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Research Article

Investigation of white matter functional networks underlying different behavioral profiles in attention-deficit/hyperactivity disorder

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Abstract

Background Cortical functional network alterations have been widely accepted as the neural basis of attention-deficit/hyperactivity disorder (ADHD). Recently, white matter has also been recognized as a novel neuroimaging marker of psychopathology and has been used as a complement to cortical functional networks to investigate brain–behavior relationships. However, disorder-specific features of white matter functional networks (WMFNs) are less well understood than those of gray matter functional networks. In the current study, we constructed WMFNs using a new strategy to characterize behavior-related network features in ADHD.

Methods We recruited 46 drug-naïve boys with ADHD and 46 typically developing (TD) boys, and used clustering analysis on resting-state functional magnetic resonance imaging data to generate WMFNs in each group. Intrinsic activity within each network was extracted, and the associations between network activity and behavior measures were assessed using correlation analysis.

Results Nine WMFNs were identified for both ADHD and TD participants. However, boys with ADHD showed a splitting of the inferior corticospinal–cerebellar network and lacked a cognitive control network. In addition, boys with ADHD showed increased activity in the dorsal attention network and somatomotor network, which correlated positively with attention problems and hyperactivity symptom scores, respectively, while they presented decreased activity in the frontoparietal network and frontostriatal network in association with poorer performance in response inhibition, working memory, and verbal fluency.

Conclusions We discovered a dual pattern of white matter network activity in drug-naïve ADHD boys, with hyperactive symptom-related networks and hypoactive cognitive networks. These findings characterize two distinct types of WMFN in ADHD psychopathology.

Keywords: attention-deficit/hyperactivity disorder; white matter function; brain network; resting-state fMRI; clustering; psychopathology

Introduction

Attention-deficit/hyperactivity disorder (ADHD) is a common neurodevelopmental disorder diagnosed in school-age children and adolescents with a worldwide prevalence of 3.4% (Polanczyk *et al.*, 2015). Numerous neuroimaging studies have outlined the role of cortical functional network abnormalities observed by resting-state functional magnetic resonance imaging (rs-fMRI) (Gao *et al.*, 2019; Sutcubasi *et al.*, 2020). For example, poor top-down control over inhibition and attention may be due to functional changes in the fronto-striato-cerebellar network while impulsivity symptoms are mainly associated with altered functional connectivity in the reward network (Faraone *et al.*, 2015).

As white matter connects gray matter regions and supports effective communication across brain networks, white matter function as detected by blood oxygen level–dependent (BOLD) signals from rs-fMRI has recently been demonstrated to have physiological significance (Ji *et al.*, 2017). The rationale for these studies depends on the fact that white matter also has the vascular capacity to support spiking-induced hemodynamic changes that can be detected by rs-fMRI, albeit on a smaller scale than hemodynamic changes in gray matter (Gawryluk *et al.*, 2014). In addition, some studies (Ding *et al.*, 2018; Li *et al.*, 2019b) further revealed that the intensity of the BOLD signal in white matter could be modulated by task stimulation and was associated with neural activity.

More recent studies have begun to demonstrate that there are reliable and stable white matter functional networks (WMFNs) that resemble gray matter functional networks in organization and small-world topology, and spatially overlap with several major white matter tracts (Peer *et al.*, 2017; Li *et al.*, 2019a; Wang *et al.*, 2022). WMFN disturbances have been reported in several neuropsychiatric disorders, such as schizophrenia and epilepsy (Jiang *et al.*, 2019a; Jiang *et al.*, 2019b; Fan *et al.*, 2020). In addition, the topological properties of WMFNs can predict the severity of depressive symptoms and discriminate depressive patients from

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controls, yielding potential clinical value (Li *et al.*, 2020). Therefore, WMFNs provide important complementary information on how intrinsic neural activity in white matter interacts and organizes itself; this information is useful for decoding human behavior and cognition, which can provide insight into the underlying neural mechanisms of psychopathology. Researchers usually define WMFNs using a clustering approach in healthy participants and patients combined. While this method amplifies the sample size and increases the statistical power of the clustering algorithm, it may obscure the unique features in patients and compromise the ability to reflect disorder-specific networks. In addition, when clustering is performed on a combined group of patients and controls with greatly imbalanced numbers, the results may heavily rely on the group with a large sample size and lead to bias.

Heterogeneity may be the most challenging of the current issues in the field of neuroimaging. Previous results of neuroimaging studies on ADHD were usually affected by gender, drug effect, illness course, and symptom profiles. For example, we found that white matter microstructure differs between boys and girls with ADHD (Lin *et al.*, 2020; Lin *et al.*, 2022). Medication is another commonly seen as a confounding factor, as both stimulants and nonstimulants, such as atomoxetine, have modulatory effects on intrinsic brain activity in patients with ADHD (Pereira-Sanchez *et al.*, 2021). Illness course and developmental trajectory are also factors that may interfere with the results. Studies conducted on patients in late adolescence or early adulthood are less likely to capture various neural substrates, since various neural processes can occur during childhood and early adolescence (Shaw and Sudre, 2021; Sudre *et al.*, 2021).

