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# Neural dissociation of visual attention span and phonological deficits in developmental dyslexia: A hub-based white matter network analysis

Tianqiang Liu[1] | Michel Thiebaut de Schotten[2,3] | Irene Altarelli[4,5] | Franck Ramus[5] | Jingjing Zhao[1]

[1]School of Psychology, Shaanxi Normal University, and Key Laboratory for Behavior and Cognitive Neuroscience of Shaanxi Province, Xi'an, China

[2]Groupe d'Imagerie Neurofonctionnelle, Institut des Maladies Neurodégénératives-UMR 5293, CNRS, CEA, University of Bordeaux, Bordeaux, France

[3]Brain Connectivity and Behaviour Laboratory, Sorbonne Universities, Paris, France

[4]LaPsyDÉ laboratory (UMR 8240), Université Paris Cité, Paris, France

[5]Laboratoire de Sciences Cognitives et Psycholinguistique (ENS, EHESS, CNRS), Département d'Etudes Cognitives, Ecole Normale Supérieure, PSL University, Paris, France

**Correspondence**
Jingjing Zhao, School of Psychology, Shaanxi Normal University, 199 South Chang'an Road, Xi'an 710062, China.
Email: jingjing.zhao@snnu.edu.cn

Franck Ramus, Laboratoire de Sciences Cognitives et Psycholinguistique (ENS, EHESS, CNRS), Ecole Normale Supérieure-PSL, 29 rue d'Ulm, Paris 75005, France.
Email: franck.ramus@ens.psl.eu

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## Abstract

It has been suggested that developmental dyslexia may have two dissociable causes—a phonological deficit and a visual attention span (VAS) deficit. Yet, neural evidence for such a dissociation is still lacking. This study adopted a data-driven approach to white matter network analysis to explore hubs and hub-related networks corresponding to VAS and phonological accuracy in a group of French dyslexic children aged from 9 to 14 years. A double dissociation in brain-behavior relations was observed. Structural connectivity of the occipital-parietal network surrounding the left superior occipital gyrus hub accounted for individual differences in dyslexic children's VAS, but not in phonological processing accuracy. In contrast, structural connectivity of two networks: the temporal–parietal-occipital network surrounding the left middle temporal gyrus hub and the frontal network surrounding the left medial orbital superior frontal gyrus hub, accounted for individual differences in dyslexic children's phonological processing accuracy, but not in VAS. Our findings provide evidence in favor of distinct neural circuits corresponding to VAS and phonological deficits in developmental dyslexia. The study points to connectivity-constrained white matter subnetwork dysfunction as a key principle for understanding individual differences of cognitive deficits in developmental dyslexia.

**KEYWORDS**
developmental dyslexia, hub, phonological deficit, visual attention span, white matter network

## 1 | INTRODUCTION

Developmental dyslexia is characterized by significant and persistent difficulties in reading acquisition with prevalence rates ranging from 1.3% to 17.5% (Di Folco et al., 2021; Shaywitz & Shaywitz, 2005). It is a common learning disorder that cannot be accounted for by intellectual disability, sensory (vision or hearing) or neurological impairment, lack of availability of education, or psychosocial adversity (ICD-11; World Health Organization, 2019).

Dyslexia has been widely acknowledged as a neurodevelopmental disorder with specific neural origins (Ramus et al., 2018). There are a plenty of neuroimaging studies that have investigated the "neurobiological biomarkers" of dyslexia. Yet, dyslexic brains vary in the pattern of their cortical abnormalities, which suggests that there might be several ways to become dyslexic, depending on which subset of the reading-related cognitive networks is affected (Ramus, 2004). In other words, there seem to be different neural pathways involved in the huge variability in the reading deficits observed in children with dyslexia. Focal cortical abnormalities of dyslexic individuals may disrupt the development of the particular cognitive function(s) that would normally recruit those areas. For example, Vandermosten et al. (2012) demonstrated a specific relation between the integrity of left arcuate fasciculus (AF) and performance on phoneme awareness and speech perception, and between left inferior fronto-occipital fasciculus and orthographic processing in dyslexia. The current study aimed to explore whether different neural pathways are involved in two important deficits in children with dyslexia—phonological deficit and visual attention span (VAS) deficit.

There are various cognitive theories about the underlying causes of dyslexia. The most influential theory of dyslexia is the phonological deficit hypothesis, which states that the core deficit of developmental dyslexia lies in phonological abilities: the representation, storage, or retrieval of speech sounds (Ramus, 2003; Shankweiler & Liberman, 1972; Vellutino, 1979). Evidence in support of the phonological theory includes results showing that dyslexic individuals perform particularly poorly on tasks requiring phonological awareness, i.e. conscious segmentation and manipulation of speech sounds, and retrieval of phonological representation, i.e. rapid automatized naming (Landerl et al., 2013; Ramus et al., 2003; Saksida et al., 2016). Poor phonological abilities lead to poor performances in grapheme-phoneme mapping, and further impact reading acquisition and performance (Ramus & Szenkovits, 2008).

