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Research Paper

Neurofunctional Correlates of Hostility in Adolescents With Externalizing Disorders: Implications for Clinical Assessment and Intervention

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ABSTRACT

Introduction: Previous neuroimaging studies have rarely investigated hostility as a distinct cognitive-emotional dimension of aggression. Most research has focused on overall aggressive behavior without differentiating hostility from other behavioral components. Specifically, the neural correlates associated with hostility in adolescents diagnosed with externalizing disorders have not been thoroughly investigated. To fill this void, the current research focused on hostility— a fundamental psychological element of violence—and its neurofunctional foundations in adolescents.

Methods: This study examined resting-state functional connectivity (rsFC) differences in adolescents with high and low hostility, focusing on brain networks related to emotion regulation, salience, and executive control using the Buss–Perry aggression questionnaire (BPAQ) scale. We utilized seed-to-Voxel and region of interest (ROI)-to-ROI functional magnetic resonance imaging (fMRI) models to examine rsFC in two groups of adolescents: 14 with externalizing disorders and 13 typically developing controls.

Results: Seed-to-Voxel analysis showed greater rsFC in low-hostility adolescents within two clusters: Left dorsolateral prefrontal cortex (dlPFC) (BA 9/46) and ventromedial prefrontal cortex (vmPFC) (BA 10/11) compared to high-hostility peers. Both target regions represent top-down emotional processing and social-affective processing, respectively, providing evidence that the lower hostility group is more efficient at regulating aggressive impulses. ROI-to-ROI analysis revealed significantly reduced connectivity in high-hostility adolescents, particularly between the dlPFC and the amygdala, and between the frontal midline and the amygdala, indicating impaired emotion regulation. Lowered links were also found between dorsal attention and salience networks, visual-limbic regions, and between cerebellar and medial prefrontal areas.

Conclusion: These differences reinforce disrupted functioning of conceptually relevant executive and attentional networks, as well as affective and socioemotional networks in adolescents with increased hostility. We perceive these findings collectively as a neurobiological contrast between low and high hostility, and note that decreased connectivity of both the prefrontal and salience networks may represent targets for neurotherapeutic interventions to reduce aggression in children and adolescents with externalizing problems.

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Highlights

- The study aimed to examine the neural basis of hostility in adolescents with externalizing disorders using rs fMRI and network level connectivity analysis.

- High hostility adolescents showed reduced functional connectivity in salience, attentional, and prefrontal–limbic networks, reflecting poor emotional salience detection and control.

- Weak coupling between dlPFC/vmPFC and amygdala indicated impaired top down emotion regulation and heightened reactive aggression.

- Findings revealed altered cerebello prefrontal communication, highlighting the cerebellum's extended role in affective processing beyond motor control.

- The distinct rsFC patterns may serve as biomarkers for early detection and intervention targeting emotional dysregulation in hostile or aggressive youth.

Plain Language Summary

This study explored the neural mechanisms underlying hostility in adolescents with externalizing disorders using resting state functional magnetic resonance imaging (rs fMRI). Whereas most previous research has treated hostility as part of general aggression, this work examined it as a distinct cognitive emotional construct characterized by mistrust, cynicism, and antagonism toward others. The goal was to determine how patterns of functional connectivity (rsFC) differ between adolescents exhibiting high and low levels of hostility. Analyses revealed that adolescents with high hostility scores showed reduced connectivity across several key brain networks that coordinate emotional and attentional control. In particular, functional links within the salience network, dorsal attention network, visual limbic system, and cerebello prefrontal circuits were significantly weaker. Such disruptions suggest inefficient processing of salient environmental cues and difficulty mobilizing regulatory mechanisms during emotionally charged or ambiguous situations. In contrast, adolescents characterized by low hostility displayed stronger intra and inter network connectivity within prefrontal control regions, notably the dorsolateral prefrontal cortex (dlPFC) and ventromedial prefrontal cortex (vmPFC). Enhanced connectivity in these areas indicates more efficient top down control over emotional and behavioral responses. Importantly, weakened coupling between the prefrontal cortex and amygdala was observed in high hostility individuals, implying ineffective regulation of subcortical emotional reactivity. Additional findings included disrupted communication in the salience network (encompassing the insula, anterior cingulate cortex, and supramarginal gyrus) and altered cerebello prefrontal links, underscoring the cerebellum's underrecognized role in emotional regulation. Overall, the findings support a network based model of hostility, in line with theories like the I^3 model of aggression, highlighting that hostility stems from dysfunctional integration among attention, salience, and inhibitory control systems. The study concludes that altered brain network organization may serve as a neural risk marker for developing aggressive or antisocial behaviors in adolescence.

Introduction

Aggression and hostility are prominent behavioral manifestations frequently observed in externalizing disorders, such as conduct disorder and oppositional defiant disorder. These conditions, which often emerge during childhood or adolescence, are characterized by persistent patterns of rule-breaking, defiance, and in many cases, overt aggression (American Psychiatric Association, 2022). Such behaviors significantly increase the risk of adverse outcomes, including academic difficulties, delinquency, and progression to adult antisocial behavior or personality pathology.

