Title: Neurofunctional Correlates of Hostility in Adolescents with Externalizing Disorders: Impli-cations for Clinical Assessment and Intervention

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Abstract:

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. This study examined resting-state functional connectivity differences in adolescents with high and low hostility, focusing on brain networks related to emotion regulation, salience, and executive control using the BPAQ scale. We utilized seed-to-voxel and ROI-to-ROI fMRI models to examine rsFC in two groups of adolescents: 14 with externalizing disorders and 13 typically developing controls.Seed-to-voxel analysis showed greater rsFC in low-hostility adolescents within two clusters: left DLPFC (BA 9/46) and vmPFC (BA 10/11) compared to high-hostility peers.. Both target regions represent top-down emotional processing and social-affective processing, respectively, providing evidence of the lower hostility group being more efficient in regulating aggressive impulses. ROI-to-ROI analysis revealed significantly reduced connectivity in high-hostility adolescents, notably between DLPFC–amygdala and frontal midline–amygdala, indicating impaired emotion regulation. Decreased links were also found between dorsal attention and salience networks, visual–limbic regions, and between cerebellar and medial prefrontal areas.. These differences reinforce disrupted functioning of conceptually relevant executive and attentional networks, as well as affective and socio-emotional networks in adolescents with increased hostility. We perceive these findings collectively as a neurobiological difference contrast between low hostility and high hostility, and notice that decreased connectivity of both the prefrontal network and salience network may represent targets for neurotherapeutic interventions to decrease aggression in children and adolescents with externalizing problems.

Keywords: Externalizing disorders, Hostility, Adolescents, Frontal lobe, Resting-state fMRI

Introduction

Aggression and hostility are prominent behavioral manifestations frequently observed in externalizing disorders such as Conduct Disorder (CD) and Oppositional Defiant Disorder (ODD). These conditions, which often emerge during childhood or adolescence, are characterized by persistent patterns of rule-breaking, defiance, and in many cases, overt aggression(First, 2013) (American Psychiatric Association, 2013). The presence of such behaviors significantly increases the risk of adverse outcomes, including academic difficulties, delinquency, and progression to adult antisocial behavior or personality pathology.

The frontal lobe alongside its prefrontal cortex (PFC) has been identified as essential for managing aggressive behavior by recent studies. The PFC which includes its four 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 cortical thickness decrease 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 MRI 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 through evidence that shows right dlPFC activation decreases proactive aggression while left dlPFC suppression intensifies reactive and proactive aggression. The bilateral stimulation of dlPFC regions produces decreases in both aggressive intentions and violent behavior justifications(Fritz et al., 2023; Singh & Gobrogge, 2024). Animal model studies of development demonstrate that early social stress through social isolation prevents proper PFC growth which results in increased aggression and modified neuronal patterns within PFC circuits(Singh & Gobrogge, 2024). During adolescence serotonin signaling within the medial PFC plays an essential function in controlling aggression because impaired serotonin transporter function in this region causes pathological aggression(Szebik et al., 2025). The hypothalamus and temporal cortex serve as important components of the aggression network because research reveals 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 and connectivity and neurochemical defects lead to harmful behavioral outcomes. Targeted interventions which include 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 while the brain is at rest, allowing for 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 regarding resting state functional connectivity (rsFC) has uncovered the brain network that underlies aggression. Emerging evidence identifies both the common and the relatively aggression subtype-specific effects on global-scale networks. For example targeting emotional dysregulation and maladaptive aggression, rsFC effects from the amygdala - ventromedial prefrontal cortex (vmPFC) that are particularly relevant to youth also demonstrated both global and aggression subtype rsFC patterns, especially the disruptive youth with increased rsFC to the amygdala and decreased 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 such as 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), Medial PFC (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, was associated with similar rsFC reductions)(Zhu et al., 2019). New findings provided empirical support for the importance of rsFC changes, including in the lateral hypothalamus (LH) that negatively correlated with aggression, along with differences of male vs female youth in rsFC LH–thalamus 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, demonstrated 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 disrupted rsFC, as well as DMN disrupted rsFC, may be suggestive of 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 ADHD, and as biomarkers for risk profiling and intervention targeting in clinical practice.