The left frontal and temporal–parietal areas have been highlighted as the key brain regions related to phonological deficit in dyslexia. Many studies have reported abnormal activation (Finn et al., 2014; Paulesu et al., 2014; Pugh et al., 2000; Richlan et al., 2009), altered brain morphometry (Altarelli et al., 2013; Altarelli et al., 2014; Eckert et al., 2016; Frye et al., 2010; Richlan et al., 2013), and changes of fractional anisotropy (FA) (Deutsch et al., 2005; Klingberg et al., 2000; Vandermosten et al., 2019; Vandermosten, Boets, Wouters, & Ghesquiere, 2012) in the left inferior frontal gyrus and the left temporo-parietal areas in dyslexic participants compared with controls, as well as altered functional connectivity (Boets et al., 2013; Finn et al., 2014; Schurz et al., 2015) and lower structural connectivity of the tracts connecting these regions (Lou et al., 2019; Su et al., 2018; Vandermosten, Boets, Poelmans, et al., 2012; Zhao et al., 2016). However, replication of all these results remains an enduring problem (Ramus et al., 2018).

As reading relies primarily on vision as well as on language, it is only logical that visual deficits should have been postulated as an alternative cause of dyslexia. Indeed, theories implicating deficiencies in the visual system have been the most influential theories of dyslexia in the early part of the 20th century (Vellutino et al., 2004). Nonetheless, in the visual or visual-attentional domain, there is also more than one theory available (Vidyasagar & Pammer, 2010), including the magnocellular theory (Stein & Walsh, 1997), the sluggish attentional shifting theory (Gori & Facoetti, 2015; Hari & Renvall, 2001), visual stress theory (Wilkins et al., 2004), and the VAS theory (Bosse et al., 2007). Given that it was not possible to test all conceivable visual theories of dyslexia, and given the debate between VAS and phonological theory in recent years (Cheng et al., 2021; Saksida et al., 2016; Ziegler et al., 2010), in the present study, we focused on the VAS theory. VAS theory proposes a deficit in dyslexia caused by a severely limited number of elements that can be processed in parallel from a brief visual display (Bosse et al., 2007). Two kinds of stimulus have been widely selected to measure VAS: verbal stimulus (e.g., letter, digit strings) and nonverbal stimulus (e.g., color dots, symbol strings). Valdois and colleagues conducted a series of studies using various stimuli and in different languages, and found that a VAS deficit might be the primary cause of some cases with dyslexia, independently of the phonological deficit (e.g., Bosse et al., 2007; Valdois et al., 2021). However, this independence has been disputed by other investigators. A few studies argue that VAS deficit in dyslexia is not independent from the phonological deficits (Cheng et al., 2021; Saksida et al., 2016; Ziegler et al., 2010). In Ziegler et al. (2010), children with dyslexia showed significant deficits in VAS task for letter and digit strings but not for symbol strings. Similar VAS deficit patterns were also revealed in Chinese dyslexia, indicating visual-to-phonological mapping impairment may be responsible for VAS deficit in dyslexia (Cheng et al., 2021). In addition, researchers also found that most dyslexic children who presented VAS deficits also had phonological deficits (Cheng et al., 2021; Saksida et al., 2016).

A number of studies have reported dysfunction of bilateral superior parietal lobules in dyslexic participants when performing VAS tasks (Lobier et al., 2014; Peyrin et al., 2011; Reilhac et al., 2013; Valdois et al., 2019). The strongest neuroimaging support for an independent VAS deficit in dyslexia might be a case study by Peyrin and colleagues (Peyrin et al., 2012). They reported two cases of dyslexia. One of them showed phonological disorder but preserved VAS, while the other one showed a VAS deficit but intact phonological ability. These two cases showed dissociated brain activation patterns when performing phonological and VAS tasks. The dyslexic case with a phonological deficit showed lower activation in the left inferior frontal gyrus, whereas the dyslexic case with a VAS deficit showed lower

**TABLE 1** Demographic data and behavioral results of visual attention span, phonological accuracy and rapid automatized naming tasks.