In light of the combined evidence, we decided to explore the organization of WMFNs in boys with and without ADHD as two separate groups. We recruited 46 medication-naïve boys with ADHD and the same number of typically developing (TD) boys (age range 7–16 years) to exclude the influence of drugs and gender. The current study bears the advantage of homogeneity of participants and exclusion of drug and gender effects. We hypothesized that ADHD would exhibit characteristic WMFNs when clustered separately and that altered functional networks would be associated with specific clinical symptoms.

Methods and Materials

Participants

All participants were recruited from the Mental Health Center, West China Hospital of Sichuan University, from June 2009 to July 2013. In total, 105 participants, including 55 boys with ADHD and 50 TD boys, were recruited in this study. The ADHD diagnoses were given according to the DSM-IV by an experienced psychiatrist. TD boys underwent screening using the Structured Clinical Interview for DSM-IV Non-patient Edition, and their first-degree relatives reported no history of psychiatric illness. All participants included were right-handed, confirmed by Annett's Hand Preference Questionnaire, and had a full-scale IQ above 80. The exclusion criteria for all participants were: (i) any current or past history of other psychiatry disorders; (ii) any history of neurologic disorders or neurosurgery; and (iii) any history of treatment for ADHD or other psychotropic medications. We excluded 11 children (ADHD = 7, TD = 4) with translation >2 mm and rotation >2° during scanning and two ADHD children with failed fMRI scanning. A final sample composed of 46 boys with ADHD and 46 TD boys was analyzed. See Table 1 for demographic and clinical details.

Prior to study, legal guardians provided written informed consent. Study approval was given by the ethics committee of West China Hospital of Sichuan University.

Clinical, behavioral and cognitive assessment

We used the Chinese version of the Conners' Parent Rating Scale (CPRS) to characterize ADHD symptoms. It includes 48 items and comprises six subscales: Conduct Problems, Psychosomatic, Anxiety, Study Problems, Hyperactivity–Impulsivity, and Hyperactivity Index.

The Chinese version of the Child Behavior Checklist (CBCL) was used to assess comprehensive behavior problems. This instrument, which records a wide range of behavioral problems and competencies of children aged 4 to 16 years, includes the following eight subscales: withdrawal, somatic complaints, anxiety/depression, social problems, thought problems, attention problems, rule-breaking behavior, and aggressive behavior.

A set of neuropsychological tests was applied to evaluate multiple aspects of executive function, including the Stroop color–word test, Wisconsin Card Sorting Test (WCST), Digit Span Test, and Verbal Fluency Test. Details about the neuropsychological tests are provided in the Supplementary Methods.

Image acquisition

Imaging data were acquired on a 3 T Siemens Trio scanner with an eight-channel head coil. T1-weighted images were acquired by using a magnetization-prepared rapid gradient-echo sequence with the following parameters: repetition time/echo time = 1900/2.5 ms; inversion time = 900 ms; flip angle = 9°; matrix = 256 × 256; field of view = 256 × 256 mm; number of sagittal slices = 176; and slice thickness = 1 mm. The resting-state fMRI images were acquired by using a 7-minute gradient-echo echo-planar functional imaging sequence with the following parameters: repetition time/echo time = 2000/30 ms; flip angle = 90°; matrix size = 64 × 64; field of view = 240 × 240 mm, number of axial slices = 30; slice thickness = 5 mm with no slice gap; and voxel size = 3.75 × 3.75 × 5 mm^3. The participants were asked to stay still and close their eyes but avoid thinking of anything or falling asleep during scanning.

Image preprocessing

Preprocessing was performed using DPABI (http://rfmri.org/dpabi) and open MATLAB scripts (https://www.neuropsychiatrylab.com/codes_wm). First, removing the first five volumes of fMRI scans. Next, slice-timing correction and realignment were performed. T1 images were then coregistered to the functional images, and then segmented into white matter, gray matter, and cerebrospinal fluid using DARTEL. Linear trends were removed from functional images to correct for signal drift. The mean cerebrospinal fluid signals and 24-parameter motion parameters (six rigid-body motion parameters, their values at the previous time point and the 12 corresponding squared values) were regressed out from functional data. The white matter and global brain signals were not regressed out to avoid elimination of important neural signals. To minimize the potential effects of head motion, we also applied temporal scrubbing using motion 'spikes' (framewise displacement >0.2 mm) as separate repressors to effectively censor the data at the spike. Bandpass filtering (0.01–0.08 Hz) was performed to reduce minimize high-frequency physiological noise. Subsequently, spatial smoothing (4-mm FWHM) on the white matter and gray matter separately to avoid mixing their signals. Finally, the functional images were then spatially normalized into the Montreal

Table 1: Demographic and clinical features of the participants.