Hostility is a critical construct of aggression and a cognitive-emotional orientation associated with suspiciousness, resentment, and a tendency to assign a 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. In 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 which had been extensively studied for aggression broadly, especially in adolescents and social interactions(Dodge & Coie, 1987). Researchers have identified five subcomponents of hostility—angry affect, hostile intent, verbal aggression, physical aggression, and relational aggression. Neurocognitive models have put forward the idea that hostile cognitions and behaviors developed and caused dysfunction due to increased limbic activity poorly regulated by prefrontal regulation of limbic activity(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, medial prefrontal cortex, anterior cingulate cortex, insula) that were related to measures of hostility and aggression when the studies were evaluated determinatively. Neuroimaging studies have shown a significant association of aggression and hostility and limbic networks across several studies (Emil F Coccaro et al., 2007; E. F. Coccaro et al., 2007).Recent work published in the Journal of Affective Disorders indicated that increased hostile attribution bias was associated with displaced aggression and underpinned by hyperactivity in limbic regions of the brain. This suggests 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 links 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 hostility-related rsFC alterations in adolescents for the ultimate aim of informing and developing specific neurobiological markers to assist in the early age assessments and interventions for youth.

Methods

Participants

The present study investigated adolescents residing in an economically-disadvantaged and intervention-needing neighbourhood and setting in Tehran. 27 adolescents were selected for study, consisting of 14 adolescents with externalizing disorders (ED) and 13 typically developing (TD) controls. This sample size was both feasible and determined correspondingly to guidelines and norms from neuroimaging (fMRI) literature. The sample size would provide adequate statistical power for resting-state functional connectivity (rsFC) statistics while simultaneously accounting for the scanning time and potential data quality and processing issues. The overall balance also allowed for more appropriate group comparisons, and control of potential confounding variables at the adolescent level in sample and evaluation designs.

Several covariates were included in the group comparisons to account for potential confounding/confounding effects and to strengthen the ability to make conclusions about whether statistically significant differences in brain connectivity groups would be specifically due to group differences in hostility. These covariates included:

Parental education Level as a measure of cultural and educational experience,

1. Parental Psychopathology as representing genetic and or environmental, risk factors converging on externalizing disorders,
2. Family socioeconomic status (SES) as a measure of environmental stress and deprivation,
3. The presence or absence of domestic violence as a direct risk factor associated with hostility and aggression in the home, and
4. Gender, age, and IQ as additional controls for biological and cognitive variability across participants.

Behavioral Analysis and Assessment

Buss–Perry Aggression Questionnaire (BPAQ)

For assessing aggressive behavior, the Buss–Perry Aggression Questionnaire (BPAQ) was administered. It was originally created in 1992, and since then it has been embraced for its capacity to assess various facets of aggression (Buss & Perry, 1992). The BPAQ is a 29-item self-report scale that measures four main dimensions: physical aggression (9 items), verbal aggression (5 items), anger (7 items), and hostility (8 items). This multi-dimensional approach permits an in-depth analysis of both the overt aggressive acts and the affective states that may be underpinning them. BPAQ has been shown to possess robust psychometric properties and has been effectively cross-translated in a wide range of culturally and linguistically diverse populations (Javela et al., 2023; Morren & Meesters, 2002; Vigil-Colet et al., 2005).fMRI

Image Acquisition

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

Image Pre-processing 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 match functional and anatomical images to 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 processes, which consisted of regression of confounding variables in addition to temporal band-pass filtering, in order to decrease 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: 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, which is a widely used parcellation framework in resting-state fMRI studies. According to the CONN atlas, the brain's seven canonical intrinsic networks include: Default Mode Network (DMN), Sensorimotor Network, Visual Network, Salience Network, Dorsal Attention Network, Auditory Network, Central Executive Network (also referred to as 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 the purpose of this study, these networks were not meant to be exclusively defined or separated as intrinsic networks in the atlases with which they are compared, but they do embody some identifiable components related to some important areas for top-down regulation including the medial prefrontal cortex (MPFC)—which is associated with affective and self-referential processing—, and the lateral prefrontal cortex, a critical hub for executive control. The cerebellar networks are typically comprised of two functional subdivisions: a motor cerebellar network involved with coordination of movement, and a cognitive cerebellar network, which contains the posterior cerebellum and supports higher-order cognitive functions or behaviour. Language regions (e.g., inferior frontal gyrus and posterior superior temporal gyrus) were included in our work due to their role in language processing and higher-order cognition. The language regions are not usually represented as separate intrinsic networks in most parcellations.