| | Control children | | Dyslexic children | | |
|---|---|---|---|---|---|
| | *N* | Mean (SD) | *N* | Mean (SD) | Test statistics |
| *Subject characteristics* | | | | | |
| Sex (male/female) | 31 | 18/13 | 26 | 13/13 | $\chi^2$ (1) = .371, $p$ = .543 |
| Handedness (left/right) | 31 | 2/29 | 26 | 3/23 | $\chi^2$ (1) = .457, $p$ = .499 |
| Age (months) | 31 | 137.90 (16.33) | 26 | 139.27 (15.77) | $t$ (55) = .320, $p$ = .751 |
| Maternal education | 31 | 2.65 (1.38) | 26 | 3.08 (1.80) | $t$ (55) = 1.029, $p$ = .308 |
| Paternal education | 31 | 2.52 (1.61) | 26 | 3.62 (1.92) | $t$ (55) = 2.352, $p$ = .022 |
| Nonverbal IQ | 31 | 110.29 (17.09) | 26 | 106.00 (15.69) | $t$ (55) = .980, $p$ = .332 |
| Verbal IQ | 31 | 123.84 (18.70) | 26 | 107.88 (18.22) | $t$ (55) = 3.246, $p$ = .002 |
| Reading age (months) | 31 | 145.94 (18.65) | 26 | 87.27 (11.43) | $t$ (55) = 13.979, $p$ < .0001 |
| *Visual attention span tasks* | | | | | |
| Global report (letters reported /100) | 31 | 89.58 (7.95) | 25 | 67.24 (12.88) | $t$ (54) = 7.589, $p$ < .0001 |
| Partial report (letters correct /50) | 31 | 44.65 (3.97) | 25 | 38.64 (5.66) | $t$ (54) = 4.486, $p$ < .0001 |
| *Phonological accuracy tasks* | | | | | |
| Phoneme deletion (/24) | 31 | 22.97 (1.38) | 26 | 17.89 (4.77) | $t$ (55) = 5.667, $p$ < .0001 |
| Spoonerism (/12) | 31 | 7.83 (2.56) | 24 | 2.29 (2.73) | $t$ (52) = 7.679, $p$ < .0001 |
| Digit span (WISC scaled score) | 31 | 10.87 (2.68) | 26 | 6.58 (2.18) | $t$ (55) = 6.554, $p$ < .0001 |
| *Rapid automatized naming tasks* | | | | | |
| RAN digits (sec) | 31 | 21.33 (3.19) | 26 | 32.60 (7.62) | $t$ (55) = 7.493, $p$ < .0001 |
| RAN objects (sec) | 31 | 35.86 (6.92) | 26 | 51.23 (9.52) | $t$ (55) = 7.043, $p$ < .0001 |

activation in the left superior parietal lobules. Besides, functional MRI meta-analyses indicate that dyslexia not only involves a ventral dysfunction route in the left temporo-parietal network responsible for phonological processing, but also a dorsal dysfunction route in the left fronto-parietal network responsible for visuo-spatial processing (Paulesu et al., 2014; Richlan et al., 2009). Similarly, recent white matter tractography studies in dyslexia also reported anomalies in both the AF, a white matter pathway connecting the temporo-parietal network (Lou et al., 2019; Vandermosten, Boets, Poelmans, et al., 2012), and the superior longitudinal fasciculus (SLF), a white matter pathway connecting the frontal–parietal network (Zhao et al., 2016). Notably, previous research also revealed some longitudinal changes in structural connectivity of AF, that might differ in children with dyslexia or at-risk of dyslexia and controls, depending on the age of participants (Van Der Auwera et al., 2021; Wang et al., 2017; Yeatman et al., 2012). Thus, there is evidence for structural anomalies in brain areas associated with both ventral and dorsal networks in dyslexia. However, whether these structural anomalies in ventral and dorsal networks (temporo-parietal network vs. frontal–parietal network) in dyslexia are compatible with the hypothesis of independent cognitive deficits corresponding to different brain structures (Vandermosten, Boets, Poelmans, et al., 2012) is remained unclear. These structural anomalies might also be explained by a broader view of the brain networks involved in reading and dyslexia, that would not assume two independent cognitive deficits.

Therefore, the overall goal of the present DTI study was to employ a brain-behavior correlation approach to explore the neural subnetworks corresponding to VAS and phonological skills in a sample of dyslexic children with one and/or the other deficit. We adopted a data-driven approach of hub-based white matter network analysis to identify the hubs and subnetworks associated with VAS and phonological deficits. We specifically aimed to identify whether VAS and phonological deficits in dyslexia relied on a shared neural network or dissociated subnetworks.

## 2 | MATERIALS AND METHODS

### 2.1 | Participants

A total of 26 dyslexic children and 31 age-matched control children (aged 109–169 months) were included in this study. All children were native French speakers with normal vision and hearing abilities and none of them was diagnosed with a history of brain damage, psychiatric, or any other cognitive disorder. Dyslexic children were referred by a clinic for reading and language disabilities and had to present a delay greater than 18 months on text reading age (based on accuracy and speed of the Alouette test, a meaningless text that assesses both reading accuracy and speed [Lefavrais, 1967]) while control children had to be no more than 12 months behind. The two groups were matched on sex, handedness, age, and nonverbal IQ. Demographics for the two groups are shown in Table 1. The study was approved by the ethics committee of Bicetre Hospital and informed consent was obtained from all children and their parents. Analyses of group

differences of white matter pathways and network connectivity, and relation between literacy skills and white matter network parameters with the same sample have been published previously (Liu et al., 2021; Lou et al., 2019; Zhao et al., 2016).

