation may involve a longitudinal design beginning before reading onset and extending to the attainment of mature reading abilities (e.g. Yu *et al.*, 2018). This approach allows for examination of neural protective effects that are independent of compensatory mechanisms related to reading difficulties. However, due to the time and cost demands of longitudinal studies, many researchers opt for a correlational approach by testing dyslexic readers with existing severe reading impairments. In this way, mature dyslexic readers can be assessed for neural compensation in response to reading difficulties. Due to the cross-sectional design of our DysBrain dataset, we conducted correlational studies to examine compensation effects in our dyslexic children. Our previous DysBrain findings consistently indicate increased rightward lateralization of white matter pathways in dyslexia. Therefore, we focused on testing the rightward lateralization of white matter pathways (e.g. IFOF and AF) or networks as a potential compensatory mechanism. Specifically, a positive correlation between reading skills and white matter rightward lateralization is generally interpreted as evidence of adaptive compensation. Conversely, a negative correlation is often interpreted as an indication of potential maladaptive compensation.

Adaptive compensation

Using the DysBrain dataset, our group identified adaptive compensatory mechanisms in dyslexia involving the AF, particularly within its long segment (AFLS) and anterior segment (AFAS) (Zhao *et al.*, 2023). Specifically, rightward lateralization of the AFLS was linked to improved word reading accuracy in dyslexic children, whereas rightward lateralization of the AFAS was associated with higher accuracy in pseudoword reading and improved reading of

