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Callous-unemotional traits in adolescents moderate neural network associations with empathy

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Abstract

Callous-unemotional (CU) traits in adolescents are a dimensional construct involving a symptom subset of empathy impairments amongst broader affective deficits. Higher CU trait scores associate with less cognitive and affective empathy; and brain regions linked with cognitive and affective empathy show aberrant function in those with CU traits. How CU traits impact the relationship between brain function and both cognitive and affective empathy in adolescents is less clear. Here we examine how functional properties of networks that support cognitive and affective empathy is moderated by CU traits. Eighty-four adolescents underwent resting-state fMRI scanning and completed self-reports for empathy (Interpersonal Reactivity Index) and CU traits (Inventory of Callous-Unemotional Traits). Analysis revealed that CU traits moderate the association between affective empathy and connectivity between the default mode-frontoparietal networks. Weaker between default mode-frontoparietal anticorrelation negatively associated with affective empathy at low to moderate CU traits. Those highest in CU traits had the lowest affective empathy; and negative associations for those highest in CU traits were insignificant as default mode-frontoparietal anticorrelation weakened. Our results indicate that functional properties of networks that support affective empathy is different at varying levels of CU traits. This novel finding demonstrates that CU traits presence changes the relationship between the brain and empathy.

Keywords

Callous-Unemotional traits; Functional Connectivity; Cognitive Empathy; Affective Empathy

1. Introduction

Callous-unemotional (CU) traits involve an impairment in prosocial emotions such as guilt, remorse, and empathy (Frick & White, 2008; Miller & Eisenberg, 1988). Empathy, the

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Conflict of Interest

The authors declare that there is no conflict of interest

capacity to understand others and share their emotions (Decety et al., 2016), encompasses only a subset of deficits underlying CU traits and does not account for the broader affective impairments such as a lack of concern for performance and restricted affect (Frick et al., 2014a, 2014b). Previous research demonstrates CU traits has a moderate negative association with both empathy (for meta-analysis: Waller et al., 2020) and neural networks underlying empathy, empirically suggesting a potential moderating effect (Sharma et al., 1981). This suggests it is plausible that the functional connectivity (FC) underlying empathy is different in the presence of increasing CU traits; however, this has yet to be examined. Thus, the present study aims to clarify how empathy may function differently in adolescent brains when CU traits are present.

Although related, CU traits encompass broader affective impairments beyond deficits in cognitive or affective empathy. *Cognitive empathy* describes the ability to adopt another's point of view to understand their thoughts and feelings; whereas *affective empathy* involves sharing another's emotional experience, which can involve *empathic concern* or feelings of concern for their emotional wellbeing (Decety, 2011; Decety & Cowell, 2015). The lack of empathy associated with CU traits is thought to account for the callous harm of others and antisocial behavior; however, these constructs are multifaceted and are not synonymous (Rijnders et al., 2021). CU traits definition involves impairment in empathy within broader affective impairments such as a lack of care for performance and emotional under responsiveness (Frick et al., 2014a, 2014b). Empirically, CU traits association with both cognitive and affective empathy is moderate (affective r= −0.33, cognitive r = −0.44; in meta-analysis: Waller et al., 2020) demonstrating CU traits are not completely accounted for by empathy impairments. And the implied variance accounted for (affective R^2= 0.11; cognitive R^2= 0.19) demonstrates that, amongst those with CU traits, there is some unaccounted variation in both cognitive and affective empathy. Both empirical and conceptual evidence suggests that these constructs are related but not synonymous. Thus, it is plausible that these two constructs have both shared and unshared neural underpinnings.