## 2.2 | Behavioral measures

Behavioral assessments included tests to determine each child's intellectual abilities, VAS and phonological abilities. Intellectual abilities were measured using the WISC-IV blocks, matrices, similarities, and comprehension subtests (Wechsler, 2005). The VAS was measured by both global letter report task and partial letter report task (Bosse et al., 2007; Saksida et al., 2016). Phonological skills were estimated by a phoneme deletion task (Sprenger-Charolles et al., 2005), a spoonerism test (Bosse & Valdois, 2009), the WISC digit span subtest (Wechsler, 2005). Phonological processing speed was estimated by rapid automatized naming (RAN) tasks for digits and objects (Plaza & Robert-Jahier, 2006). Parental education was recorded as the highest diploma obtained, coded on a 1–6 scale, from 1: postgraduate diploma to 6: neither high-school diploma nor professional certificate. Handedness was based on children's writing hand.

For the purpose of hub-based white matter subnetwork analyses, we defined three composite measures by averaging z scores as follows (in keeping with our previous study (Zhao et al., 2016)): VAS from global and partial letter report; phonological processing accuracy (PHONO) from phoneme deletion, spoonerisms, and digit span; and phonological processing speed from rapid automatized naming (RAN) of digit and object. Signs were adjusted such that positive z-scores represented above-average performance.

## 2.3 | Image acquisition

All children were scanned on a 3 T MRI system (Tim Trio, Siemens Medical Systems, Erlangen, Germany), equipped with a whole-body gradient (40 m T/m, 200 T/m/s) and a 32-channel head coil. For T1-weighted structural MRI scans, a MPRAGE sequence with following parameters: acquisition matrix $= 230 \times 230 \times 224$, repetition time (TR) $= 2300$ ms, echo time (TE) $= 3.05$ ms, flip angle $= 9°$, field of view (FOV) $= 230$ mm, voxel size $= 0.9 \times 0.9 \times 0.9$ mm$^3$ was used. For diffusion MRI scans, a spin-echo single-shot EPI sequence was used, with parallel imaging (GRAPPA reduction factor 2), partial Fourier sampling (factor 6/8), and bipolar diffusion gradients to reduce geometric distortions. The whole brain was imaged with an isotropic spatial resolution of 1.7 mm$^3$ (matrix size $= 128 \times 128$, field of view $= 218$ mm), and 70 interleaved axial slices. Diffusion gradients were applied along 60 orientations, uniformly distributed, with a diffusion weighting of $b = 1400$ s/mm$^2$ (repetition time $= 14{,}000$ msec, echo time $= 91$ msec). Additionally, three images were acquired with no diffusion gradient applied ($b = 0$). Each sequence took about 6 min, resulting in a total acquisition time of 18 min.

## 2.4 | Diffusion tensor imaging analysis

Processing of the diffusion MRI dataset was implemented using a pipeline toolbox, PANDA (Cui et al., 2013) (http://www.nitrc.org/projects/panda), programmed based on FSL6.0.1 (http://fsl.fmrib.ox.ac.uk/fsl/fslwiki). Raw DICOM files were firstly converted to 4D NIFTI files using dcm2nii tool embedded in MRIcron. A brain mask for each individual was then generated by removing the skull to extract the brain tissues. Head motion was corrected using eddy-current method by registering the diffusion-weighted images to the *b*0 image with affine transformation of diffusion gradient direction adjusted accordingly. FA metrics of each individual were calculated by fitting diffusion tensors to the native head-motion corrected diffusion-weighted image of each participant. Then, each individual's FA image in the native space was normalized to the MNI space using a standard FA template (FMRIB58_FA). Finally, whole brain tractography was performed using the deterministic fiber tracking method employed by Continuous Tracking (FACT) algorithms (http://trackvis.org/dtk) with the angle threshold of 45° and the FA threshold of $0.2 \sim 1$.

## 2.5 | Network node definition

Nodes of the white matter network were defined by automated anatomical labeling (AAL) atlas (Tzourio-Mazoyer et al., 2002). According to the AAL atlas, the whole-brain gray matter (excluding cerebellum) of each child was divided into 90 regions of interest (ROIs). To obtain better brain extraction results, a utility in PANDA named *Brain Extraction (T1)* with parameters of *eye & optic nerve clean up* and *bias field & neck cleanup* was used to extract T1 images. The T1 image of each individual was then coregistered with the FA image in the diffusion tensor imaging (DTI) space using a linear transformation. Then, the transformed T1 image was normalized to the ICBM152 template in the MNI space using a nonlinear transformation. Finally, the AAL mask from the MNI space was warped to the DTI native space using the resulting inverse transformation.

## 2.6 | Defining the backbone network in the control group children

To identify the highly consistent cortical connections, we firstly computed the backbone network in the control group according to Gong's method (Gong et al., 2009). A nonparametric one-tailed sign test was applied with a null hypothesis of no existing connection for each pair of cortical nodes (fiber bundle number $= 0$). Results were Bonferroni corrected for multiple comparisons ($C_{90}{}^2 = 4005$ pairs of regions). Finally, a symmetric binarized matrix with 396 tracts survived the threshold (Figure S1). The sparsity of the resultant network (9.89%) was similar to that reported in previous adult studies (Chen et al., 2020; Gong et al., 2009).