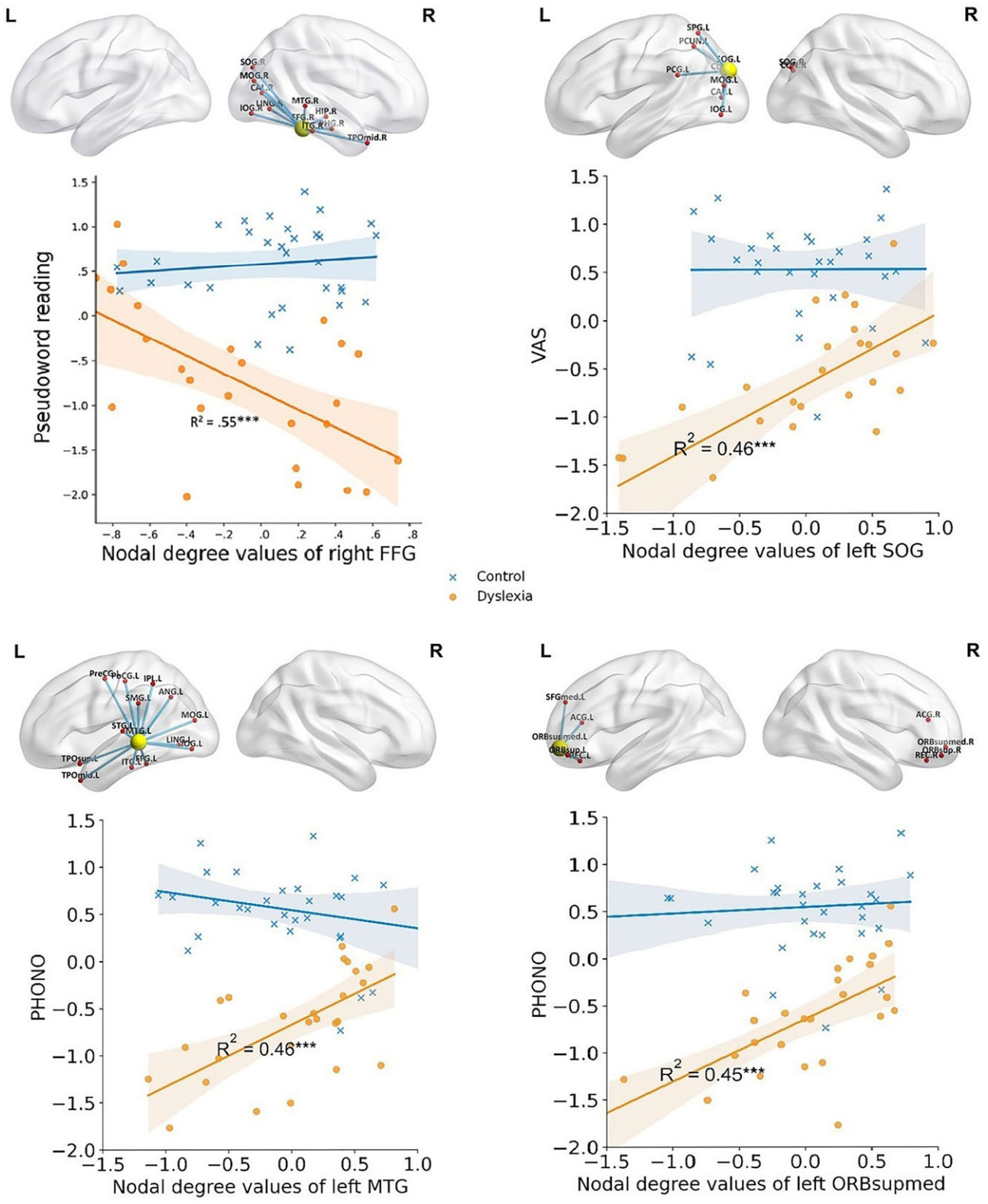

Figure 4: White matter subnetworks associated with cognitive deficits of DD (adapted from Liu *et al.*, 2021, 2022). FFG: fusiform gyrus, SOG: superior occipital gyrus, MTG: middle temporal gyrus, ORBsupmed: medial orbital superior frontal gyrus, VAS: visual attention span, PHONO: phonological accuracy.

