Research Paper

Impulse control deficits among patients with nonsuicidal self-injury: a mediation analysis based on structural imaging

Ya Xie*, MMed; Sichu Wu*, MD; Jian Li*, PhD; Congjie Zhang, MPsy; Yumin Zhang, MPsych (Clin); Yaming Hang, MPsy; Nan Lang, MPsy; Zhangwei Lv, MPsych (Clin); Pei Zhang, MPsych (Clin); Minlu Liang, MPsych (Clin); Bo Yu, MMed; Jing Long, MBBS; Yuan Liu, MMed; Suhong Wang, MD; Lichen Ouyang, MMed; Liping Zhang, MMed; Yun Wu, MPsych (Clin); Chun Wang, MD, PhD

Background: Nonsuicidal self-injury (NSSI) is posited to arise from a complex interaction of biopsychosocial factors, with impulsivity playing a critical role. Given that current research on the neural mechanisms underlying this hypothesis remains inconsistent and limited in scope, we sought to explore how NSSI behaviours are associated with impulsivity resulting from structural brain alterations. **Methods:** We recruited patients with NSSI behaviours and healthy controls from 11 psychiatric hospitals. We assessed the differences in impulse control between the 2 groups using the Barratt Impulsiveness Scale version 11 and the Attention Network Test. We also conducted T_1-weighted magnetic resonance imaging (MRI) and diffusion tensor imaging. Finally, we analyzed the associations among brain structure, psychological characteristics, and self-injurious behaviour among patients with NSSI. **Results:** We included 293 patients with NSSI behaviours and 140 healthy controls. Among them, 182 patients with NSSI and 95 controls underwent the T_1-weighted MRI and diffusion tensor imaging. Patients with NSSI showed increased impulsivity and alerting function, with the strongest correlation between NSSI frequency and motor impulsivity. Compared with controls, patients with NSSI exhibited decreased grey matter volume and increased white matter volume, with no significant difference in cortical thickness. Pathway analysis demonstrated that motor impulsivity significantly mediated the association between white matter volume and the NSSI frequency in the right superior frontal gyrus and right inferior parietal lobe. When examining the connecting fibre tracts in the right frontoparietal area, patients with NSSI showed decreased integrity of white matter microstructure in the right cingulum, right superior corona radiata, and the splenium of the corpus callosum. **Limitations:** Accurately measuring executive control linked to NSSI is challenging in cognitive behavioural tasks, as impulsive tendencies during NSSI occurrence are not effectively captured. **Conclusion:** Our findings suggested that motor impulsivity, a prominent psychopathological characteristic of NSSI, is primarily modulated by the frontoparietal regions. These results provide empirical neuroimaging evidence for the impaired impulse control observed in NSSI.

Introduction

Nonsuicidal self-injury (NSSI) refers to a series of direct, deliberate, and repetitive acts of harm to oneself that are done without suicidal intent and do not result in death. Adolescents and young adults are particularly susceptible to NSSI behaviour. A multicentre clinical epidemiological study from China found a 14.3% prevalence of NSSI behaviour among adolescents aged 10–19 years.[1] Other studies involving nonclinical samples found a pooled prevalence of NSSI behaviour of 17.2% among adolescents, 13.4% among young adults, and 5.5% among adults.[2,3] A combination of biological, psychological, and social factors contribute to NSSI, with impulsivity being a critical, often underestimated determinant of NSSI behaviours.[4] Individuals engaging in NSSI often describe the onset of such behaviour as driven by uncontrollable impulses.[5]

Correspondence to: C. Wang, Clinical Non-pharmacological Treatment Center, The Affiliated Brain Hospital of Nanjing Medical University, 264 Guangzhou Road, Nanjing, Jiangsu, China; chun_wang@njmu.edu.cn.

*The authors contributed equally.

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Impulsivity is a multidimensional construct, with varied conceptualizations across different fields.[6] Barratt and colleagues[7] proposed 3 dimensions of impulsivity based on personality traits, namely attentional impulsivity (difficulty concentrating and cognitive instability), motor impulsivity (motor activity and lack of perseverance), and nonplanning impulsivity (lack of self-control and cognitive complexity). Studies have shown that people with NSSI behaviours exhibit higher levels of nonplanning and motor impulsivity on the Barratt Impulsiveness Scale version 11 (BIS-11),[8] and a meta-analysis suggested greater self-reported impulsivity among those with NSSI.[9] A prospective study further found that self-reported impulsivity was a substantial predictor of self-injurious behaviours.[10] Although the association between impulsivity and NSSI has been established, most studies rely on self-report scales. In contrast, task-based measures have recently been proposed as alternative methods for evaluating impulsivity.[11] For example, a nonverbal Stroop task study identified target control deficits among patients with NSSI.[12] However, in NSSI studies, the assessment of impulse control has been inconsistent between self-reports and task-based measures, which may stem from differing conceptualizations of impulsivity, as well as variations in the cortical and subcortical structures involved.[13,14] Therefore, combining these 2 types of measures may better reveal the neural mechanisms underlying the diverse impulsivity traits in NSSI.

Currently, the neural mechanisms underlying impulse control among patients with NSSI remain unclear. Dahlgren and colleagues[15] found that, compared with healthy controls, patients with NSSI exhibited increased activation in the cingulate cortex and decreased activation in the dorsolateral prefrontal cortex during a multiple-source interference task. However, this study was limited by its small sample size.[15] In contrast, structural brain abnormalities have rarely been identified in relation to impulsivity in NSSI behaviours. Evidence suggests that different dimensions of impulsive traits are associated with distinct corticostriatal neural circuits.[16,17] In the field of cognitive neuroscience, impulsivity is primarily viewed as a manifestation of impaired executive control, involving both structural and functional abnormalities in multiple cortical and subcortical brain regions.[18] The prefrontal cortex, inferior frontal gyrus, supplementary motor area, anterior cingulate gyrus, and subcortical basal ganglia collectively contribute to the processes of impulsive detection, projection, correction, and stopping.[19,20] Additionally, weak connectivity within the frontoparietal network (FPN) has been linked to impairments in error detection, contributing to impulsive behaviour.[21]

Therefore, we hypothesized that different dimensions of impulsivity, characterized by NSSI behaviours, would be reflected in structural alterations in different brain regions. To acquire a holistic picture of brain–behavioural relationships among people with NSSI, we sought to examine how NSSI behaviours are associated with impulsivity resulting from structural brain alterations among patients with NSSI and to explore the key factors influencing NSSI, as well as its neurostructural basis.