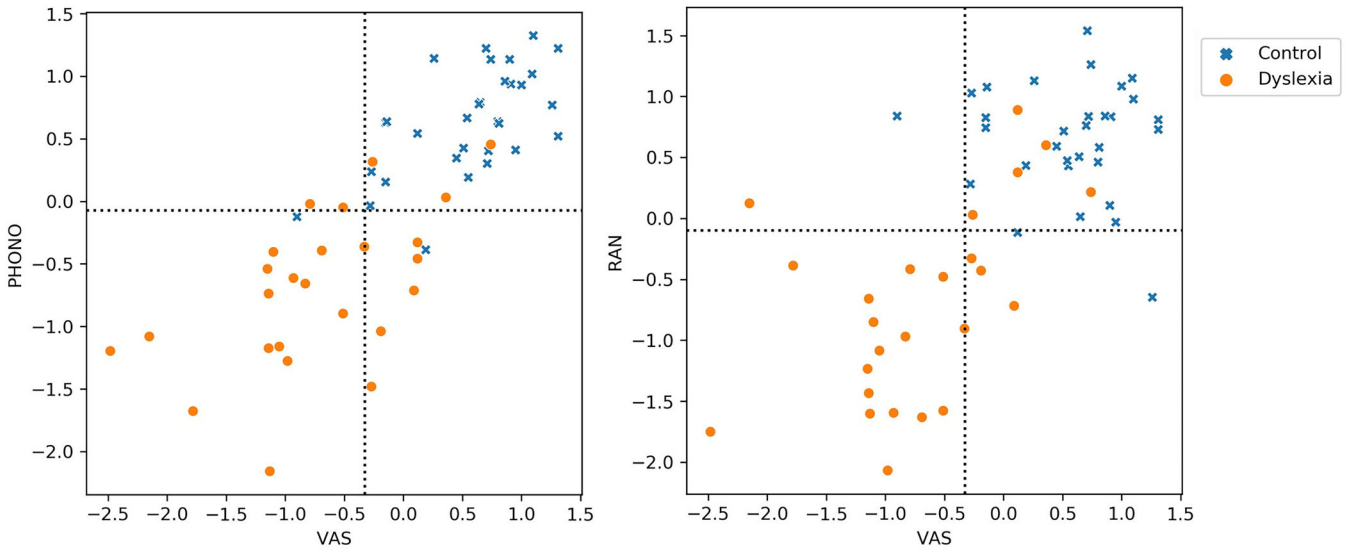


**FIGURE 1** Individual scatter plots showing the distribution of dyslexic and control children according to their visual attention span (VAS), phonological accuracy (PHONO), and rapid automatized naming (RAN). Dotted lines correspond to the 1.65 standard deviations below the mean values of the control group.

### 2.7 | Identifying anatomical hubs in dyslexia

Using the backbone network mask that we identified in the control group, we then extracted the mean FA of the backbone network in both dyslexic and control groups and computed the nodal degree of each AAL region, as the sum FA values of all the edges connected to the node. To identify neural hubs for the VAS and phonological deficits in dyslexia, partial correlation between each AAL nodal degree and the behavioral *z* scores were performed in the dyslexic group and the control group respectively: VAS for VAS hubs, PHONO for phonological accuracy hubs, and RAN for phonological processing speed hubs, by regressing out the effects of sex, handedness, age, and parental education level. Results were corrected for multiple testing with Bonferroni correction ($p < .05/90 = .00056$).

## 3 | RESULTS

### 3.1 | Behavioral results

Descriptive statistics for behavioral measures for dyslexic and control groups are shown in Table 1. As expected, dyslexic children had a significantly worse performance than controls on all measures of VAS, phonological processing accuracy, and phonological processing speed. In order to identify the overlap between VAS and phonological deficits in developmental dyslexia, deficit thresholds were calculated by applying a cutoff criterion of 1.65 standard deviations below the control group's mean z score of each component. Scatter plots representing individual performance in VAS, PHONO, and RAN are shown in Figure 1, revealing substantial comorbidity between VAS and phonological deficits (PHONO or RAN) in dyslexic children.

### 3.2 | Hub regions

The partial correlation analyses between the z scores of VAS, PHONO, RAN, and the FA nodal degree values of the 90 AAL regions yielded three significant correlations with Bonferroni correction ($p < .05/90 = .00056$) in the dyslexic group (see a full correlation table in supplementary Table S1). Positive correlations were observed between the left superior occipital gyrus (SOG) and the VAS score ($r = 0.68$, $p < .0005$) as well as between the left middle temporal gyrus (MTG) and the left medial orbital superior frontal gyrus (ORBsupmed) and the PHONO score (left MTG: $r = 0.68$, $p < .0005$; left ORBsupmed: $r = 0.67$, $p < .0005$) in the dyslexic group. RAN tasks for the dyslexic group showed no significant correlation with any hub (see supplementary Table S1). No significant correlation was found in the control group either (see supplementary Table S1).