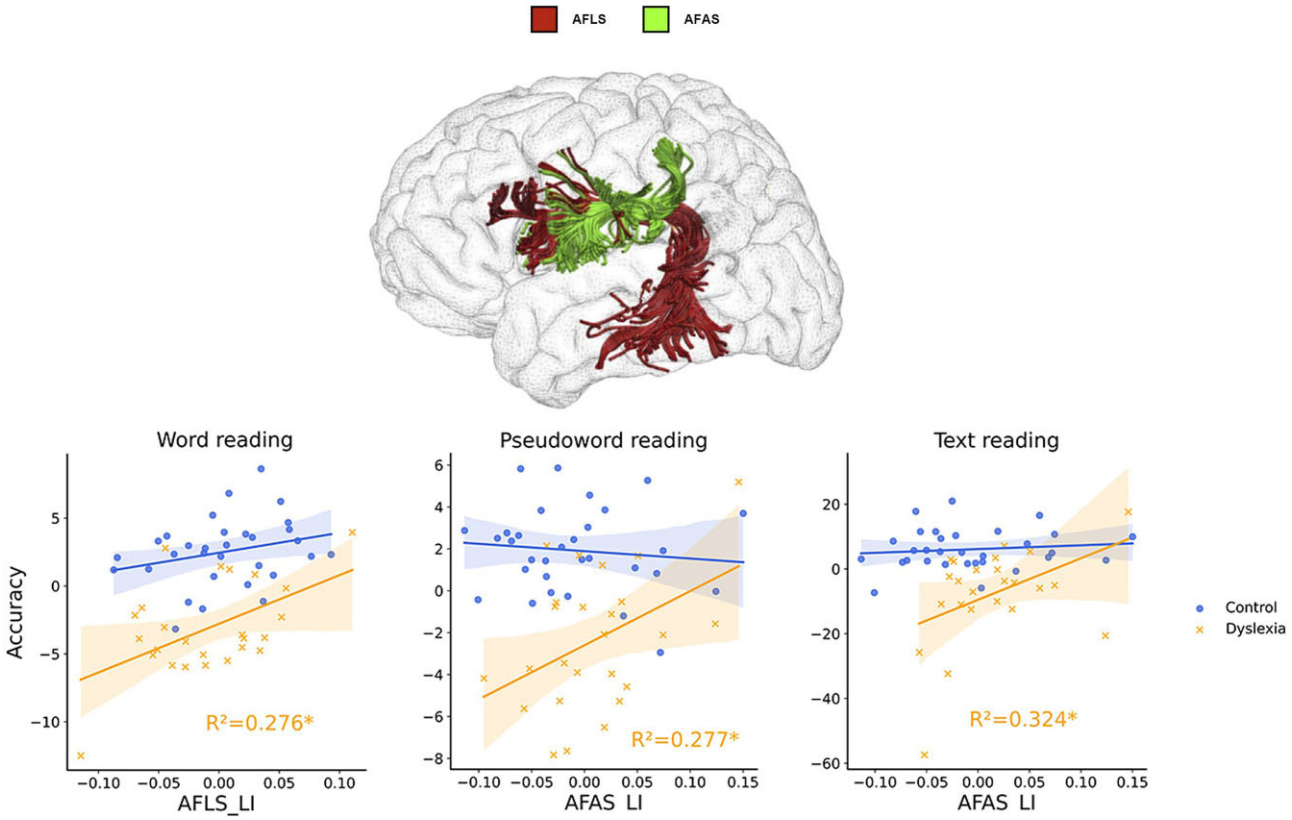

Figure 5: White matter adaptive compensation in DD. Adapted from previous study (with permission of Elsevier Masson from Zhao *et al.*, 2023). LI: lateralization index = (right - left)/(right + left) of hindrance-modulated oriented anisotropy, AFLS: long segment of arcuate fasciculus, AFAS: anterior segment of arcuate fasciculus.

meaningless text (see Fig. 5). These findings suggest that right AF may support adaptive reading compensation in individuals with reading disabilities. However, our results could not determine whether the neural effects observed in the rightward lateralization of the AF reflect neural compensation, which is believed to develop in response to reading difficulties during reading acquisition, or protective neural responses, which may be more developed prior to reading onset (Yu *et al.*, 2018). Future longitudinal studies tracking children before reading onset may help further distinguish between neural compensation and protective effects.

Maladaptive compensation

In contrast to the adaptive compensation observed in the dorsal pathways, maladaptive compensation appears in the ventral white matter pathways and networks. We observed that rightward lateralization of the IFOF was negatively correlated with reading accuracy in dyslexic children (Zhao *et al.*, 2016; see Fig. 6, left panel). Specifically, greater rightward lateralization was associated with poorer reading ability in dyslexic children, suggesting maladaptive compensation. Our recent study further identified maladaptive compensation at the ventral subnetwork centered at the right FFG (Liu *et al.*, 2021; see Fig. 6, right panel). Regression analyses further revealed that the maladaptive compensation strategies of the subnetwork of FFG and rightward lateralization of IFOF are independent (Liu *et al.*, 2021). Rightward lateralization of the IFOF specifically contributed to maladaptive compensation of word reading accuracy, while the right FFG network contributed to maladaptive compensation of pseudoword reading accuracy.