Variable	ADHD ($n = 46$)	TD boys ($n = 46$)	t	P value
Age	10.14 (2.38)	10.93 (2.21)	1.66	0.1
Sex				
boy	46	46		
Conners' Parent Rating Scale				
Conduct problem	0.98 (0.62)	0.44 (0.42)	4.9	<0.001
Psychosomatic	0.23 (0.28)	0.07 (0.15)	3.48	0.001
Anxiety	0.38 (0.39)	0.43 (0.44)	−0.63	0.54
Study problem	1.80 (0.69)	0.78 (0.69)	7.04	<0.001
Impulsivity–hyperactivity	1.41 (0.79)	0.52 (0.49)	6.47	<0.001
Hyperactivity index	1.35 (0.59)	0.53 (0.51)	7.1	<0.001
Children Behavior Checklist				
Withdrawn	3.59 (3.03)	3.26 (3.44)	48	0.63
Somatic complaints	1.32 (1.83)	0.89 (1.56)	1.23	0.22
Anxiety/depressive	5.15(4.43)	2.30 (3.26)	3.51	0.001
Social problems	5.00 (3.00)	2.67 (2.50)	3.99	<0.001
Thought problems	2.17 (2.53)	0.50 (0.81)	4.27	<0.001
Attention Problems	9.48 (3.68)	3.61 (3.18)	8.19	<0.001
Rule-breaking behavior	5.04 (3.37)	1.98 (1.82)	5.43	<0.001
Aggressive behavior	14.07 (7.79)	5.35 (4.56)	6.48	<0.001
Stroop color–word test				
Interference time (s)	182.25 (92.61)	101.11 (36.65)	5.52	<0.001
Wisconsin Card Sorting Test				
Total correct matches	29.63 (10.52)	35.11 (5.87)	−3.08	0.003
Categories completed	4.30 (1.95)	5.22 (1.43)	−2.56	0.012
Total errors	16.33 (12.35)	10.02 (7.31)	2.97	0.004
Perseverative errors	4.87 (5.34)	1.91 (2.09)	3.5	0.001
Nonperseverative errors	11.46 (8.10)	8.11 (5.64)	2.29	0.025
Digit Span	7.20 (2.02)	8.13 (1.64)	−2.45	0.016
Verbal Fluency Test	17.83 (7.76)	18.83 (5.14)	−0.73	0.47

Neurologic Institute space by T1 segmentation and were resampled into $3 \times 3 \times 3$ mm^3. We also analyzed the head motion differences between boys with ADHD and TD boys using a two-sample t test, and the result showed no difference in head motion between the two groups (Supplementary Table 1).

Construction of WMFNs

In this study, we used a data-driven approach, clustering, to construct WMFNs separately for the ADHD and TD groups. The analysis pipeline and details were described in previous studies (Peer *et al.*, 2017; Bu *et al.*, 2020). First, white matter masks were created for two groups separately to select voxels for clustering. Specifically, only voxels identified as white matter and contained functional data across >80% of participants were retained. Then, a voxel-wise Pearson's correlation matrix of white matter was computed for each participant and was averaged for each group to create two group-level correlation matrixes. Finally, K-means clustering was performed separately on the two correlation matrixes to identify the clusters of white matter voxels with similar functional connectivity patterns. The number of clusters ranged from 2 to 22, and the stability of clustering was evaluated by Dice's coefficient (the threshold was set at 0.90).

Activity of WMFNs

Amplitudes at each frequency were extracted using Fourier transform from each white matter network for each participant. The frequency–power graph for each network was produced by averaging across participants in each group. For each white matter network, the averaged amplitudes were obtained for each participant to represent the network activity and were then used to perform a two-sample t test. We used Bonferroni correction to address multiple comparisons across several white matter networks. $P < 0.05$ was used to determine significance.

Correlations between WMFNs and clinical measures

We further examined the associations between significant network activity and several clinical measures (CPRS scores, CBCL scores, and cognitive function scores) by Pearson correlation analysis. We used false discovery rate (FDR) correction to address multiple correlation analysis. $P < 0.05$ was used to determine significance.

Results

Organization of WMFNs

Using K-means clustering on the white matter correlation matrixes, we identified nine stable and reasonable white matter functional networks for ADHD and TD boys according to Dice's coefficient (Fig. 1). Then, we qualitatively defined the white matter networks according to the correspondence between our white matter networks and the known resting-state gray matter networks (Yeo *et al.*, 2011). The identified networks were the default-mode network (DMN), the somatomotor network (SMN), the dorsal

