

Frequency-Specific Cognitive Control Network Hypoconnectivity in Adolescents With Elevated Externalizing Symptoms

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Abstract

Background

Externalizing symptoms during adolescence are associated with behavioral difficulties and an increased risk of adverse developmental outcomes, and are often accompanied by difficulties in self-regulation across cognitive and emotional domains. Although prior neuroimaging studies have reported altered brain connectivity in externalizing psychopathology, most evidence comes from functional magnetic resonance imaging, leaving frequency-specific electrophysiological network dynamics poorly understood. We examined resting-state electroencephalographic (EEG) functional connectivity in a community-based sample of 35 adolescents aged 13–15 years with relatively high or low parent-reported externalizing symptoms. Source-level connectivity was estimated using exact low-resolution electromagnetic tomography and lagged phase synchronization across delta-to-gamma frequency bands within the cognitive control network (CCN) and default mode network (DMN).

Results

Adolescents with higher externalizing symptoms showed reduced connectivity in five beta1- and gamma-band CCN connections, whereas no significant group differences were observed within the DMN. After adjustment for internalizing symptoms, bilateral anterior-insula connectivity in the beta1 band and left anterior insula–right dorsolateral prefrontal cortex connectivity in the gamma band remained significant. Lower left dorsolateral prefrontal cortex–right inferior frontal junction beta1 connectivity was also associated with greater self-reported physical aggression.

Conclusions

Elevated externalizing symptoms during adolescence may be linked to altered functional coordination within the cognitive control network. These findings highlight the potential value of frequency-resolved EEG connectivity for characterizing the neural correlates of adolescent externalizing symptoms.

1 Background

Adolescence is a developmental period marked by rapid changes in cognitive, emotional, and social functioning, while large-scale brain networks continue to undergo substantial reorganization across this stage of development [1]. Externalizing symptoms, including impulsive, oppositional, and aggressive behaviors, are particularly relevant in this context because difficulties in self-regulation may involve not only controlling overt behavior but also regulating emotional states. Consistent with this broader view, emotion dysregulation has been associated with externalizing symptoms across the normative–clinical continuum [2], while cognitive-control impairments have also been linked to important dimensions of externalizing psychopathology [3]. Developmental evidence further indicates that early trajectories of emotion dysregulation are associated with later conduct problems as well as attention deficit and hyperactivity disorder (ADHD) and internalizing symptoms [4]. Together, these findings suggest that

externalizing symptoms are better understood in relation to interacting cognitive and affective regulatory processes than through behavioral control alone. Examining the neural networks that support these processes during adolescence may therefore provide a useful window into the neurobiological organization associated with externalizing symptoms.

At the network level, two systems are especially relevant to this broader view of externalizing psychopathology: the cognitive control network (CCN) and the default mode network (DMN). The CCN supports the flexible adjustment and maintenance of goal-directed behavior and is therefore central to cognitive control processes relevant to behavioral regulation [5, 6]. The DMN, in contrast, is more strongly engaged in internally directed and self-referential processing [7, 8], making it potentially relevant to the affective and interpersonal dimensions that often accompany externalizing behavior. Consistent with this possibility, altered DMN connectivity has been linked to specific externalizing-related phenotypes in youth, including callous-unemotional traits in a large community sample [9] and aggression-related dimensions in children and adolescents with disruptive behavior [10]. Taken together, these findings suggest that externalizing symptoms may involve abnormalities across systems supporting both externally directed control and internally oriented processing, providing a rationale for examining the CCN and DMN in parallel rather than focusing on a single regulatory network.

Although network-level alterations in externalizing psychopathology are increasingly recognized, the available evidence is methodologically uneven. In adolescents, resting-state functional magnetic resonance imaging (fMRI) studies have linked externalizing and disruptive behavior problems to altered connectivity and dynamic organization across cognitive-control, affective, and internally oriented networks [10–12]. Electroencephalography (EEG) findings likewise indicate that externalizing behavior is accompanied by frequency-specific alterations in neural activity: a meta-analysis of 62 resting-state EEG studies found higher delta and theta power and lower beta power in externalizing samples, while alpha and gamma effects were less consistent [13]. However, much of the EEG literature has focused on spectral power and event-related potential analyses rather than functional connectivity [14–16], and connectivity studies have been concentrated largely in disorder-specific samples such as ADHD. Source-level resting-state EEG work illustrates the additional information provided by connectivity analyses: using eLORETA and lagged phase synchronization, Ahmadi et al. [17] identified frequency-dependent network alterations in children with ADHD, including widespread reductions in beta-band connectivity. Such findings suggest that externalizing-related neural differences may involve not only where brain activity differs, but also how distributed regions coordinate within specific oscillatory frequency bands. However, to our knowledge, few studies have examined frequency-specific source-level connectivity in community-based adolescents selected based on broader externalizing symptom severity, leaving it unclear whether connectivity alterations observed in disorder-specific samples extend beyond formally diagnosed clinical populations.

A source-level EEG approach is particularly useful for addressing this gap because it retains the temporal and frequency resolution of EEG while reducing some of the interpretive limitations of scalp-level connectivity. Connectivity estimated directly between scalp electrodes can be distorted by volume

conduction, such that apparent coupling may partly reflect activity spreading from common neural sources rather than interaction between distinct regions [18]. Source reconstruction provides a more anatomically informed basis for examining functional interactions, while lagged connectivity measures reduce the contribution of instantaneous components that are particularly susceptible to field spread [19]. Together, these approaches enable examination of large-scale network interactions across distinct oscillatory frequency bands, providing a useful framework for identifying frequency-specific patterns of dysconnectivity associated with externalizing symptoms. This frequency-resolved information cannot be obtained directly from fMRI connectivity, which does not capture oscillatory interactions at the temporal scale measured by EEG.

The present study therefore examined resting-state, source-level EEG functional connectivity within the CCN and DMN in a community-based sample of adolescents with relatively high and low levels of externalizing symptoms. Connectivity was quantified using the exact Low Resolution Electromagnetic Tomography (eLORETA) and lagged phase synchronization (LPS) across canonical frequency bands, allowing us to test whether externalizing-related differences varied across the two networks and across oscillatory frequency bands. Given prior evidence implicating both cognitive-control and default-mode systems in externalizing psychopathology, we hypothesized that adolescents with higher externalizing symptoms would show altered functional connectivity within one or both networks compared with adolescents with lower symptom levels. We further examined whether any identified connectivity differences were associated with self-reported aggression and evaluated the robustness of the findings after accounting for co-occurring internalizing symptoms. By focusing on a community-based, symptom-defined adolescent sample, the study tested whether network-level alterations reported primarily in disorder-specific populations are also detectable outside formally diagnosed clinical groups.

2 Methods

2.1 Participants

We recruited participants from an initial pool of 50 adolescents (aged 13–15 years) referred by school counselors from several middle schools in Tehran. As part of the screening process, parents completed the Child Behavior Checklist (CBCL) [20] via the online platform Porsline. The CBCL assesses a broad range of behavioral and emotional problems in children and adolescents. This questionnaire evaluates several subdomains, including thought problems, attention difficulties, and aggressive behaviors. Additionally, the CBCL yields scores for internalizing problems (e.g., anxiety, depressive symptoms, and social withdrawal), externalizing problems (e.g., oppositionality and rule-breaking), and overall behavioral difficulties. In the current study, we used the Persian version of the CBCL, which has been validated for use in the Iranian population and demonstrates adequate validity and reliability [21]. In the present sample, Cronbach's α was .88 for the Externalizing Problems scale and .81 for the Internalizing Problems scale. Responses were analyzed using the Child Behavior Checklist Analyzer (Version 2.4)[22], and participants were selected based on their externalizing T-scores for further EEG functional connectivity assessment.