Recent studies have identified the frontal lobe, particularly its prefrontal cortex (PFC), as an essential center for managing aggressive behavior. The PFC, with its 4 subregions, the dorsolateral (dlPFC), ventromedial (vmPFC), ventrolateral (vlPFC), and orbitofrontal cortex (OFC), leads emotional and impulsive control from the top down to control aggression (Kolb & Whishaw,

2009). The reduction of gray matter volume, together with a decrease in cortical thickness in the vmPFC, leads to increased aggression levels based on structural imaging studies, and dlPFC lesions cause elevated physical aggression in trauma-exposed populations, including war veterans (Fritz et al., 2023; Singh & Gobrogge, 2024). The research using functional magnetic resonance imaging (fMRI) demonstrates that patients with impulsive aggression show abnormal communication between the medial PFC (mPFC) and subcortical regions such as the amygdala, which is typical for individuals with intermittent explosive disorder (IED) (Singh & Gobrogge, 2024). Neuromodulation research verifies the PFC's control function by showing that right dlPFC activation decreases proactive aggression, while left dlPFC suppression intensifies reactive and proactive aggression. Bilateral stimulation of dlPFC regions decreases both aggressive intentions and justifications for violent behavior (Fritz et al., 2023; Singh & Gobrogge, 2024). Animal model studies of development demonstrate that early social stress, through social isolation, impairs PFC development, leading to increased aggression and altered neuronal patterns within PFC circuits (Singh & Gobrogge, 2024). During adolescence, serotonin signalling in the mPFC plays an essential role in controlling aggression, as impaired serotonin transporter function in this region leads to pathological aggression (Szebik et al., 2025). The hypothalamus and temporal cortex are important components of the aggression network, as research shows that lateral hypothalamic connectivity shifts and temporal lobe dysfunction occur in people who lose control of their anger (Yao et al., 2024). The PFC functions as a fundamental control center of aggression neural networks, since structural, connectivity, and neurochemical defects lead to harmful behavioral outcomes. Targeted interventions, including neurostimulation and pharmacological modulation, now offer opportunities to restore regulatory control over aggression.

Resting-state functional connectivity (rsFC) has emerged as a powerful tool in neuroimaging for examining the brain's intrinsic functional architecture. Unlike task-based paradigms, rsFC measures low-frequency fluctuations in neural activity at rest, allowing the identification of stable patterns of interregional communication (Fair et al., 2007). This method has proven valuable in detecting aberrant connectivity patterns in psychiatric populations, where dysfunction in intrinsic brain networks may serve as potential biomarkers of psychopathology. Research on rsFC has identified the brain network underlying aggression. Emerging evidence identifies both common and relatively aggressive subtype-specific effects on global-scale networks. For example, targeting emotional dysregulation and maladaptive aggression, rsFC effects from the amygdala-vmPFC that are particularly relevant to youth also demonstrate both global and aggression-subtype rsFC patterns, especially in disruptive youth, who show increased rsFC to the amygdala and decreased rsFC to the vmPFC (Sukhodolsky et al., 2022). Research also identified reactive aggression (linked to aggressive/environmental triggers) and proactive aggression (more intent-based on the youth's aggression subtype; e.g. callous-unemotional youth), with differences in rsFC across subtypes, including the central gyrus, precuneus, and volatile patterns in the temporal lobe (Werhahn et al., 2023). Changes in the default mode network (DMN), regulated by reductions in rsFC between the dorsolateral PFC to the inferior parietal lobule (IP) and posterior cingulate cortex (PCC), MPFC/anterior cingulate cortex (ACC) that had associated empirical higher proactive aggression overall (e.g. proactive aggression was noted when there was poorer moral reasoning, which was associated with similar rsFC reductions) (Zhu et al., 2019). New findings provided empirical support for the significance of rsFC changes, including in the lateral hypothalamus (LH), which negatively correlated with aggression, as well as differences in male vs female youth in LH–thalamus rsFC connectivity (Yao et al., 2025). Using the cerebellum, which has limited prior aggression rsFC research, our preliminary findings suggest that this region may modulate aspects of aggression, with the medial region associated with impulsivity and the posterolateral region associated with premeditated aggression and possible lateralization effects (Kruithof, 2025).

Additionally, research in violent offenders and intimate partner violence (IPV) perpetrators, including studies by researchers studying psychopathy and IPV, demonstrates hyperconnectivity between the amygdala and discriminative-perceptual or speech/receptive language networks involving the inferior frontal/superior temporal regions, but also shown increased rsFC network involvement between the amygdala, the ACC, and right cerebellum, possibly indicating their salience and emotion regulation circuits (Romero-Martínez et al., 2024). Lastly, the supramarginal gyrus that disrupts rsFC and DMN that disrupts rsFC, may suggest neurodevelopmental immaturity and heightened vulnerability to violent behavior for the juvenile offenders (Wei & Xia, 2023). Collectively, these findings provide converging evidence that we can differentiate subtypes of aggression using rsFC signatures, likewise acknowledge comorbidities, such as attention-deficit hyperactivity disorder (ADHD), and as biomarkers for risk profiling and intervention targeting in clinical practice.

