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The Inferior Parietal Lobule Links Childhood Trauma to Non-Suicidal Self- Injury via Anxiety

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Abstract

Background

Non-suicidal self-injury (NSSI) refers to deliberate self-harm without suicidal intent, and the neural mechanisms underlying disturbed bodily self-consciousness (BSC) in NSSI remain unclear. This study examined cortical thickness across BSC-related regions and their mediating role in the childhood trauma–NSSI relationship.

Methods

A total of 173 young participants (aged 10–24 years) were classified into NSSI (n = 117) and non-NSSI (n = 56) groups based on DSM-5 criteria; structural MRI data with complete surface-quality metrics were available for 128. Cortical thickness was measured bilaterally in five BSC regions: inferior parietal lobule (IPL), supramarginal gyrus, insula, postcentral gyrus, and posterior cingulate cortex. ANCOVA with permutation-based inference and FDR correction, serial mediation analyses with bias-corrected bootstrap confidence intervals, and an exploratory whole-cortex analysis across 34 regions were conducted.

Results

Only the IPL exhibited significant group differences after FDR correction ($p = 0.010$, $pFDR = 0.049$; $d = -0.42$), with the NSSI group displaying thinner cortex; in the whole-cortex analysis, the IPL showed the largest cortical thinning of all 34 regions. Childhood trauma correlated with thinner IPL ($r = -0.19$, $p = 0.039$). Serial mediation revealed a significant indirect pathway from childhood trauma to NSSI through IPL thickness and anxiety (indirect effect = 0.012, 95% CI [0.001, 0.040]); the anxiety-alone pathway was more substantial (34.3%), and full mediation was observed.

Conclusions

Among the five BSC regions, only the IPL was associated with NSSI. Altered higher-order body representation may constitute a neural pathway linking early adversity to NSSI through heightened anxiety.

Background

Non-suicidal self-injury (NSSI), defined as the deliberate destruction of one's body tissue without suicidal intent, is a significant public health concern, with lifetime prevalence rates reaching 17% among adolescents [1]. NSSI emerges during early adolescence, peaks in mid-adolescence, and subsides by early adulthood [2], indicating that adolescence constitutes a period of heightened vulnerability. Studies

have identified numerous risk factors, including childhood adversity [3]; however, the neurobiological mechanisms underlying their increased NSSI risk remain unclear.

NSSI's defining feature - deliberate damage to one's body - raises fundamental questions about how individuals with NSSI perceive and relate to their physical selves. Bodily self-consciousness (BSC), that is, the conscious experience of owning a body, inhabiting it, and perceiving the world from its perspective [4], is central to these questions. Behavioral studies support the presence of BSC disturbances in NSSI. Individuals exhibit elevated pain thresholds, altered interoceptive accuracy [5, 6], and diminished "body trust" [7, 8], while adolescents with NSSI demonstrate enhanced susceptibility to the rubber hand illusion, suggesting more malleable body ownership boundaries [9]. BSC's neural substrates form a hierarchical processing system [4, 10]. Primary somatosensory and interoceptive processing occurs in the postcentral gyrus and insula. Intermediate multimodal integration occurs in the supramarginal gyrus. Higher-order body ownership and self-location are supported by the inferior parietal lobule (IPL) and posterior cingulate cortex (PCC) [11–13]. Disruption at any level could contribute to altered bodily self-experience in NSSI. We previously reported reduced IPL thickness in this cohort within mentalizing and suicide-risk frameworks [14, 15]; however, no study has examined cortical structure across multiple levels of the BSC hierarchy within a single a priori framework, or tested whether BSC-related structure links childhood trauma to NSSI.

Childhood trauma is one of the most robust predictors of NSSI, with meta-analyses demonstrating significantly higher rates among individuals with NSSI history [16]. Evidence suggests that childhood abuse may alter the neurodevelopment of body-related brain circuits [17–19], leading to enduring difficulties in accepting the body as one's own and integrated. Furthermore, dissociative symptoms involving disturbances in body experience integration are elevated in trauma-exposed individuals and rank among the strongest correlates of NSSI [20, 21].

If childhood trauma affects BSC-related brain regions, anxiety may link these neural alterations to NSSI behavior. Theoretical models reveal that NSSI leads to emotional regulation, providing relief from intense negative affect [22]. Particularly, anxiety is relevant because it is a body-based experience characterized by physiological arousal, with regions involved in BSC overlapping with circuits implicated in interoceptive awareness of bodily states [23, 24]. Clinical descriptions emphasize the need for relief from anxious arousal as a key motivator for self-injury. However, despite anxiety disorders co-occurring in 29–53% of NSSI samples across neuroimaging studies [25, 26], their role as a neural mediator has not been systematically examined.

To address this gap, we propose an integrative model in which childhood trauma affects the development of BSC-related cortical structures, which manifest as heightened anxiety; thus, increasing vulnerability to NSSI. This study tested this model by examining cortical thickness in five regions of interest (ROIs), selected based on the hierarchical BSC framework: IPL, supramarginal gyrus, insula, postcentral gyrus, and PCC. Specifically, we assessed whether: (1) any of these regions reveal group differences between NSSI and non-NSSI groups; (2) regions showing significant differences relate to

childhood trauma; and (3) IPL thickness and anxiety mediate the relationship between childhood trauma and NSSI. Given the age-specific nature of NSSI and the protracted maturation of BSC-related cortical regions from early adolescence to early adulthood, we conducted exploratory developmental analyses to examine whether brain-NSSI associations varied by age.

Methods

Participants

Participants were young people (aged 10–24 years) recruited from Beijing Huilongguan Hospital, China. Their age range encompassed adolescents (10–19 years) and youth (15–24 years), as defined by the World Health Organization [27], covering the period of highest NSSI risk. All the participants presented mental health concerns and received structured clinical assessments. Inclusion criteria were: (1) aged 10–24 years, (2) ability to provide informed consent/assent, and (3) no contraindications for magnetic resonance imaging (MRI). Exclusion criteria included a neurological disorder, a current substance use disorder, or intellectual disability.

This study included only psychiatric patients; no healthy controls were enrolled. Participants were classified according to the Diagnostic and Statistical Manual of Mental Disorders, 5th Edition (DSM-5) criteria for NSSI history. Those with ≥ 5 NSSI episodes in the past 12 months were classified as the NSSI group, exhibiting clinically significant NSSI, while those with < 5 episodes formed the non-NSSI group [28]. Nine members of the non-NSSI group lacked a documented episode count; their allocation rested on the psychiatrist-conducted enrollment interview, which confirmed that the five-episode threshold had not been reached over the preceding year [15]. This enabled the examination of NSSI-specific neural correlates in a clinically relevant population while controlling for transdiagnostic effects of general psychopathology.