A total of 37 students (20 females, 17 males) met the inclusion criteria. Participants were divided into two groups. Those with T-scores below 59 were classified as the low-externalizing symptoms group (low-EXT), whereas those scoring 64 or above were categorized as the high-externalizing symptoms group (high-EXT). Exclusion criteria included any clinical diagnosis of intellectual disability, psychotic or mood disorders, anxiety, obsessive-compulsive disorder, epilepsy, developmental disorders, traumatic brain injury, chronic headaches, or Tourette’s syndrome. During EEG preprocessing, data from two participants were excluded due to excessive noise, resulting in a final sample of 35 adolescents (low-EXT, $n = 18$; high-EXT, $n = 17$) (see Table 1 for demographic characteristics). Socioeconomic status and gender identity were not systematically assessed.

Table 1
Demographic and Behavioral Characteristics by Externalizing Group

Variable	Overall ($N = 35$)	low-EXT ($n = 18$)	high-EXT ($n = 17$)	p
Age, years	14.0 (0.8)	14.0 (0.8)	14.1 (0.8)	.50
Female sex, n (%)	18 (51%)	11 (61%)	7 (41%)	.20
CBCL Externalizing	60.5 (9.2)	53.1 (5.3)	68.3 (4.8)	< .001
CBCL Internalizing	59.3 (8.5)	55.7 (8.5)	63.1 (6.8)	.009
BPAQ Total	84.8 (11.5)	83.9 (11.8)	85.8 (11.4)	.50
Physical Aggression	23.5 (6.3)	21.6 (6.1)	25.5 (6.0)	.074
Verbal Aggression	16.1 (3.2)	16.6 (3.5)	15.7 (3.0)	.593
Anger	20.0 (3.1)	19.7 (3.3)	20.3 (2.9)	.539
Hostility	24.3 (5.4)	25.3 (5.8)	23.2 (4.8)	.260
<i>Note.</i> Values are M (SD) unless otherwise indicated. Sex is reported as n (%). CBCL = Child Behavior Checklist; BPAQ = Buss–Perry Aggression Questionnaire; low-EXT = low externalizing symptoms; high-EXT = high externalizing symptoms. p values are from Wilcoxon rank-sum tests for continuous variables and Fisher’s exact or Pearson’s chi-square tests for categorical variables, as appropriate.				

To examine the association between aggression and functional connectivity, we administered the Buss–Perry Aggression Questionnaire (BPAQ) [23] a self-report measure assessing four dimensions of aggression: Physical Aggression (9 items), Verbal Aggression (5 items), Anger (7 items), and Hostility (8 items). Participants rated each statement on a 5-point Likert scale ranging from 1 (“very unlike me”) to 5 (“very much like me”), indicating how accurately each item reflected their typical behaviors and emotional tendencies. The validated Persian version of the BPAQ, which has demonstrated acceptable reliability and construct validity in Iranian samples [24], was used in the present study. In the present sample, Cronbach’s α was .75 for the total BPAQ score and .56, .37, .47, and .65 for Physical Aggression, Verbal Aggression, Anger, and Hostility, respectively.

The study was approved by the Ethics Committee of Shahid Beheshti University, Iran (approval No. IR.SBU.REC.1402.025). Before participation, the study procedures were explained in detail to the adolescents and their legal guardians. Written informed consent was obtained from the legal guardians, and verbal assent was obtained from the adolescents prior to participation. Participants received a small gift as a token of appreciation after the EEG session.

2.2 EEG recording and analysis

Data collection took place at the National Brain Mapping Lab. Each participant attended a pre-scheduled session accompanied by a parent. Upon arrival, we obtained informed consent and performed a routine medical check-up. Participants were seated in a semi-reclined position in a quiet, well-lit room. EEG signals were recorded from 64 scalp electrodes (AFz, Fp1, Fp2, AF3, AF4, F7, F3, Fz, F4, F8, FC5, FC1, FC2, FC6, T7, C3, Cz, C4, T8, CP5, CP1, CP2, CP6, P7, P3, Pz, P4, P8, PO3, PO4, O1, O2, A2, AF7, AF8, F5, F1, F2, F6, FT7, FC3, FCz, FC4, FT8, C5, C1, C2, C6, TP7, CP3, CPz, CP4, TP8, P5, P1, P2, P6, PO7, Poz, PO8, Oz, P9, P10, TP9) using active sintered Ag/AgCl electrodes mounted in a g.GAMMAcap and connected to an 80-channel g.Hamp biosignal amplifier (g.tec medical engineering GmbH, Austria), with 24-bit analog-to-digital conversion and a sampling rate of 512 Hz. Electrode positions followed the international 10–10 system. The right earlobe electrode (A2) served as the online reference, and Fpz served as the ground electrode. Conductive gel was applied, and electrode–skin impedances were maintained below 5 k Ω . No user-defined online filters were applied during acquisition. Participants remained seated with their eyes closed for 5 min during resting-state EEG recording.

EEG preprocessing was performed using EEGLAB (version 2023.1) implemented in MATLAB R2022a. Continuous EEG data were band-pass filtered from 1 to 40 Hz using EEGLAB's Basic FIR filter; no notch filter was applied. No offline re-referencing was performed, and the data therefore retained the original A2 reference. Channels were initially inspected visually in the frequency domain, and channels showing markedly atypical spectral profiles were subsequently examined in the time domain. Channels exhibiting persistent excessive noise or flat activity were removed; typically, two to three channels were excluded per participant. The continuous EEG was then visually inspected, and segments containing prominent movement, muscle, or other gross artifacts were manually rejected. No fixed amplitude threshold was used for artifact rejection.

Independent component analysis was subsequently performed using the runica algorithm. Artifactual independent components were identified primarily through visual inspection of their scalp topographies, time courses, and power spectra, with ICLabel used in some cases as an additional aid to classification. Components reflecting common non-neural artifacts, including ocular and muscular activity, were removed. No dedicated electrooculography channels were recorded. Following ICA-based artifact correction, previously removed channels were reconstructed using spherical interpolation. The data were then downsampled from 512 to 256 Hz and segmented into non-overlapping 2-s epochs. The resulting epochs were visually inspected again, and epochs containing residual artifacts were excluded. No baseline correction was applied. For each participant, the last 20 artifact-free epochs were retained for

connectivity analysis, providing 40 s of resting-state EEG and preferentially sampling the later portion of the recording after participants had had time to acclimate to the resting-state condition.

For EEG source-based functional connectivity analysis, we used eLORETA software (developed by Roberto Pascual-Marqui at The KEY Institute for Brain-Mind Research, University Hospital of Psychiatry, Zurich), a validated method for estimating distributed cortical electrical activity in three-dimensional space. eLORETA provides a linear, weighted minimum-norm solution to the EEG inverse problem and shows strong spatial correspondence with other neuroimaging modalities, including fMRI [25, 26]. The method assumes that functionally connected neuronal populations exhibit synchronous activity, allowing identification of cortical sources and their underlying neural dynamics [19].

A total of 18 regions of interest (ROIs) for connectivity analysis were selected based on previous eLORETA studies [27, 28]. Of these, 7 ROIs belonged to the DMN and 11 to the CCN (see Supplementary Table S1). ROIs were defined by seed coordinates, encompassing all gray matter voxels within a 12 mm radius of each seed point. For functional connectivity analysis, a nonlinear approach, lagged phase synchronization (LPS), was used in the delta (1.5–4 Hz), theta (4–8 Hz), alpha (8–13 Hz), beta1 (13–18 Hz), beta2 (18–21 Hz), beta3 (21–30 Hz), and gamma (30–40 Hz) frequency bands. This method evaluates signal similarity in the frequency domain using normalized Fourier transforms. By decomposing total connectivity into instantaneous and delayed (“lagged”) components, it effectively removes spurious instantaneous connectivity caused by volume conduction and low spatial resolution, yielding a more accurate estimate of proper functional connectivity [29]. The 20 retained artifact-free epochs from each participant were submitted to eLORETA for frequency-domain source-connectivity analysis. The Fourier transform was computed using the fast Fourier transform algorithm implemented in eLORETA with a Hann window (taper).