Methods

Participants

We recruited patients with NSSI and healthy controls aged 10–45 years from 11 different hospitals across China. At least 1 professional psychiatrist evaluated patients using the diagnostic criteria for NSSI outlined in the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* (DSM-5).[22] We included patients who, in the last year, on 5 or more days, had engaged in intentional self-inflicted damage to the surface of their body such that it was likely to induce bleeding, bruising, or pain, with the expectation that the injury would lead to only minor or moderate physical harm (i.e., no suicidal intent). We excluded patients with neurologic or severe somatic disorders, those who were pregnant or breastfeeding, and those who were not right-handed. We excluded controls with neurologic, psychiatric, or severe somatic disorders, those with a psychiatric family history, those who had taken psychotropic drugs within 3 months, those who were pregnant or breastfeeding, and those who were not right-handed. We informed all participants about the study in detail. Participants or a legal guardian (for those younger than 18 yr) signed informed consent forms.

Standardized psychometric assessments

For the NSSI group, we used the Ottawa Self-injury Inventory (OSI) to assess the frequency, severity, and time interval between thinking about and performing an NSSI, as well as the power to stop self-injury behaviours.[23] We also used the Hamilton Depression Scale (HAMD24), the Mood Disorders Questionnaire (MDQ)[24], and the Borderline Symptom List (BSL-23)[25] to assess the severity of depressive, manic, and borderline symptoms. All participants completed the Barratt Impulsiveness Scale version 11 (BIS-11) to measure 3 dimensions of impulsivity.[7] We converted all subscale scores to a scale of 0–100 using the formula $[(x - 10)/40] \times 100$.

We assessed participants' cognitive control by completing the Attention Network Task (ANT).[26] This cognitive paradigm combines a cued-target task and a Flanker task to evaluate the functions of alerting, orienting, and executive control by measuring the response time of participants under different target cue stimuli (Figure 1). Alerting efficiency was the response time to no cue minus the response time to a double cue. Orienting efficiency was calculated as the central cue response time minus the spatial cue response time. Executive control efficiency was the response time to an incongruent conditional target stimulus RT minus that to a congruent conditional target stimulus. The experimental program was implemented and presented using E-Prime 3.0 on a Windows 10 64-bit processor.

We excluded data with single-trial response times that exceeded 3 standard deviations from the mean for each participant. We also excluded participants with a no-response rate greater than 10% or accuracy less than 30%.

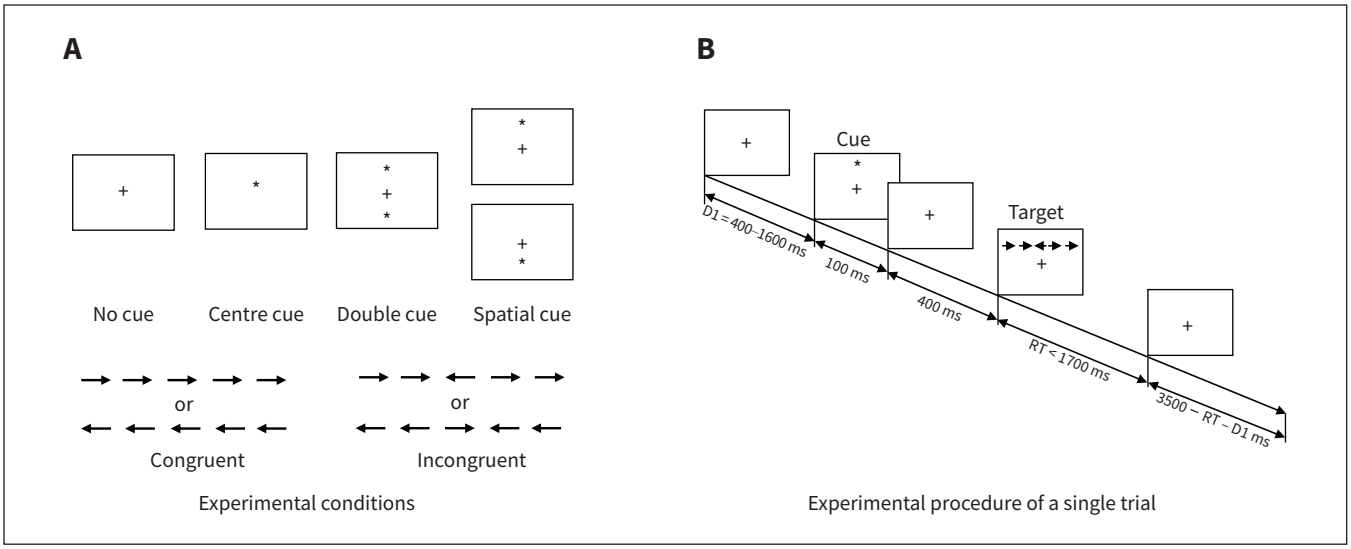

Figure 1: Schematic diagram of the Attention Network Test. The functions of alerting, orienting, and executive control are evaluated by measuring the response time (RT) of participants under different target cue stimuli of no cues (+), centre, spatial, or double cues (*), and target arrows shown in either congruent (all oriented in the same direction) or incongruent (mix of directions) sequences. DI = decision stage 1.

Magnetic resonance imaging

Six hospitals participated in MRI data collection. We acquired structural brain images using the 3.0 T resting-state MRI scanner (Siemens 3.0T Magnetom Verio, Medical Solutions). For the 3-dimensional T_1-weighted sequence, we used a repetition time of 1900 ms, echo time of 2.48 ms, matrix of 256 × 256, flip angle of 9°, slice thickness of 1.0 mm, slice gap of 0.5 mm, and 176 sagittal slices. For diffusion tensor imaging, the diffusion sensitizing gradients involved the baseline image with no diffusion weighting (b = 0 s/mm^2) along with diffusion-weighted images (b = 1000 s/mm^2) along 30 nonlinear directions, with a repetition time of 6600 ms, echo time of 93 ms, field of view of 256 × 256 mm, matrix of 128 × 128, flip angle of 90°, slice thickness of 3.0 mm, slice gap of 0 mm, and 70 axial slices.

We instructed each participant to remain motionless with their eyes closed during the MRI, as well as to use foam pads and earplugs to reduce head movement and noise.

Image processing

We converted DICOM files to NIFTI format using MRIcron. We preprocessed all the T_1-weighted images in MATLAB 2016b using the Computational Anatomy Toolbox (CAT12; https://neuro-jena.github.io/cat12-help/) available in SPM12 (https://www.fil.ion.ucl.ac.uk/spm/software/spm12/). Next, we corrected bias-field inhomogeneities on T_1-weighted images before segmentation, which included normalization to a template space and segmentation into grey matter, white matter, and cerebrospinal fluid. We then estimated the central surface and cortical thickness by projection-based distance measurements. Finally, we smoothed the grey matter and white matter images with an 8-mm full width at half maximum Gaussian kernel, and resampled and smoothed surface-based cortical thickness data with a 12-mm Gaussian kernel.

We conducted quality checks of all segmented image data. In addition, we quantified grey matter volume and white matter volume in 90 regions based on the Anatomic Automatic Labelling atlas,[27] and quantified cortical thickness in 72 regions based on the Desikan–Killiany 40 atlas.[28] We excluded samples with regional volumes exceeding 5 standard deviations from the group-level average from the analysis.[29]

We performed 2-sample t tests using SPM12 to compare the 3 morphometric measures (grey matter volume, white matter volume, cortical thickness) between the 2 distinct groups. Age, sex, educational level, hospitals, and total intracranial volume were included as covariates (cortical thickness comparison excluded total intracranial volume). For between-group differences, we applied a false discovery rate (FDR) correction with a threshold set at p_{FDR} less than 0.05 and a voxel threshold of 30 or higher.