Hostility is a critical construct in aggression and a cognitive-emotional orientation associated with suspiciousness, resentment, and a tendency to attribute hostile intent to others. Hostility is distilled into cognitive (e.g. hostile attribution), emotional (e.g. chronic anger), and behavioral (e.g. verbal or relational aggression) responses. Regarding the motive, the cognitive dimension includes hostile attributional bias (i.e. the tendency of people to view others' ambiguous actions as intentional harms or insults) and the emotional dimension reflects a predisposition toward affective states of anger, irritability, or resentment. The behavioral dimension engages expressions of aggression which are primarily verbal or relational and not physical (i.e. sarcasm, gossip, social exclusion). The cognitive, emotional, and behavioral components contribute to a pattern of hostile responding that has been extensively studied for aggression broadly, especially in adolescents and social interactions (Dodge & Coie, 1987). Researchers have identified 5 subcomponents of hostility: angry affect, hostile intent, verbal aggression, physical aggression, and relational aggression. Neurocognitive models have proposed that hostile cognitions and behaviors developed and caused dysfunction due to increased limbic activity poorly regulated by prefrontal control (Smeijers et al., 2018). Neuroimaging research on clinical populations, such as borderline personality disorder, antisocial personality disorder, and schizophrenia, has shown altered activity and connectivity in limbic structures (e.g. amygdala, mPFC, ACC, insula) that are related to measures of hostility and aggression when the studies are evaluated conclusively. Neuroimaging studies have shown a significant association of aggression and hostility and limbic networks across several studies (Coccaro et al., 2007). Recent work published in the Journal of Affective Disorders indicates that increased hostile attribution bias is associated with displaced aggression and underpinned by hyperactivity in limbic regions of the brain. This issue suggests that unfortunate, angry, and aggressive social cognition may have a biological basis (Zhu et al., 2022).

Despite a broad implementation of more explicit neuroscience investigations of aggression, the specific neurofunctional configuration of hostility—an independent and central construct on the aggression continuum—is underexamined. Given the strong association of hostility with psychosocial impairment and its predictive nature for antisocial behaviors, identifying the intrinsic functional connectivity profiles related to hostility is imperative. The current study will seek to contribute to knowledge about hostility by isolating rsFC alterations associated with hostility in adolescents, with the ultimate aim of informing and developing specific neurobiological markers to support early-age assessments and interventions for youth.

Materials and Methods

Study participants

The present study investigated adolescents residing in an economically disadvantaged and intervention-needing neighbourhood and setting in Tehran City, Iran. Twenty-seven adolescents were selected for the study, including 14 with externalizing disorders (ED) and 13 typically developing (TD) controls. This sample size was both feasible and determined in accordance with guidelines and norms from the neuroimaging fMRI literature. The sample size would provide adequate statistical power for rsFC analyses while accounting for scanning time and potential data quality and processing issues. The overall balance also enabled more appropriate group comparisons and control of potential confounding variables at the adolescent level in the sample and evaluation designs.

Several covariates were included in the group comparisons to account for potential confounding and to strengthen the ability to determine whether statistically significant differences in brain connectivity were specifically due to group differences in hostility. These covariates were as follows: parental education level as a measure of cultural and educational experience, parental psychopathology as representing genetic and or environmental risk factors converging on externalizing disorders, family socioeconomic status (SES) as a measure of environmental stress and deprivation, the presence or absence of domestic violence as a direct risk factor associated with hostility and aggression in the home, and gender, age, and intelligence quotient (IQ) as additional controls for biological and cognitive variability across participants.

Behavioral analysis and assessment

Buss–Perry aggression questionnaire (BPAQ)

To assess aggressive behavior, the BPAQ was administered. It was created in 1992, and since then, it has been embraced for its capacity to assess various facets of aggression. The BPAQ is a 29-item self-report scale that measures 4 main dimensions: physical aggression (9 items), verbal aggression (5 items), anger (7 items), and hostility (8 items). This multidimensional approach permits an in-depth analysis of both overt aggressive acts and the affective states that may underpin them. BPAQ

has been shown to possess robust psychometric properties and to have been effectively cross-translated across a wide range of culturally and linguistically diverse populations.

fMRI image acquisition

Neuroimaging data were acquired on a 3 Tesla (T) Siemens MAGNETOM Prisma scanner. All scanning sessions began with a localization sequence, followed by a high-resolution structural image acquired with a T1-weighted MPRAGE protocol. The scanning parameters for the anatomical procedure were as follows: repetition time (TR) of 1800 ms, echo time (TE) of 3.5 ms, inversion time (TI) of 1100 ms, flip angle of 7°, and isotropic Voxel resolution of 1 cm3. Functional imaging was performed with a T2*-weighted echo-planar imaging sequence, synchronized with the anatomical slices. The functional series was specified with a TR of 3000 ms, a TE of 30 ms, a flip angle of 90°, a field of view (FOV) of 192 mm, and an isotropic Voxel size of 3 mm. A resting-state scan comprising 120 volumes, lasting 6 min and 11 s, was administered to all participants. During this scan, the subjects were requested to remain motionless with their eyes open and to gaze at a centrally located crosspoint.

Image preprocessing of functional data was conducted using the default pipeline provided by the CONN toolbox, a MATLAB-based software package used for functional connectivity analysis. The pipeline involved a series of sequential operations: motion correction in the form of realignment and unwarping, slice timing correction to account for differences in acquisition times between slices, and detection of artifacts using the artifact detection tools (ART) to detect outlier volumes based on motion and signal intensity deviations. Spatial normalization was applied to align functional and anatomical images with the Montreal Neurological Institute (MNI) standard template. Structural segmentation was also conducted to segment tissue types into gray matter, white matter, and cerebrospinal fluid. Spatial smoothing was also accomplished by convolving a Gaussian kernel with a full width at half maximum (FWHM) of 6 mm to improve signal detectability. The final preprocessing step included denoising, consisting of regression of confounding variables and temporal band-pass filtering, to reduce physiological noise and improve the accuracy of subsequent connectivity measures. This rigorous preprocessing pipeline ensured high-quality data and consistent outputs, enabling reliable group-level comparisons.