2.3 Declaration of generative AI and AI-assisted technologies

During the preparation of this work, the authors used OpenAI's ChatGPT to assist with English-language editing, wording and clarity, and literature-search support. After using this tool, the authors reviewed and edited the content as needed and take full responsibility for the publication's content.

2.4 Statistical analysis

Group comparisons for demographic variables were conducted using Fisher's exact test or Pearson's chi-squared test for categorical variables and the Wilcoxon rank-sum test for continuous variables. We assessed group differences in functional connectivity obtained with eLORETA in MATLAB (R2022a) using a custom implementation of Statistical Non-Parametric Mapping (SnPM), following the non-parametric permutation framework described by Nichols and Holmes [30]. Unlike conventional parametric inference, this approach estimates statistical significance empirically by repeatedly permuting group labels to generate the null distribution of the test statistic, thereby avoiding distributional assumptions and providing robust control of family-wise error rates in neuroimaging data.

We evaluated statistical significance using a max-statistic permutation framework with 5,000 random permutations. For each permutation, the maximum absolute t -value across all ROI-to-ROI connections and all frequency bands was retained to construct a single empirical null distribution. Family-wise error rate (FWER)-corrected p -values were then obtained by comparing the observed absolute t -statistics with this permutation-derived maximum distribution. We performed multiple-comparison correction separately for CCN and DMN. Statistical significance was defined as a two-tailed FWER-corrected $p < .05$. Effect sizes were quantified using Cohen's d for each ROI-to-ROI connection. The spatial distribution of significant CCN connectivity effects was visualized using BrainNet Viewer (version 1.7) [31].

As a sensitivity analysis, internalizing symptom scores were included as a nuisance covariate because they differed significantly between groups (Table 1) and could potentially influence the observed connectivity effects. Covariate-adjusted group differences were tested using the Freedman–Lane permutation procedure within a general linear model framework [32, 33]. For each ROI-to-ROI connection and frequency band, the observed group effect was estimated from a full model including an intercept, group, and internalizing symptom score. A reduced model containing only the intercept and internalizing score was then fitted, and its residuals were permuted across participants. For each permutation, the permuted residuals were added back to the fitted values from the reduced model to generate a pseudo-dataset, after which the full model was refitted, and the t -statistic corresponding to the group effect was obtained. The same participant permutation was applied simultaneously across all ROI-to-ROI connections and frequency bands within each network. For each permutation, the maximum absolute group t -statistic across all connections and frequency bands was retained to construct the empirical null distribution. FWER-corrected p -values were derived from this maximum-statistic distribution using 5,000 permutations, with correction performed separately for the CCN and DMN. Statistical significance was defined as a two-tailed FWER-corrected $p < .05$.

To assess the behavioral relevance of the identified connectivity alterations, Pearson correlation analyses were performed between connectivity values from the five connections surviving FWER correction in the primary analysis and the BPAQ total score and four subscale scores, yielding 25 correlations. Multiple comparisons were controlled using the Benjamini–Hochberg false discovery rate (FDR) procedure across all 25 tests.

Because the primary connectivity analyses used a max-statistic permutation procedure with FWER correction across multiple ROI-to-ROI connections and frequency bands, conventional power calculations based on an uncorrected two-sample test do not directly represent the sensitivity of the full inferential procedure. Simulation-based approaches provide a practical means of estimating statistical sensitivity for high-dimensional EEG analyses when the inferential procedure includes permutation-based multiple-comparison correction [34, 35]. We therefore performed a simulation-based sensitivity analysis using the same group sizes and max-statistic permutation framework as the primary analyses [30]. The analysis was conducted separately for the CCN and DMN, consistent with the network-specific FWER correction used in the primary analysis.

Participant-level connectivity values from the final sample were used to preserve the empirical multivariate dependence structure among connections and frequency bands. For each network, the observed group mean difference was first removed for each ROI-to-ROI connection and frequency-band combination by centering participants' values around their respective group means. The resulting residualized values were then standardized feature-wise to unit variance and pooled to provide an empirical null background. Entire participant-level feature vectors were bootstrap-resampled with replacement, thereby retaining the observed dependence among connectivity measures across connections and frequency bands within each simulated dataset.

Standardized between-group effects were then introduced into a single ROI-to-ROI connection $\times$ frequency-band combination in each simulated dataset. Effect locations were selected randomly from all tests within the relevant network, rather than being restricted to connections that were significant in the observed data. This strategy was used to avoid conditioning the sensitivity estimate on the locations of the observed significant effects. Simulated effects corresponded to Cohen's d values of .40, .60, .80, 1.00, 1.20, 1.40, 1.60, 1.80, and 2.00. For each simulated effect, high-EXT and Low-EXT values were shifted symmetrically in opposite directions so that the expected standardized difference matched the specified Cohen's d . The direction was specified as lower connectivity in the high-EXT group, although detection probabilities are unaffected by effect direction because the inferential procedure was two-tailed.

For each effect size, 1,000 Monte Carlo datasets were generated while retaining the original group sizes. Each simulated dataset was analyzed using the same procedure as the primary connectivity analysis. Equal-variance two-sample t statistics were computed for all unique ROI-to-ROI connections across all seven frequency bands, and 5,000 random permutations of group labels were used to construct an empirical null distribution of the maximum absolute t statistic. A single participant-label permutation was applied simultaneously to all connections and frequency bands within a given permutation, preserving the dependence structure among tests. FWER-corrected p values for the injected effect were calculated by comparing its observed absolute t statistic with the permutation distribution of the maximum absolute t statistic. An injected effect was considered detected when its FWER-corrected p value was $< .05$.

Empirical power was defined as the proportion of the 1,000 simulated datasets in which the injected effect survived max-statistic FWER correction. Binomial 95% confidence intervals around the estimated detection probabilities were calculated using Wilson intervals. Because FWER correction was performed separately for the CCN and DMN in the primary analysis, separate sensitivity curves were estimated for the two networks. The CCN analysis included 55 unique ROI-to-ROI connections across seven frequency bands (385 tests), whereas the DMN analysis included 21 unique connections across seven frequency bands (147 tests). The standardized effect corresponding to an 80% detection probability was estimated by linear interpolation between adjacent simulated effect sizes that bracketed 80% power.

3 Results

3.1 Demographic variables statistical analysis

The low-EXT and high-EXT groups did not differ significantly in age or sex distribution (all p -values $\geq .20$). In contrast, questionnaire data showed apparent group differences on the CBCL. Adolescents in the high-EXT group had significantly higher parent-reported externalizing and internalizing scores than those in the low-EXT group (Wilcoxon rank-sum tests, both $p < .01$). For self-reported aggression, no significant group differences were observed in the BPAQ total score or its subscales (all $p \geq .074$). Physical Aggression scores were numerically higher in the high-EXT group, although this difference did not reach statistical significance ($p = .074$).

3.2 EEG resting-state functional connectivity analysis

No ROI-to-ROI connection within the DMN differed significantly between groups in any of the seven frequency bands, either in the primary (unadjusted) model or after adjustment for internalizing symptoms (all $p \geq .05$, FWER-corrected). The full pattern of observed between-group t statistics across DMN connections and frequency bands is shown in Supplementary Figure S3.