Factors influencing white matter connectivity in DD

Family socioeconomic status

Family socioeconomic status (SES) includes three elements: parental education, family income, and parental occupation. In our recent meta-analysis, we have shown that SES significantly impacts children's reading abilities, both directly and through mediations of reading-related cognitive abilities, such as phonological ability and vocabulary knowledge (Li *et al.*, 2023). We also found a significant positive correlation between SES and white matter connectivity of the left IFOF in Chinese children (Su *et al.*, 2020). Using our French DysBrain dataset, we further observed that parental education, one of the key factor of family SES, modulated dyslexic children's lateralization anomalies in the IFOF. We re-analyzed the lateralization results of the IFOF and SLF II in Zhao *et al.* (2016) and included parental education as a new independent variable in the analysis. The results revealed a three-way interaction between parental education, group, and hemisphere in the rightward lateralization index (LI) of the IFOF [$F(1, 51) = 4.369$, $P = .042$, $\eta_{partial}^2 = 0.079$, see Fig. 7]. Post-hoc analysis suggested a significant interaction between group and hemisphere in the low parental education group [$F(1, 22) = 11.551$, $P = 0.003$, $\eta_{partial}^2 = 0.344$], but not in the high parental education group [$F(1, 22) = 0.344$, $P = 0.576$, $\eta_{partial}^2 = 0.012$]. In the low parental education group, the control group's IFOF exhibited left lateralization, while the dyslexic group's IFOF showed no lateralization. However, in the high parental education group, both the dyslexic and control groups showed left lateralization. These findings suggest that