For each significant region/hub, we have plotted the connections in Figure 2 (top panel). The left SOG hub is connected to nine regions, including the left superior parietal gyrus (SPG), middle occipital gyrus (MOG), calcarine fissure and surrounding cortex (CAL), inferior occipital gyrus (IOG), precuneus (PCUN), posterior cingulate gyrus, CUN, and the right SOG and CUN, as shown in Figure 2 (left top). The left MTG is connected to 13 regions, including the left superior temporal gyrus (STG), supramarginal gyrus (SMG), angular gyrus (ANG), fusiform gyrus (FFG), temporal pole: STG (TPOsup), temporal pole: middle temporal gyrus (TPOmid), inferior temporal gyrus (ITG), MOG, IOG, lingual gyrus, inferior parietal gyrus, precentral gyrus (PreCG), and postcentral gyrus, as shown in Figure 2 (middle top). The left ORBsupmed is connected to eight regions, including the left superior frontal gyrus: medial (SFGmed), superior frontal gyrus: orbital part (ORBsup), anterior cingulate and paracingulate gyri (ACG), and rectus gyrus (REC), and the right ACG, superior frontal gyrus: medial orbital (ORBsupmed), superior frontal gyrus: orbital part (ORBsup), and REC,

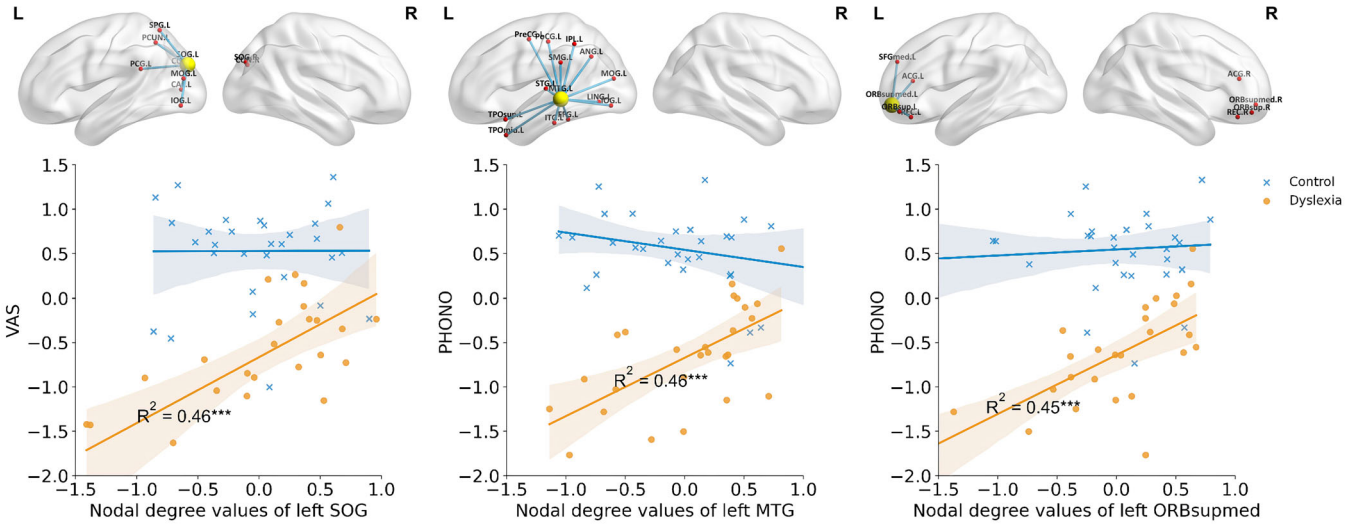


**FIGURE 2** Left-top: The hub and subnetwork corresponding to visual attention span (VAS)–left superior occipital gyrus (SOG), connected with the left superior parietal gyrus (SPG), middle occipital gyrus (MOG), calcarine fissure and surrounding cortex (CAL), inferior occipital gyrus (IOG), precuneus (PCUN), posterior cingulate gyrus (PCG), cuneus (CUN), and the right superior occipital gyrus (SOG) and cuneus (CUN). Left-bottom: Scatterplot of the correlation between residuals of the nodal degree values of the left SOG and residuals of the VAS score (controlling for sex, handedness, age, and parental education level). Middle-top: The hub and subnetwork corresponding to phonological processing accuracy (PHONO)–left middle temporal gyrus (MTG), connected with the left superior temporal gyrus (STG), supramarginal gyrus (SMG), angular gyrus (ANG), fusiform gyrus (FFG), temporal pole: Superior temporal gyrus (TPOsup), temporal pole: Middle temporal gyrus (TPOmid), inferior temporal gyrus (ITG), middle occipital gyrus (MOG), inferior occipital gyrus (IOG), lingual gyrus (LING), inferior parietal gyrus (IPL), precentral gyrus (PreCG), and postcentral gyrus (PoCG). Middle-bottom: Scatterplot of the correlation between residuals of the nodal degree values of the left MTG and residuals of the PHONO score (controlling for sex, handedness, age, and parental education level). Right-top: The hub and subnetwork corresponding to phonological processing accuracy (PHONO)–left ORBsupmed, connected with the left superior frontal gyrus: Medial (SFGmed), superior frontal gyrus: Orbital part (ORBsup), anterior cingulate and paracingulate gyri (ACG), and rectus gyrus (REC), and the right anterior cingulate and paracingulate gyri (ACG), superior frontal gyrus: Medial orbital (ORBsupmed), superior frontal gyrus: Orbital part (ORBsup), and rectus gyrus (REC). Right-bottom: Scatterplot of the correlation between residuals of the nodal degree values of the left ORBsupmed and residuals of the PHONO score (controlling for sex, handedness, age, and parental education level).