Separate studies suggest neural networks underlying empathy show aberrant connectivity in higher levels of CU traits, but little is known about how the presence of CU traits may change the relationship between the brain and empathy. Adolescent studies demonstrate that during cognitive empathy tasks (perspective taking) regions of the default mode network (DMN; i.e., medial prefrontal cortex and posterior cingulate cortex) and the frontoparietal network (FPN; i.e., the inferior parietal lobule and dorsolateral prefrontal cortices) are active (D'Argembeau et al., 2007; Gallagher et al., 2000; Kral et al., 2017); whereas affective empathy tasks (e.g. viewing others' pain) the salience network (SAL; e.g. anterior insula, anterior cingulate and rostral prefrontal cortex) is active (Decety & Michalska, 2010; Decety et al., 2008). Previous work with the dataset used in the current study revealed FC within the DMN associated with cognitive empathy (Winters, Pruitt, et al., 2021). The extant literature shows that CU traits associate with lower connectivity in the DMN (Cohn et al., 2015; Umbach & Tottenham, 2020) and SAL (Yoder et al., 2016), as well as with higher connectivity in the FPN (Cohn et al., 2015). Furthermore, where typically developing brains demonstrate anticorrelation between the DMN-FPN (Uddin et al., 2009), which has been implicated in empathy (Xin & Lei, 2015), those with CU traits demonstrate a lower anticorrelation between the DMN-FPN (Pu et al., 2017; Winters, Sakai, et al.,

2021). The different directions in the same networks suggests, empirically, that a "quasi" moderation exists because CU traits have both an independent effect but plausibly affects the relationship between these variables (Sharma et al., 1981). Despite the clear connections in the literature and the relevance for identifying mechanisms underlying core impairments in CU traits, research has rarely examined how CU traits moderate the functional relationship between the brain and empathy.

The present study addresses an important gap in the literature by examining network FC underlying empathy at different levels of CU traits. This study builds on prior research with the same sample examining functional connections underlying empathy in adolescents, (Winters, Pruitt, et al., 2021), by testing how these associations are different in the presence of CU traits. As suggested by previous literature, we hypothesized that stronger within DMN and FPN connectivity would associate with higher cognitive empathy, stronger within SAL connectivity would associate with higher affective empathy, and CU traits would moderate these within-network associations with empathy. Also, we explored the relationship between DMN-FPN connectivity for both cognitive and affective empathy. We test these hypotheses in a community sample of adolescents as a considerable amount of support exists that CU traits is dimensionally present in the community, and they are associated with a range of the same neurocognitive correlates (Viding & McCrory, 2012) and similar brain abnormalities (Seara-Cardoso et al., 2022) as clinical or forensic samples. Furthermore, community samples have the advantage of more variation in symptoms (fewer ceiling effects), which affords greater ability to parse comorbid symptomology (e.g., conduct problems). Because socio-affective impairments are thought to drive antisocial behavior amongst youth with CU, investigating mechanisms underlying core social impairments is a critical incremental step that identifies potential mechanisms used for larger goals of targeting antisocial behavior in these youth. The present study aims to identify mechanisms underlying differences observed in cognitive and affective empathy amongst adolescents with CU traits.

2. Methods

2.1. Sample

Participants for the current study consisted of a sample of community adolescents (ages 13–17) drawn from the Nathan Kline Institute's Rockland dataset (for study procedures see: Nooner et al., 2012) using the 1000 connectomes project website (www.nitrc.org/projects/fcon_1000/). Institutional review boards at the Nathan Kline Institute and Montclair State University approved the study and written informed consent/assent was obtained from all parties including the child, parents, and legal guardians (see: Nooner et al., 2012). There were a total of 122 participants between the ages of 13–17. To ensure participants understood self-report measures, we excluded 10 participants with a WAIS-II IQ score < 80 (α = .96; Wechsler, 2011). To ensure proper image quality, we excluded 24 participants for motion (i.e., movement > 3mm in any direction) and four participants for invalid scans (> 20% of invalid volumes). Leaving a total of 84 participants for analysis that were predominantly White (White= 63%, Black = 24%, Asian = 9%, Indian = 1%, other= 3%) balanced between sex (female = 45%) and a mean age of 14.59±1.48.

2.2. Measures

Interpersonal Reactivity Index (IRI).—Cognitive and affective empathy were assessed using the perspective taking and empathic concern subscales (respectively) of the IRI (Davis, 1980, 1983). The cognitive empathy subscale (α=.74) measured the tendency to adopt the psychological point of view of others (e.g., "I try to look at everybody's side of a disagreement before I make a decision"). The affective empathy subscale (α=.79) measured the tendency to experience other's feelings and have concern for them (e.g., "When I see someone being taken advantage of, I feel kind of protective towards them"). Each subscale consisted of seven items that were rated on a five-point scale ranging from 0 ("does not describe me") to 4 ("describes me well"). Higher scores indicate higher levels of dispositional empathy.