We considered the brain regions with statistically significant differences in the 3 morphometric structures described above as regions of interest (ROIs) for further discussion of the brain function. The spherical ROIs were plotted with the peak of each cluster as a centre and a radius of 6 mm. Finally, we performed the same covariate regression to all extracted ROI values using the ComBat Harmonization algorithm (ComBatHarmonization/Matlab at master · Jfortin1/ComBatHarmonization · GitHub) to harmonize for confounders.

Diffusion tensor imaging analysis

We processed all diffusion tensor imaging data using the Functional Magnetic Resonance Imaging of the Brain (FMRIB) Software Library (FSL) (https://fsl.fmrib.ox.ac.uk/). First, we corrected the NIFTI images for head motion and eddy currents, removed non-brain tissues, and obtained the brain template. Next, we performed tensor calculation using the DTIFit function in FSL to acquire fractional anisotropy images for

each participant, which we then registered to the Johns Hopkins University atlas. Finally, we extracted specific white matter fibre ROIs from the atlas and computed the mean fractional anisotropy, mean diffusivity, and radial diffusivity values for each fibre tract. We conducted an analysis of variance (ANOVA) to compare the fibre bundle values between the 2 groups, with age, sex, education, and hospital as covariates.

Statistical analysis

We analyzed the demographic and clinical data using IBM SPSS Statistics 27. We applied a 2-sample t test to compare age in the 2 groups and χ^2 tests to compare sex and education. To compare the scale and ANT data between the 2 groups, we used either the 2-sample t test or the Mann–Whitney U test in accordance with the normality of the data. We explored associations between the scale, ANT, and imaging data using Pearson or Spearman correlation analysis. We conducted parallel mediation analysis to examine the relationship between different impulsivity dimensions, ROIs, and NSSI frequency based on 5000 bootstrap samples using Amos26 (https://www.ibm.com/products/structural-equation-modeling-sem). The significance level was set at p value less than 0.05.

Ethics approval

This study protocol was reviewed and approved by the Ethics Committee of The Affiliated Brain Hospital of Nanjing Medical University (no. 2019-KY043–01).

Results

Demographics and clinical features

We recruited 293 patients with NSSI and 140 healthy controls. We excluded 43 participants in the NSSI group (25 with nonresponses > 10% and 18 with accuracy < 30%) and 47 participants in the control group (36 with nonresponses > 10% and 11 with accuracy < 30%) because of invalid data. There were no significant group differences in age ($t = –1.632$, $p = 0.1$), sex ($\chi^2 = 2.751$, $p = 0.1$), or education ($\chi^2 = 4.185$, $p = 0.1$). The primary diagnoses of patients with NSSI included depressive disorder (58.7%), bipolar disorder (18.8%), and borderline personality disorder (18.8%) (Appendix 1, Table 1, available at www.jpn.ca/lookup/doi/10.1503/jpn.240129/tab-related-content). The OSI revealed that 54.3% of patients had engaged in NSSI monthly or more often over the past year; 53.6% of patients progressed from thinking about self-injury to committing the act within minutes, while only 11.9% believed they had a strong motivation to stop self-injury (Table 1).

Impulse assessment

The NSSI group had significantly higher mean total scores on the BIS-11 than the control group ($t = 12.940$, $p < 0.001$), with notably higher scores in the subdimensions of nonplanning impulsivity ($t = 11.697$, $p < 0.001$), motor impulsivity ($t = 11.734$, $p < 0.001$), and attentional impulsivity ($t = 7.762$, $p < 0.001$). Compared with the control group, the NSSI group had higher mean response times across cues in the ANT task.

Table 1: Features of self-injury behaviour among patients with nonsuicidal self-injury (NSSI)

Measure	No. (%) of patients with NSSI* $n = 293$
OSI	
NSSI frequency in the past month	
Not at all	53 (18.1)
At least once	120 (41.1)
Weekly	78 (26.6)
Daily	42 (14.3)
NSSI frequency in the past 6 months	
Not at all	0 (0.0)
1–5 times	129 (44.0)
Monthly	66 (22.5)
Weekly	75 (25.6)
Daily	23 (7.8)
NSSI frequency in the past year	
Not at all	0 (0.0)
1–5 times	134 (45.7)
Monthly	84 (28.7)
Weekly	54 (18.4)
Daily	21 (7.2)
Time to perform NSSI	
A few seconds	49 (16.7)
A few minutes	108 (36.9)
Less than half hour	90 (30.7)
Half to 1 hour	20 (6.8)
Less than 1 day	16 (5.5)
More than 1 day	10 (3.4)
Power to control NSSI occurrence	
Not at all	49 (16.7)
A little	108 (36.9)
Somewhat	101 (34.5)
Greatly	27 (9.2)
Extremely	8 (2.7)
HAMD24, mean ± SD	33.87 ± 12.75
MDQ, mean ± SD	4.20 ± 2.32
BSL-23, mean ± SD	2.36 ± 0.92
Psychiatric diagnoses†	
Depressive disorder	172 (58.7)
Bipolar disorder	55 (18.8)
Borderline personality disorder	55 (18.8)
Anxiety disorder	10 (3.4)
Obsessive–compulsive disorder	5 (1.7)
Posttraumatic stress disorder	2 (0.7)
Schizoaffective disorder	2 (0.7)
Adolescent mood disorders	2 (0.7)
Pathological gambling	2 (0.7)
Not meeting diagnostic criteria	10 (3.4)

BSL = Borderline Symptoms List; HAMD = Hamilton Depression Scale; MDQ = Mood Disorders Questionnaire; OSI = Ottawa Self-injury Inventory; SD = standard deviation.
*Unless indicated otherwise.
†Each NSSI participant could have several diagnoses.

Regardless of group, response times were longer when target stimuli were incongruent rather than congruent. For cue type, reaction times were shortest for the double-cue condition and longest for the no-cue condition. The NSSI group exhibited greater efficiency in alerting function (t = –2.616, p = 0.009), but no significant differences were observed between the 2 groups in orienting function (t = –0.540, p = 0.6) and executive control (t = –1.727, p = 0.08) function (Table 2).

Correlation between impulse control and NSSI symptoms

The frequency of self-injury among patients with NSSI correlated positively with their BIS-11 scores. Among the 3 BIS-11 subdimensions, motor impulsivity exhibited the strongest correlation with the NSSI frequency over the past month (r = 0.203, p < 0.001) (Figure 2).