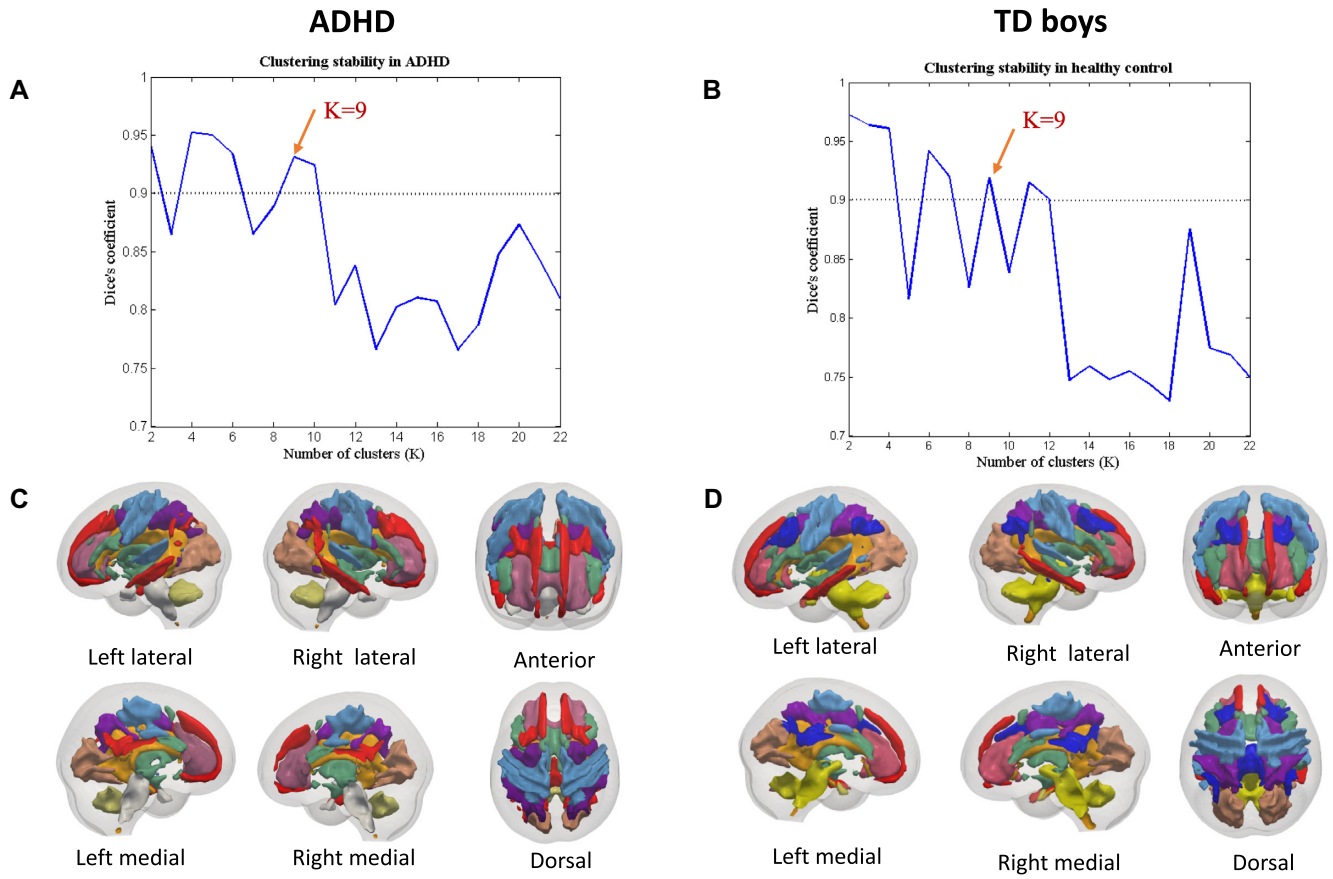

Figure 1: Stable white matter functional networks for ADHD and TD boys according to Dice's coefficient: (A, C) for ADHD and (B, D) for TD boys.

attention network (DAN), the visual network, the deep frontoparietal network (FPN), the deep frontal network, the frontostriatal network, the inferior corticospinal–cerebellar network, and the cognitive control network (CCN). Notably, compared with TD boys, the inferior corticospinal–cerebellar network in ADHD split into two subnetworks, and the CCN was not present in ADHD. The detailed organization of the nine networks is presented in Fig. 2.

Activity of WMFNs

The amplitudes in WMFNs are shown in Fig. 3. Most networks displayed a decreasing trend in amplitude with increased frequency, suggesting greater activity in white matter at lower frequencies, except for the DAN and CCN in TD boys exhibiting consistent activity across the whole frequency.

To compare the network activity between the two groups, we combined the amplitudes of two subnetworks in ADHD, namely the inferior corticospinal network and cerebellum network. Compared with TD boys, boys with ADHD showed increased amplitudes in the SMN, DAN, and visual network (Fig. 3A) but reduced amplitudes in the deep FPN, frontostriatal network, and inferior corticospinal–cerebellar network (Fig. 3B).

Correlation between WMFNs and clinical phenotypes

We next explored the relationship of altered network activity with symptoms, behavior, and cognitive function across all participants. We found that increased activity of the SMN correlated positively with the hyperactivity–impulsivity score ($r = 0.25$, P-FDR corrected $= 0.004$) and the hyperactivity index ($r = 0.33$, P-FDR corrected $= 0.007$) in the CPRS; increased activity of the DAN correlated positively with study problems in the CPRS ($r = 0.37$, P-FDR corrected $= 0.003$) and attention problems in the CBCL ($r = 0.45$, P-FDR corrected < 0.001) (Fig. 4A, B).