Region of interest (ROI)

In our study, for the functional connectivity analysis, 32 predefined ROIs were selected from major large-scale brain networks based on the CONN network atlas, a widely used parcellation framework in resting-state fMRI studies. According to the CONN atlas, the brain's 7 canonical intrinsic networks include the DMN, sensorimotor network, visual network, salience network, dorsal attention network, auditory network, and central executive network (also referred to as the frontoparietal control network). The prefrontal control networks, the executive, affective (i.e. limbic), and attentional control networks were highlighted because of their fundamental role in top-down regulation of cognition, emotion, and attention. For this study, these networks were not intended to be defined or separated exclusively as intrinsic networks in the atlases with which they are compared. Still, they do embody identifiable components in important areas of top-down regulation, including the mPFC, which is associated with affective and self-referential processing, and the lateral PFC, a critical hub for executive control. The cerebellar networks are typically comprised of 2 functional subdivisions: a motor cerebellar network involved in the coordination of movement, and a cognitive cerebellar network, which includes the posterior cerebellum and supports higher-order cognitive functions and behaviour. Language regions (e.g. the inferior frontal gyrus and posterior superior temporal gyrus [pSTG]) were included in our work due to their roles in language processing and higher-order cognition. Language regions are not typically represented as separate intrinsic networks in most parcellations.

Resting-state fMRI data were preprocessed and analyzed using the CONN functional connectivity toolbox (version 22a) implemented in MATLAB. After standard preprocessing steps (realignment, slice-timing correction, normalization to MNI space, and spatial smoothing with a 6 mm FWHM Gaussian kernel), denoising was performed using the CompCor method to remove physiological and motion-related confounds. The resulting residual BOLD time series were band-pass filtered (0.008–0.09 Hz) before functional connectivity analysis.

Seed-to-Voxel analysis

To identify Voxel-wise differences in connectivity between groups, a seed-to-Voxel analysis was conducted. Three principal seeds were defined based on their established roles in emotional and executive control: the DMN, the amygdala, and the frontal control network (including the dlPFC and vmPFC regions). Each seed

region's average BOLD time course was correlated with all other Voxels in the brain to generate individual subject-level connectivity maps. Group-level contrasts were then computed (low hostility > high hostility) using two-sample t-tests with cluster-level false discovery rate (FDR) correction (P<0.05).

ROI-to-ROI analysis

Complementary ROI-to-ROI analyses were performed to assess functional integration between large-scale brain networks. ROIs were defined according to the CONN standard parcellation atlas, encompassing the following networks and MNI coordinates:

DMN: mPFC: (1, 55, −3), lateral parietal (left): (−39, −77, 33), lateral parietal (right): (47, −67, 29), PCC: (1, −61, 38),

Sensorimotor network: lateral (left): (−55, −12, 29), lateral (right): (56, −10, 29), superior: (0, −31, 67),

Visual network: medial: (2, −79, 12), occipital: (0, −93, −4), lateral (left): (−37, −79, 10), lateral (right): (38, −72, 13),

Salience network: ACC: (0, 22, 35), anterior insula (Left): (−44, 13, 1), anterior insula (right): (47, 14, 0), rostral PFC (right): (32, 46, 27), supramarginal gyrus (left): (−60, −39, 31), supramarginal gyrus (right): (62, −35, 32),

Dorsal attention network: frontal eye field (FEF) (left): (−27, −9, 64), FEF (right): (30, −6, 64), intraparietal sulcus (IPS) (left): (−39, −43, 52), IPS (right): (39, −42, 54),

Frontoparietal network: lateral PFC (left): (−43, 33, 28), posterior parietal cortex (left): (−46, −58, 49), lateral PFC (right): (41, 38, 30), posterior parietal cortex (right): (52, −52, 45),

Language network: inferior frontal gyrus (left): (−51, 26, 2), inferior frontal gyrus (right): (54, 28, 1), pSTG (left): (−57, −47, 15), pSTG (right): (59, −42, 13), and