Correlation between brain structure alterations and impulsive assessment

After quality control, we included imaging data from 182 patients with NSSI and 95 controls for further analysis. There were no statistical differences in age (t = –1.311, p = 0.2), sex (χ^2 = 1.202, p = 0.3), or education (χ^2 = 9.927, p = 0.007) between the NSSI and control groups. Compared with the control group, the NSSI group showed significantly lower grey matter volume in the left superior frontal gyrus (SFG) medial division, left supplementary motor area, right middle frontal gyrus, bilateral precentral gyrus, bilateral putamen, and right pallidum (Figure 3A and Appendix 1, Table 2). After extracting statistically different brain regions as ROIs, we found no correlation between alterations in grey matter volume among patients with NSSI and impulsivity dimensions. Patients with NSSI exhibited greater white matter volume in regions of the frontal, parietal, and temporal lobes, as well as hippocampus (Figure 3B and Appendix 1, Table 3). Of these, 11 regions demonstrated associations between higher BIS-11 scores, alerting function efficiency, and white matter volume, including the bilateral dorsolateral superior frontal gyrus, opercular part of inferior frontal gyrus, bilateral inferior parietal lobe (IPL), bilateral supramarginal gyrus, right hippocampus and parahippocampal gyrus, and bilateral inferior temporal gyrus. The strongest negative correlation was observed between the right supramarginal gyrus and nonplanning impulsivity (r = –0.215, p = 0.003), while the only positive correlation occurred between the right IPL and motor impulsivity (r = 0.168, p = 0.02) (Figure 3C). The correlation was no longer significant after Bonferroni correction. In the surface-based morphometry analysis, there was no significant difference in cortical thickness between the 2 groups after FDR correction. When we reincorporated the HAMD scores, MDQ scores, and BSL-23 scores of patients with NSSI into the ComBat Harmonization model, the results indicated that, after reharmonization, the ROIs for grey matter volume remained uncorrelated with impulsivity scores and alerting efficiency. Regarding white matter volume, the correlation between the left dorsolateral SFG and attentional impulsivity disappeared. Moreover, there were no

Table 2: Impulse control differences between patients with nonsuicidal self-injury (NSSI) and controls

Measure	Patients with NSSI n = 293	Controls n = 140	t or z	p value
BIS-11, mean ± SD				
Nonplanning impulsivity	58.51 ± 18.36	37.09 ± 16.65	11.697	< 0.001
Motor impulsivity	52.06 ± 19.33	30.21 ± 15.28	11.734	< 0.001
Attentional impulsivity	48.66 ± 16.98	35.46 ± 15.59	7.762	< 0.001
Total score	53.08 ± 14.67	34.26 ± 13.00	12.940	< 0.001
ANT,* median (IQR)				
No cue correct rate	1.00 (0.91 to 1.00)	1.00 (0.80 to 1.00)	–0.192	0.8
No cue reaction time	641.54 (558.52 to 773.52)	591.25 (543.82 to 679.32)	–3.153	0.002
Double cue correct rate	1.00 (0.89 to 1.00)	1.00 (0.73 to 1.00)	–0.342	0.7
Double cue reaction time	597.85 (522.62 to 726.29)	563.92 (521.32 to 638.83)	–2.017	0.04
Centre cue correct rate	1.00 (0.85 to 1.00)	1.00 (0.79 to 1.00)	–0.081	0.9
Centre cue reaction time	623.41 (532.23 to 729.67)	566.79 (526.45 to 648.78)	–2.610	0.009
Spatial cue correct rate	0.50 (0.42 to 0.58)	0.50 (0.42 to 0.58)	–1.286	0.2
Spatial cue reaction time	606.38 (514.25 to 729.90)	562.21 (499.25 to 620.08)	–2.913	0.004
Consistency cue correct rate	0.85 (0.79 to 0.89)	0.86 (0.80 to 0.91)	–0.898	0.4
Consistency cue reaction time	598.73 (509.60 to 687.96)	535.07 (488.23 to 604.03)	–3.432	< 0.001
Inconsistency cue correct rate	0.83 (0.74 to 0.89)	0.84 (0.61 to 0.90)	–0.141	0.9
Inconsistency cue reaction time	652.32 (565.36 to 786.69)	616.47 (570.55 to 669.21)	–2.232	0.03
Alerting	37.89 (–8.25 to 86.80)	16.92 (–23.91 to 55.51)	–2.616	0.009
Orienting	12.71 (–25.51 to 51.67)	17.21 (–19.53 to 46.81)	–0.540	0.6
Executive control	60.13 (28.70 to 97.24)	68.59 (49.24 to 101.16)	–1.727	0.08

ANT = Attention Network Test; BIS = Barratt Impulsiveness Scale; IQR = interquartile range; SD = standard deviation.
*Inlcudes 250 patients with NSSI and 93 controls.