Of the 173 participants with clinical data (NSSI: n = 117; non-NSSI: n = 56), 28 did not complete MRI scanning (declined or unable to tolerate the procedure); quality control removed a further 13 of the 145 acquired scans—nine because of marked motion-related artifacts and four because the FreeSurfer reconstruction could not be repaired—so that usable imaging data were available for 132 participants, as detailed for this cohort in [14]. The present analyses included the 128 participants (NSSI: n = 85; non-NSSI: n = 43) with complete cortical-thickness and surface-quality (surface-hole count) data; because of this additional data requirement, the imaging subsample differs slightly from those of our previous reports [14, 15], and regional estimates are therefore not numerically identical across these papers. Partial correlations used all participants with available data for each pair of variables (n = 115–124 per pair); the serial mediation analysis included participants with complete MRI, CTQ, HAMA, and NSSI-frequency data (n = 114); the simple mediation model, which did not require CTQ data, included 115 participants. The study was approved by the Institutional Review Board of Beijing Huilongguan Hospital (approval number: 2021-23). All adult participants provided written informed consent; participants under 18 provided assent with parental consent.

Measures

Psychiatric diagnoses were established using the Structured Clinical Interview for DSM-IV (SCID-IV), administered by trained clinicians. NSSI was assessed with the Adolescent Non-Suicidal Self-Injury Assessment Questionnaire, a validated instrument for Chinese adolescents [29]. The questionnaire captures the number of self-injury episodes over the preceding 12 months—on which the ≥ 5 vs. <5 episode grouping was based—together with a 13-item checklist covering distinct methods of self-injury. Throughout this paper, NSSI frequency denotes the behavior-frequency composite score used in our earlier work [15]: the 12-month frequency of each of the 13 methods was rated from 0 (never) to 4 (always), and the ratings were summed to yield a total between 0 and 52. The score therefore captures both how regularly and through how many different methods a person self-injures, and should not be read as a count of discrete episodes. Childhood trauma was assessed using the Childhood Trauma Questionnaire (CTQ), a 28-item retrospective self-report measure [30]. It assessed five types of childhood maltreatment: emotional abuse, physical abuse, sexual abuse, emotional neglect, and physical neglect. Items were rated on a 5-point scale, with higher scores indicating greater trauma exposure. The CTQ demonstrated good reliability and validity in Chinese adolescent samples [31]. Anxiety symptoms were measured using the Hamilton Anxiety Rating Scale (HAMA-14) [32], a clinician-administered measure of anxiety severity, with established reliability and validity [33]. Total scores ranged from 0 to 56, with higher scores indicating greater anxiety.

MRI acquisition and processing

Structural MRI data were acquired using a 3.0 Tesla Siemens Prisma scanner (Siemens Healthineers, Erlangen, Germany) equipped with a 64-channel head coil. High-resolution T1-weighted anatomical images were obtained using a three-dimensional magnetization-prepared rapid gradient-echo sequence with the following parameters: repetition time = 2300 ms, echo time = 2.98 ms, flip angle = 9°, field of view = 256 × 256 mm, matrix size = 256 × 256, slice thickness = 1.0 mm (no gap), number of slices = 192, voxel size = 1.0 × 1.0 × 1.0 mm^3, total acquisition time = 5 min 21 s.

Images were processed using FreeSurfer v7.1 [34]. The automated pipeline included motion correction, skull stripping, gray/white matter segmentation, and cortical surface reconstruction. Cortical thickness, computed as the shortest distance between white matter and pial surfaces, was extracted for five bilateral ROIs based on the Desikan-Killiany atlas [35]: IPL, supramarginal gyrus, postcentral gyrus, insula, and PCC (Fig. 1). Bilateral averages were computed in accordance with ENIGMA consortium protocols [36]. Quality control followed a two-stage protocol pairing an automated check of reconstruction quality with visual inspection by two trained raters blind to group status (Cohen's κ = 0.89); full criteria are reported in [14]. Scans showing marked motion-related artifacts were removed. For display, the five ROIs and the regional effect sizes of the whole-cortex analysis were rendered on a partially inflated fs_LR 32k template surface [37] carrying the Desikan–Killiany parcellation; because bilateral means were analyzed, values are shown on the left-hemisphere template.

Statistical analysis

Differences in cortical thickness were examined using analysis of covariance (ANCOVA), with group (NSSI vs. non-NSSI) as the independent variable and age and sex as covariates. Homogeneity of variance and normality were assessed using Levene's test and the Shapiro–Wilk test; because thickness distributions departed from normality in the NSSI group, primary inference for group differences was based on permutation tests (100,000 random permutations of group labels applied to covariate-adjusted residuals), with parametric F tests, heteroscedasticity-consistent (HC3) standard errors, and the rank-based Mann–Whitney U test reported alongside. The false discovery rate (FDR) correction was applied across the five ROIs to the permutation p values; parametric FDR-corrected values are reported in Table S1 [38]. Effect sizes were calculated as Cohen's *d* (from the unadjusted group means and the pooled standard deviation), with 95% confidence intervals (CIs). To assess regional specificity, an exploratory whole-cortex analysis was additionally performed across all 34 Desikan–Killiany regions using the same ANCOVA model, with FDR correction applied to the parametric p values across the 34 regions.

For regions showing significant group differences, associations with childhood trauma using partial correlations, controlling for age and sex, were examined. Subsequently, serial mediation analysis was conducted using bootstrap methods (200,000 iterations) to test the hypothesized pathway from childhood trauma (X) through cortical thickness (M1) and anxiety (M2) to NSSI frequency (Y). Additionally, a simple mediation analysis was performed to determine whether anxiety mediated the relationship between cortical thickness and NSSI frequency. Both mediation models were adjusted for age and sex. The indirect effect was considered significant if the 95% bias-corrected CI did not include zero [39].

Furthermore, the potential developmental sensitivity of significant brain-behavior associations was examined, which included testing the age × group interaction on cortical thickness and, where appropriate, performing Johnson–Neyman analysis to identify age thresholds at which associations transitioned from significant to non-significant [40]. These analyses were exploratory, given the cross-sectional design and sample size limitations. Sensitivity analyses included: covariate adjustment for borderline personality disorder (BPD) diagnosis; additional adjustment for current mood and anxiety disorders, intracranial volume, global mean cortical thickness, depressive symptom severity (Hamilton Depression Rating Scale, HAMD), and surface reconstruction quality (number of surface holes, a proxy for the Euler number); propensity score matching for age and sex (1:1 nearest-neighbour matching on the logit of the propensity score, caliper 0.2 SD, without replacement); and E-value calculation to assess robustness to unmeasured confounding [41]. E-values were derived from the age- and sex-adjusted odds ratio for NSSI per 1-SD decrease in IPL thickness, converted to an approximate risk ratio by taking its square root because the outcome was common. All analyses were conducted in Python (version 3.10) using NumPy, SciPy, pandas, statsmodels, and pingouin packages.