Resting-state fMRI data were pre-processed and analysed using the CONN functional connectivity toolbox (v.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) prior to 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 role in emotional and executive control: Default Mode Network (DMN), Amygdala, Frontal Control Network (including 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 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:

Default Mode Network (DMN): Medial Prefrontal Cortex (MPFC): (1, 55, −3), Lateral Parietal (Left): (−39, −77, 33), Lateral Parietal (Right): (47, −67, 29), Posterior Cingulate Cortex (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: Anterior Cingulate Cortex (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 (Left): (−27, −9, 64), Frontal Eye Field (Right): (30, −6, 64), Intraparietal Sulcus (Left): (−39, −43, 52), Intraparietal Sulcus (Right): (39, −42, 54)

Fronto-Parietal Network: Lateral Prefrontal Cortex (Left): (−43, 33, 28), Posterior Parietal Cortex (Left): (−46, −58, 49), Lateral Prefrontal Cortex (Right): (41, 38, 30), Posterior Parietal Cortex (Right): (52, −52, 45)

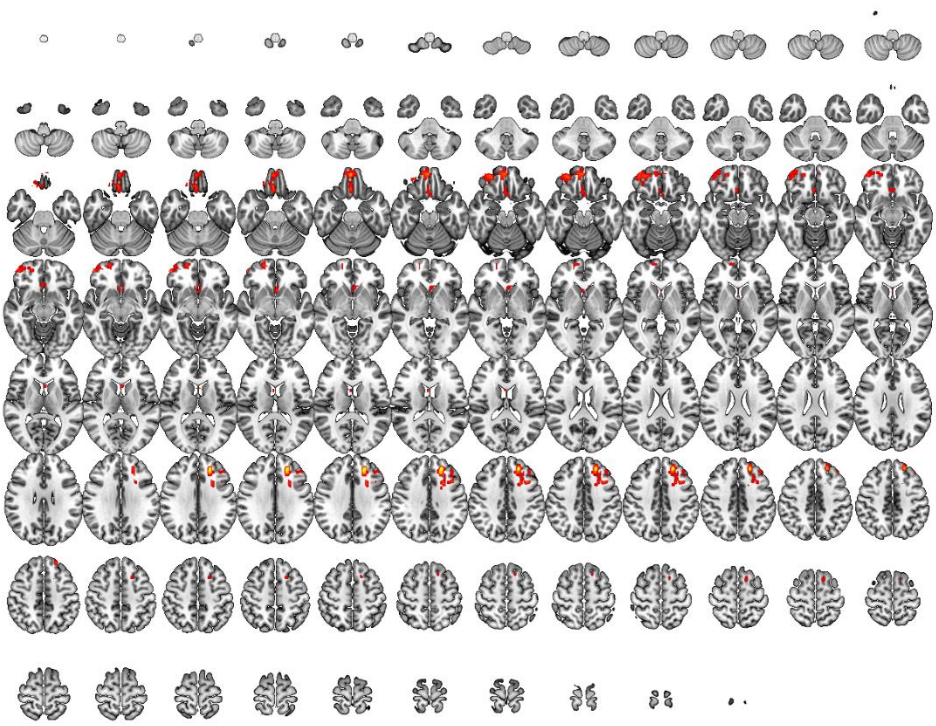
Language Network: Inferior Frontal Gyrus (Left): (−51, 26, 2), Inferior Frontal Gyrus (Right): (54, 28, 1), Posterior Superior Temporal Gyrus (Left): (−57, −47, 15), Posterior Superior Temporal Gyrus (Right): (59, −42, 13)

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, FDR-corrected 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