Inventory of Callous-Unemotional Traits (ICU).—The ICU is a 24-item assessment of CU traits (Frick, 2004). The ICU has a confirmed factor structure and demonstrates convergent and divergent validity (Kimonis et al., 2008) and the overall score had adequate reliability in the present sample (α=.72). Participants rate items on a four-point Likert scale from 0 ("not true at all") to 3 ("definitely true") on items such as "I do not show my emotions to others". Higher scores indicate greater level of CU traits. In comparison to other studies with community samples (e.g., Byrd et al., 2013; Essau et al., 2006) the present sample mean total ICU scores were within one standard deviation of comparable samples (Boys= 23.11±7.56; Girls= 22.27±10.31; Total= 22.71±8.93).

Covariates and Demographics.—The youth self-report (YSR) is a measure for behavior problems in youth ages 11–18 (Achenbach & Rescorla, 2001). The externalizing (α=.87) subscale was used to control for conduct issues in the present analysis. Items such as 'I feel a lack of guilt after misbehaving' are rated on a three-point scale (0 not true – 2 very true) indicating how much they agree with the statement for the previous 6-months. Higher scores indicate higher externalizing symptoms. Validity and reliability of the YSR externalizing measure are within acceptable standards (Achenbach & Rescorla, 2001). Raw scores were used as recommended for research purposes by Achenbach and Rescorla (2001).

Pubertal development was measured by the genital and breast development subscales of the Tanner assessment (α = .77), in which parents rated pictures representing development of secondary sex characteristics on a scale of 1 (pre-pubertal) to 5 (full maturity) (Petersen et al., 1988). Higher scores indicate greater developmental maturity.

Self-report of age and sex were included as nuisance covariates. We had no theory to support differences in hypothesized associations according to race – therefore race was not included as a covariate.

2.3. Imaging Procedures

Imaging Acquisition.—Resting state images were collected with a Siemens TimTrio 3T scanner using a blood oxygen level dependent (BOLD) contrast with an interleaved multiband echo planar imaging (EPI) sequence. Participants were instructed to keep their eyes closed without falling asleep and to not think of anything in particular while they let

their mind wander. Each participant received an fMRI scan during resting state (260 EPI volumes; repetition time (TR) 1400ms; echo time (TE) 30ms; flip angle 65°; 64 slices, Field of view (FOV) = 224mm, voxel size 2mm isotropic, duration = 10 minutes) and a magnetization prepared rapid gradient echo (MPRAGE) anatomical image (TR= 1900ms, flip angle 9°, 176 slices, FOV= 250mm, voxel size= 1mm isotropic). T1 stabilization scan removal was not necessary given that the Siemens sequence collects images after saturation is achieved.

Imaging Preprocessing.—Preprocessing was conducted using Statistical Parametric Mapping (SPM version 12; Penny et al., 2011) using the standard preprocessing pipeline via the CONN toolbox (version 18b; Whitfield-Gabrieli & Nieto-Castanon, 2012). The standard preprocessing pipeline involves using the first scan as a reference image to co-register realigned and unwarped scans. Using the Artifact Detection Tools (ART; http://www.nitrc.org/projects/artifact_detect), motion outliers were flagged for correction if composite motion > 0.5mm and then regressed out of the time series using binary motion covariates. No timing correction was used due to the short TR and multiband sequence used for acquisition. Functional and anatomical data was standardized into MNI space and segmented into grey matter, white matter, and CSF tissue using the unified segmentation and normalization procedure (Ashburner & Friston, 2005). Physiologic CSF and white matter noise was regressed out of the BOLD signal using anatomic component-based noise correction method (aCompCor) (Whitfield-Gabrieli & Nieto-Castanon, 2012). aCompCor is an alternative to global signal regression that does not artificially introduce anticorrelations (Whitfield-Gabrieli & Nieto-Castanon, 2012). Co-registered MPRAGE and EPI images were normalized to an MNI template. Image smoothing was conducted using a 6mm Gaussian kernel. Finally, to correct for drift and preserve meaningful resting state associations, the data was bandpass filtered to between .008 and .09Hz (Satterthwaite et al., 2013).