Results

Sample characteristics

Sample characteristics are presented in Table 1. The NSSI group reported higher childhood trauma (CTQ: 48.78 ± 14.49 vs. 42.75 ± 14.14, $p = 0.013$) and anxiety (HAMA: 17.90 ± 11.06 vs. 11.82 ± 9.74, $p = 0.001$), along with higher rates of BPD (41.9% vs. 7.1%, $p < 0.001$). The groups did not differ in age, sex, or intracranial volume.

Table 1
Sample characteristics

Demographic variables	NSSI (n = 117)	Non-NSSI (n = 56)	χ^2/t	P
Sex, female	83 (70.9%)	36 (64.3%)	0.50	0.479
Age, years	15.90 ± 2.82	15.68 ± 3.08	0.46	0.643
Education, years	11.1 ± 2.8	10.6 ± 3.4	1.02	0.310
Duration of illness (in years)	1.27 ± 1.3	1.36 ± 1.5	−0.41	0.686
Clinical assessments				
NSSI frequency, 12 months	15.47 ± 8.12	2.98 ± 5.56	9.91	< 0.001***
HAMA-14	17.90 ± 11.06	11.82 ± 9.74	3.36	0.001**
CTQ total	48.78 ± 14.49	42.75 ± 14.14	2.51	0.013*
Emotional abuse	12.54 ± 5.15	10.04 ± 4.52	3.02	0.003**
Physical abuse	7.50 ± 3.15	6.69 ± 2.98	1.57	0.119
Sexual abuse	5.00 ± 0.00	5.00 ± 0.00	—	—
Emotional neglect	13.77 ± 4.92	12.21 ± 5.12	1.87	0.063
Physical neglect	9.01 ± 3.75	8.10 ± 2.89	1.56	0.121
Psychiatric conditions				
Mood disorders	91 (77.8%)	42 (75.0%)	0.05	0.832
Anxiety disorders	44 (37.6%)	13 (23.2%)	2.93	0.087
Eating disorders	1 (0.9%)	1 (1.8%)	Fisher	0.544
Borderline personality disorder	49 (41.9%)	4 (7.1%)	19.90	< 0.001***

Note. Values are shown as n (%) or mean ± standard deviation. CTQ = Childhood Trauma Questionnaire; HAMA = Hamilton Anxiety Rating Scale; NSSI = non-suicidal self-injury. Non-NSSI group = participants with < 5 NSSI episodes. *p < 0.05, **p < 0.01, ***p < 0.001. The sexual abuse subscale was at the scale minimum for all participants with available data and was not tested. Instruments were not completed by all participants; valid n (NSSI/non-NSSI): HAMA 114/50, CTQ 114/52, NSSI frequency 115/50. Test statistics use valid cases only. NSSI frequency refers to the behavior-frequency composite score, which aggregates 13 method-specific frequency ratings over the past 12 months. Continuous variables were compared with pooled-variance (Student's) t tests and categorical variables with Yates-corrected χ2 tests.

Group differences in cortical thickness

The ANCOVA results are presented in Fig. 2A and Table S1. Only the IPL revealed significant group differences after FDR correction across the five ROIs ($F(1, 124) = 6.82$, parametric $p = 0.010$; permutation $p = 0.010$, $pFDR = 0.049$; partial $\eta^2 = 0.052$; parametric $pFDR = 0.051$). The NSSI group displayed a thinner IPL cortex (2.556 ± 0.170 mm vs. 2.622 ± 0.125 mm; $d = -0.42$, 95% CI [− 0.79, − 0.05]; Fig. 2B). Other ROIs showed no significant differences (all $pFDR \geq 0.45$). The IPL difference was supported by heteroscedasticity-robust inference (HC3 $p = 0.005$), whereas the rank-based Mann–Whitney U test was attenuated to a trend ($p = 0.075$); the effect was present in both hemispheres (left: $d = -0.42$, $p = 0.018$; right: $d = -0.35$, $p = 0.027$). In the exploratory whole-cortex analysis (Fig. 3; Table S2), the IPL showed the largest cortical thinning of all 34 Desikan–Killiany regions ($d = -0.42$), with a nominal reduction in the entorhinal cortex ($d = -0.40$, $p = 0.025$); the transverse temporal gyrus showed the largest absolute difference overall, in the opposite direction (thicker in NSSI; $d = 0.68$, $pFDR = 0.021$), surviving FDR correction across the 34 regions.

Associations between IPL and clinical variables

As only IPL revealed significant group differences, subsequent analyses focused on this region. Partial correlations, controlling for age and sex, are presented in Fig. 2C. IPL thickness revealed significant correlations with childhood trauma (CTQ total: $r = -0.19$, $p = 0.039$; Fig. 4), anxiety ($r = -0.31$, $p < 0.001$), and NSSI frequency ($r = -0.33$, $p < 0.001$). Childhood trauma was also correlated with anxiety ($r = 0.33$, $p < 0.001$) and NSSI frequency ($r = 0.28$, $p = 0.002$), and anxiety with NSSI frequency ($r = 0.45$, $p < 0.001$). Among CTQ subscales, emotional neglect had the strongest association with IPL thickness ($r = -0.20$, $p = 0.023$), followed by physical neglect ($r = -0.18$, $p = 0.048$), whereas physical abuse ($r = -0.15$, $p = 0.112$) and emotional abuse ($r = -0.14$, $p = 0.113$) were not significantly associated. The sexual abuse subscale showed no variance in this sample (all participants with available data scored the scale minimum of 5) and was therefore not analysed.

Serial mediation analysis: childhood trauma → IPL → anxiety → NSSI

Serial mediation analysis tested the hypothesized pathway from childhood trauma to NSSI frequency through IPL thickness and anxiety (Fig. 5; $n = 114$), with a significant total effect ($c = 0.178$, $p = 0.002$). Furthermore, the serial indirect effect through IPL thickness and anxiety was significant ($a_1 \times d_{21} \times b_2 = 0.012$, bias-corrected 95% CI [0.001, 0.040]; percentile 95% CI [− 0.001, 0.031]), accounting for 6.6% of the total effect. Additionally, the specific indirect effect through anxiety alone was substantial and significant ($a_2 \times b_2 = 0.061$, bias-corrected 95% CI [0.020, 0.135]), accounting for 34.3% of the total effect. However, the specific indirect effect through IPL thickness alone was not significant ($a_1 \times b_1 = 0.020$, bias-corrected 95% CI [− 0.001, 0.070]). The direct effect of childhood trauma on NSSI frequency did not reach significance after accounting for the mediators ($c' = 0.085$, $p = 0.125$), consistent with full mediation. Hence, collectively, the indirect pathways accounted for approximately 52% of the total effect, with the anxiety pathway being the largest contributor.