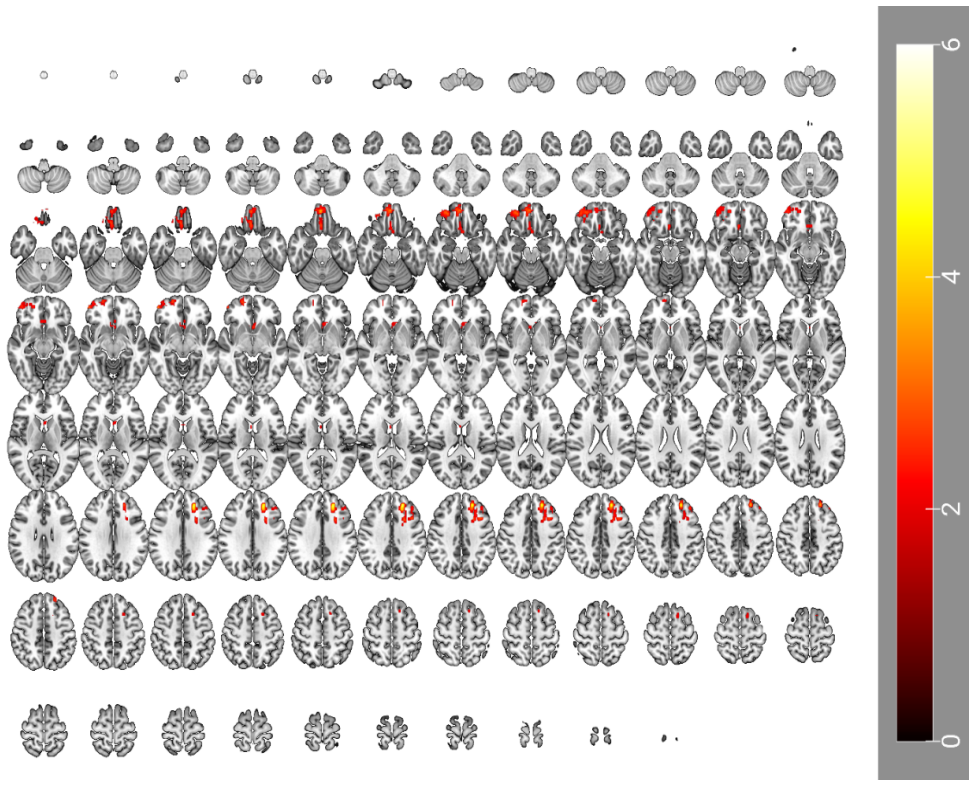
Cerebellar network: anterior: (0, −63, −30), posterior: (0, −79, −32).

For each participant, Fisher Z-transformed correlation matrices were computed across all ROIs, and between-group contrasts (low > high hostility) were assessed using two-sample t tests, with FDR correction for multiple comparisons. This approach enabled a detailed examination of network-level disruptions in adolescents with higher hostility—particularly decreased connectivity between salience, dorsal attention, visual-limbic, and cerebello-prefrontal circuits.

Results

In our study, low-hostility adolescents vs high-hostility adolescents (i.e. low > high), 2 significant clusters of increased rsFC were identified (Figure 1). The first cluster included 776 Voxels ($P=5.11\times10^{-6}$). This location was in the left dorsolateral PFC (dlPFC), given previous literature which we would characterize as being in peaks significant compared to coordinates: −20, 36, 36 (peak MNI coordinates: approximately Brodmann area: 9/46; also 2 additional negative peaks were located at (−24, 12, 36) and (−20, 38, 46) when we assessed more peak validity across the dlPFC). This region has a strong link with emotional and executive function. The increase in rsFC in this region for low-hostility individuals may suggest more effective top-down control over aggressive impulses. The second cluster included 1244 Voxels ($P=9.65\times10^{-5}$). Peaks were in the ventromedial PFC (vmPFC) (peak MNI coordinates: 4, 54, − 024; BA 10/11; significant negative peaks at (48, 50, -10) (28, 48, −20)). The vmPFC is a significant brain region supporting social behavior, emotion valuation, and emotion regulation. The increase in rsFC in vmPFC suggests that lower hostility in adolescents is associated with greater engagement of their affective control circuits. The implications of these findings reinforce the importance of the prefrontal control network's role in mitigating aggressive tendencies, supported by neurobiological differences in adolescents with varying levels of hostility.

A circular ROI-to-ROI connectivity analysis detected several significant decreases in rsFC in high-hostility adolescents compared with low-hostility adolescents. Interestingly, the left amygdala showed reduced connectivity with dlPFC and frontal midline regions, suggesting impaired top-down affective regulation in aggressive subjects. Additional disruptions were observed between dorsal attention networks (FEF and IPS) and sensorimotor and salience-related nodes (ACC and anterior insula) (Figure 2). These findings may indicate impaired attentional control and the detection of stimulus salience. Furthermore, reduced connectivity between visual lateral areas and medial prefrontal regions demonstrates dysfunction in socio-perceptual integration. Disrupted cerebello-prefrontal connectivity also manifested, highlighting the underappreciated role of the cerebellum in emotional and executive modulation. Collectively, these findings indicate a widespread dysfunction within the executive, affective, and attentional control networks in adolescents with higher hostility that may be involved in the maladaptive control of aggressive behavior.



Cluster	Voxel	P
+04 +54 -24	1244	0.003673
-20 +36 +36	776	0.07852

Figure 1. Functional connectivity differences in low vs high hostility adolescents, seed to Voxel analysis NEUROSCIENCE