Region of Interest Selection.—Brain imaging studies of both empathy (Decety & Michalska, 2010; Decety et al., 2008; Fan et al., 2011; Kral et al., 2017; Lamm et al., 2011) and CU traits (Pu et al., 2017; Umbach & Tottenham, 2020; Yoder et al., 2016) supported focus on the DMN, SAL, and FPN in the current analysis. We defined all regions of interest that represent the DMN, SAL, and FPN networks anatomically using the Harvard-Oxford atlas that is included in the CONN toolbox, which includes the entire atlas defined region. The regions included the medial prefrontal cortex, posterior cingulate cortex, and angular gyri for the DMN; bilateral anterior insulae, anterior cingulate, and bilateral rostral prefrontal cortices for the SAL; and the bilateral lateral prefrontal and posterior parietal cortices for the FPN (MNI coordinates: Table 1).

Extracting Connectivity Parameters.—BOLD time-series of each ROI defined by the Harvard-Oxford Atlas was extracted from the 4D preprocessed resting state scan. Then, using the defined ROI time series, we calculated the strength of connection for all pairwise connections within and between each network, averaged for all pairwise connections representing the within and between network connection, and extracted this value for each participant for each within and between network connection separately. These participant-level connections were converted to a Z value using Fisher's r-to-z transformation. The

result was a single value representing the strength of connection for each within and between network connection (three within variables and three between variables) for each individual participant. These connectivity values were used in subsequent analyses.

2.4. Analysis

All analyses were conducted with the statistical language R (Version 4.02; R Core Team, 2021). To reduce multiple comparisons, we conducted a path analysis using the 'lavaan' package (Rosseel, 2012). Prior to analysis, the data met assumptions of distribution normality of residuals (based on histogram of model residuals, Q-Q plot, and residual plot), autocorrelation (based on Durbin-Watson test [$p<0.05$ and statistic approximately 2]), and multicollinearity (based on variance inflation factor [<2.5] and tolerance [>0.4]). Additionally, we examined scatterplots for non-linear associations which revealed none and linearity was established for all variables in the analysis. Assumptions for multivariate normality were met, thus deemed appropriate for maximum likelihood estimation. Additionally, we examined if there were differences between the participants who were excluded compared to those included in the final sample on demographics and variables of interest using t tests, which revealed no significant differences.

A path analysis was fit to test the study's hypotheses, which afforded us the advantage of estimating paths to multiple dependent variables in one model simultaneously (reducing multiple comparison concerns) while evaluating confidence in estimated parameters by evaluating overall model fit (Figure 1). The model was evaluated for adequate fit using criteria of both TLI and CFI ≥ 0.90, and RMSEA ≤ 0.05 (Hu & Bentler, 1999; Mulaik et al., 1989). We modelled functional connectivity as the independent variable and empathy as the outcome variable with CU traits as the moderator. This is in line with contemporary theory that the brain and its FC drives behavior (Nielsen et al., 2018; Raichle & Gusnard, 2005), which has been used to predict dispositional personality features in previous research (e.g., Friedel et al., 2015; Lai et al., 2020) as well as features of CU traits (Pu et al., 2017).

The CU traits moderating term was derived using the residual centering approach proposed by Little et al. (2007), which was implemented in the R package 'semTools' (Jorgensen et al., 2018). This method orthogonalizes the moderating term from items in the model comprising the moderation term, which retains model assumptions of independence between independent variable residuals while allowing us to interpret both moderating term as well as direct path coefficients in one model (Little et al., 2007). Moderating effects were modeled by calculating simple slopes at − 1 SD, mean SD, and +1 SD of ICU for significant moderating effects observed in the model; and confidence intervals of moderating effects were estimated using bootstrapping with 1000 resamples.

Finally, we examine the unique effects of CU traits in relation to functional connectivity by controlling for conduct problems as has been demonstrated in previous studies with community samples (e.g., Umbach & Tottenham, 2020) while also running a separate model without conduct problems to ensure we are not capturing a suppression effect (e.g., Hyde et al., 2016; Lozier et al., 2014). Because results of the first analyses were identical to the models that controlled for overlap between conduct problems and CU traits – suggesting no

suppression or impact to model coefficients – we only report on the model that controls for conduct problems.