Simple mediation: IPL thickness → anxiety → NSSI frequency

To further characterize the IPL-NSSI relationship, we tested anxiety as a mediator of this relationship (Figure S1; n = 115). The total effect of IPL on NSSI was significant (c = − 18.608, p < 0.001). Thinner IPL was associated with higher anxiety (a = − 22.132, p < 0.001), which predicted more NSSI, controlling for IPL (b = 0.328, p < 0.001). The indirect effect through anxiety was significant (ab = − 7.257, bias-corrected 95% CI [− 14.124, − 2.487]), accounting for 39.0% of the total effect; the direct effect remained significant (c' = −11.351, p = 0.030), consistent with partial mediation.

Exploratory developmental analyses

We examined potential developmental sensitivity of the IPL-NSSI association (Figure S2). The age × group interaction effect on IPL thickness did not reach statistical significance (p = 0.303). Nevertheless, given the theoretical interest in developmental effects, we conducted follow-up exploratory analyses with appropriate caveats about interpretation.

The Johnson–Neyman analysis suggested that the IPL group difference was significant for ages below 17.4 years. Descriptive age-stratified patterns revealed the largest effect in early adolescence (10–14 years, n = 46: d = − 0.69, p = 0.089), with attenuated effects observed in mid-adolescence (15–18 years, n = 59: d = − 0.31, p = 0.141) and non-significant effects in emerging adulthood (19–24 years, n = 23: d = − 0.24, p = 0.904); none of the stratum-level contrasts reached statistical significance. The non-NSSI group exhibited expected normative cortical thinning with age (β = −16.6 μm/year, p = 0.005) [42, 43], while the NSSI group exhibited a flattened trajectory (β = −6.2 μm/year, p = 0.335). These exploratory findings suggested that the IPL-NSSI association may be more pronounced during adolescence, although replication in larger samples with longitudinal designs is needed.

Sensitivity analyses

Results were generally robust across sensitivity analyses (Table S3). Controlling for BPD diagnosis did not attenuate the IPL group difference (F = 9.41, p = 0.003). The difference also remained significant after additionally adjusting for current mood and anxiety disorders (F = 8.55, p = 0.004), intracranial volume (F = 7.72, p = 0.006), and global mean cortical thickness (F = 11.78, p < 0.001), and was attenuated to marginal significance after adjusting for depressive symptom severity (HAMD; F = 3.16, p = 0.079, n = 104) and for surface reconstruction quality (number of surface holes; F = 3.76, p = 0.055; Figure S3). Propensity score matching (1:1 nearest-neighbour matching on the logit of the propensity score for age and sex, caliper 0.2 SD, without replacement) yielded 43 matched pairs, with the IPL difference remaining significant (d = − 0.58, paired t-test p = 0.004). In logistic regression adjusted for age and sex, the odds of NSSI increased by a factor of 1.82 (95% CI 1.14–2.90) per 1-SD decrease in IPL thickness (approximate risk ratio 1.35, 95% CI 1.07–1.70). The corresponding E-value was 2.03 (1.34 for the CI bound), indicating that an unmeasured confounder would need to be associated with both IPL thickness and NSSI by a risk

ratio of at least 2.03 to fully explain the observed association, suggesting moderate robustness to unmeasured confounding.

Discussion

This study examined cortical thickness across five BSC regions in young people with and without clinically significant NSSI. Three key findings emerged. First, among the five BSC regions examined, only IPL exhibited significant group differences, suggesting a selective involvement of higher-order body representations in NSSI. Second, IPL thickness was associated with childhood trauma, particularly emotional and physical neglect. Third, serial mediation analysis revealed that the relationship between childhood trauma and NSSI operated through IPL thickness and anxiety, with the latter accounting for the largest proportion of this mediated effect.

The five regions serve different purposes within the body representation system: primary sensory processing (postcentral gyrus), interoception (insula), self-referential processing (PCC), and multimodal integration (supramarginal gyrus and IPL). Hence, the selective association with IPL has important implications for understanding how NSSI relates to the BSC hierarchy. It suggests that NSSI relates specifically to disturbances at the integrative apex of the BSC hierarchy rather than to primary sensory or interoceptive processing. The IPL integrates diverse sensory signals into coherent body representations [44–46], thereby supporting the conscious experience of the body as a unified entity that belongs to oneself [4, 47]. Our findings suggest that this integrative function may be compromised in NSSI.

The absence of significant differences in other BSC regions warrants consideration. The insula, often considered the hub of interoceptive processing [48], did not differ between groups despite theoretical expectations. This suggests that NSSI primarily affects higher-order integrative processes rather than basic interoceptive signaling. Furthermore, it suggests that insular involvement in NSSI is primarily functional rather than structural, consistent with meta-analyses reporting altered insula activation and variable structural findings [49, 50]. Additionally, our psychiatric non-NSSI comparison group controls for transdiagnostic effects that may explain some previously reported differences when comparing NSSI individuals to healthy controls [51].

Beyond the a priori BSC regions, the whole-cortex analysis identified greater transverse temporal (Heschl's gyrus) thickness in the NSSI group, the largest absolute difference of any region. This replicates the most robust group marker in our previous analysis of this cohort [15] and converges with a report of increased left transverse temporal thickness in adolescents with depression and NSSI [52]. Because this region lies outside the body-representation framework tested here and the direction of the difference is opposite to the IPL finding, it likely reflects a distinct mechanism - possibly atypical maturation of auditory cortex implicated in inner speech - rather than compromising the interpretation of IPL-specific thinning; nevertheless, it underscores that NSSI-related structural variation is not confined to BSC circuitry.

The association between childhood trauma and reduced IPL thickness extends neurodevelopmental models of adversity. Early-life stress affects brain development through multiple mechanisms, including altered synaptic pruning, glucocorticoid-mediated effects, and disruptions to experience-dependent plasticity [17, 18, 53]. Our findings suggest that BSC-related cortical regions, particularly the IPL, may be vulnerable to these effects. This is consistent with theoretical accounts hypothesizing that childhood maltreatment disrupts the development of integrated body-self representations. A UK Biobank study revealed that childhood maltreatment influenced adult brain structure, including temporo-parietal regions, through immune, metabolic, and psychosocial factors [54]. Notably, emotional and physical neglect exhibited the strongest associations with IPL thickness, aligning with evidence that chronic relational trauma is particularly detrimental to self-related neural circuits.