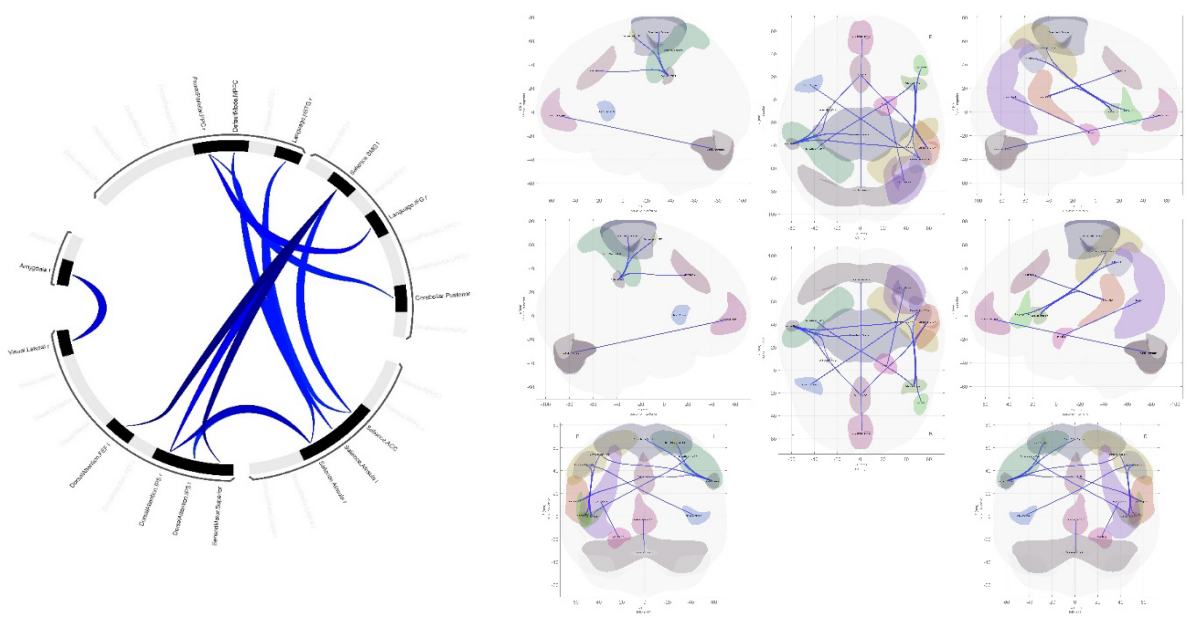


Figure 2. ROI-to-ROI functional connectivity results: low hostility > high hostility NEUROSCIENCE

Table 1. ROI-to-ROI connectivity analysis

Brain Region Connections	Statistics	P
Dorsal attention network – left FEF (−27, −9, 64) ↔ salience network – left supramarginal gyrus (−60, −39, 31)	$T_{(12)}$=-3.24	0.0071
Sensorimotor network – superior region (0, −31, 67) ↔ salience network – left supramarginal gyrus (−60, −39, 31)	$T_{(12)}$=-3.08	0.0095
Dorsal attention network – right IPS (39, −42, 54) ↔ salience network – right anterior insula (47, 14, 0)	$T_{(12)}$=-2.75	0.0174
Visual network – right lateral occipital region (38, −72, 13) ↔ amygdala – right hemisphere	$T_{(12)}$=-2.65	0.0212
Dorsal attention network – right IPS (39, −42, 54) ↔ salience network – left supramarginal gyrus (−60, −39, 31)	$T_{(12)}$=-2.55	0.0253
Language network – right inferior frontal gyrus (54, 28, 1) ↔ frontoparietal network – right posterior parietal cortex (52, −52, 45)	$T_{(12)}$=-2.44	0.0310
Salience network – ACC (0, 22, 35) ↔ salience network – left supramarginal gyrus (−60, −39, 31)	$T_{(12)}$=-2.32	0.0389
Salience network – right anterior insula (47, 14, 0) ↔ language network – right posterior superior temporal gyrus (59, −42, 13)	$T_{(12)}$=-2.31	0.0391
Salience network – left anterior insula (−44, 13, 1) ↔ frontoparietal network – right posterior parietal cortex (52, −52, 45)	$T_{(12)}$=-2.29	0.0406
Cerebellar network – posterior cerebellum (0, −79, −32) ↔ DMN – medial prefrontal cortex (1, 55, −3)	$T_{(12)}$=-2.28	0.0418
Dorsal attention network – left IPS (−39, −43, 52) ↔ salience network – right anterior insula (47, 14, 0)	$T_{(12)}$=-2.20	0.0484
Salience network – ACC (0, 22, 35) ↔ language network – right posterior superior temporal gyrus (59, −42, 13)	$T_{(12)}$=-2.19	0.0487

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An ROI-to-ROI functional connectivity (Table 1) shows significantly diminished connectivity in adolescents with high hostility compared to those with low hostility (contrast: low hostility > high hostility). The most substantial changes involved disrupted connectivity within the salience, attentional, and executive networks. The left FEF showed significantly reduced connectivity with the left supramarginal gyrus (SMG) ($T_{(12)}$=-3.24, P=0.007), indicating impaired integration of top-down attentional control and saliency processing. In the same vein, diminished coupling between sensorimotor superiority and SMG ($T_{(12)}$=-3.08, P=0.009) suggests a reduced predisposition to employ adaptive responses to meaningful stimuli. Reductions in the connectivity were also shown to occur between the right IPS and right anterior insula (AI) ($T_{(12)}$=-2.75, P=0.017). The visual lateral cortex (R) and right amygdala ($T_{(12)}$=-2.65, P=0.021), confirming the disrupted affective-perceptual significance of stimuli. The connections between the salience ACC and language (pSTG), the salience insula (I), and frontoparietal executive networks were also reduced in the high hostility group. A dissociated connection was also identified to emerge between the posterior cerebellum and the mPFC ($T_{(12)}$=-2.28, P=0.041), thereby implicating cerebellar-prefrontal dysregulation of emotional control. Collectively, this evidence supports the view that higher levels of hostility are associated with lower levels of functional integration across cognitive control, salience detection, and emotion regulation systems.