3. Results

The hypothesized model adequately fit the data without need for further respecification ($X^2(88) = 90.02$, CFI = 0.983, TLI= 0.978, RMSEA= 0.017). Path analysis indicates affective empathy positively associates with SAL within network FC and negatively associates with the DMN-FPN between network FC after adjusting for controlling variables ($\gamma = 5.64$, $p = 0.004$; $\gamma = -7.94$, $p = 0.023$); however, cognitive empathy did not associate with either DMN or FPN within network FC as expected ($p = 0.253$; $p = 0.886$). Thus, we followed up on this by examining zero order correlations which revealed cognitive empathy did associate significantly with DMN FC (r= 0.27, p= 0.012) but not with FPN FC (r= 0.01, p= 0.911). Other results indicated that CU traits negatively associated with both cognitive and affective empathy ($\gamma = -0.272$, $p < 0.001$; $\gamma = -0.431$, $p < 0.001$) and females report higher levels of cognitive empathy than males when adjusting for control variables ($\gamma = -1.984$, $p = 0.041$), but this was not found for affective empathy ($p = 0.064$) (Table 2; Figure 1). Of primary interest, CU traits moderated the association between affective empathy and DMN-FPN between network connectivity ($\gamma = 0.673$, $p = 0.035$) but no other moderations were found. As depicted in Figure 2, adolescents who score higher (1 SD above) on CU traits did not show a significant association between affective empathy and DMN-FPN FC ($\gamma = -0.235$, $p = 0.659$), whereas those who score lower (1 SD below) and average (mean SD) on CU traits do show a negative association between affective empathy and DMN-FPN between network connectivity ($\gamma = -1.630$, $p = 0.001$; $\gamma = -0.933$, $p = 0.011$).

4. Discussion

The current results demonstrate neural correlates of empathy are moderated by the presence of CU traits in adolescents. The association of DMN–FPN between-network connectivity with affective empathy was fundamentally different in the presence of CU traits. Previous research in individuals with higher CU traits shows 1) weaker anticorrelation in DMN–FPN between network connectivity (e.g., Pu et al., 2017; Winters, Sakai, et al., 2021) and 2) weaker associations with affective social processing (for meta-analysis: Waller et al., 2020). Our study extends these previous findings by demonstrating that the relationship between empathy and the brain is different in the presence of CU traits – specifically in the association between DMN–FPN anticorrelation and affective empathy.

Examining DMN-FPN between network FC revealed that larger anticorrelation was associated with greater affective empathy and this association was different as CU traits increased. An anticorrelation between the DMN and FPN is a consistent finding in typical brain function, and deviation in this neural association has significant implications for aberrant neural function (Menon, 2011). Although previously implicated in a multitude of processes (e.g., Hampson et al., 2010; Long et al., 2016; Stern et al., 2012), the DMN-FPN anticorrelation here shows a fundamentally different relationship with affective empathy as CU traits change. Blair et al. (2014) posit that the profound impairment in affective social processing associated with CU traits accounts for persistence in social behavior. The

present finding supports previous results that functional coupling between the DMN-FPN is implicated in affective social processing (e.g., Xin & Lei, 2015) and extend this to identify CU traits as a moderator; thus, it is plausible that connectivity between the DMN-FPN may underlie affective social processing impairments associated with CU traits during affective empathy.

However, our analysis did not find a significant slope for those who were high in CU traits. First, this finding must be interpreted with caution given that only 8 participants (9.5%) qualified for a clinical rating of CU traits based on the established cut off criteria (Kemp et al., 2019), which may limit the necessary power for detecting the anticipated effect. Second, this may reflect a difference amongst those with higher levels of CU traits such that between DMN-FPN connectivity does not account for socio-affective processing impairments. Given that those with higher CU traits started much lower on affective empathy – this may indicate other impairments for those higher in CU traits. One example may be impairments in affective arousal involved in subcortical regions such as the amygdala (for review: DeLisi et al., 2009). A weakening of the DMN-FPN anticorrelation may have some utility in accounting for a lower threshold of affective empathy; however, there may be additional considerations in more extreme levels of CU traits. Future studies would need to confirm the present results with samples that include those at the higher end of CU traits, which may be achieved by sampling forensic samples or oversampling for higher CU traits in the community, as well as those in the normative range of CU traits.