On the other hand, decreased activity of the deep FPN and frontostriatal network was linked to deficits in cognition (including poor inhibitory control), working memory, and verbal fluency (Fig. 4C, D): activity of the deep FPN was correlated negatively with interference time in the Stroop test ($r = -0.34$, P-FDR corrected $= 0.003$), and positively with the digit span ($r = 0.28$, P-FDR corrected $= 0.042$) and verbal fluency score ($r = 0.30$, P-FDR corrected $= 0.009$); similarly, activity of the frontostriatal network correlated negatively with interference time in the Stroop test ($r = -0.34$, P-FDR corrected $= 0.003$), and positively with the digit span ($r = 0.26$, P-FDR corrected $= 0.042$) and verbal fluency score ($r = 0.31$, P-FDR corrected $= 0.009$).

Discussion

In the current study, we characterized the functional organization and activity of white matter networks in boys with and without ADHD based on BOLD signals from white matter. The current study yielded two main findings. First, nine WMVNs were identified for both ADHD and TD boys; however, ADHD showed the breakdown of the inferior corticospinal–cerebellar network and the lack of CCN compared with TD boys. Second, compared with TD boys, ADHD boys showed higher amplitudes of BOLD signals in the SMN, DAN, and visual network and lower amplitudes in the deep FPN, frontostriatal network and inferior corticospinal network. Moreover, the SMN and DAN positively

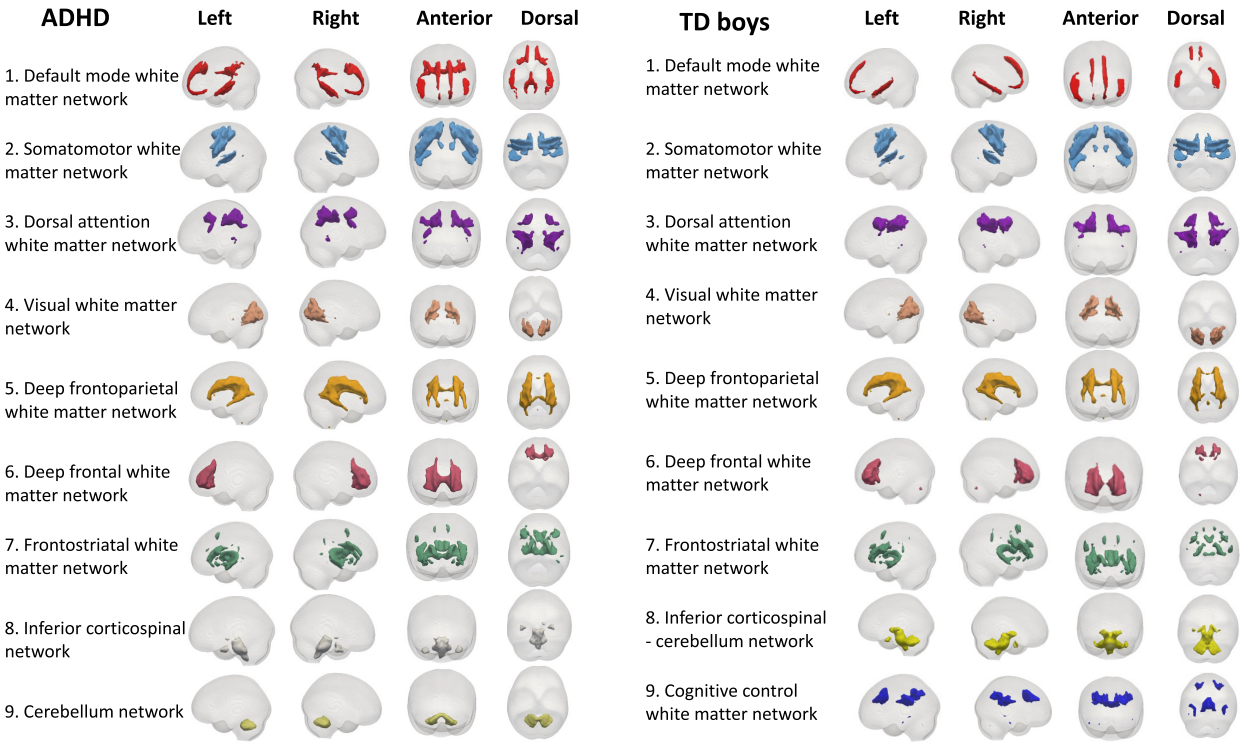

Figure 2: The nine WMFNs identified for ADHD and TD boys using the *K*-means clustering method. Seven shared networks were found in both groups of participants: (1) the default-mode white matter network, (2) the somatomotor white matter network, (3) the dorsal attention white matter network, (4) the visual white matter network, (5) the deep frontoparietal white matter network, (6) the deep frontal white matter network, and (7) the frontostriatal white matter network. In ADHD, however, the inferior corticospinal–cerebellar network was split into two subnetworks, and the cognitive control network was not present.

correlated with symptom scores, while the FPN and frontostriatal network were associated with cognitive performance. In summary, our findings demonstrated symptom- and cognition-related WM networks, which may be the specific neural mechanism underlying ADHD.

Disrupted WMFN organization

Previous studies have demonstrated alterations in white matter microstructure in several fasciculi and cortical networks, and we further explored the functional activity of white matter in patients with ADHD from a network point of view. The number of networks (*K*) for which the Dice coefficient was >0.9 was $K = 2$, 4, 6, and 9 for both groups. We defined the white matter network organization according to Yeo's canonical gray matter functional network and chose nine white matter functional networks as our target networks for further analysis. ADHD boys differ from TD boys in that the inferior corticospinal–cerebellar network is split into two subnetworks and the CCN is absent.