as shown in Figure 2 (right top). A scatter plot of the correlation between the nodal degree of the left SOG and VAS is shown in Figure 2 (left bottom). A scatter plot of the correlation between the nodal degree of the MTG and PHONO is shown in Figure 2 (middle bottom). A scatter plot of the correlation between the nodal degree of the left ORBsupmed and PHONO is shown in Figure 2 (right bottom).

### 3.3 | Regression analyses

In order to further assess the white matter subnetwork dissociation between VAS and phonological processing accuracy deficits in the dyslexic group, we carried out hierarchical linear regression analyses. VAS and PHONO scores of the dyslexic children were entered into the model as dependent variables respectively. Sex, handedness, age, and education were included in the regression analyses in the first step. Nodal degree values of the three hubs of the dyslexic group were entered in the second step simultaneously.

As shown in Table 2, when sex, handedness, age, and education variables were controlled for, only the nodal degree of the left SOG was a significant predictor of VAS ($\beta = .576$, $p = .026$) in the dyslexic group, while the nodal degrees of the left MTG and the left ORBsupmed were not significantly associated with VAS ($\beta = .047$, $p = .829$; $\beta = .126$, $p = .541$). In contrast, phonological processing accuracy was significantly associated with nodal degrees of the left MTG ($\beta = .551$, $p = .004$) and the left ORBsupmed ($\beta = .518$, $p = .004$) in the dyslexic group, but not with the left SOG ($\beta = -.207$, $p = .270$).

## 4 | DISCUSSION

Using a data-driven hub-based white matter subnetwork approach, we have shown that distinct white matter hubs and subnetworks corresponded to VAS and phonological processing performance in developmental dyslexia. Brain-behavior analyses reveal three distinct hubs: one, located in the left SOG and connecting with visual occipital-parietal subnetworks, accounts for individual differences in VAS but not in phonological processing in dyslexic children; two other hubs account for phonological processing accuracy but not VAS: one in the left middle temporal gyrus connecting with temporal-occipital-parietal subnetworks, and one in the medial orbital superior frontal gyrus connecting with frontal subnetworks. Our findings therefore suggest that

**TABLE 2** Hierarchical regression models of visual attention span and phonological accuracy predicted by the nodal degree values of the left superior occipital gyrus (SOG), middle temporal gyrus (MTG), and medial orbital superior frontal gyrus (ORBsupmed)

| | | Visual attention span | | | | | | Phonological accuracy | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | Unstandardized coefficients | | Standardized coefficients | | | | Unstandardized coefficients | | Standardized coefficients | |
| Step | | $\Delta R^2$ | Adjusted $R^2$ | *B* | SE | Beta | *p* | $\Delta R^2$ | Adjusted $R^2$ | *B* | SE | Beta | *p* |
| 1 | Control variables | 0.141 | −0.031 | | | | | 0.154 | −0.007 | | | | |
| | Age | | | .003 | .010 | .069 | .755 | | | .005 | .008 | .137 | .520 |
| | Sex | | | −.071 | .341 | −.048 | .837 | | | .004 | .267 | .003 | .989 |
| | Handedness | | | −.702 | .539 | −.303 | .207 | | | .089 | .422 | .047 | .835 |
| | Parental education | | | −.034 | .048 | −.153 | .482 | | | −.065 | .038 | −.359 | .098 |
| 2 | Nodal degree FA values | 0.406[a] | 0.360[a] | | | | | 0.563[c] | 0.607[c] | | | | |
| | Left SOG | | | .640 | .263 | **.576**[a] | **.026** | | | −.186 | .163 | −.207 | .270 |
| | Left MTG | | | .062 | .281 | .047 | .829 | | | .584 | .175 | **.551**[b] | **.004** |
| | Left ORBsupmed | | | .160 | .257 | .126 | .541 | | | .533 | .159 | **.518**[b] | **.004** |

*Note*: $\Delta R^2$ is the $R^2$ change at each hierarchical step.