The serial mediation pathway – childhood trauma → IPL thickness → anxiety → NSSI – provides an integrative account, linking early adversity to self-injury through neural structure and affective processes. Notably, the specific indirect effect through anxiety alone (34.3%) was substantially larger than that through IPL and anxiety (6.6%), although the specific indirect effect through IPL alone was not significant. This suggests that anxiety represents the primary mechanistic link to NSSI behavior and that the contribution of the IPL operates largely through its association with anxiety. Thus, we suggest that childhood adversity may disrupt the development of stable body representations, manifesting as reduced IPL integrity and contributing to heightened anxiety through compromised interoceptive processing or unstable body ownership. The resulting anxiety subsequently drives NSSI as a maladaptive regulatory strategy [22].

The finding that anxiety significantly mediated the IPL-NSSI relationship replicates our previous single-mediator result in this cohort [15] and embeds it within a serial framework that begins with childhood trauma. It aligns with research demonstrating that interoceptive deficits exert indirect effects on self-injurious behaviors [55, 56]. The overlap between BSC circuits and interoceptive awareness networks provides a plausible neural substrate for this mediation [23]. However, cross-sectional data limit causal inference, and alternative models (e.g., anxiety affecting IPL and NSSI) cannot be excluded. Hence, longitudinal studies with repeated assessments of all variables would establish temporal precedence and strengthen causal inference.

Although exploratory developmental analyses suggested that the IPL-NSSI association was more pronounced in adolescence, these findings should be interpreted with caution, given that the age × group interaction did not reach statistical significance. If replicated, such a pattern would be consistent with the known developmental trajectory of NSSI [2] and the protracted maturation of the parietal cortex through adolescence, with higher-order association areas maturing after the primary sensory cortices [42, 43]. Recent research on adolescent brain development highlighted the importance of cortical excitation-inhibition ratio maturation [57]. The NSSI group's flattened developmental trajectory, with thinner IPL from early ages, is more consistent with pre-existing vulnerability than developmental deviation. However, longitudinal research is needed to establish temporal precedence.

These findings have several clinical implications. The role of childhood trauma in the pathway to NSSI underscores the importance of early intervention and trauma-informed care. Screening for early adversity among young people presenting NSSI behavior could identify people with neurobiologically mediated vulnerability. Furthermore, the mediating role of anxiety supports the use of anxiety-focused treatments (e.g., dialectical behavior therapy and exposure-based interventions) for NSSI [58]. The substantial mediation through anxiety (accounting for over one-third of the IPL-NSSI relationship) suggests that anxiety is a particularly important treatment target. Moreover, interventions targeting bodily self-awareness (e.g., mindfulness-based approaches and body-oriented therapies) could address the hypothesized mechanism by strengthening the coherence of body representation [59, 60].

Nonetheless, several limitations warrant consideration. The cross-sectional design precludes causal inference [61]. The mediation analysis, despite being theoretically motivated and statistically significant, cannot establish causality. Hence, longitudinal studies are needed to determine whether IPL differences precede, follow, or co-develop with NSSI. All the participants were psychiatric patients, which enhances the clinical relevance of the findings, yet limits their generalizability to community samples [62]. Without a healthy control group, we cannot determine whether IPL differences represent NSSI-specific effects or broader markers of psychopathology. Moreover, the sample was predominantly female (69%), limiting conclusions about sex differences. Childhood trauma was assessed retrospectively, potentially introducing recall bias [63]. The sample size limited statistical power for some analyses, particularly the exploratory developmental analyses and tests of moderation [64]. In addition, the present analyses are based on a cohort reported in our previous publications [14, 15, 51]; although the research questions are distinct, the overlapping samples mean that the IPL findings across these papers are not independent replications. Future studies should employ longitudinal designs to track developmental trajectories, include healthy controls to establish specificity, and examine functional connectivity patterns to complement structural findings.

Conclusions

Among the five BSC regions examined, IPL was selectively associated with NSSI. Serial mediation analysis revealed that IPL thickness and anxiety statistically mediated the relationship between childhood trauma and NSSI frequency, with anxiety accounting for a larger proportion of the effect. These findings suggest that early adversity may affect higher-order body representation, contributing to elevated anxiety and increased NSSI risk, with exploratory analyses suggesting that adolescence may represent a window of heightened vulnerability. Hence, anxiety-focused and body-oriented interventions may address these underlying mechanisms.

Abbreviations

ANCOVA
Analysis of covariance
BPD

Borderline personality disorder
BSC
Bodily self-consciousness
CI
Confidence interval
CTQ
Childhood Trauma Questionnaire
DSM-5
Diagnostic and Statistical Manual of Mental Disorders, 5th Edition
FDR
False discovery rate
HAMA
Hamilton Anxiety Rating Scale
HAMD
Hamilton Depression Rating Scale
HC3
Heteroscedasticity-consistent standard errors
IPL
Inferior parietal lobule
MRI
Magnetic resonance imaging
NSSI
Non-suicidal self-injury
PCC
Posterior cingulate cortex
ROI
Region of interest
SCID-IV
Structured Clinical Interview for DSM-IV
SD
Standard deviation.

Declarations

Ethics approval and consent to participate

All procedures contributing to this work complied with the ethical standards of the relevant national and institutional committees on human experimentation and the Declaration of Helsinki of 1975, as revised in 2013. This study was approved by the Institutional Review Board of Beijing Huilongguan Hospital (approval number: 2021-23). Written informed consent was obtained from all adult participants; participants under 18 years provided written assent together with written consent from a legal guardian.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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Author Contribution

Y.C. and Y.T. conceptualized the study. Y.C., X.L., X.W., Z.D., Y.L., Z.W., S.P., X.G.L., H.D., L.T., X.H.L. and C.S.R.L. developed the methodology. Y.C., N.L., P.Z. and Y.T. conducted the investigation. Y.C., W.C., M.G. and T.Y. developed the software. Y.C. and Y.T. performed the formal analysis, and Y.C., X.W. and Y.T. curated the data. J.W., W.C., Y.Y., M.G., W.L., J.T., T.X., T.Y., Y.L. and X.Y. provided resources. X.W. supervised the study, and Y.T. administered the project. Z.D., S.C., X.Y., S.P. and Y.T. acquired funding. Y.C. wrote the original draft of the manuscript, and Y.C., T.T. and Y.T. reviewed and edited it. All authors read and approved the final manuscript.

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Data Availability

Data supporting the findings of this study are available from the corresponding author, YT, upon reasonable request.