The splitting of the inferior corticospinal–cerebellar network into two subnetworks suggested disruption of functional connectivity within the motor–cerebellar system in boys with ADHD, which also represents functional segregation in the motor–cerebellar system in ADHD. The corticospinal–cerebellar network, which starts from the cortex, passes through the pons and ends at the cerebellum, and plays an important role in motor and cognition-related information processing. Previous fMRI studies detected reduced connectivity not only in the cerebellum but also between the cerebellum and various regions of the cerebral cortex in ADHD (Tomasi and Volkow, 2012; O'Halloran *et al.*, 2018). Damage to the cerebellum and its related circuit/network during development can have negative long-term effects on movement, cognition, and affective regulation (Stoodley, 2016). Our findings on the breakdown of the white matter corticospinal–cerebellar network suggested that dysfunction of structural connectivity within the cortico-cerebellar circuit may contribute to the inability to integrate and process information within the circuit and result in poor motor coordination and cognitive flexibility.

Lack of CCN in WMFNs is another important finding in the current study. It provides direct evidence for the network alteration related to the deficits in cognitive control that underline the core symptoms in ADHD. Decreased GM functional connectivity in the CCN correlated with lower accuracy during the go/no-go task and can be used to efficiently differentiate ADHD patients from healthy controls (Cai *et al.*, 2021), while increased functional connectivity in the CCN has been associated with symptom relief in remitted ADHD (Francx *et al.*, 2015). The lack of a CCN WMFN in boys with ADHD revealed in the current study using a data-driven method gave additional evidence for the disruption of this network. It also emphasized that this network may have a fundamental role in the neural pathology of boys with ADHD.

Hyperactive white matter networks

We found two symptom-related high-function white matter networks in boys with ADHD, which are increased SMN activity linked to impulsivity/hyperactivity symptoms and DAN activity related to poor attention. Dysfunction of the gray matter sensorimotor network has been shown to contribute to ADHD pathophysiology. Patients with ADHD had higher values of both degree centrality and functional connectivity in the motor and visual cortices (Wang *et al.*, 2009; Di Martino *et al.*, 2013). Referring to the motor network of gray matter, McLeod *et al.* (2016) reported stronger within-hemisphere functional connections among several motor