As expected, the SAL within-network connectivity positively associated with affective empathy; but, while cognitive empathy positively associated with DMN in zero-order correlation, FPN did not significantly associate with cognitive empathy. Neural development of affective processing regions of empathy develop much sooner than regions associated with cognitive empathy (Singer, 2006). Although this developmental context may need to be considered in future studies, the present results suggest that the FPN does not associate with cognitive empathy in adolescents.

The present study must be interpreted under some limitations. First this study involved a cross-sectional design, which did not allow us to assert causality. Further examination of this model in a longitudinal design is needed that can improve inferences and parse out developmental effects. Second, we used a predefined atlas for our sample; however, this may improve bias induced by improperly placed ROIs for analysis. Third, a relatively low number of participants were high on CU traits (n=13 or 15% of the sample), which may make inferences for these participants less stable. Future studies would benefit from oversampling from those at higher levels of CU traits. Finally, the present approach accentuated network level associations which may miss out on important individual ROI associations. This was an intentional choice to test specific hypotheses at the network level as opposed to and exploratory characterization of our sample.

Conclusions.

The present results are a novel contribution to the literature on the neural underpinnings of CU traits by demonstrating how CU traits may be a contextual factor for the neural mechanisms underlying affective empathy in adolescents. Specifically, examining functional

connectivity between the DMN-FPN is important for understanding how CU traits changes the association between the brain connectivity and affective empathy. Future studies could improve this line of research by oversampling those who are deemed at a higher level of CU traits, which may reveal more stable inferences on the differences between youth at high CU traits from those who are moderate to low. The differences in adolescent brains between those with high CU traits from those at low to moderate CU traits is an important step for understanding differences in brain connectivity that support empathy at different levels of CU traits. Future investigations of affective social processing impairments associated with CU traits could target connectivity between the DMN and FPN to improve our mechanistic understanding of CU traits.

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Highlights

- Default mode-frontoparietal network anticorrelation underlies more affective empathy
- Callous-unemotional traits moderate the brains association with affective empathy
- Greatest change in brain association at moderate levels of callous-unemotional traits

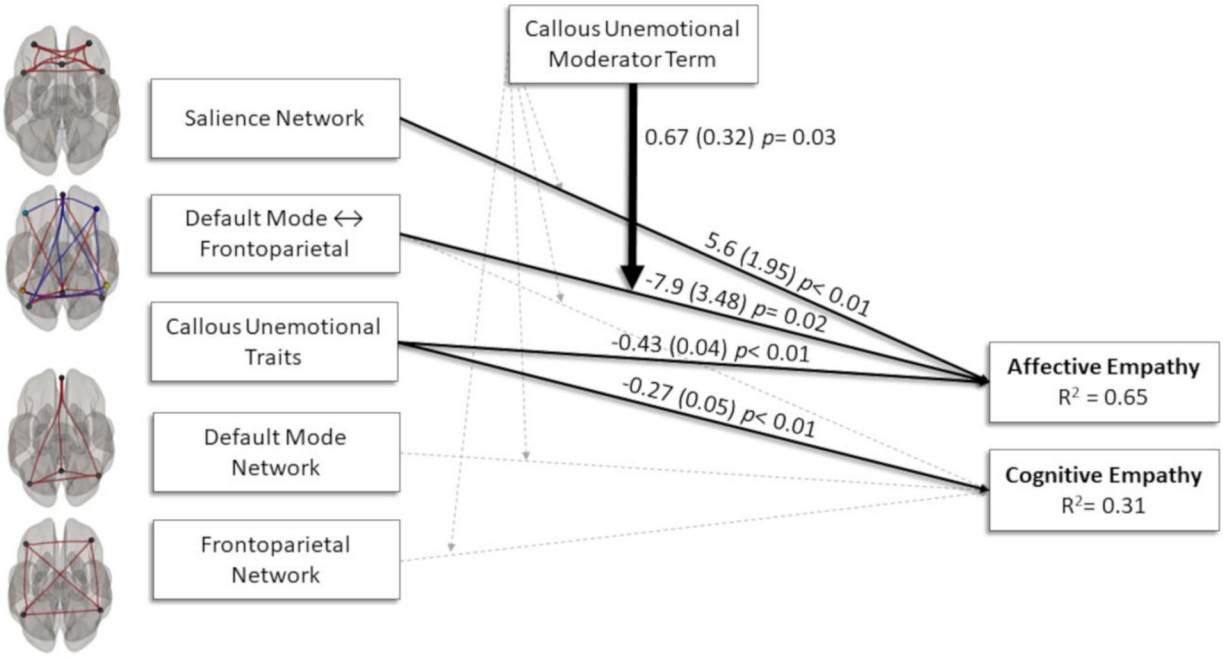

Figure 1.