Discussion

The present study investigated rsFC patterns in adolescents with high versus low hostility levels. Using seed-to-Voxel and ROI-to-ROI analyses, our findings revealed significant neurofunctional differences between these 2 groups, underscoring the critical roles of prefrontal control, salience detection, attentional engagement, and socio-affective integration in modulating aggressive behavior. Unlike prior neuroimaging studies that primarily focused on overt aggression or generalized externalizing behaviors (e.g. Amaoui et al., 2022; Coccaro et al., 2011; Edalati et al., 2023), the present research uniquely targeted the hostility dimension—a latent cognitive-affective component of aggression that has rarely been examined independently, particularly in adolescents. This study thus addresses a clear gap in the literature by isolating hostility as a core construct linked to impulsive and violent tendencies, providing one of the first rsFC-level characterizations of this trait within a developmental neurocriminological framework (Abravani et al., 2025; Anderson et al., 2025).

Prefrontal connectivity and hostility regulation

Increased rsFC in the dlPFC and vmPFC of adolescents in the low hostility category suggests a regulatory role for those regions in emotional and behavioral control. The dlPFC is well-established for its role in facilitating executive function, specifically inhibitory control and conflict monitoring (Kolb & Whishaw, 2009; Boisgueheneuc et al., 2006). Increased dlPFC connectivity in our group of low hostility individuals is consistent with previous reports that the dlPFC modulates impulsivity and facilitates cognitive-emotional integration (Sukhodolsky et al., 2022; Zhu et al., 2019). Among other things, the vmPFC is engaged in the value of emotions, social cognition, and moral reasoning. The vmPFC connectivity was higher in less hostile participants. This area integrates affective data to discriminate among emotions and regulate responses in the limbic system, including, but not limited to, the amygdala (Murphy et al., 2018; Yao et al., 2024). The current findings suggest that adolescents in the low hostility categories tend to have better integration in the prefrontal-limbic area, which may support top-down regulation of emotional reactivity (Coccaro et al., 2007; Coccaro et al., 2011). Our results expand on Grecucci et al., who found that decreased dlPFC-ACC connectivity predicted greater anger expression, by showing that adolescents with lower hostility demonstrate more coherent prefrontal–limbic integration (Grecucci et al., 2022). This pattern aligns with Coccaro et al. (2011) and Romero-Martínez et al. (2024), who emphasized top-down modulation of limbic reactivity as a protective factor against aggression.

Impaired amygdala-prefrontal connectivity in high-hostility adolescents

The largest disruption in high-hostility adolescents was a decrease in rsFC between the left amygdala and frontal control regions, particularly the dlPFC. This finding aligns with models of reactive aggression that attribute proposed deficits in regulatory control over subcortical emotional processes as a primary mechanism underlying reactive aggression (Coccaro et al., 2011). The amygdala is involved in the processing of threat and emotional salience (Barlow et al., 2016). Consequently, decreased coupling with the dlPFC could reflect a decreased capacity for emotional regulation, which could manifest behaviourally as irritability and impulsive aggression. Furthermore, decreased connectivity between the amygdala and the lateral visual cortices suggests a reduced capacity to evaluate social and affective cues in the environment. This finding further extends the work of Zhu et al. (2022), who found that dysfunction in visual-amygdala circuits was linked to hostile attribution bias, a major cognitive distortion of aggressive people. Furthermore, the reduced connectivity between the amygdala and visual cortices parallels Zhu et al. (2019), who demonstrated that dysfunction in visual–amygdala circuits predicts hostile attribution bias—a cognitive distortion underlying aggressive misinterpretation of social cues. This pattern resonates with findings in adolescent and adult offenders, suggesting that decreased amygdala–prefrontal and visual–limbic connectivity constitutes a neurobiological marker of hostility-related aggression (Amaoui et al., 2022).

Dysfunctional salience and attention networks

The salience network exhibited substantial rsFC disruptions in high-hostility adolescents, specifically in the anterior insula, ACC, and SMG. These areas provide critical signal detection for outcome-relevant stimuli and initiation of appropriate control processes (Petersen & Sporns, 2015; Uddin et al., 2019). A complete breakdown of salience network connectivity is likely to lead to switching deficits between default mode and central executive networks, contributing to a reduced ability to regain attention towards relevant emotional or social stimuli (Abravani et al., 2025; Abravani et al., 2023; Werhahn et al., 2023; Zhou et al., 2016). Furthermore, reduced coupling between dorsal attention nodes (FEF, IPS) and either salience or sensorimotor nodes indicates the breakdown of goal-directed attention. This disruption may diminish situational awareness and regulation of impulses, processes that are significant contributors to high-hostility adolescents with oppositional and conduct disorders (Dugré & Potvin, 2021; Ibrahim et al., 2022). From a neurocriminological perspective, these findings suggest that hostility disrupts attention-salience integration, leading to distorted threat perception, impaired moral reasoning, and impulsive behavioral responses (Anderson et al., 2025). Complementing our findings, Edalati et al. (2023) showed that weakened salience network connectivity mediates the relationship between social adversity and later psychopathology, reinforcing the developmental vulnerability of these circuits in hostile adolescents.

Cerebello-prefrontal integration: an underestimated contributor

One of the unique findings of our study was the disrupted rsFC between the posterior cerebellum and mPFC in the high hostility group. While the cerebellum has typically been associated only with motor coordination, there has been growing recognition that it is involved in

executive processing and emotion regulation (Colombari et al., 2024; Kruithof, 2025). Our results contribute to the emerging literature that suggests cerebello-prefrontal circuits may be involved with emotional dysregulation and aggression; therefore, more research should be undertaken in neurodevelopmental psychopathology. Our results extend this emerging literature by revealing cerebello–prefrontal dysconnectivity as a novel correlate of hostility-related emotional dysregulation, suggesting potential cross-network mechanisms in neurodevelopmental psychopathology.