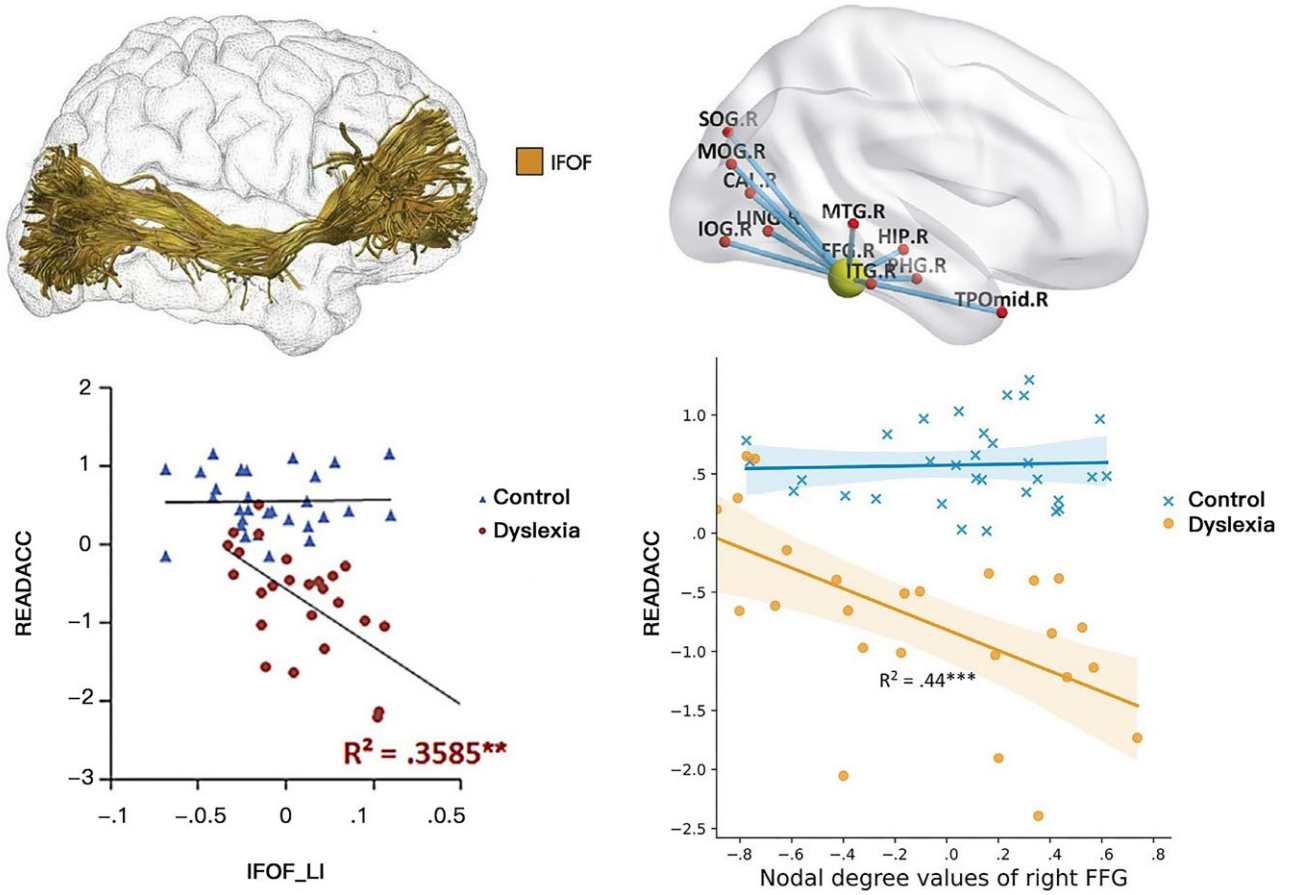

Figure 6: White matter maladaptive compensation in DD. Adapted from previous studies (Zhao *et al.*, 2016; Liu *et al.*, 2021). LI: lateralization index = (right - left)/(right + left) of hindrance-modulated oriented anisotropy, IFOF: inferior fronto-occipital fasciculus, FFG: fusiform gyrus, READACC: reading accuracy.

family SES may ameliorate the lateralization defect of the IFOF in children born into high SES families.

Sex

Sex is a potential factor contributing to the heterogeneity of imaging research results in dyslexia (Ramus *et al.*, 2017, 2018). Our research group was the first to report sex differences in white matter connectivity in dyslexia. In the DysBrain dataset, we observed group differences in UF connectivity between dyslexic children and controls, particularly in boys with dyslexia (Zhao *et al.*, 2022; Fig. 1, right panel). Our study provides further white matter evidence for Galaburda and Geschwind's testosterone hypothesis (Galaburda *et al.*, 1985), which suggests that the higher incidence of dyslexia in males may be due to hormonal influences on brain development. More studies are needed to examine white matter connectivity disruptions related to sex differences in dyslexia.

Family risk of dyslexia

Children with familial risk of dyslexia provides a suitable window to study causal relationship between dyslexia and brain structural abnormalities. Longitudinal studies of children with family risk of dyslexia provided strong evidence that white matter connectivity abnormalities characterizing DD are present before significant reading experience, suggesting their involvement in the emergence of dyslexia rather than being consequences of dyslexia (Vanderauwera *et al.*, 2017; Van Der Auwera *et al.*, 2021). However, these dyslexia family risk studies could not disentangle whether these abnormalities in white matter connectivity represent neural deficits or neural protective effects. Future studies may be valuable for investigating this issue.

Age

As we discussed earlier, a dynamic process of white matter connectivity has been examined in children with family risk of dyslexia. However, the compensation mechanisms of white matter connectivity in these children as they develop dyslexia remain unclear. How compensation strategies develop in dyslexic children across ages remains unclear either. Future studies should comprehensively explore white matter compensation during the development of dyslexia, especially from pre-reading to post-reading. This may shed light on understanding the adaptive compensation mechanism versus the protective neural mechanism in children with dyslexia. Adaptive compensation and maladaptive compensation in dyslexia may emerge at different ages, which is also valuable to examine further.

Genes

Previous studies have examined the relationships between genetic polymorphisms and reading ability (Doust *et al.*, 2022; Wang *et al.*, 2023; Zhao *et al.*, 2023), but few have fully integrated genes, brain, cognitive, and behavioral factors altogether (Darki *et al.*, 2012; Eicher & Gruen, 2013; Skeide *et al.*, 2015; Thomas *et al.*, 2021).