Figure depicting path analysis model. Significant paths are indicted by solid lines where insignificant paths are denoted by dotted lines. All paths included controls for sex, age, pubertal stage, and externalizing symptoms (not depicted). Moderation term was orthogonalized from the rest of the model by centering the residuals (Little et al., 2007); thus, independence of residuals is not violated, and direct paths are also interpretable.

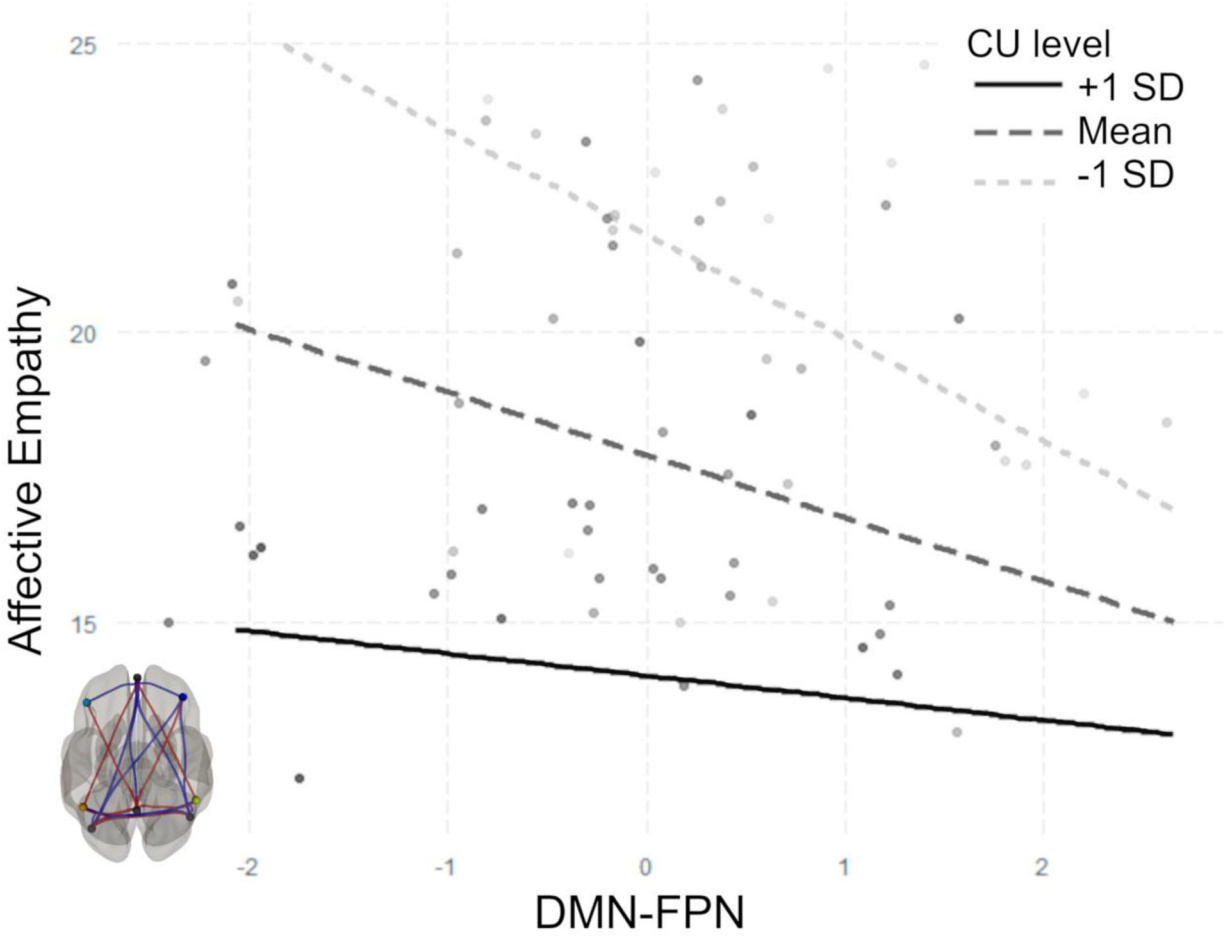

Figure 2.