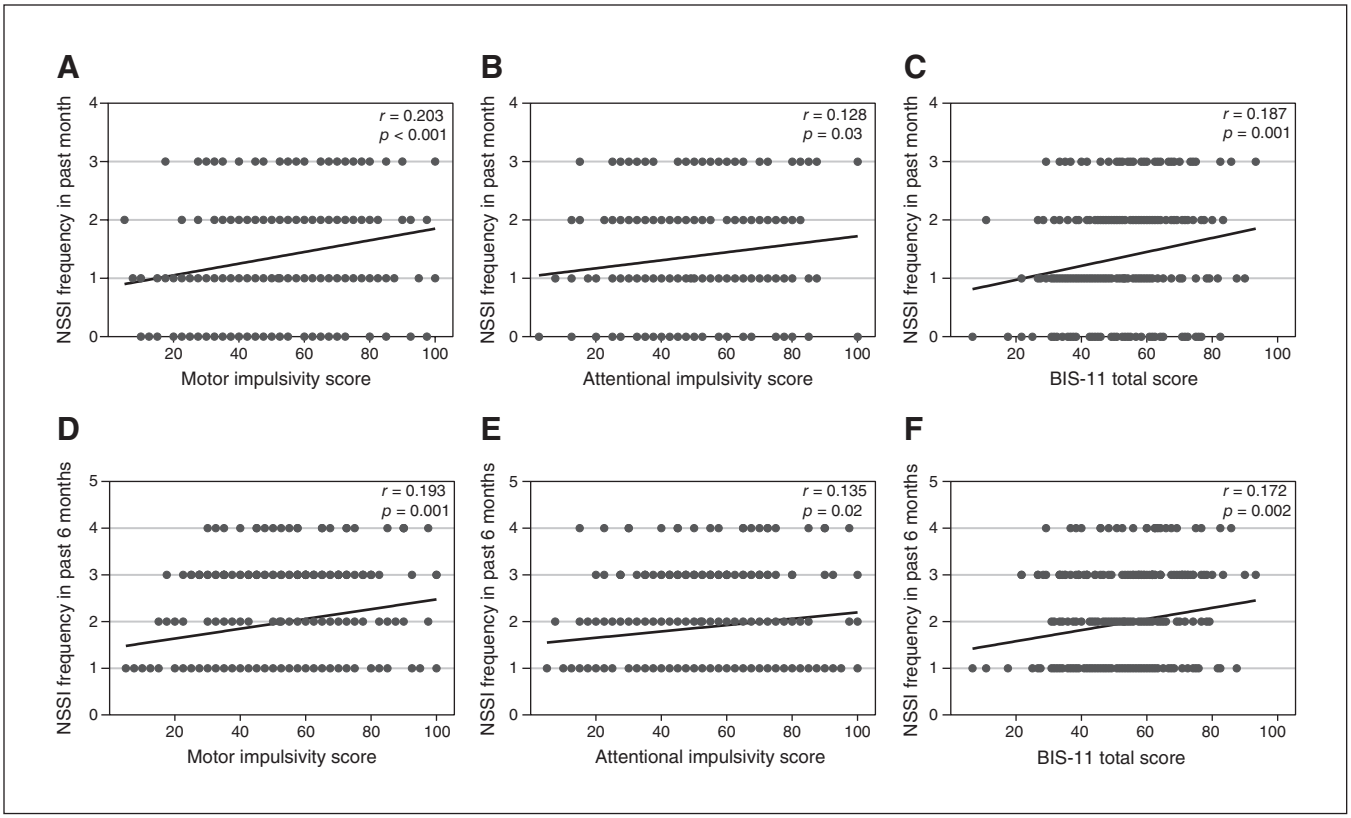

Figure 2: Correlation analysis between frequency of nonsuicidal self-injury (NSSI) and dimensions of impulse control, including NSSI frequency over the past month and (A) motor impulsivity ($r = 0.203$, $p < 0.001$), (B) attentional impulsivity ($r = 0.128$, $p = 0.03$), and (C) total score on the Barratt Impulsiveness Scale (BIS) version 11 ($r = 0.187$, $p = 0.001$), as well as NSSI frequency over the past 6 months and (D) motor impulsivity ($r = 0.193$, $p = 0.001$), (E) attentional impulsivity ($r = 0.135$, $p = 0.02$), and (F) total BIS-11 scores ($r = 0.172$, $p = 0.002$).

significant changes in the brain regions previously correlated with different dimensions of impulsivity, with the only positive correlation still in the right IPL ($r = 0.169$, $p = 0.02$) (Appendix 1, Figure 1).

Motor impulsivity as a mediator of the brain and NSSI behaviour

The mediation model indicated that motor impulsivity mediated the association between the frequency of NSSI behaviours and white matter volume in the right dorsolateral SFG and IPL. Among patients with NSSI, white matter volume in the right IPL positively mediated the effect on NSSI frequency through motor impulsivity (β = 1.552, 95% confidence interval [CI] 0.029 to 5.171). In contrast, white matter volume in the right dorsolateral SFG exerted a significant negative indirect effect on NSSI frequency through motor impulsivity (β = –1.753, 95% CI –5.728 to –0.060) (Figure 4). Furthermore, grey matter volume in the left medial SFG (β = 1.372, 95% CI 0.119 to 2.900), right precentral gyrus (β = 4.349, 95% CI 1.274 to 8.131), and bilateral rectus gyrus (left: β = 3.037, 95% CI 0.268 to 6.545; right: β = 2.400, 95% CI 0.335 to 5.023) had a direct positive effect on the NSSI frequency. Meanwhile, white matter volume in the left dorsolateral SFG (β = –3.627, 95% CI –7.951 to –0.333), and left precentral gyrus (β = –4.222, 95% CI –9.547 to –0.363) had a direct negative effect on the frequency of NSSI behaviours. Furthermore, after incorporating HAMD, MDQ, and BSL-23 scores for harmonization of these ROIs, these direct and indirect effects remained consistent.

White matter microstructural changes among patients with NSSI

Based on our findings, we selected white matter fibre tracts connecting the right frontal lobes and parietal lobe as ROIs. These included the corpus callosum, corona radiata, right superior longitudinal fasciculus, right superior frontal–occipital fasciculus, and right cingulum bundle. Covariance analysis showed that, compared with controls, patients with NSSI exhibited significantly lower fractional anisotropy values in the right cingulum bundle ($F = 2.585$, $p = 0.03$). They also showed higher mean diffusivity values in the splenium of the corpus callosum ($F = 3.121$, $p = 0.01$) and the right superior corona radiata ($F = 2.566$, $p = 0.03$). We also observed higher radial diffusivity values in the splenium of the corpus callosum ($F = 2.549$, $p = 0.03$), the right cingulum bundle ($F = 2.363$, $p = 0.04$), and the right superior corona radiata ($F = 2.774$, $p = 0.02$) compared with controls (Figure 5).