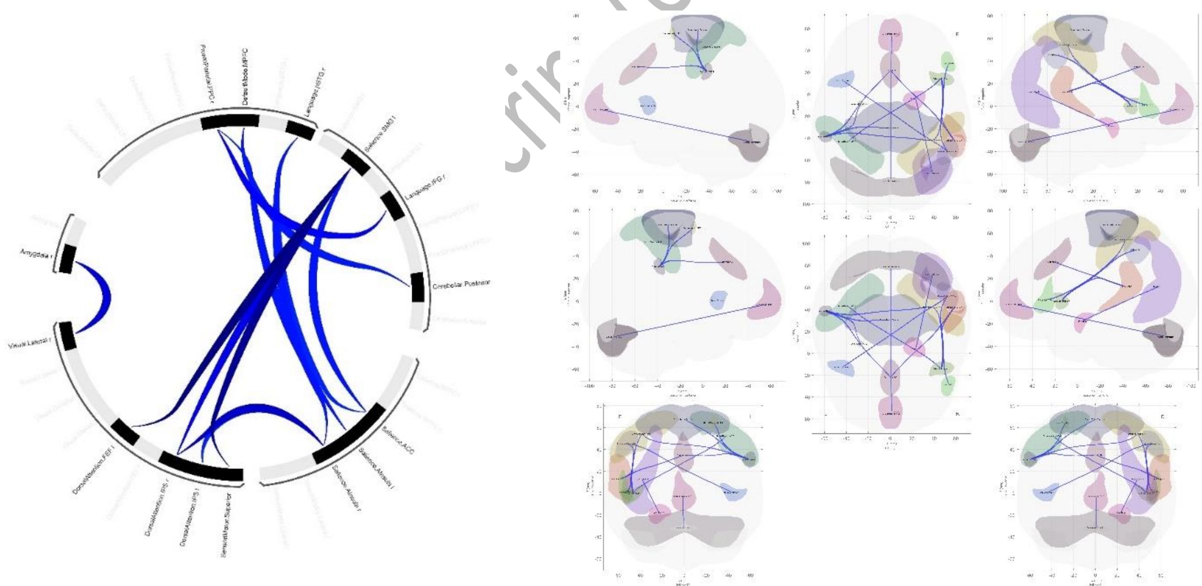
Fig 1. Functional Connectivity Differences in Low vs. High Hostility Adolescents, Seed to voxel analysis



clusters	voxels	p- value
+04 +54 -24	1244	0.003673
-20 +36 +36	776	0.07852

In our study, low hostility adolescents vs. high hostility adolescents (i.e., Low > High), two significant clusters for increased resting-state functional connectivity (rsFC) were identified. The first cluster included 776 voxels (p value = 5.11×10^{-6}). This location was in the left dorsolateral prefrontal cortex (DLPFC), given previous literature which we would characterize as being in peaks significant in compared to coordinates: −20, 36, 36 (peak MNI coordinates: approximately Brodmann Area: 9/46; also two 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 within 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 value = 9.65×10^{-5}). Peaks were in the ventromedial prefrontal cortex (vmPFC) (peak MNI coordinates: 4, 54, − 024; BA 10/11; significant negative peaks at (48, 50,-10) (28, 48 , −20). The vmPFC represents a significant brain region in supportive function for social behavior, emotion valuation, and emotion regulation. The increase in rsFC in vmPFC suggests the lower hostility adolescents are more engaged with their affective control circuits. The implications of these findings reinforce the importance of the prefrontal control network roles in mitigating aggressive behavior tendencies supported by a neurobiological difference in adolescents with different levels of hostility.

Fig 2. ROI-to-ROI Functional Connectivity Results: Low Hostility > High Hostility



Circular ROI-to-ROI connectivity analysis detected a number of significant decreases in resting-state functional connectivity (rsFC) in high hostility adolescents, as compared to low hostility adolescents. Interestingly, left amygdala showed reduced connectivity with dorsolateral prefrontal cortex (DLPFC) and frontal midline regions, which is indicative of impaired top-down affect regulation among aggressive subjects. Additional disruptions were found between dorsal attention networks (FEF and IPS) and sensorimotor and salience-related nodes (ACC, anterior insula). These findings may be indicative of impaired attentional control and detection of salience of stimuli. 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 maladaptive control of aggressive behavior.