In contrast to the DMN, the CCN showed significant group differences in resting-state connectivity, concentrated in the beta1 and gamma bands. Five ROI-to-ROI connections survived FWER correction applied jointly across all connections and frequency bands ($p < .05$, corrected; Table 2, Fig. 3). All significant connections reflected hypoconnectivity in the high-EXT group relative to the Low-EXT group. Within the beta1 band, hypoconnectivity was observed between the left and right anterior insula (AI), between the left dorsolateral prefrontal cortex (DLPFC) and right inferior frontal junction (IFJ), and between the left and right IFJ. Within the gamma band, hypoconnectivity was observed between the left AI and right DLPFC and between the left AI and right rostralateral prefrontal cortex (RLPFC). Participant-level distributions of LPS values for the five significant connections are shown in Fig. 1, while the corresponding standardized between-group effect sizes and 95% confidence intervals are summarized in Fig. 2. The spatial distribution of the significant connections across the CCN is illustrated in Fig. 3. The full pattern of observed between-group t statistics across all CCN connections and frequency bands is shown in Supplementary Figure S2, with FWER-significant connections indicated in the beta1 and gamma bands.

Table 2
Significant ROI-to-ROI Hypoconnectivity Within the Cognitive Control Network

ROI 1	ROI 2	Frequency band	t	FWER-corrected <i>p</i>	Cohen's <i>d</i>	95% CI for <i>d</i>
Left AI	Right AI	Beta1	5.519	.002	-1.866	[-2.66, -1.06]
Left IFJ	Right IFJ	Beta1	4.246	.035	-1.436	[-2.17, -0.68]
Left DLPFC	Right IFJ	Beta1	4.199	.038	-1.420	[-2.16, -0.67]
Left AI	Right DLPFC	Gamma	4.896	.007	-1.656	[-2.42, -0.87]
Left AI	Right RLPFC	Gamma	4.174	.041	-1.412	[-2.15, -0.66]
Note. All reported connections were lower in the high-EXT group than in the low-EXT group and survived family-wise error rate (FWER) correction across all ROI-to-ROI connections and frequency bands within the CCN. AI = anterior insula; DLPFC = dorsolateral prefrontal cortex; IFJ = inferior frontal junction; RLPFC = rostrolateral prefrontal cortex.						

3.3 Sensitivity analysis: adjustment for internalizing symptoms

Because internalizing symptoms differed significantly between groups (Table 1), we repeated the full CCN and DMN connectivity analyses while adjusting for internalizing symptom scores using the Freedman–Lane permutation procedure. The same max-statistic framework was applied across all ROI-to-ROI connections and frequency bands within each network. No significant DMN connections emerged after adjustment. Within the CCN, two connections remained significant after FWER correction: left AI–right AI connectivity in the beta1 band ($|t| = 5.28, p = .002$) and left AI–right DLPFC connectivity in the gamma band ($|t| = 4.64, p = .013$). Both effects reflected lower connectivity in the high-EXT group relative to the low-EXT group. The remaining three connections identified in the primary analysis did not survive FWER correction after adjustment for internalizing symptoms: left DLPFC–right IFJ connectivity in beta1 ($|t| = 4.08, p = .056$), left IFJ–right IFJ connectivity in beta1 ($|t| = 4.03, p = .064$), and left AI–right RLPFC connectivity in gamma ($|t| = 3.69, p = .150$) (Table 3; Fig. 3).

Table 3
Sensitivity Analysis Adjusting for Internalizing Symptoms

Functional connectivity	Frequency band	Adjusted t	FWER-corrected <i>p</i>
Left DLPFC–Right IFJ	Beta1	4.077	.056
Left IFJ–Right IFJ	Beta1	4.027	.064
Left AI–Right AI	Beta1	5.282	.002
Left AI–Right DLPFC	Gamma	4.639	.013
Left AI–Right RLPFC	Gamma	3.690	.150

Note. Internalizing symptom scores were included as a nuisance covariate using the Freedman–Lane permutation procedure. FWER correction was applied across all ROI-to-ROI connections and frequency bands within the CCN. Values in bold remained significant after adjustment ($p < .05$). AI = anterior insula; DLPFC = dorsolateral prefrontal cortex; IFJ = inferior frontal junction; RLPFC = rostralateral prefrontal cortex.

3.4 Associations between CCN connectivity and self-reported aggression

Pearson correlations were computed between connectivity values at the five connections identified in the primary analysis and the BPAQ total and subscale scores, yielding 25 correlations. After Benjamini–Hochberg FDR correction across all 25 tests, only one association remained significant: lower left DLPFC–right IFJ connectivity was associated with higher Physical Aggression scores ($r = -.648$, 95% CI $[-.81, -.40]$, $q = .001$; Table 4; Fig. 4). No other correlations survived FDR correction (all $q \geq .278$). Full correlation results, including uncorrected and FDR-adjusted *p*-values for all 25 tests, are provided in Supplementary Table S2.

Table 4
Associations Between Significant Cognitive Control Network (CCN) Connectivity Measures and BPAQ Scores

Functional connectivity	BPAQ Total	Physical Aggression	Verbal Aggression	Anger	Hostility
Left DLPFC–Right IFJ	–.27	–.65	.02	–.02	.15
Left IFJ–Right IFJ	–.15	–.36	.18	.00	.07
Left AI–Right AI	–.09	–.22	.02	–.02	.12
Left AI–Right DLPFC	–.06	–.27	.11	–.03	.21
Left AI–Right RLPFC	–.27	–.37	.00	–.09	.11

Note. Values are Pearson correlation coefficients (r). Benjamini–Hochberg false discovery rate (FDR) correction was applied across all 25 correlations. The left DLPFC–right IFJ $\times$ Physical Aggression association survived FDR correction ($q < .001$); no other association survived correction (all $q \geq .278$). AI = anterior insula; BPAQ = Buss–Perry Aggression Questionnaire; DLPFC = dorsolateral prefrontal cortex; IFJ = inferior frontal junction; RLPFC = rostralateral prefrontal cortex.

3.5 Simulation-based sensitivity analysis

Under the same max-statistic FWER procedure used for the primary connectivity analyses, approximately 80% detection probability was reached for standardized effects of Cohen's $d = 1.70$ in the CCN and $d = 1.57$ in the DMN. Thus, with the present group sizes, the corrected connectivity analyses were primarily sensitive to large between-group effects. The full simulation-based sensitivity curves and detection probabilities across the evaluated effect sizes are shown in Supplementary Figure S1.

4 Discussion

The primary objective of this study was to examine whether resting-state EEG functional connectivity within the CCN and DMN differed between adolescents with relatively high and low levels of externalizing symptoms across frequency bands. Compared with the low-EXT group, adolescents in the high-EXT group showed selective reductions in connectivity among several CCN regions. These differences were restricted to the beta1 and gamma bands, rather than reflecting a generalized disruption of connectivity across frequencies. No significant between-group differences were observed within the DMN. Importantly, the pattern was further refined after adjustment for co-occurring internalizing symptoms: although five CCN connections were significant in the primary analysis, only bilateral anterior-insula connectivity in the beta1 band and left anterior insula–right DLPFC connectivity in the gamma band remained significant after FWER correction. Together, these findings point to a frequency- and network-specific pattern of CCN dysconnectivity, while also indicating that some of the initially observed effects may not be fully independent of internalizing symptomatology.