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Figures

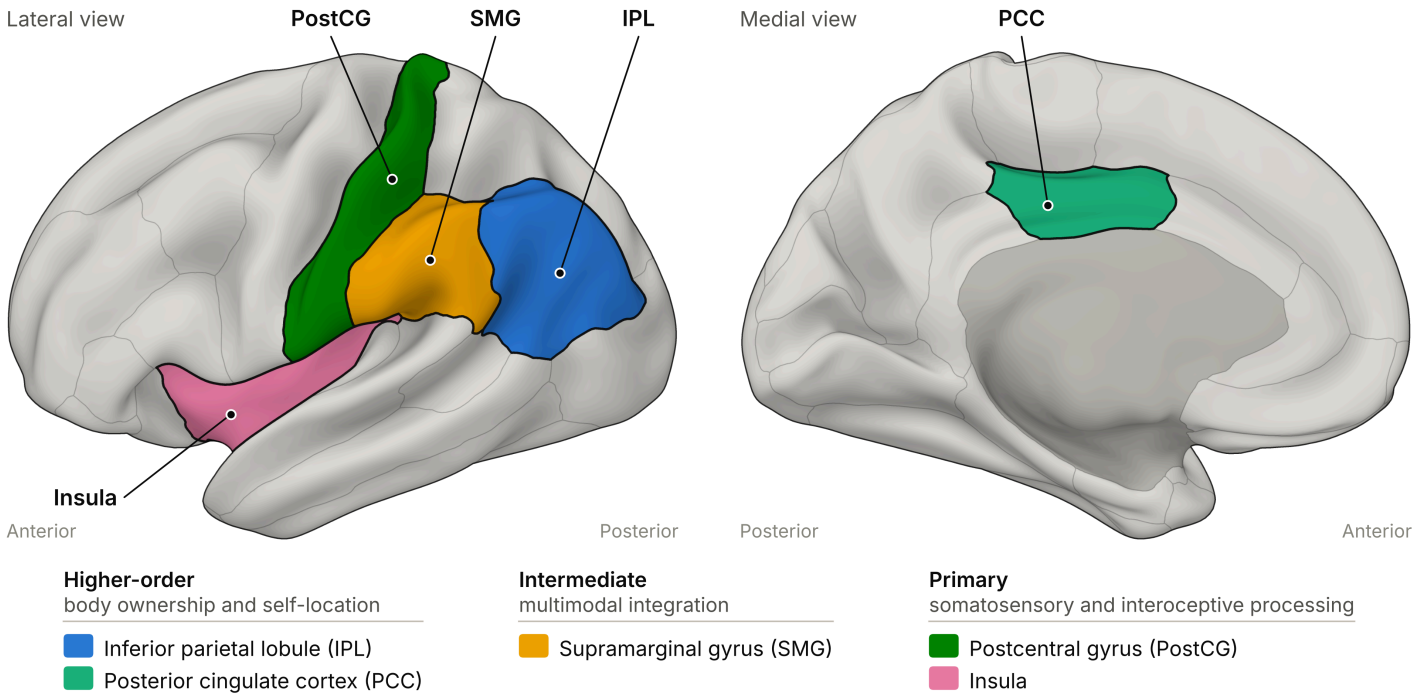


Figure 1

A priori regions of interest within the bodily self-consciousness (BSC) hierarchy. The five Desikan–Killiany regions are shown on a partially inflated left-hemisphere template surface (fs_LR 32k; lateral and medial views) and grouped by the level of the BSC hierarchy to which they were assigned. Each region is colored only in the view in which it is mainly visible. Thin gray lines mark the boundaries of the remaining atlas regions, and the medial wall is shown in darker gray. BSC = bodily self-consciousness; IPL = inferior parietal lobule; PCC = posterior cingulate cortex; PostCG = postcentral gyrus; SMG = supramarginal gyrus.

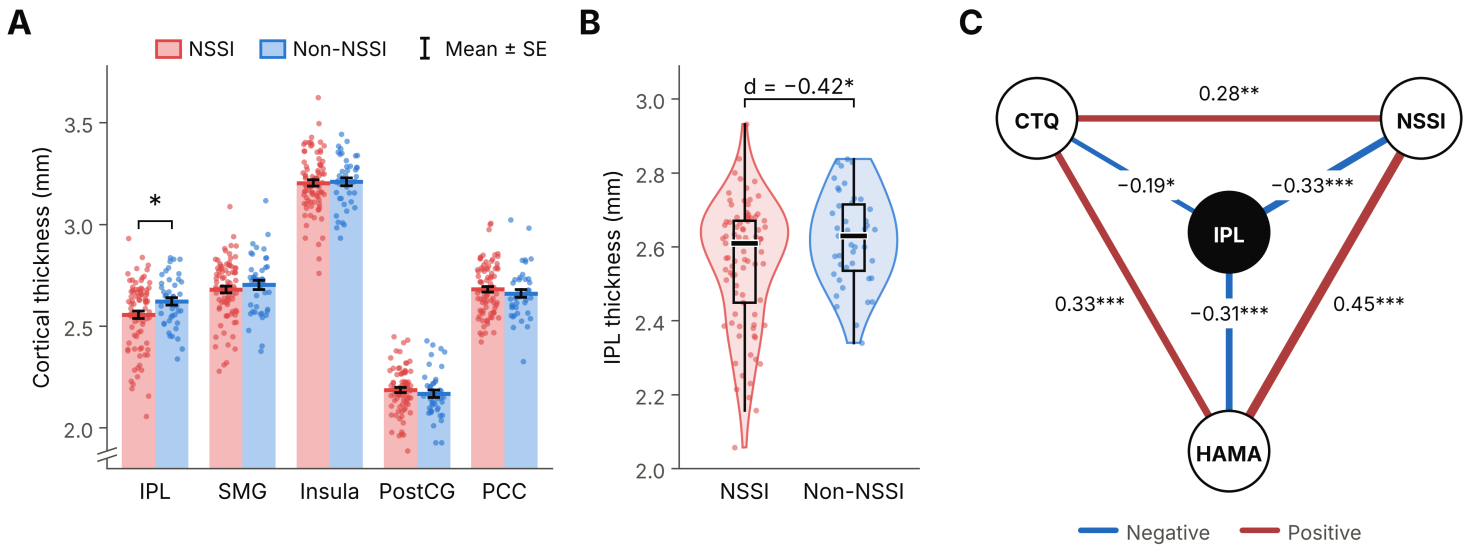


Figure 2

Cortical thickness in BSC regions and associations with clinical variables. (A) Mean cortical thickness (±standard error; horizontal lines with error bars) in five BSC regions for NSSI (n = 85) and non-NSSI (n = 43) groups, with individual data points overlaid. (B) Distribution of IPL thickness by group (violin plots with overlaid box plots and individual data points). (C) Partial correlations among CTQ total score, IPL thickness, HAMA score, and NSSI frequency (n = 115–124 per pair), controlling for age and sex. Line thickness represents correlation magnitude, with blue and red indicating negative and positive correlations, respectively. *pFDR < 0.05 (A, B); *p < 0.05, **p < 0.01, ***p< 0.001 (C). BSC = bodily self-consciousness; CTQ = Childhood Trauma Questionnaire; HAMA = Hamilton Anxiety Rating Scale. IPL = inferior parietal lobule; NSSI = non-suicidal self-injury; PostCG = postcentral gyrus; PCC = posterior cingulate cortex; SMG = supramarginal gyrus.