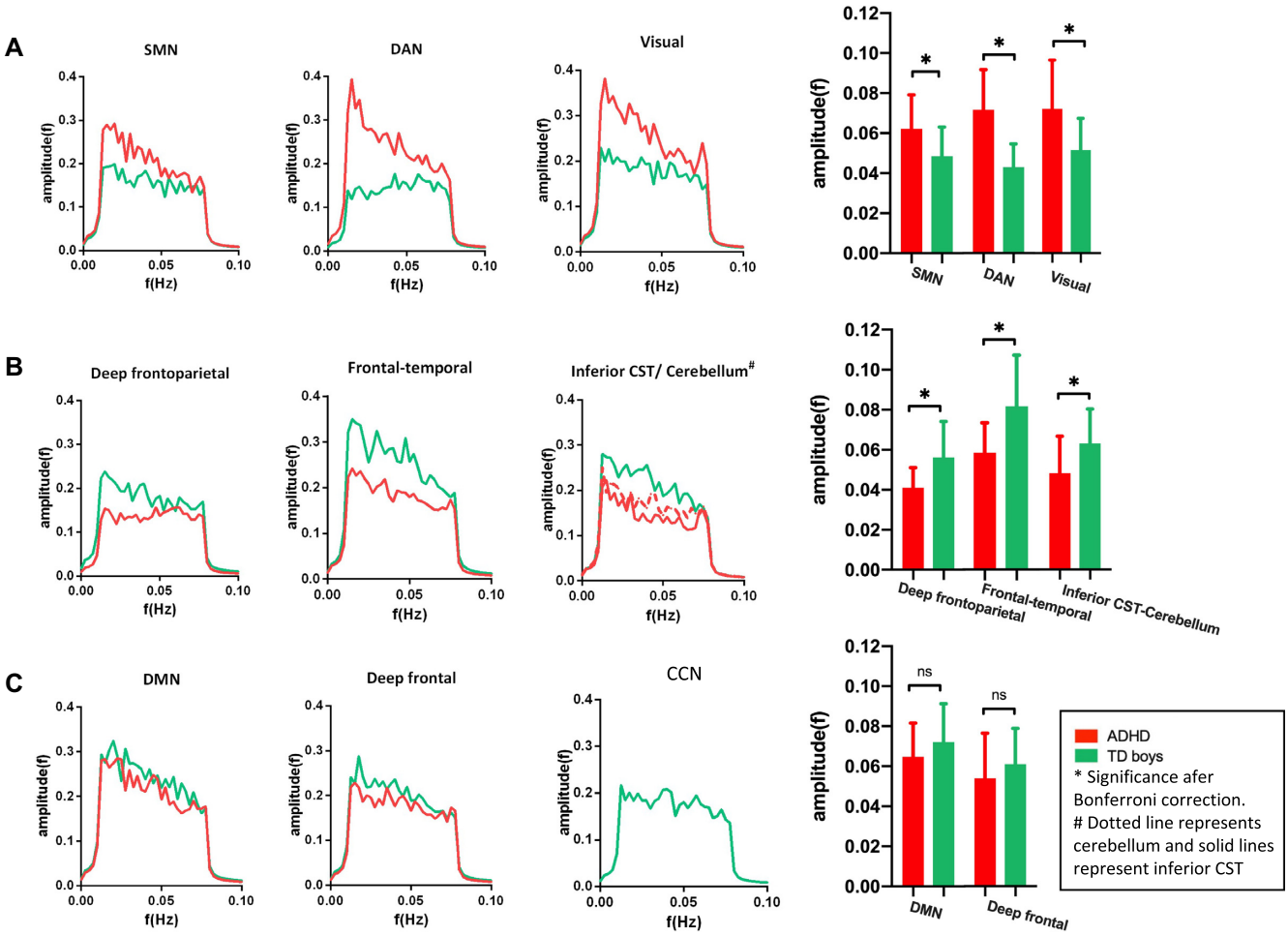

Figure 3: Signal amplitude for hyperactive (A), hypoactive (B), and nonsignificant (C) WMFNs. The left panel illustrates power–frequency graphs showing spontaneous neural activity in each white matter network. The right panel shows differences in the average amplitude of each network between ADHD and TD boys. Asterisks represent significant differences (*$P < 0.05$, Bonferroni corrected).

cortices, including primary and sensory motor cortices and precentral and postcentral gyri. We observed higher intrinsic function in the motor and visual WMFNs, and it correlated with a higher score of impulsivity–hyperactivity. This highlights the role of the SMN in ADHD core symptoms, such as hyperactivity, from the function of the structural network.

The DAN is involved in top-down attention control and is usually activated during high-demand situations. In the current study, we detected elevated function in white matter within the DAN and its correlation with study and attention problems in boys with ADHD. We assume this represents the increased demand of this particular network for ADHD. Individuals with ADHD are prone to distraction and mind wandering; thus, they may need more activity from the attention network to sustain the concentration, which leads to increased activity in white matter DAN. The more severe the inattention symptoms that exist, the higher intrinsic activity in the DAN network will be needed. This compensatory higher neural activity in ADHD may result from the low efficiency of the neural system, as patients need to consume more energy to complete a task (Fassbender and Schweitzer, 2006).

Hypoactive white matter networks

Apart from hyperactive WMFNs, we also observed hypoactivity in the FPN and frontostriatal network, which are cognition-related functional networks. It has been suggested that executive function can be distinguished as cool and hot. Cool executive function is predominantly involved in non-emotional cognitive control and is regulated by the FPN. The FPN comprises the lateral frontal pole, dorsal lateral prefrontal cortex, intraparietal sulcus, precuneus, anterior insula, and dorsal anterior cingulate cortex (Vincent *et al.*, 2008). This network is linked to cognitive control processes, including goal-directed behavior, response inhibition, working memory, and set shifting (Menon, 2011). Impairment in executive control is one of the characteristic presentations in ADHD. Previous studies demonstrated that ADHD patients showed decreased activity in FPN regions during tasks that place high demands on executive control (Durston *et al.*, 2007; Smith *et al.*, 2008; Rubia, 2011). Our finding of lower intrinsic activity in white matter within the FPN and its correlation with worse performance on the Stroop and digit span tasks gave direct evidence of functional disruption of this network, which contributes to response inhibition and working memory deficits.

In contrast, hot executive function relates to highly motivating and emotional situations that depend on the frontostriatal network, comprising the ventral prefrontal cortex, orbitofrontal cortex, striatum, and limbic system (Cubillo *et al.*, 2012). It is postulated that due to motivation deficits, ADHD individuals have delay aversion, which means preferring immediate rewards to long-term rewards and ultimately leads to impulsivity and