[a] $p < .05$.

[b] $p < .01$.

[c] $p < .001$.

VAS and phonological deficits in developmental dyslexia are subtended by distinct neural networks.

The present work represents a methodological advance over previous diffusion studies in dyslexia by applying a data-driven hub-based network analysis approach to examine white matter subnetworks corresponding to visual and phonological deficits in developmental dyslexia. The hub-based subnetwork approach allowed us for the first time to identify hubs whose structural connectivity is associated with cognitive skills that are deficient in dyslexia, namely phonological deficit and VAS deficit. That is, to test how connectivity of structural networks surrounding hub regions are involved in a specific cognitive deficit in dyslexia.

One of the main results of the present study was that we observed an independent subnetwork corresponding to VAS but not phonological skills in dyslexic children. This subnetwork included a hub in the left SOG, and connections from the hub to the parietal and occipital cortices including the left SPG, PCUN, SOG, CAL, and IOG. It is consistent with previously reported results in functional MRI studies suggesting that in typical readers, when performing visual-attentional tasks, activation mainly distributed in the bilateral superior parietal lobules (SPLs), in particular the left superior parietal lobule; in contrast, dyslexic individuals showed less activation in SPL in VAS tasks (Lobier et al., 2014; Peyrin et al., 2011, 2012; Reilhac et al., 2013; Valdois et al., 2019). In particular, our results provide a complement to the two-case functional neuroimaging study by Valdois and colleagues indicating independent neural bases corresponding to VAS deficit in dyslexia (Peyrin et al., 2012). In sum, we provide the first piece of anatomical evidence for a distinct white matter network associated with VAS deficit in dyslexia.

Concerning subnetworks specifically corresponding to phonological deficits in dyslexia, our results showed significant correlations between phonological accuracy and subnetworks based on hubs of left MTG and left ORBsupmed in dyslexic children. These subnetworks included regions of the left STG, SMG, ANG, FFG, and SFG. These results are largely consistent with well-established previous findings that functional and structural connectivity of frontal and temporal subnetworks are involved in pseudoword reading and/or phonological processing (Boets et al., 2013; Vandermosten, Boets, Poelmans, et al., 2012), and are significantly decreased in dyslexic individuals (Klingberg et al., 2000; Vandermosten, Boets, Poelmans, et al., 2012). Although the present study was not focused on the reconstruction of white matter tracts, we speculate that the subnetworks for phonological processing may include the left AF, inferior longitudinal fasciculus (ILF), part of the SLF and the inferior fronto-occipital fasciculus. Disruption of these tracts has previously been reported to impact phonological processing and reading in dyslexic participants (Lou et al., 2019; Vandermosten, Boets, Poelmans, et al., 2012; Yeatman et al., 2012; Zhao et al., 2016). Overall, results from our study are consistent with previous findings and provide new information regarding individual differences of brain-behavior correlations in the phonological processing subnetwork of dyslexic children.

One limitation of the present study may be that our study is correlational. We therefore cannot rule out the possibility that a

phonological deficit initially induces reading disability, which in turn delays the development of VAS. Similarly, it cannot be ruled out that a VAS deficit might in turn engender relatively poor phonological performance, through the induced delay in reading acquisition. Only longitudinal studies of phonological processing ability, VAS, and reading ability would allow disentangling the causal pathways. Another limitation might be the fact that the majority of participants showed double deficits in our study. A stronger representation of pure VAS deficit and pure phonological deficit groups would have been desirable. This would have allowed us to better assess the correspondence between neural and cognitive dissociation. Unfortunately, the prevalence of cases with pure deficits was not under our control, and indeed such cases have previously been found to be quite rare (Cheng et al., 2021; Saksida et al., 2016). Finally, cognitive interventions targeting different subtypes of dyslexia to further test the correspondence between those specific neural subnetworks and the associated cognitive skills in dyslexia are needed to validate this hypothesis.

Overall, our study suggests that the severity of the VAS deficit and the phonological accuracy deficit in dyslexic children is associated with connectivity disruptions in distinct white matter subnetworks, located surrounding the hub of left SOG and the hubs of the left middle temporal gyrus and superior frontal gyrus, respectively. These results are consistent with the hypothesis of VAS and phonological deficits as two dissociable causes of reading disability, each with its own neural basis.

## AUTHOR CONTRIBUTIONS

Jingjing Zhao conceived of the presented idea. Irene Altarelli and Franck Ramus collected the behavioral, MRI, and DTI data. Tianqiang Liu and Jingjing Zhao analyzed the behavioral data, performed the MRI and DTI analyses, and wrote the main manuscript. Michel Thiebaut de Schotten and Franck Ramus verified the analytical methods and supervised the findings of this work.

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## CONFLICT OF INTEREST

No conflicts of interest.

## DATA AVAILABILITY STATEMENT

Data will be made available on reasonable request.

## ORCID

*Jingjing Zhao* https://orcid.org/0000-0001-9000-6227

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## SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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