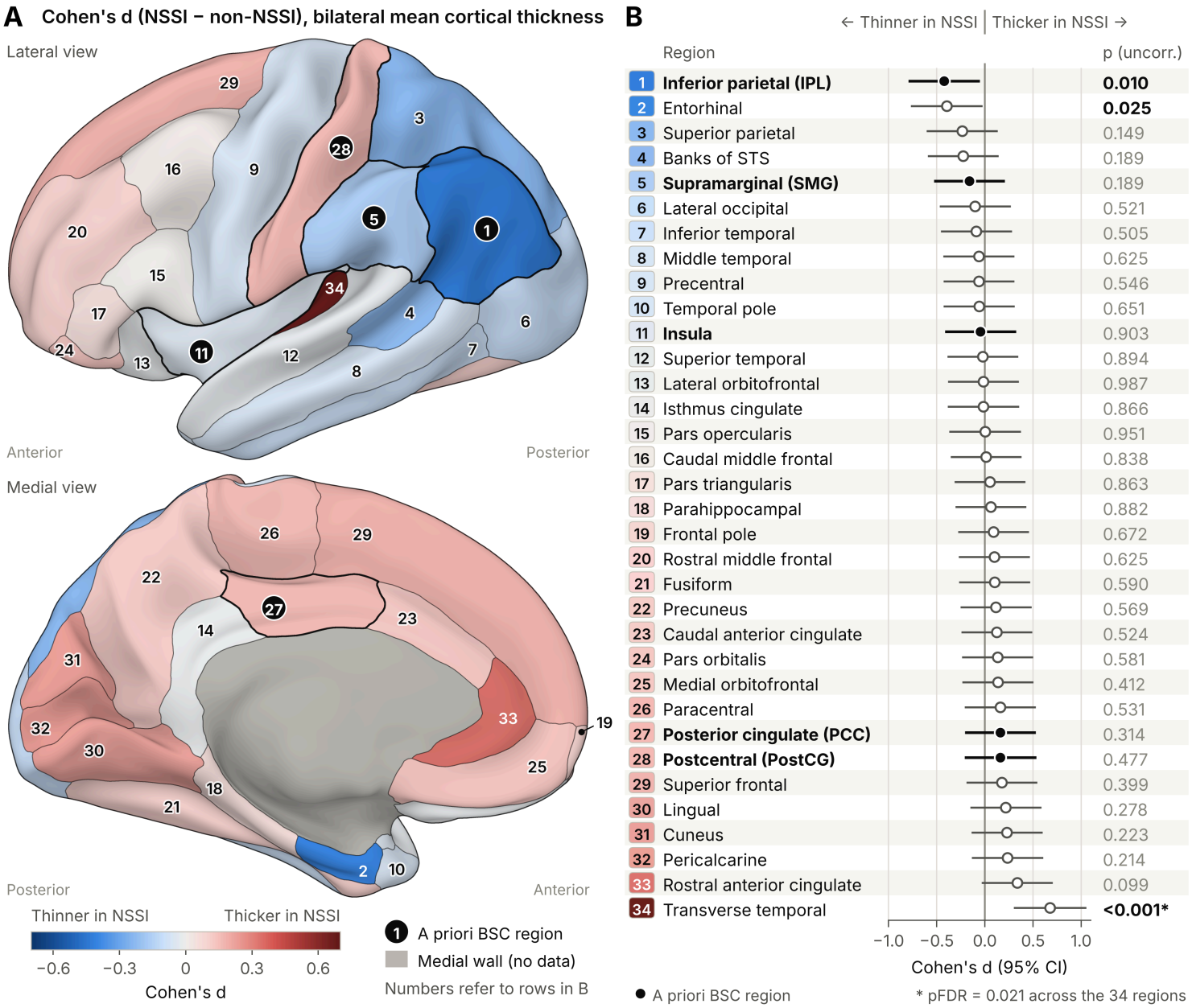


Figure 3

Whole-cortex comparison of cortical thickness between the NSSI and non-NSSI groups. (A) Cohen's d (NSSI − non-NSSI) for bilateral mean cortical thickness in the 34 Desikan–Killiany regions, displayed on a partially inflated left-hemisphere template surface (fs_LR 32k); because bilateral means were analyzed, one hemisphere is shown. The lateral view is seen from 20° above and the medial view from 10° below the horizontal to expose the supratemporal plane and the ventral temporal lobe. Blue indicates thinner and red thicker cortex in the NSSI group. The medial wall is shown in gray. Numbers identify the regions and correspond to the rows in (B); circled numbers and heavier outlines mark the five a priori BSC regions of interest. (B) Cohen's d (pooled standard deviation) with 95% CI for all 34 Desikan–Killiany regions, ordered by effect size (n = 123–128 per region). Filled markers denote the five a priori BSC regions of interest. Uncorrected p values from ANCOVAs controlling for age and sex are listed on the right; *pFDR < 0.05 across all 34 regions. BSC = bodily self-consciousness; CI = confidence interval; IPL = inferior parietal lobule; NSSI = non-suicidal self-injury; PCC = posterior cingulate cortex; PostCG = postcentral gyrus; SMG = supramarginal gyrus.