The frequency-specific pattern observed in the present study is broadly consistent with previous EEG evidence indicating altered high-frequency functional coordination in conditions characterized by behavioral dysregulation. In children with ADHD, source-level resting-state EEG studies using eLORETA and lagged phase synchronization have reported reduced beta-band connectivity, including decreases involving frontal control regions [17]. Similar reductions in frontal beta connectivity have also been observed in children with ADHD with slower cognitive processing speed [36], while an eLORETA-LPS study in adults with opioid use disorder identified reduced frontal synchronization in the beta1 and beta2 bands [37]. Taken together, these findings suggest that disrupted beta-band coordination may represent one electrophysiological expression of altered temporal coordination within frontal control systems across phenotypically different forms of behavioral dysregulation. Evidence for gamma-band abnormalities in externalizing-related psychopathology remains comparatively limited. In children with ADHD, resting-state EEG studies have reported reduced gamma activity [38] and reduced frontal interhemispheric gamma coherence [39], providing some precedent for diminished high-frequency neural coordination in conditions characterized by impaired attentional and behavioral regulation. However, the direction of gamma-band alterations does not appear to be uniform across clinical populations. Park et al. [40], for example, reported increased intrahemispheric gamma coherence in individuals with internet gaming disorder (IGD). In contrast, individuals with alcohol use disorder (AUD) did not show the same gamma pattern and instead exhibited only a marginal tendency toward increased theta coherence relative to healthy controls. Although IGD is not conventionally classified as an externalizing disorder, it overlaps with externalizing-related characteristics such as impulsivity, ADHD symptoms, and aggression [41, 42], whereas AUD is more directly embedded within the broader externalizing spectrum. Taken together, these findings suggest that high-frequency synchronization may be altered across conditions involving behavioral dysregulation, but the direction and frequency specificity of these alterations likely depend on the phenotype and neural systems examined. Accordingly, the gamma hypoconnectivity observed in the present study should not be interpreted as an established externalizing-specific signature, but rather as preliminary evidence of altered high-frequency coordination within cognitive-control circuitry that requires replication in larger samples. The beta-band findings appear to converge more clearly with the existing literature. In contrast, the gamma hypoconnectivity observed here may reflect a more context-dependent aspect of CCN dysconnectivity that requires replication.

A closer look at the spatial organization of these effects suggests that the observed hypoconnectivity was distributed across several regions of the CCN rather than being confined to a single frontal locus. Within this pattern, the anterior insula emerged as a particularly prominent node, participating in the bilateral anterior-insula beta1 effect and in both gamma-band connections identified in the primary analysis. The involvement of the insula is consistent with previous evidence linking insula-centered network organization to externalizing tendencies. In a large community sample, Abram et al. [43] found that lower coherence within an anterior insula–ventral striatum–anterior cingulate network was associated with greater general disinhibition, suggesting that altered coordination of insula-centered networks may be relevant even outside clinically diagnosed populations. Meta-analytic evidence from adolescents with aggressive behavior further identified the left insula as one of the regions in which

structural and functional abnormalities converged across studies [44]. Taken together, these findings raise the possibility that the anterior-insula effects observed here reflect disruption not simply within a single control region, but in its coordination with other regions involved in cognitive control and behavioral regulation.

The sensitivity analysis further refined the pattern of CCN dysconnectivity. When internalizing symptoms were included as a covariate across the full connectivity analysis, only two effects remained significant after FWER correction: bilateral anterior insula connectivity in the beta1 band and left anterior insula–right DLPFC connectivity in the gamma band. The attenuation of the remaining three effects suggests that not all of the connectivity differences observed in the primary analysis were independent of co-occurring internalizing symptoms. This is plausible given the well-established overlap between internalizing and externalizing psychopathology during childhood and adolescence, with evidence that the two domains are linked by multiple symptom-level associations rather than functioning as entirely independent dimensions [45]. More broadly, large-scale work in emerging adolescence supports a hierarchical organization in which internalizing and externalizing dimensions retain distinct correlates while also sharing variance with a general psychopathology factor [46]. Accordingly, some of the CCN alterations observed in the unadjusted analysis may reflect neural variance shared across broader psychopathology rather than variance uniquely associated with externalizing symptoms. At the same time, the loss of FWER-corrected significance does not necessarily imply that the underlying associations were eliminated, particularly given the modest sample size and stringent correction framework. Notably, both effects that remained significant involved the anterior insula, raising the possibility that insula-centered CCN dysconnectivity represents a comparatively robust component of the externalizing-related pattern observed here. This interpretation nevertheless requires replication in larger samples that can more clearly separate shared from symptom-specific neural variance.

The behavioral association observed for the left DLPFC–right IFJ connection provides a further clue to the potential functional relevance of the CCN alterations. Lower beta1 connectivity between these regions was associated with higher self-reported physical aggression, and this association remained significant after correction for multiple comparisons. Notably, however, the corresponding group difference was attenuated after adjustment for internalizing symptoms and no longer survived FWER correction. The brain–behavior association should therefore be viewed as complementary evidence for the potential relevance of this circuit to physical aggression, rather than as evidence for an externalizing-specific connectivity effect. Both regions are strongly implicated in cognitive control, although they appear to contribute to somewhat different components of that process. The IFJ is consistently recruited across tasks requiring task switching, interference resolution, and updating of task representations, suggesting a role in flexibly reconfiguring behavior when current demands change [47, 48]. The DLPFC, in turn, forms part of the broader prefrontal circuitry supporting goal-directed control, and converging evidence links weaker prefrontal control to greater aggressive tendencies. Notably, experimental stimulation of the DLPFC has been shown to reduce intentions to engage in aggressive behavior, providing causal evidence that prefrontal regulatory processes can influence aggression-related responses [49]. Against this background, the present association may indicate that physical aggression

is related not simply to altered activity in a single prefrontal region, but to weaker coordination between frontal nodes that together support the flexible regulation of behavior. A similar network-level relationship has been reported in children with ADHD, in whom reduced frontoparietal connectivity was associated with greater impulsivity and oppositional-defiant symptoms as well as poorer response inhibition and attentional control [50]. Importantly, however, the present data do not establish that impaired inhibitory control mediates the relationship between connectivity and aggression; rather, they suggest that disrupted communication within frontal control circuitry may be one neural correlate of individual differences in overt aggressive behavior.

The absence of clear group differences in self-reported aggression deserves separate consideration, particularly because group classification was based on parent-reported externalizing symptoms. Parent and adolescent reports of behavioral problems are not expected to correspond perfectly, and discrepancies between informants are common in developmental psychopathology; importantly, such discrepancies may reflect differences in context, observability, and perspective rather than measurement error alone. Longitudinal work on adolescent aggression, for example, has shown that the magnitude and even direction of parent-adolescent discrepancies can change across adolescence [51]. The BPAQ introduces an additional consideration because it asks adolescents to characterize their own aggressive tendencies. Self-reports of aggression can be influenced by socially desirable responding, and this effect has been documented specifically for the Aggression Questionnaire [52, 53]. Measurement reliability may also have contributed to the pattern observed here. Several BPAQ subscales showed relatively low internal consistency in the present sample, which may have attenuated associations involving these dimensions and should be considered when interpreting the brain–behavior analyses. These factors may help explain why the substantial group difference in parent-rated externalizing symptoms was not mirrored by the BPAQ total score or most of its subscales. At the same time, the lack of a group-level BPAQ difference should not be taken to imply that the measure was behaviorally uninformative in the present sample: individual variation in Physical Aggression was strongly associated with left DLPFC–right IFJ connectivity. This contrast—weak separation at the group level but a clear brain–behavior association at the individual level—suggests that the categorical CBCL grouping and continuous self-report measure may be capturing partly different aspects of externalizing behavior.