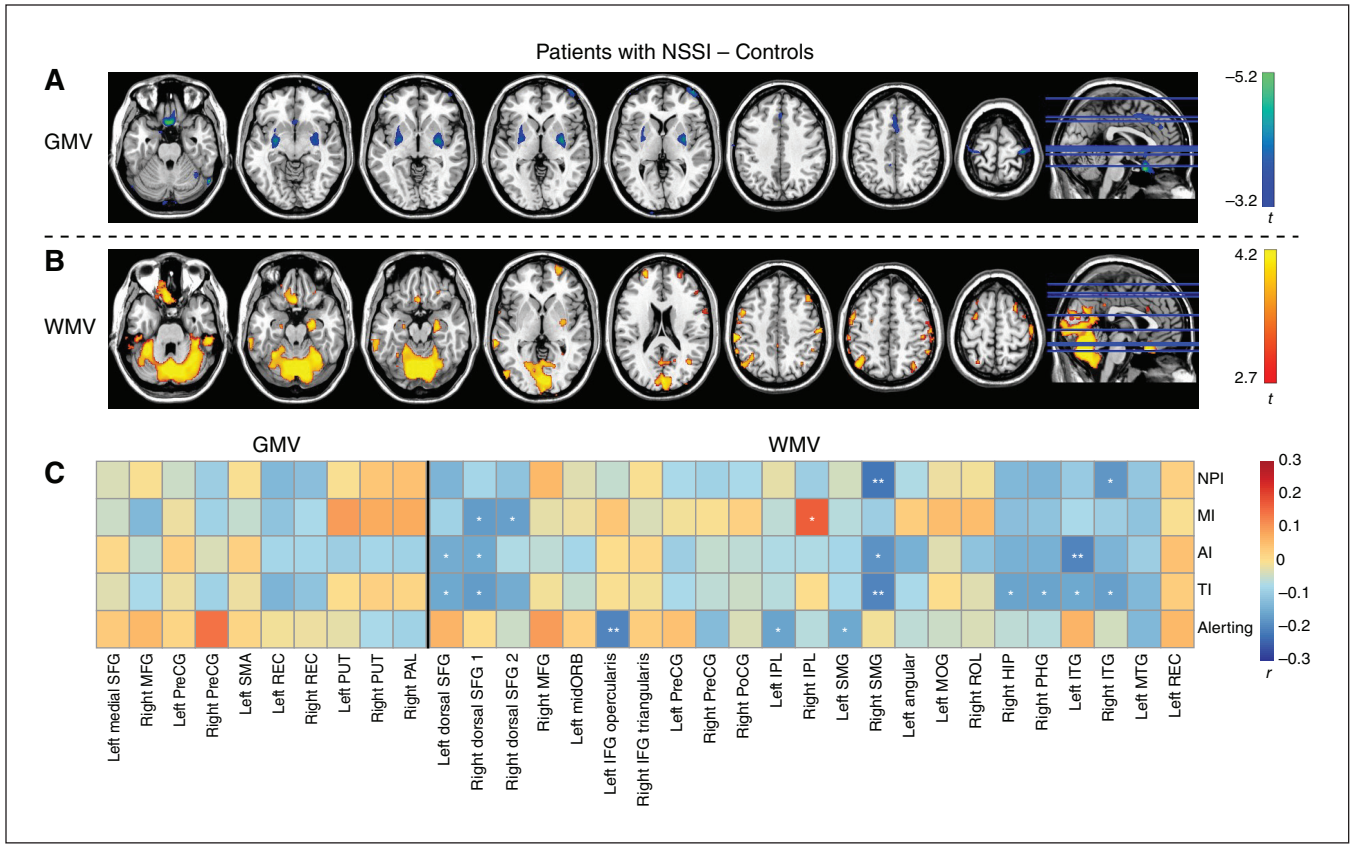

Figure 3: (A) Compared with healthy controls, patients with nonsuicidal self-injury (NSSI) showed lower grey matter volume (GMV) in the bilateral putamen (PUT), right pallidum (PAL), right middle frontal gyrus (MFG), left superior frontal gyrus (SFG) medial division, left supplementary motor area (SMA), and bilateral precentral gyrus (PreCG) ($p < 0.05$, voxels ≥ 30, with correction for false discovery rate [FDR]). (B) Patients with NSSI also showed higher white matter volume (WMV) in the frontoparietal temporal lobes, hippocampus (HIP), and cerebellar regions ($p < 0.05$, voxels ≥ 30, FDR correction). (C) Associations between impulsivity and brain structural alterations among patients with NSSI. We observed significant negative correlations between nonplanning impulsivity (NPI) scores and WMV in the right inferior temporal gyrus (ITG) and right supramarginal gyrus (SMG); significant negative correlations between motor impulsivity (MI) scores and WMV in both subregions of the right dorsal SFG and positive correlations between MI scores and the right inferior parietal lobule (IPL); and significant negative correlations between attentional impulsivity (AI) scores and WMV in the left dorsal SFG, subregion 1 of the right dorsal SFG, the right SMG, and the left ITG. We also observed significant negative correlations between total impulsivity scores (TI) and WMV in the left dorsal SFG, subregion 1 of the right dorsal SFG, the right SMG, the right HIP, the right parahippocampalgyrus (PHG), and the bilateral ITG, and between alerting function scores and WMV in the left inferior frontal gyrus (IFG) opercularis, the left IPL, and the left SMG. *$p < 0.05$; **$p < 0.01$. MidORB = orbital part of middle frontal gyrus; MOG = middle occipital gyrus; MTG = middle temporal gyrus; PoCG = postcentral gyrus; REC = gyrus rectus; ROL = rolandic operculum.

Discussion

In this study, we used the ANT and BIS-11 to assess impulsivity across multiple dimensions, aiming to illustrate the relationship between structure abnormalities in specific brain regions, impulse control deficits, and NSSI behaviours through a mediation model. We discovered that motor impulsivity significantly mediated the relationship between the right-sided IPL, dorsolateral SFG, and NSSI behaviour. Furthermore, among patients with NSSI, we observed a reduction in microstructural integrity of white matter fibre bundles connecting the frontal and parietal lobes. These findings suggest that structural abnormalities in white matter of the right IPL and dorsolateral SFG are involved in modulating NSSI behaviour and impulsivity enhancement.

Current research has demonstrated that self-reported impulsivity, particularly the subtraits of motor impulsivity, plays an important role in the development of NSSI. Previous studies suggested that correlation coefficients between motor impulsivity and cognitive control were higher than those for the other 2 BIS dimensions.[30] These aspects of the BIS-11 may be dissociable because they focus on different cognitive processes.[31] The BIS-11 assessment of motor impulsivity centres on quantifying impulsive behaviour, which is closely related to response inhibition, aligning with the uncontrollable impulsiveness often reported by patients with NSSI.[32,33] Conversely, attentional impulsivity is associated with an inefficient conflict detection system, which is less stable than other subtraits.[7]

In ANT, we found that the efficiency of the alerting function was better among patients with NSSI than healthy controls.

However, this function did not significantly correlate with NSSI behaviours. This finding aligns with those of Mirabella and colleagues, which identified enhanced response inhibition among adolescents with NSSI, suggesting that this heightened inhibition may suppress self-protective instincts.[34] Alertness refers to a heightened sensitivity to potential stimuli, promoting quick and accurate task responses.[35] We propose that, although patients with NSSI display impulsivity in self-injury behaviours, chronic stress dysregulation may render them more sensitive to external stimuli and more alert. Notably, participants in our study were generally in a calm state during ANT experiments, and behavioural tasks typically capture a brief snapshot of decision-making behaviour under the current emotional state.[10] Without a specific negative context imposed