Table 1. ROI-to-ROI Connectivity Analysis

Brain Region Connections	**Statistic**	**p-value**
Dorsal Attention Network – Left Frontal Eye Field (−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 Intraparietal Sulcus (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 Intraparietal Sulcus (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 – Anterior Cingulate Cortex (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) ↔ Default Mode Network – Medial Prefrontal Cortex (1, 55, −3)	T(12) = -2.28	0.0418
Dorsal Attention Network – Left Intraparietal Sulcus (−39, −43, 52) ↔ Salience Network – Right Anterior Insula (47, 14, 0)	T(12) = -2.20	0.0484
Salience Network – Anterior Cingulate Cortex (0, 22, 35) ↔ Language Network – Right Posterior Superior Temporal Gyrus (59, −42, 13)	T(12) = -2.19	0.0487

A ROI-to-ROI functional connectivity table demonstrated significantly diminished connectivity for adolescents with high hostility compared to adolescents with low hostility (contrast: Low Hostility > High Hostility). The most substantial points of change included disrupted connectivity within the salience network, attentional network, and executive networks. The left frontal eye field (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 connectivity in coupling between sensorimotor superiority and SMG (T(12) = -3.08, p = 0.009), suggests reduced predisposition to employ adaptive responses to meaningful stimuli. Reductions in the connectivity were also shown to occur between the right intraparietal sulcus (IPS) and right anterior insula (AI) (T(12) = -2.75, p = 0.017), and the visual lateral cortex (R) and right amygdala (T(12) = -2.65, p = 0.021), confirm disrupted affective-perceptual significance of stimuli. The connections between salience anterior cingulate cortex (ACC) and language (posterior superior temporal gyrus [pSTG]), salience insula (I) and frontoparietal executive networks, were also reduced for the high hostility group. A dissociated connection was also identified to emerge between posterior cerebellum and medial prefrontal cortex (MPFC) (T(12) = -2.28, p = 0.041), therefore implicating cerebellar-prefrontal dysregulation of emotional control. Collectively, this evidence articulates evidence toward the view that higher levels of hostility are affiliated with lower levels of functional integration across cognitive control, salience detection, and emotion regulation systems.

Discussion and Conclusion

The present study investigated resting-state functional connectivity (rsFC) patterns in adolescents with high versus low levels of hostility. Using seed-to-voxel and ROI-to-ROI analyses, our findings revealed significant neurofunctional distinctions between these two groups, emphasizing the critical roles of prefrontal control, salience detection, attentional engagement, and socio-affective integration in the modulation of aggressive behavior. Unlike prior neuroimaging studies that primarily focused on overt aggression or generalized externalizing behaviors (e.g., Coccaro et al, Amaoui et al.,Edalati et al)(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

Increases rsFC in the dorsolateral prefrontal cortex (DLPFC) and ventromedial prefrontal cortex (vmPFC) of adolescents in the low hostility category implies a regulatory role of those regions in emotional and behavioral control. The DLPFC is well-established for its role which facilitates executive function, and 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 is engaged in the integration of affective data for discriminating among emotions and the regulation of responses in the limbic system, which includes but is not limited to, the amygdala(Murphy et al., 2018; Yao et al., 2024). The current finding suggests 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(Emil F Coccaro et al., 2007; Romero-Martínez et al., 2019). Our results expand upon Grecucci et al, who found that decreased DLPFC and ACC connectivity predicted higher anger expression, by showing that adolescents with lower hostility demonstrate more coherent prefrontal–limbic integration(Grecucci et al., 2022). This pattern supports Coccaro et al and Romero-Martínez et al, who emphasized top-down modulation of limbic reactivity as a protective factor against aggression.