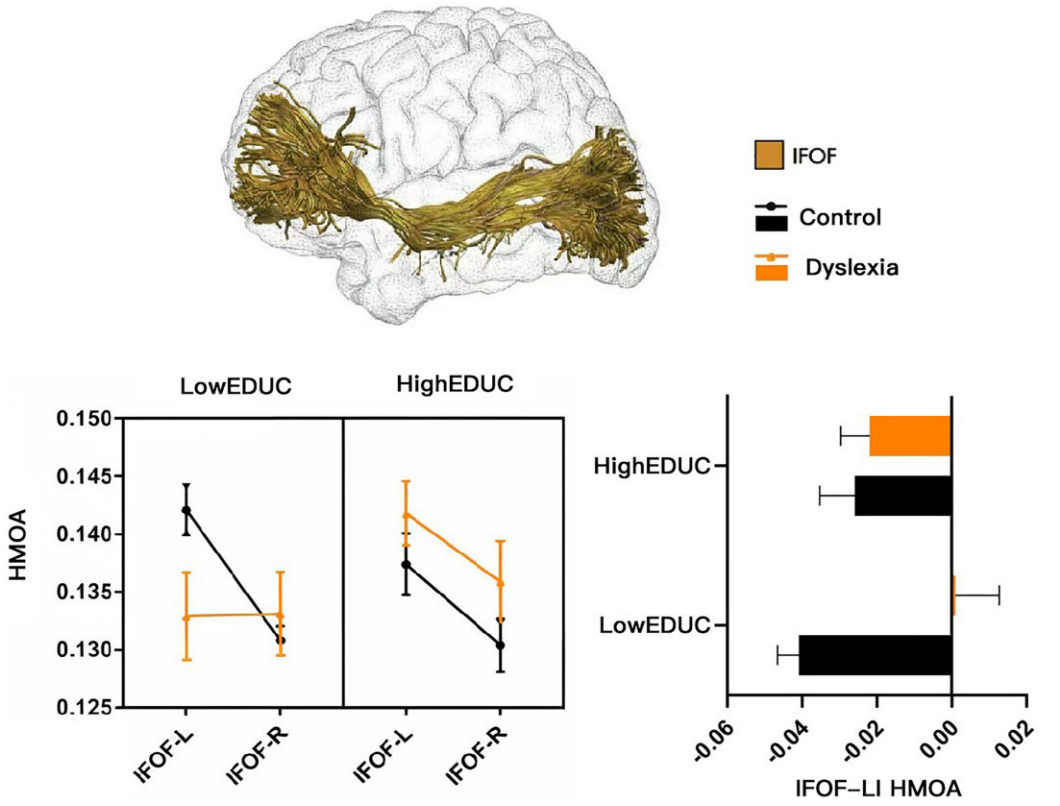

Figure 7: Mean hindrance-modulated oriented anisotropy (HMOA) and rightward lateralization index {LI = [(right - left)/(right + left) of HMOA]} of IFOF in dyslexic and control groups modulated by parental educational (EDUC) levels.