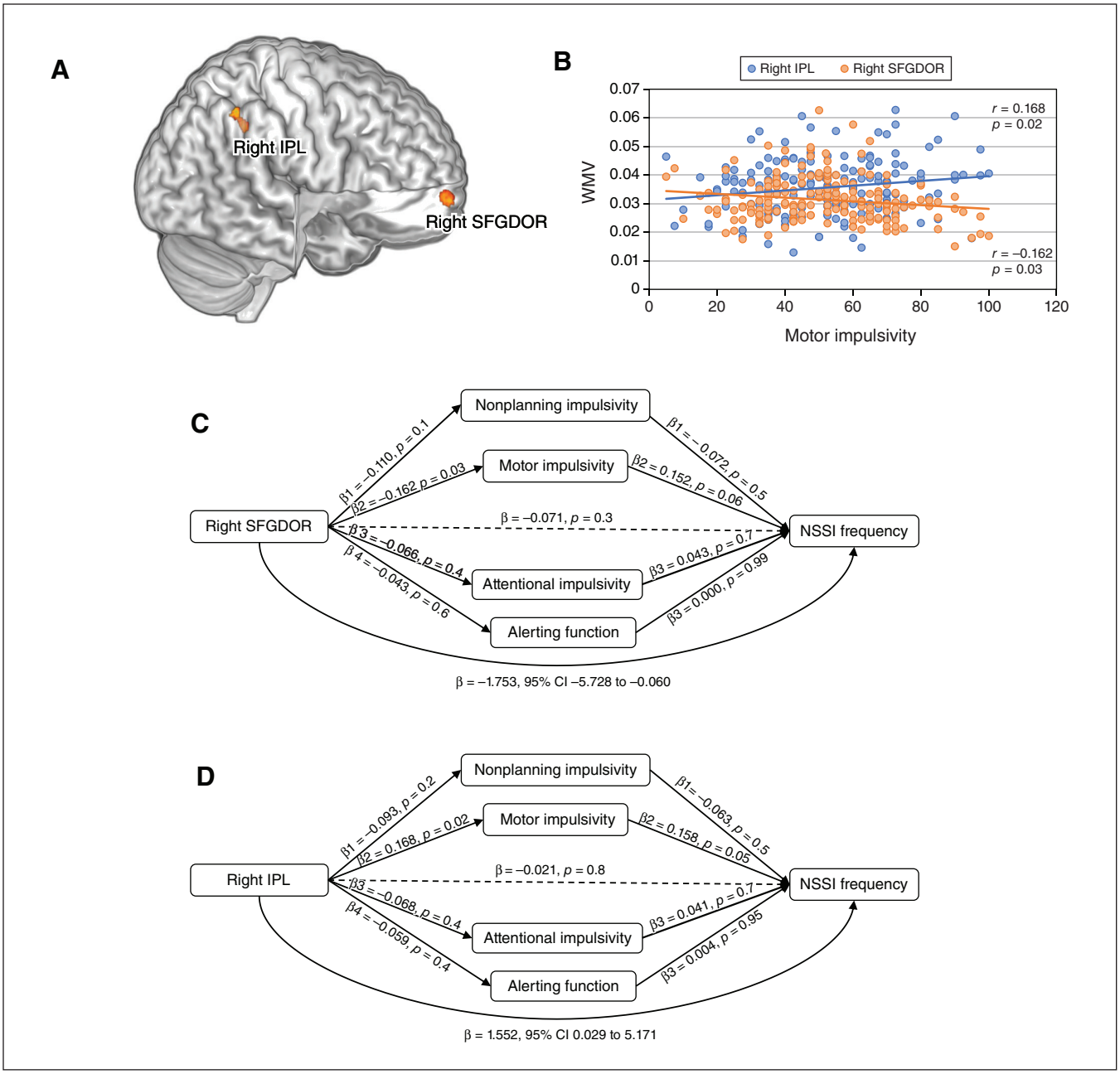

Figure 4: The mediating role of impulse control between altered brain regions and the frequency of nonsuicidal self-injury (NSSI) behaviours among patients with NSSI. (A) Image showing the location of the right inferior parietal lobe (IPL) and the right dorsolateral superior frontal gyrus (SFGDOR). (B) Correlations between motor impulsivity and white matter volume (WMV) of the right IPL (r = 0.168, p = 0.02) and right SFGDOR (r = –0.162, p = 0.03). (C) Among patients with NSSI, WMV in the right SFGDOR exerted a significant negative indirect effect (IE) on NSSI frequency through motor impulsivity (β = –1.753, 95% confidence interval [CI] –5.728 to –0.060), while (D) WMV in the right IPL positively mediated the effect on NSSI frequency through motor impulsivity (β = 1.552, 95% CI 0.029 to 5.171).

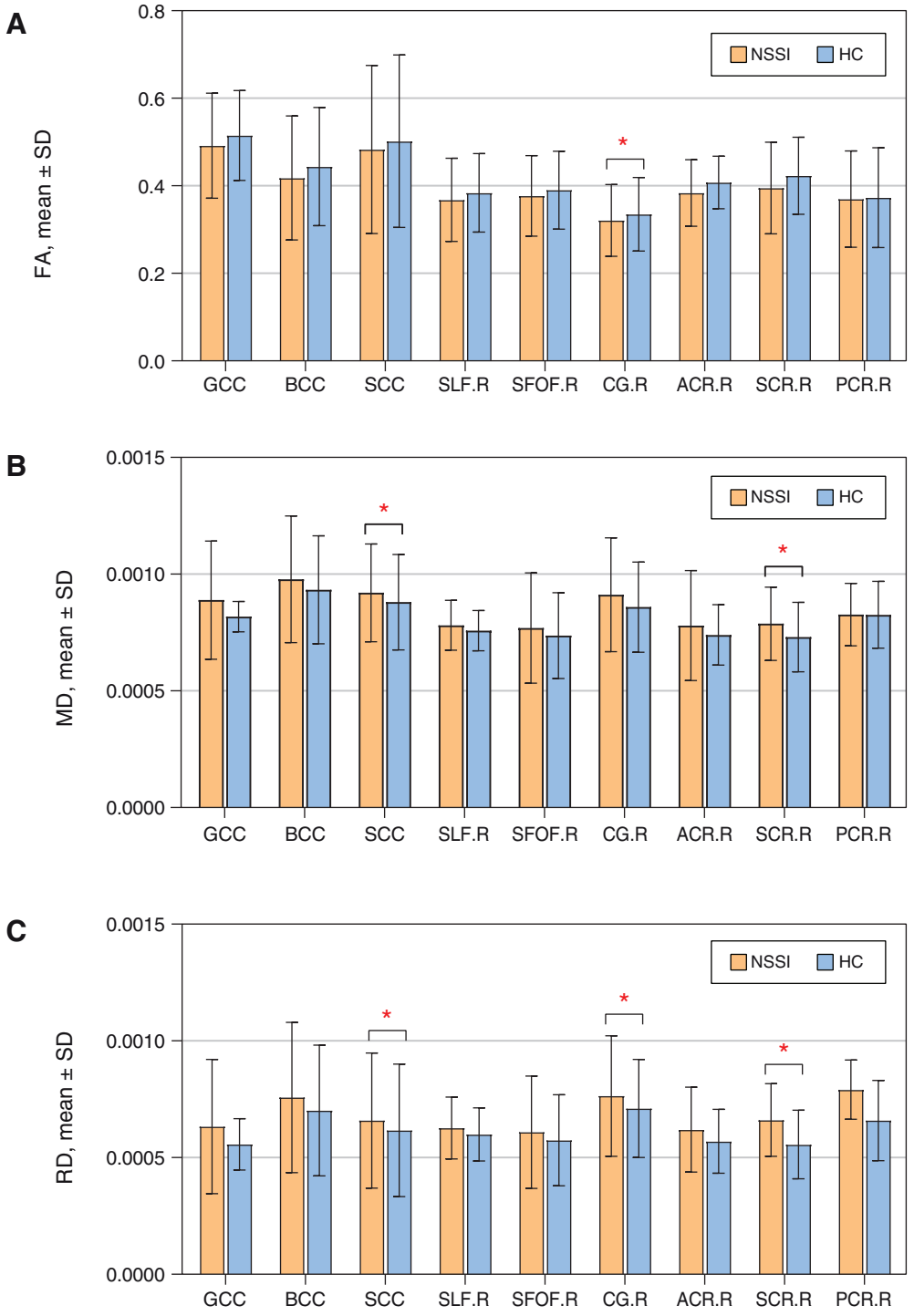

Figure 5: Impulsivity-related white matter fibre damage to (A) fractional anisotropy (FA), (B), mean diffusivity (MD), and (C) radial diffusivity (RD) among patients with nonsuicidal self-injury (NSSI) and healthy controls (HCs). Patients with NSSI had significantly lower FA in the right cingulate gyrus (CG.R), significantly higher MD in the splenum of the corpus callosum (SCC) and the right superior corona radiata (SCR.R), and significantly higher RD in the SCC, CG.R, and SCR.R. ACR.R = right anterior corona radiata; BCC = body of the corpus callosum; GCC = genu of the corpus callosum; PCR.R = right posterior corona radiata; SD = standard deviation; SFOF.R = right superior fronto-occipital fasciculus; SLF.R = right superior longitudinal fasciculus. *$p < 0.05$.