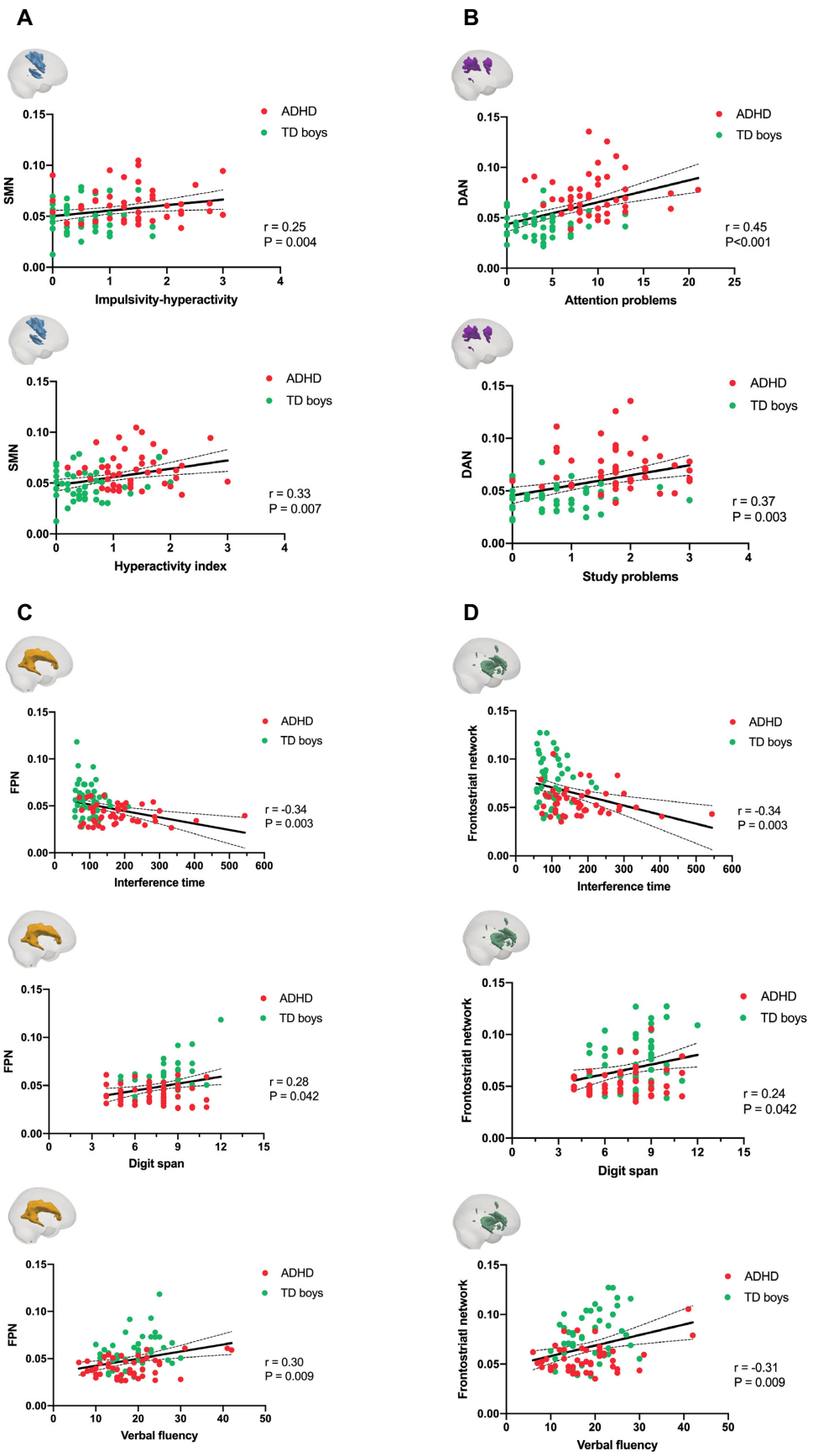

Figure 4: Correlation between hyperactive networks and symptoms (A, B) and between hypoactive networks and cognitive deficits (C, D). ($P < 0.05$, FDR corrected).

hyperactivity symptoms (Sonuga-Barke, 2005). Previous fMRI studies reported lower functional activity within the ventral frontostriatal network during reward tasks (Plichta and Scheres, 2014). Our finding of reduced intrinsic activity of white matter within the frontostriatal network and its correlation with poor performance in the Stroop task, digit span, and verbal fluency test suggested involvement of the frontostriatal network in cool executive function, while its contribution to hot executive function remains to be investigated in the future. The hypoactivity of white matter in both the FPN and the frontostriatal network highlights the essential role they play in cognitive function, especially in the etiology of cognitive dysfunction in ADHD.

Limitations

Several limitations should be considered in the current study. First, to reduce confounding effects, we included drug-naïve boys without any comorbidities. Although this helps to discover the fundamental role of specific networks in ADHD, it may be difficult to generalize to routine clinical patients. Second, our sample size is relatively small for the clustering algorithm, which may hinder the robustness of the results. However, despite the small sample size, the results withstood correction for multiple comparisons, etc., which also supports the reliability of our results. Of course, further validation with a larger sample will be needed to prove the stability of our findings. Third, given the complexity of white matter fibers, it is difficult to determine exactly which fasciculus is the source of the white matter BOLD signal. Combining tractography with white matter network analysis will help researchers identify the relationship between white matter fasciculi and WMFNs.

Conclusions

In the current study, we proposed a modified data-driven method to cluster white matter intrinsic networks. We proposed symptomatology-related networks and cognitive networks in boys with ADHD. We found that boys with ADHD presented a characteristic disruption of both the inferior corticospinal–cerebellar network and CCN. In addition, we demonstrated hyperactive symptom-related networks (i.e. the SMN and DAN) and hypoactive cognition-related networks (i.e. the FPN and frontostriatal network), which may underline the neural mechanism of core symptoms in boys with ADHD.

Supplementary Data

Supplementary data are available at *Pyschoradiology* online.

Conflict of Interest

One of the authors, Xiaoqi Huang, is also the executive editor of *Psychoradiology*. She was blinded from reviewing or making decisions on the manuscript.

Acknowledgements

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