Figure depicts moderation effect at high (+1SD), medium (Mean), and low (−1SD) callous-unemotional traits of affective empathy regressed on between default mode (DMN) – frontoparietal (FPN) network connectivity.

Table 1.

MNI coordinates for Network ROIs

Network	Region	MNI coordinates (x, y, z)
Default Mode Network	Medial Prefrontal Cortex	1, 55, −3
	Angular Gyrus (L)	−39, −77, 33
	Angular Gyrus (R)	47, −67, 29
	Posterior Cingulate Cortex	1, −61, 38
Salience Network	Anterior Cingulate Cortex	0, 22, 35
	Anterior Insula (L)	−44,13, 1
	Anterior Insula (R)	47, 14, 0
	Rostral Prefrontal Cortex (L)	−32, 45, 27
	Rostral Prefrontal Cortex (R)	32, 46, 27
Frontoparietal Network	Lateral Prefrontal Cortex (L)	−43, 33, 28
	Lateral Prefrontal Cortex (R)	41, 38, 30
	Posterior Parietal Cortex (L)	−46, −58, 49
	Posterior Parietal Cortex (R)	52, −52, 45

Note: (L) = left, (R) = right

Table 2.

Path analysis results

	Unstandardized Estimate	Standard Error	Standardized Estimate	Z score	P value	Bootstrapped 95% CI Lower	Bootstrapped 95% CI Upper
Affective Empathy (R^2 = 0.65)							
CU traits	−0.431*	0.040	−0.738	−10.682	<0.000	−0.436	−0.431
SAL	5.641*	1.950	0.193	2.893	0.004	5.469	5.777
DMN-FPN	−7.941*	3.480	−0.166	−2.282	0.023	−8.529	−8.110
Sex (Male)	−1.314	0.718	−0.124	−1.832	0.067	−1.515	−1.417
Tanner	0.091	0.423	0.017	0.215	0.830	0.160	0.221
Age	−0.004	0.290	−0.001	−0.012	0.990	−0.030	0.005
YSR Externalizing	−0.013	0.065	−0.013	−0.200	0.841	−0.015	−0.006
Moderator CU/ DMN–FPN	0.673*	0.320	0.138	2.105	0.035	0.569	0.618
Moderator CU/SAL	−0.251	0.207	−0.079	−1.210	0.226	−0.201	−0.163
Cognitive Empathy (R^2 = 0.31)							
CU traits	−0.272*	0.054	−0.488	−5.048	0.000	−0.281	−0.274
DMN	3.194	2.794	0.106	1.143	0.253	3.352	3.743
FPN	−0.358	2.495	−0.013	−0.143	0.886	−0.418	−0.049
DMN-FPN	−4.740	4.555	−0.104	−1.040	0.298	−5.034	−4.401
Sex (Male)	−1.984*	0.973	−0.195	−2.039	0.041	−2.159	−2.021
Tanner	0.789	0.575	0.150	1.371	0.170	0.706	0.790
Age	−0.467	0.389	−0.126	−1.201	0.230	−0.448	−0.394
YSR Externalizing	0.023	0.087	0.025	0.267	0.789	0.026	0.038
Moderator CU/ DMN–FPN	0.135	0.385	0.033	0.350	0.726	0.104	0.160
Moderator CU/DMN	−0.094	0.258	−0.033	−0.366	0.714	−0.220	−0.176
Moderator CU/FPN	−0.105	0.438	−0.023	−0.240	0.811	−0.128	−0.064
Significant Moderation Term Slopes							
−1 SD – Low CU	−1.630*	0.536	−0.32	−3.043	0.002	−2.623	−0.467
Mean – Mean CU	−0.933*	0.365	−0.17	−2.558	0.011	−1.696	−0.239
+1 SD – High CU	−0.235	0.533	−0.03	−0.442	0.659	−1.516	0.595

Model fit= $X^2(88) = 90.02$, CFI = 0.983, TLI= 0.978, RMSEA= 0.017

*= $p < 0.05$