on participants, such task-based measures may fail to capture the complexity of NSSI behaviours and their related perceptions of impulse control.[13]

We found that white matter volume in the right IPL had an indirect positive effect on the frequency of NSSI behaviours through mediation of motor impulsivity. The parietal lobe serves as a crucial node in the inhibition network.[36,37] Huang and colleagues found that high motor impulsivity was associated with a large proactive control effect in the IPL, consistent with our findings.[32] Similarly, white matter hyperintensity in the right parietal lobe has been implicated in executive function.[38] In our study, patients with NSSI exhibited low fractional anisotropy values, and high mean and radial diffusivity values in white matter fibre bundles, indicating reduced fibre integrity. The splenium of the corpus callosum and the superior corona radiata connect the parietal lobe to other brain regions, playing critical roles in cognitive functions. Studies have shown that injuries or lesions to the superior corona radiata can impair parietal lobe functions, causing sensory, motor, and perception deficits.[39] The anterior part of the cingulum bundle connects to prefrontal areas involved in emotion regulation and decision-making, while the middle part links to parietal regions responsible for emotion processing and cognitive control.[40] Likewise, the right dorsolateral SFG is a key brain structure for impulse modulation and response inhibition, acting as a brake on impulsive behaviours.[41] Research on NSSI confirms that the dorsolateral SFG plays a key role in the cognitive processes underlying self-injurious behaviour.[42,43] In addition, cortical thickness in the dorsolateral SFG has been linked to differences in impulsivity and strategic behaviour.[44] As a prominent physiotherapeutic target, transcranial direct current stimulation of the right dorsolateral SFG may enhance response inhibition by reducing decision-making biases and discrimination.[45]

Interestingly, we found that motor impulsivity played a negative modulatory role in white matter structures of the dorsolateral SFG and NSSI behaviours. Patients with NSSI exhibit increased volume but decreased fibre integrity of white matter, which may represent a compensatory but maladaptive response to NSSI-related neurologic changes. As the disease progresses, white matter proliferation may contribute to enhanced cognitive control.[46,47] Human growth trajectories during adolescence and early adulthood demonstrate a steady decline in grey matter volume, accompanied by a gradual increase in white matter volume.[48] Patients with NSSI exhibit reduced integrity of fibre bundle microstructures, which could disrupt brain network functions associated with impulse control. Conversely, the increase in white matter volume may indicate an adaptive or reparative process, such as glial cell proliferation or alterations in myelination.[49,50]

These findings further suggest that the onset of NSSI behaviour is closely associated with structural changes in the frontoparietal lobe. Consequently, it is plausible to assume that NSSI behaviour occurring in a highly impulsive state is associated with structural and connectivity abnormalities in the FPN of patients with NSSI. The FPN — which encompasses the dorsolateral SFG, dorsal frontal cortex, IPL, precuneus, and cingulate — has been implicated in active, adaptive cognitive control.[51] The FPN and subcortical areas form the core of the executive network.[52] This network is responsible for detecting whether individuals are successful in the process of behavioural inhibition during the correction process,[53,54] which is critical for the effective suppression of impulsive behaviour.[55] A study on the white matter microstructure revealed that decreased fractional anisotropy within the FPN may be linked to inhibition-related cognitive impairments.[56]

In addition to indirect effects, we found that reduced grey matter volume in the right precentral gyrus, left medial SFG, and bilateral rectus of patients with NSSI had a direct negative effect on NSSI behaviours, while increased white matter volume in the left dorsolateral SFG and precentral gyrus exhibited a direct positive influence. The precentral gyrus is a critical brain region of substantial interest. Several studies have demonstrated that patients with NSSI exhibit cortical structural changes and enhanced functional activation in the precentral gyrus.[57–59] The primary function of the precentral gyrus is motor control, which participates directly in motor planning and the generation of movement instructions.[60] Moreover, the precentral gyrus works in concert with regions of the FPN to engage in conflict processing, facilitating the brain's flexible switching and integration between motor and cognitive control.[60–62] These findings suggest that patients with NSSI may experience abnormalities in inhibiting self-injury impulses and generating self-injury–related instructions.

Limitations

Accurately measuring executive control linked to NSSI is challenging in such a cognitive-behavioural task, as impulsive tendencies during NSSI occurrence are not effectively captured. The lack of suitable measurement tools limits our understanding of impulsivity in NSSI. Future studies should consider using task-based impulsivity measures or targeted cognitive-behavioural paradigms. The relationship between brain structure and behaviour is reciprocal. This cross-sectional study demonstrated only the current associations between brain regions and their functions in NSSI. The causal and compensatory links between brain region alterations and impulse control among patients with NSSI require further exploration. Future prospective studies will provide deeper insights into the neural mechanisms underlying NSSI behaviours. We conducted a transdiagnostic study. In future studies, collecting more detailed data on illness severity and analyzing NSSI in the context of different psychiatric diagnoses would help provide a clearer and more robust assessment of the causal relationship.

Conclusion

We investigated the neural mechanisms underlying impulse control in relation to NSSI behaviours. Our findings suggested that white matter structure abnormalities exhibited by patients with NSSI, particularly in the frontoparietal region, were involved in the exacerbation of impulsivity and worsening of NSSI behaviours. These results may provide valuable evidence for identifying therapeutic targets for intervention in NSSI behaviour.

Affiliations: From the Clinical Non-pharmacological Treatment Center, The Affiliated Brain Hospital of Nanjing Medical University, Nanjing, Jiangsu, China (Xie, Wu, Li, Y. Zhang, Hang, Lang, Lv, P. Zhang, Liang, Ouyang, L. Zhang, Wu, C. Wang); the School of Psychology, Nanjing Normal University, Nanjing, Jiangsu, China (C. Zhang, Hang, Lang, Wu, C. Wang); the Third People's Hospital of Mianyang, Youxian District, Mianyang, Sichuan, China (Yu); the Tianjin Anding Hospital, Hexi District, Tianjin, China (Long); the Xuzhou Oriental People's Hospital, Yunlong District, Xuzhou, Jiangsu, China (Liu); the First People's Hospital of Changzhou, Tianning District, Changzhou, Jiangsu, China (S. Wang).

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