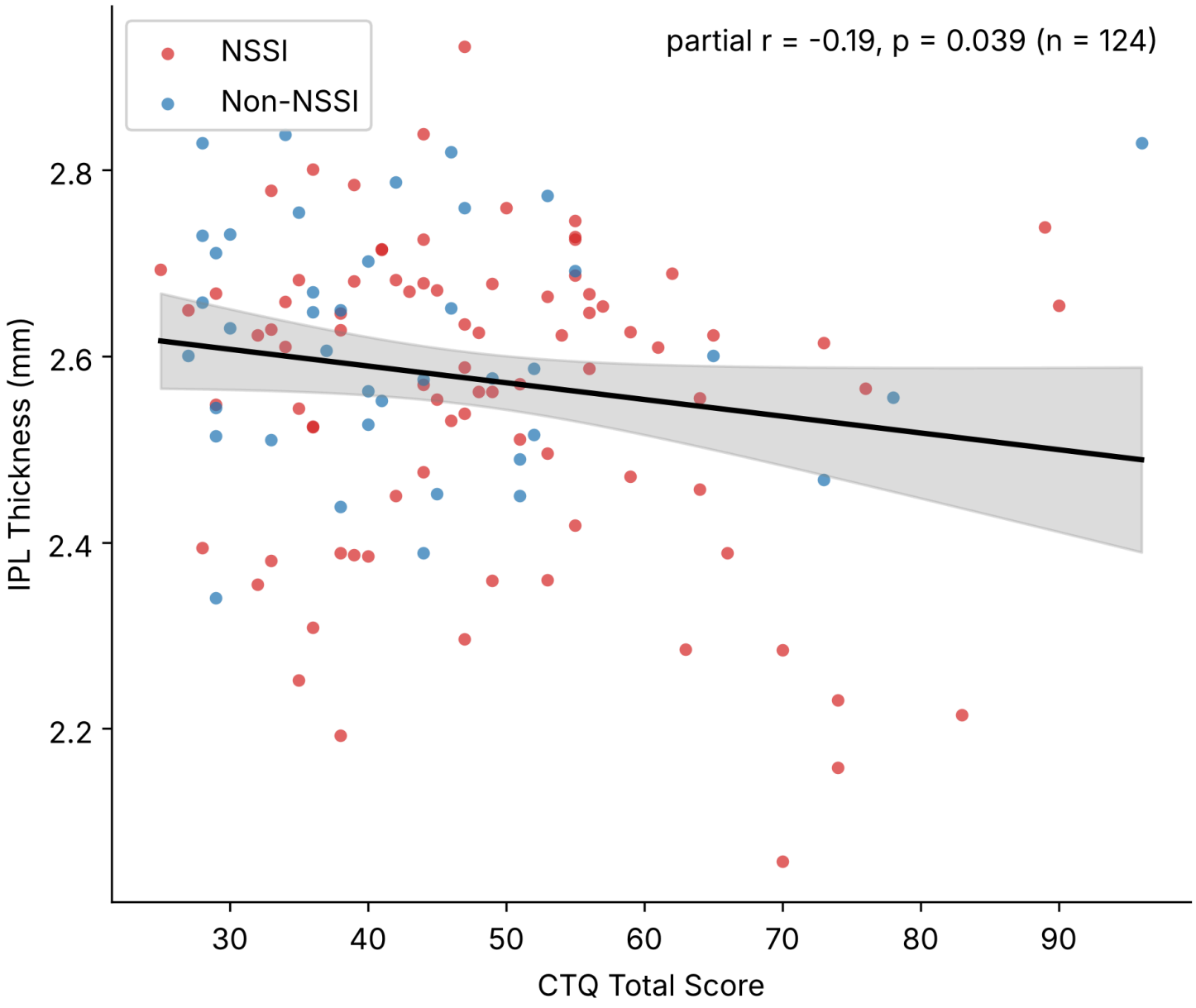


Figure 4

Association between childhood trauma and IPL thickness. IPL cortical thickness is plotted against the CTQ total score (unadjusted values; n = 124); the line shows the linear fit and the shaded area its 95% CI. The partial correlation controlling for age and sex is given in the panel. Red = NSSI group; blue = non-NSSI group. CTQ = Childhood Trauma Questionnaire; IPL = inferior parietal lobule; NSSI = non-suicidal self-injury.

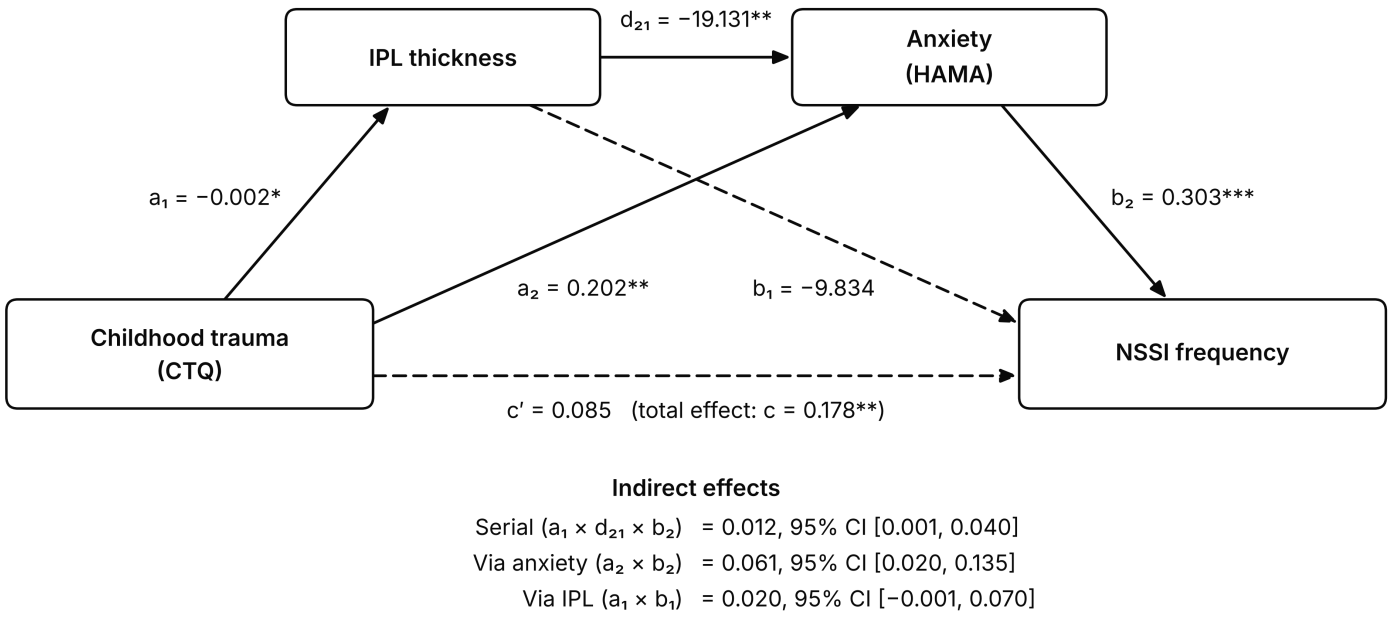


Figure 5

Serial mediation model: Childhood trauma → IPL thickness → Anxiety → NSSI frequency. Path coefficients are unstandardized and adjusted for age and sex (n = 114); indirect effects are reported with bias-corrected bootstrap 95% CIs (200,000 resamples). Solid lines indicate significant paths; dashed lines indicate non-significant paths; c = total effect, c' = direct effect. CTQ = Childhood Trauma Questionnaire; IPL = inferior parietal lobule; HAMA = Hamilton Anxiety Rating Scale; NSSI = non-suicidal self-injury. *p < 0.05, **p < 0.01, ***p < 0.001.

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