A network-based framework of aggression

Our findings give strong support for network-based approaches to aggression, including the I³ model (Finkel & Hall, 2018). The I³ model considers the effects of instigators, impellers, and inhibitors on the development of aggressive behavior. The observed impairments in prefrontal inhibitory networks and hypo-connectivity of attention and salience systems suggest that adolescents with high hostility lack the neurofunctional components necessary for appropriate response inhibition and effective emotional processing. Overall, our findings support the recent meta-analyses by Wang et al. (2024) and Werhahn et al. (2021), which suggest that aggression-related disorders are best characterized not only by behavioral symptoms but also by dysfunction within large-scale brain networks. The neural signatures we documented could also be useful for stratifying risk and for targeting interventions. From a neurocriminological standpoint, these early disruptions may represent neural risk markers for violent and antisocial tendencies (Abravani et al., 2025; Anderson et al., 2025). Reduced functional coupling within DMN (notably PCC and precuneus) and between prefrontal-limbic circuits supports the hypothesis that high-hostility youth exhibit compromised self-referential processing, diminished social reflection, and attenuated inhibitory control—all central to aggression models in neurocriminology.

Clinical and developmental implications

From a developmental neuroscience perspective, adolescence can be considered an essential period of PFC maturation and socio-emotional reorganization (Fairchild et al., 2010; Kolb & Whishaw, 2009). The patterns of functional disconnection observed in high-hostility adolescents may reflect a neurodevelopmental delay or alteration that compromises emotional regulation and social adaptation. This reasoning is especially relevant for populations in need of interventions, as detrimental childhood exposures can exacerbate the underdevelopment of prefrontal control circuits (Ayano et al., 2023; Saxbe et al., 2018). At a clinical level, the findings suggest that rsFC markers may identify at-risk adolescents and inform interventions. Neuromodulatory methods such as transcranial magnetic stimulation or neurofeedback targeting dlPFC–amygdala pathways may offer opportunities to develop self-regulation skills in youth with aggression-related disorders (Knehans et al., 2022; Martín-Luengo et al., 2023). Although our results suggest a network-based dysfunction in hostile adolescents, there are other plausible explanations for our findings. For example, Coccaro et al. (2011) suggested that aggression might not be a behavioral outcome of reduced connectivity between prefrontal and amygdala circuits, but rather of higher limbic brain volume or hyperresponsivity of the limbic system. Here, hyperresponsivity occurs without cortical modulation. In addition, some research (Siever, 2008; Matthies et al., 2012) has not found lower dlPFC connectivity from aggression and consistently found different connectomes for both aggressive and non-aggressive youth, thus suggesting heterogeneity in the functional neural substrates underlying aggression (Matthies et al., 2012; Siever, 2008). Finally, other socio-environmental factors could confound or partially mediate these processes, which included trauma, substance use, and peer factors. As such, multimodal neuroimaging, behavioral measures, and possible contextual variables, such as socio-environmental factors, may further elucidate the neurodevelopmental processes underlying hostility.

Conclusion

In summary, this study provides compelling evidence that adolescents with high levels of hostility exhibit widespread disruptions in large-scale brain networks, including prefrontal-limbic, salience, attention, and cerebellar systems. These neural disconnections may underlie the emotional dysregulation, impulsivity, and impaired social cognition commonly observed in this population. The results emphasize the potential of rsFC as a biomarker for identifying at-risk youth and tailoring individualized interventions. Future longitudinal studies are warranted to determine whether these connectivity profiles predict long-term behavioral outcomes or treatment response. Investigation offsets an informative void in existing scholarship by revealing that aggression—a principal cognitive-affective ingredient—impinges on far-reaching disruptions across prefrontal-limbic, salience, attentional, and cerebellar circuitry. These results bring together neuroscientific, developmental, and criminal observations, contributing to one of the first functional connectivity portraits of hostility found among ad-

olescents. They indicate that high-hostility adolescents have reduced neural coordination in emotion regulation, inhibitory control, and social cognition networks—both theoretically and clinically, generating preventive and neurotherapeutic interventions for externalizing and aggressive behavior.

Study limitations and future directions

While the present study presents substantial findings, it has important limitations. First, the study's small sample size limits generalizability; therefore, longitudinal studies are needed to assess whether the directions of the aforementioned rsFC patterns actually create hostile responses. Future researchers should also consider sex differences and the effects of disorders with high comorbidity with aggression, such as attention-deficit/hyperactivity disorder and anxiety. Furthermore, including a behavioral or task fMRI paradigm could inform the current resting-state findings by examining more dynamic functional regulation during emotionally primed or inhibitory tasks.

Ethical Considerations

Compliance with ethical guidelines

Data availability

Data supporting this study's findings are available upon a reasonable request from the corresponding author.

Declaration of generative AI and AI-assisted technologies in the writing process

No AI tool influenced the scientific content, data analysis, or conclusions of this work.

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Authors' contributions

All authors contributed equally to the conception and design of the study, data collection and analysis, interception of the results and drafting of the manuscript. Each author approved the final version of the manuscript for submission.

Conflict of interest

The authors declared no conflict of interest.

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