We did not observe significant group differences in DMN connectivity across any frequency band, either before or after adjustment for internalizing symptoms. This null finding should not be interpreted as evidence that the DMN is unrelated to externalizing psychopathology. Rather, existing work suggests that DMN alterations may be more closely tied to particular dimensions of externalizing behavior than to broad externalizing severity itself. For example, in a large community sample of children, higher callous-unemotional traits were associated with reduced within-DMN connectivity even after accounting for co-occurring externalizing problems [9]. Other adolescent studies likewise suggest that the neural correlates of externalizing problems vary according to the behavioral dimension being examined. In a resting-state fMRI study, broad externalizing symptoms and conduct-specific problems were associated with distinct patterns of network connectivity, indicating that these dimensions may not share a single connectivity signature [54]. Moreover, longitudinal evidence indicates that DMN involvement in changes

in externalizing problems may differ by sex, with distinct connectivity patterns predicting symptom change in adolescent boys and girls [11]. Seen in this context, the absence of a DMN effect in the present study may reflect the relatively broad way in which externalizing symptoms were defined rather than a general preservation of DMN function. Our findings therefore point more clearly toward CCN hypoconnectivity in this sample, while leaving open the possibility that DMN alterations would emerge when more specific externalizing phenotypes, such as callous-unemotional traits or conduct-related dimensions, are considered.

Several limitations should be considered when interpreting these findings. First, the relatively small sample size limits the precision and generalizability of the observed effects. Simulation-based sensitivity analyses incorporating the same max-statistic FWER procedure used in the primary connectivity analyses indicated that approximately 80% detection probability was reached for standardized effects of Cohen's $d \approx 1.70$ in the CCN and $d \approx 1.57$ in the DMN. Thus, the present analyses had greater sensitivity to large effects than to small-to-moderate connectivity differences. Consequently, nonsignificant findings, particularly within the DMN, should not be interpreted as evidence for the absence of more modest effects. The observed effect sizes should likewise be interpreted cautiously given the limited sample and the uncertainty associated with effect-size estimation in small samples. Replication in larger samples will be necessary both to detect smaller effects and to obtain more precise estimates of the magnitude of the observed connectivity differences. Second, participants were divided into high- and low-externalizing groups using CBCL cut-off scores rather than being characterized continuously across the full externalizing spectrum. Although this grouping strategy provided a clear contrast between adolescents with relatively low and elevated symptom levels, externalizing psychopathology is increasingly conceptualized as dimensional rather than as a set of sharply bounded categories [55, 56]. Accordingly, dichotomizing symptom severity may obscure variability within groups and limits our ability to determine whether the observed connectivity alterations change continuously with externalizing severity. Finally, because the study was cross-sectional, the present results cannot establish whether altered CCN connectivity precedes the emergence of externalizing symptoms, develops alongside them, or reflects consequences of behavioral and environmental experiences associated with externalizing problems. Longitudinal studies will be necessary to distinguish among these possibilities and to determine the developmental stability of the observed network differences.

A further limitation is that formal intelligence testing was not included in the study. This is relevant because individual differences in cognitive ability can covary with large-scale functional connectivity during development. For example, resting-state connectivity measures have been associated with IQ in children and adolescents, including within frontoparietal systems relevant to the present analysis [57]. Without a standardized IQ measure, we therefore cannot determine whether differences in cognitive ability contributed to the observed group differences in CCN connectivity. Future studies should therefore include standardized measures of cognitive ability to determine whether these differences contribute to variation in CCN connectivity. Methodological constraints inherent to EEG connectivity analysis should also be considered. Although source-space estimation and lagged phase synchronization reduce the influence of volume conduction and instantaneous signal mixing, they do not

eliminate all uncertainty associated with source reconstruction, spatial resolution, and residual source leakage [18, 19]. The present connectivity effects should therefore be interpreted primarily at the level of distributed network interactions rather than as evidence of highly precise anatomical coupling between individual cortical regions. Replication using complementary modalities with higher spatial resolution, particularly fMRI or multimodal EEG–fMRI designs, would help establish the spatial robustness of these network-level findings. The relatively narrow age range of the sample should also be considered when interpreting the developmental significance of these findings. Functional connectivity continues to undergo substantial reorganization from childhood through adolescence, with age-related changes in both within- and between-network dynamics [1]. Accordingly, the connectivity pattern observed in adolescents aged 13–15 years cannot be assumed to generalize to younger children, later adolescence, or adulthood. At the same time, restricting the sample to a relatively narrow developmental window reduces some of the heterogeneity that would arise from combining participants at markedly different stages of neurodevelopment. Future longitudinal studies spanning a broader age range will be important for determining whether the beta- and gamma-band CCN alterations identified here represent a transient feature of early-to-mid adolescence or a more stable correlate of externalizing symptoms across development.

Conclusion

Taken together, the present findings suggest that elevated externalizing symptoms in adolescence are associated with selective, frequency-specific alterations in resting-state connectivity within the cognitive control network. The pattern was concentrated in beta1- and gamma-band connections among CCN regions, with two anterior-insula-related connections remaining significant after adjustment for co-occurring internalizing symptoms. At the same time, the absence of significant DMN differences and the attenuation of several CCN effects after covariate adjustment underscore that these neural alterations are not uniformly expressed across networks or independent of broader psychopathology. By combining source-level EEG connectivity with a community-based and symptom-defined approach, this study adds to evidence that externalizing symptoms may be accompanied by altered coordination among frontal control systems in adolescents drawn from the community rather than from a clinically diagnosed sample. Replication in larger longitudinal samples will be necessary to determine the stability, specificity, and developmental significance of these effects, and to establish whether they can ultimately contribute to a more mechanistic understanding of behavioral dysregulation in adolescence.

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competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Availability of data and materials

The data supporting the findings of this study are available from the corresponding author upon reasonable request, subject to ethical and privacy restrictions. The custom MATLAB code used for the statistical analyses is available at <https://doi.org/10.5281/zenodo.22147141>.

Authors' contributions

Conceptualization, S.S., M.R.B.; methodology, S.S., M.R.B., P.A.; software, S.S.; formal analysis, S.S.; investigation, S.S.; data curation, S.S.; writing—original draft preparation, S.S.; writing—review and editing, S.S., M.R.B., P.A.; visualization, S.S.; supervision, M.R.B., P.A.; project administration, M.R.B. All authors have read and agreed to the published version of the manuscript.

Ethics approval and consent to participate

The study protocol was approved by the Research Ethics Committee of Shahid Beheshti University, Iran (Approval Number: IR.SBU.REC.1402.025). The study was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from the adolescents' legal guardians, and assent was obtained from all adolescent participants prior to inclusion.