Impaired Amygdalo-Prefrontal Connectivity in High Hostility Adolescents

The biggest disruption found for high-hostility adolescents was a decrease in the rsFC between the left amygdala and frontal control regions, especially the DLPFC. This finding aligns with models of reactive aggression that attribute proposed deficits in regulatory control over subcortical, emotional process as a primary mechanism underlying reactive aggression(Emil F Coccaro et al., 2007; Fair et al., 2007; Sukhodolsky et al., 2022). The amygdala is involved in the processing of threat and emotional salience(Barlow et al., 2016), consequently, decreased coupling with the DLPFC could represent a decreased capacity for emotional regulation, which could behaviourally appear as irritability and impulsive aggression. Furthermore, the decreased connectivity between the amygdala and visual lateral cortices suggests a reduced capacity to evaluate social and affective cues from the environment. This finding further extends the work of Zhu et al, who found that dysfunction in visual-amygdala circuits was linked to hostile attribution bias, a major cognitive distortion of aggressive people(Zhu et al., 2022). Furthermore, the reduced connectivity between the amygdala and visual cortices parallels Zhu et al. , who demonstrated that dysfunction in visual–amygdala circuits predicts hostile attribution bias—a cognitive distortion underlying aggressive misinterpretation of social cues(Zhu et al., 2019). This pattern resonates with findings in adolescent and adult offenders, suggesting that decreased amygdalo–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, anterior cingulate cortex (ACC), and supramarginal gyrus (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. 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(Edalati et al., 2023).

Cerebello-Prefrontal Integration: An Underestimated Contributor

One of the unique findings of our study was the disrupted rsFC between the posterior cerebellum and medial prefrontal cortex (MPFC) in the high hostility group. While the cerebellum has typically only been associated with motor coordination, there has been a growing recognition that the cerebellum 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 I^3 model(Finkel & Hall, 2018). The I^3 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 Wang et al. and Werhahn et al.'s recent meta-analyses that suggest aggression-related disorders are best characterized, not only in terms of behavioral symptoms, but also related to dysfunction within large-scale brain networks(Wang et al., 2024; Werhahn et al., 2021). The neural signatures we documented could also be useful in stratifying risk and intervention targeting. 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 the view of developmental neuroscience, adolescence can be considered an essential period of maturation of the prefrontal cortex and socio-emotional reorganization(Fairchild et al., 2010; Kolb & Whishaw, 2009). The patterns of functional disconnection found in high hostility adolescents may represent some neurodevelopmental delay or alteration that results in compromised emotional regulation and social adaptation. This reasoning is especially relevant in populations needing interventions, since having detrimental childhood exposures can heighten the underdevelopment of prefrontal control circuits(Ayano et al., 2023; Saxbe et al., 2018). At a clinical level, the findings offer the potential for rsFC markers to identify at-risk adolescents and then inform interventions. Neuromodulatory methods such as transcranial magnetic stimulation (TMS) or neurofeedback that target DLPFC–amygdala pathways may provide opportunities to develop self-regulation skills in youth presenting 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 finding. For example, Coccaro et al. suggested that aggression might not be a behavioral outcome of lower connectivity between prefrontal and amygdala circuits, but higher limbic brain volume or hyperresponsivity of the limbic system (Coccaro et al., 2011). Here, this hyperresponsivity occurs without cortical modulated regulation. In addition, some research (e.g., 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, multi-modal neuroimaging with behavioral measures and possible contextual variables such as socio-environmental measures may further explain the neurodevelopmental processes underlying hostility.

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 resting-state functional connectivity 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 response to treatment. Investigation offsets an informative void within existing scholarship by revealing that aggression—a principal cognitive-affective ingredient of aggression—impinges on far-reaching disruptions within prefrontal-limbic, salience, attentional, and cerebellar circuitry. These results bring together neuroscientific, developmental, as well as criminal, observations, contributing one of the first functional connectivity portraits of hostility found among adolescents. They indicate that high-hostility adolescents have lowered neural coordination for emotion regulation, inhibitory control, and social cognition networks—both theoretically as well as clinically generating preventive as well as neurotherapeutic interventional bearings toward externalizing behavior as well as aggressive behavior.

Limitations and Future Directions

While the present study presents substantial findings, there are important limitations to note. 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 creates hostile responses. Future researchers should also consider sex differences and the effects of disorders with high co-morbidity with aggression such as attention-deficit/hyperactivity disorder (ADHD) and anxiety. Furthermore, including a behavioral or task fMRI paradigm could potentially inform the current resting-state findings to examine more dynamic functional regulation during emotionally primed or inhibitory tasks.

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Compliance with Ethical Standards

Ethical approval for this study was granted by the Research Ethics Committee of Shahid Beheshti University (Approval ID: IR.SBU.REC.1401.089) on 2022-09-03. Written informed consent/assent was obtained from all participants, and parental consent was secured for participants under the age of 18.

Conflict of Interest

The authors declare no conflict of interest related to this study.

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