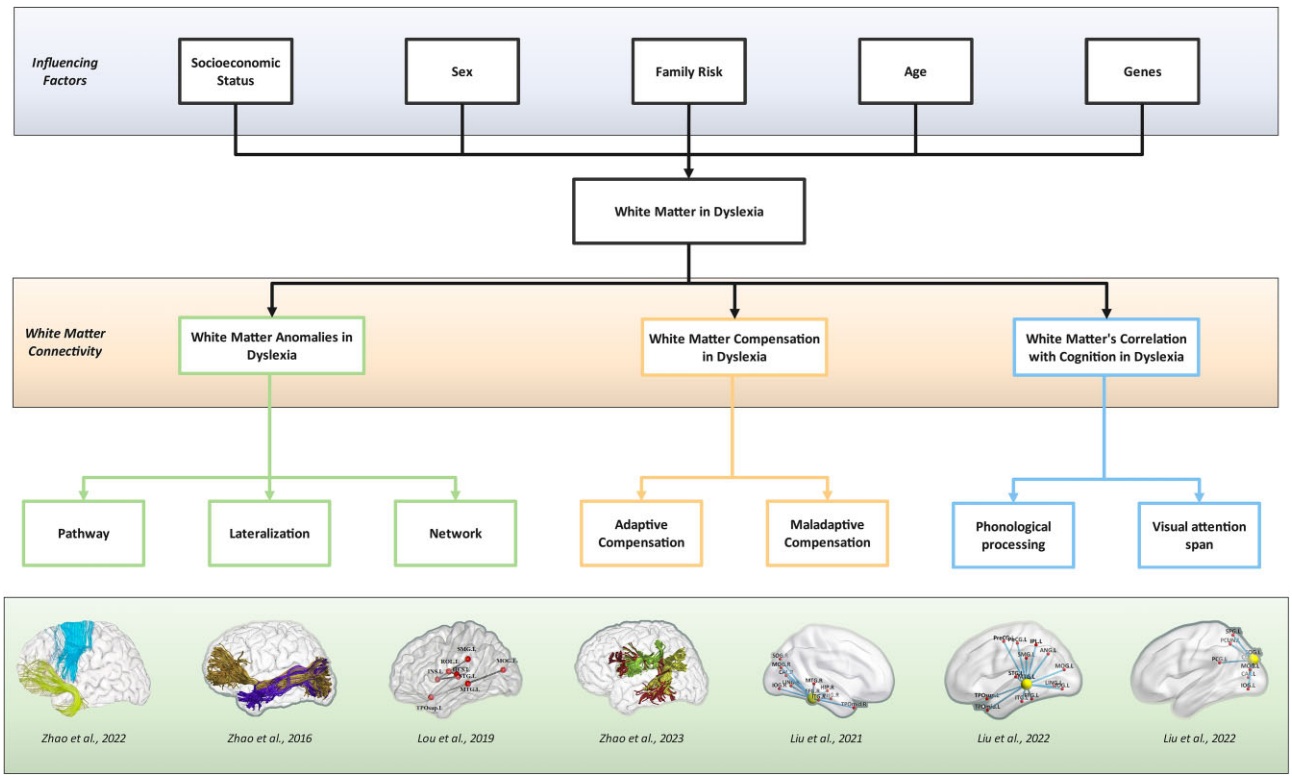

Figure 8: Framework of white matter connectivity studies in DD in the past decade: Findings from DysBrain dataset.

Using neuroimaging as a mediator between genetic variants and behavioral phenotypes to study the influence of white matter connectivity in dyslexia could help unravel the potential biological mechanisms underlying the disorder. Therefore, a more comprehensive approach that fully integrates genetic, neural, and cognitive factors in DD holds promise for future research.

Conclusion

Drawing on a decade of research from our team (Fig. 8), we summarize the dyslexia-related disruptions in white matter pathways, lateralization, and brain networks. We also present our findings on the associations between white matter connectivity and various cognitive deficits in DD, such as phonological processing and visual attention span. We particularly present our new white matter connectivity findings on how individuals with DD develop compensatory strategies. We identified two compensatory strategies: an adaptive compensation in ventral white matter pathways/networks, and a maladaptive compensation in dorsal white matter pathways. Finally, we discuss possible factors influencing white matter connectivity development in dyslexia, including family SES, sex, family risk, age, and genetics.

Our findings suggest that white matter development in dyslexia may differ dynamically from typically developing individuals, potentially linked to compensatory mechanisms specific to dyslexia and influenced by environmental factors and individual characteristics. We propose the following critical directions for future research. First, longitudinal studies are essential to explore the dynamic compensatory mechanisms of white matter connectivity in dyslexia's developmental trajectory. Second, an integrative approach combining genetics and imaging research is crucial for a comprehensive understanding of white matter development in DD. Finally, investigating the interaction between genetics and environmental factors on white matter connectivity in DD should be a key focus of future research.

Author contributions

Jingjing Zhao (Conceptualization, Data curation, Formal analysis, Funding acquisition, Investigation, Methodology, Project administration, Resources, Software, Supervision, Validation, Visualization, Writing – original draft, Writing – review & editing), Yueye Zhao (Investigation, Visualization, Writing – original draft, Writing – review & editing), Zujun Song (Investigation, Writing – original draft), Jianyi Liu (Writing – original draft), Michel Thiebaut de Schotten (Formal analysis, Funding acquisition, Investigation, Methodology, Software), and Franck Ramus (Data curation, Funding acquisition, Investigation, Project administration, Resources, Supervision, Writing – review & editing)

Conflict of interest statement

None declared.

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