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Figures

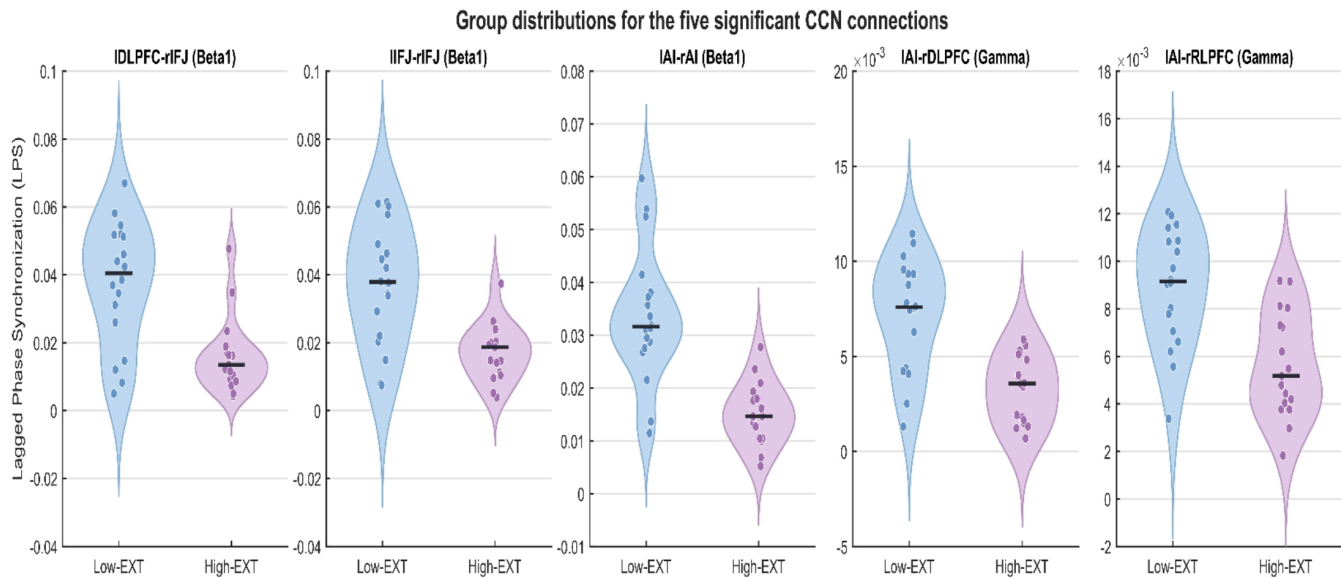


Figure 1

Distribution of lagged phase synchronization (LPS) values for the five cognitive control network (CCN) connections surviving the primary max-statistic FWER correction. Violin plots show the participant-level distributions of resting-state LPS separately for the low-EXT and high-EXT groups. Individual points represent participants, violin widths represent kernel density estimates of the observed distributions, and horizontal black lines indicate group medians. The five displayed connections comprise three beta1 effects (left DLPFC–right IFJ, bilateral IFJ, and bilateral anterior insula) and two gamma effects (left anterior insula–right DLPFC and left anterior insula–right RLPFC). All five connections showed lower connectivity in the high-EXT group in the primary analysis.

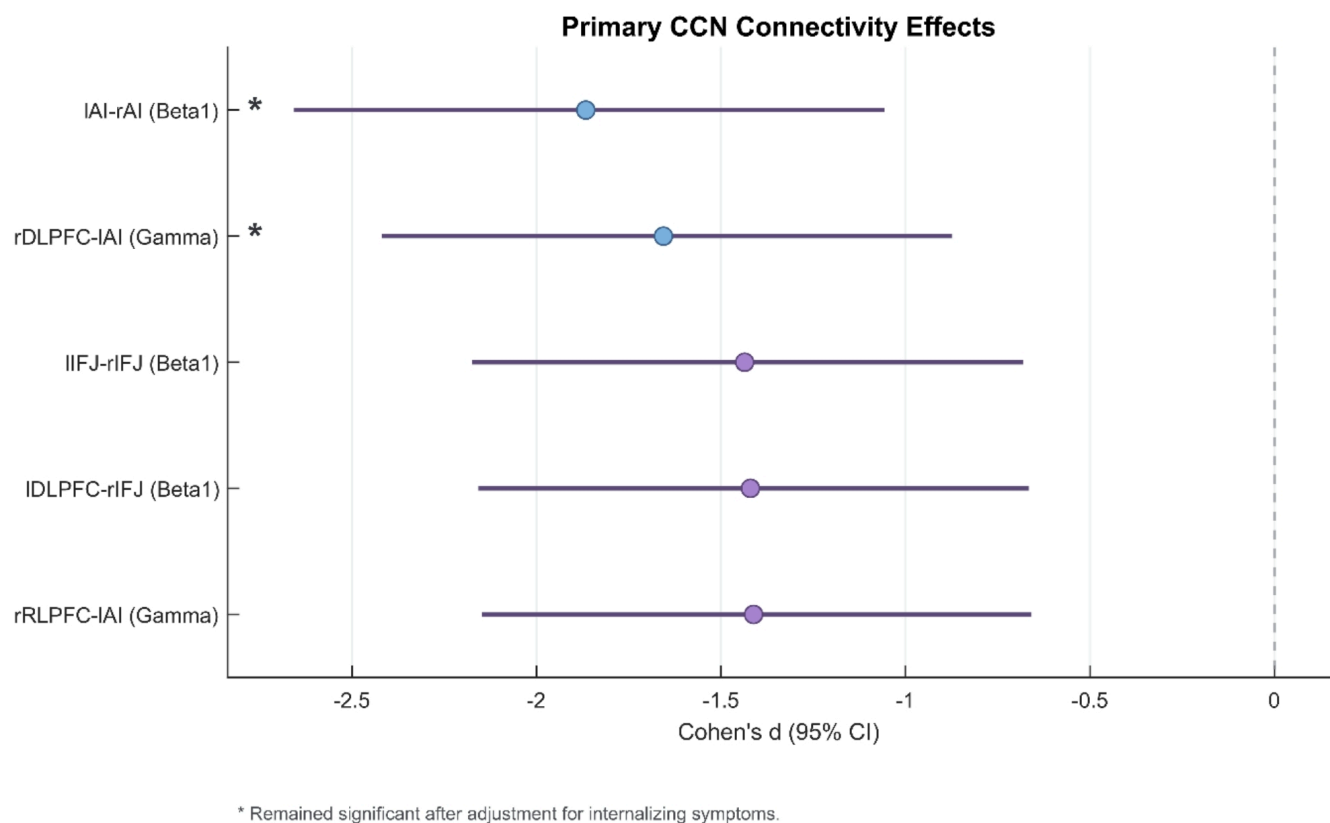


Figure 2

Standardized effect sizes for the five cognitive control network (CCN) connections surviving the primary max-statistic FWER correction. Abbreviations: r, right; l, left; AI, anterior insula; dACC, dorsal anterior cingulate cortex; DLPFC, dorsolateral prefrontal cortex; RLPFC, rostrolateral prefrontal cortex; IFJ, inferior frontal junction; PPC, posterior parietal cortex. Points represent Cohen's *d* for the high-EXT versus low-EXT group difference, and horizontal lines represent 95% confidence intervals. Negative values indicate lower LPS in the high-EXT group. All five primary effects were large and involved beta1 or gamma connectivity. Asterisks identify the two effects that remained significant after adjustment for internalizing symptoms using the Freedman–Lane permutation analysis: bilateral anterior insula connectivity in the beta1 band and left AI–right DLPFC connectivity in the gamma band. The adjusted analyses were used to evaluate robustness to internalizing symptoms; the plotted Cohen's *d* values and confidence intervals correspond to the primary unadjusted group comparisons.

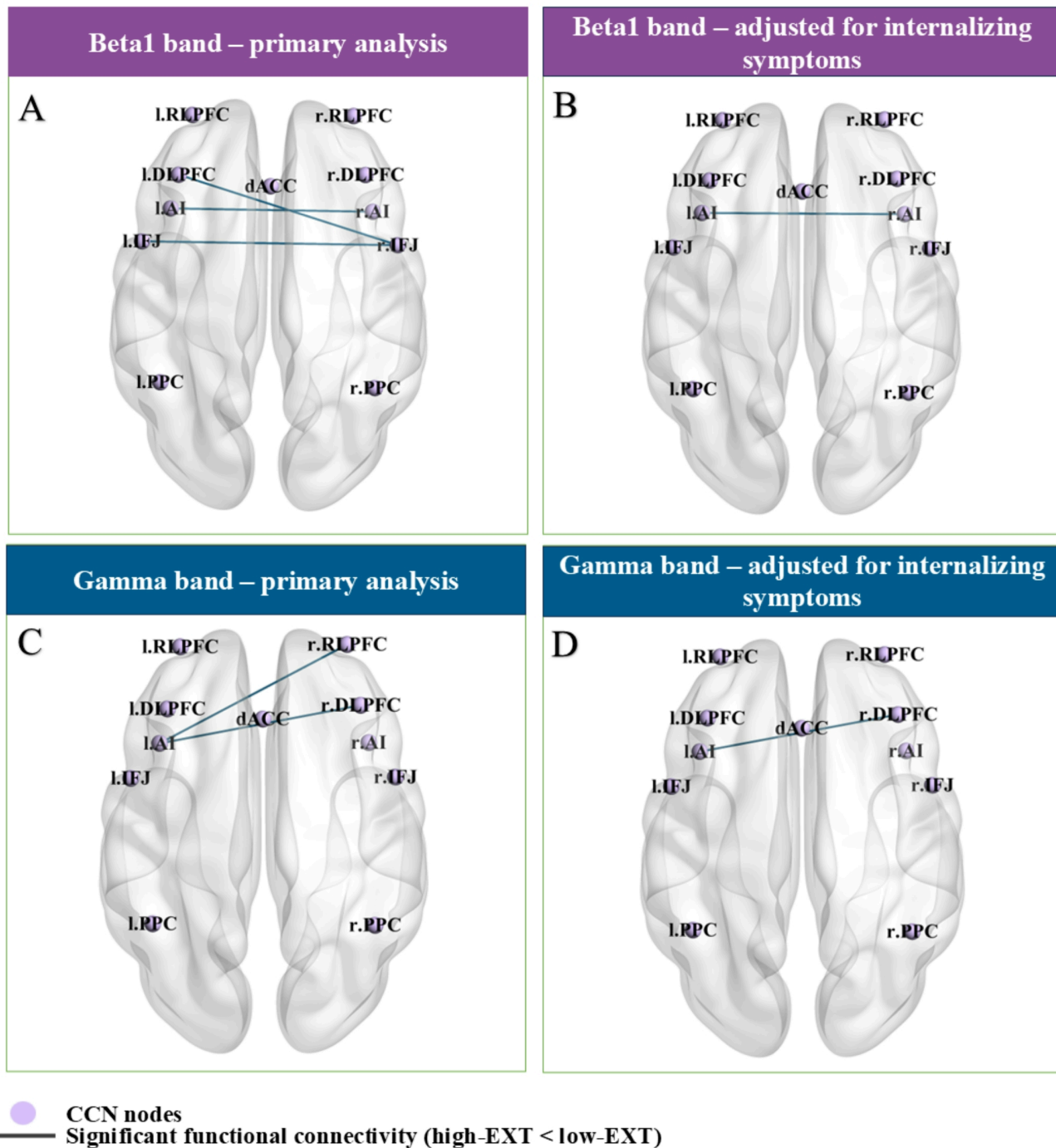


Figure 3

Significant cognitive control network (CCN) connectivity differences between the high-EXT and low-EXT groups before and after adjustment for internalizing symptoms. (A) Beta1-band connections significant in the primary analysis. (B) Beta1-band connections remaining significant after adjustment for internalizing symptoms. (C) Gamma-band connections significant in the primary analysis. (D) Gamma-band connections remaining significant after adjustment. All displayed effects reflect lower

connectivity in the high-EXT group relative to the low-EXT group. All results are FWER-corrected at $p < .05$. Abbreviations: r, right; l, left; AI, anterior insula; dACC, dorsal anterior cingulate cortex; DLPFC, dorsolateral prefrontal cortex; RLPFC, rostrolateral prefrontal cortex; IFJ, inferior frontal junction; PPC, posterior parietal cortex.

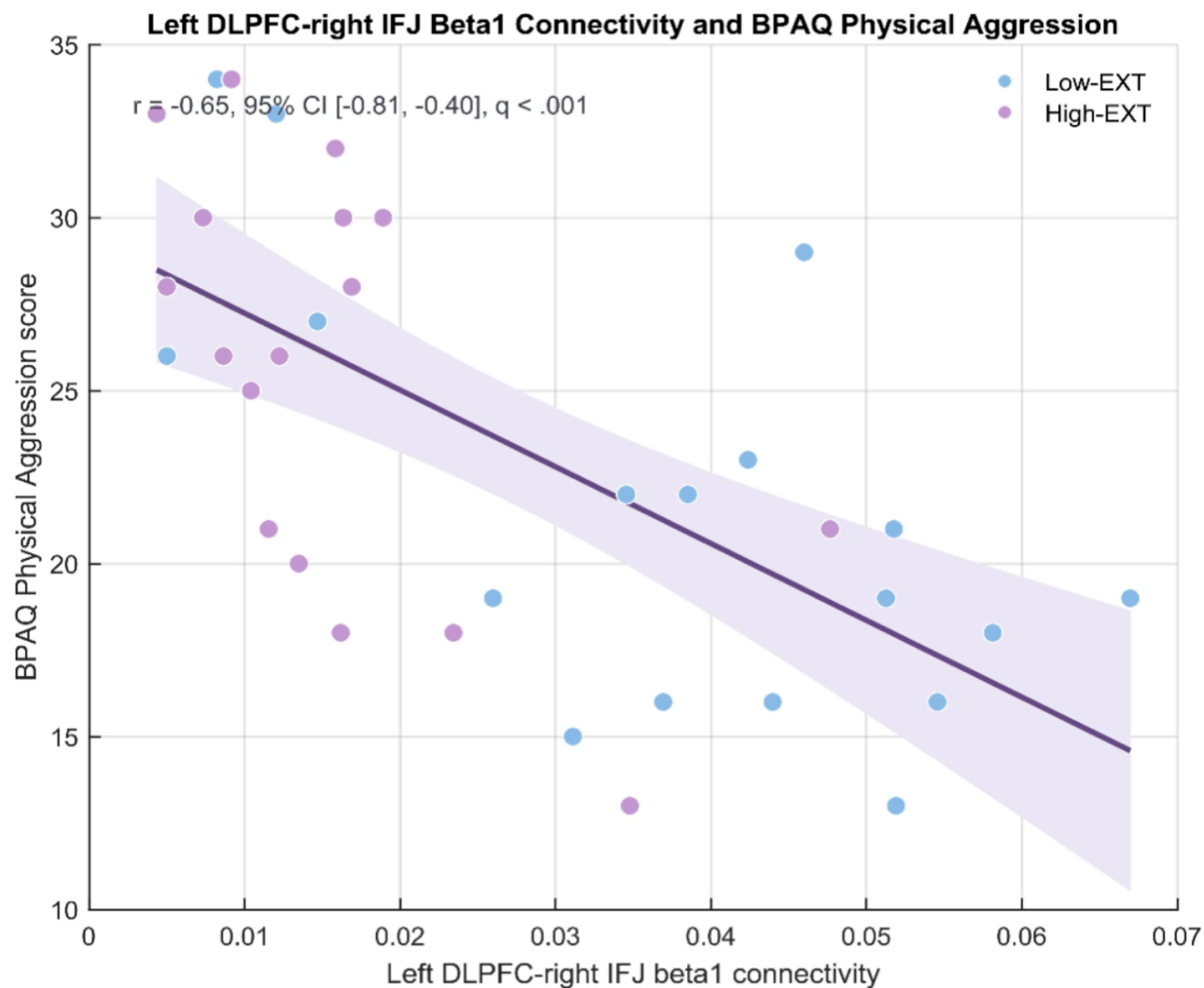


Figure 4

Association between left dorsolateral prefrontal cortex (DLPFC)–right inferior frontal junction (IFJ) beta1 connectivity and self-reported physical aggression. Lower resting-state LPS between the left DLPFC and right IFJ was associated with higher Physical Aggression scores on the Buss–Perry Aggression Questionnaire (BPAQ), $r = -.65$, 95% CI [-0.81, -0.40], FDR-adjusted $q < .001$. Individual observations are shown separately for the low-EXT and high-EXT groups. The solid line represents the ordinary least-squares regression fitted across the full sample, and the shaded region represents its 95%

confidence interval. The association was tested as part of the prespecified set of 25 connectivity–BPAQ correlations, with Benjamini–Hochberg false discovery rate correction applied across all 25 tests.

Supplementary Files

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