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Antisocial behavior is associated with reduced frontoparietal network efficiency in youth

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Abstract

Youth antisocial behavior (AB) is associated with deficits in socioemotional processing, reward and threat processing and executive functioning. These deficits are thought to emerge from differences in neural structure, functioning and connectivity, particularly within the default, salience and frontoparietal networks. However, the relationship between AB and the organization of these networks remains unclear. To address this gap, the current study applied unweighted, undirected graph analyses to resting-state functional magnetic resonance imaging data in a cohort of 161 adolescents (95 female) enriched for exposure to poverty, a risk factor for AB. As prior work indicates that callous-unemotional (CU) traits may moderate the neurocognitive profile of youth AB, we examined CU traits as a moderator. Using multi-informant latent factors, AB was found to be associated with less efficient frontoparietal network topology, a network associated with executive functioning. However, this effect was limited to youth at *low or mean* levels of CU traits, indicating that these neural differences were specific to those high on AB but not CU traits. Neither AB, CU traits nor their interaction was significantly related to default or salience network topologies. Results suggest that AB, specifically, may be linked with shifts in the architecture of the frontoparietal network.

Keywords: youth antisocial behavior; callous-unemotional traits; frontoparietal network; resting-state fMRI; graph analysis

Youth antisocial behavior (AB) encompasses a broad spectrum of behaviors (e.g. aggression and violence) and imposes significant economic, emotional and health burdens to victims and their families, as well as to those engaging in these behaviors, their families and society at large (Rivenbark *et al.*, 2018; Roberts *et al.*, 2018). Moreover, youth AB is a major risk factor for substance use disorders, major depressive episodes, poor physical health outcomes and continuing engagement in AB into adulthood (Laub and Vaillant, 2000; Simonoff *et al.*, 2004; McGue and Iacono, 2005; Colman *et al.*, 2009). Unfortunately, AB during adolescence is quite common (Moffitt, 1993), underscoring the public health priority to understanding the etiology of AB.

Youth AB and neurocognitive functioning

Youth AB has been consistently associated with deficits across several domains of neurocognitive functioning, including socioemotional processing, reward processing and executive functioning (see Blair *et al.*, 2014 for review). For example, youth with high rates of AB have difficulties identifying others' emotions (see Hawes and Dadds, 2012; Tillem *et al.*, 2020; Chang *et al.*, 2021 for reviews), may view ambiguous social cues as threatening (see Martinelli *et al.*, 2018 for review) and show differences in neural activity in the medial prefrontal cortex, precuneus and amygdala during socioemotional processing tasks (Dalwani *et al.*, 2014; Zhou *et al.*, 2016; Dotterer *et al.*, 2017). These youth also perseverate on previously rewarding behavioral patterns (see Estrada *et al.*, 2018 for review) and show blunted neural responses in the anterior insula, anterior cingulate cortex and caudate during reward processing tasks (White *et al.*, 2013, 2014). Finally, youth AB is related to differences in decision-making, sustained attention and response inhibition, particularly when under stress (Fairchild *et al.*, 2009; Hobson *et al.*, 2011; Schoorl *et al.*, 2018), with reduced neural responses in the dorsolateral prefrontal cortex during executive functioning tasks (Rubia *et al.*, 2009; Crowley *et al.*, 2010). Moreover, AB (including symptoms of conduct disorder and oppositional defiant disorder (ODD), as well as lower-level rule breaking and aggression) is part of the externalizing metafactor, which is marked by inhibitory control deficits (Iacono *et al.*, 2008).

Though prior research has identified structural and functional differences in several discrete brain regions related

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to these behavioral differences, work in basic neuroscience has highlighted that complex behaviors are supported by the coordination of large-scale neural networks, rather than activity in single regions of interest (ROIs) (Meehan and Bressler, 2012). Specifically, the default, salience and frontoparietal networks are thought to be critical to understanding neurocognitive functioning and complex behaviors across mental health diagnoses, generally (Menon, 2011), and youth AB, specifically (Cohn *et al.*, 2015). Furthermore, these networks may be of particular importance to youth AB as they are believed to primarily support socioemotional functioning (e.g. interpreting other agents' affective cues; Spreng and Grady, 2010; Jack *et al.*, 2013), salience processing (e.g. fixating on potentially rewarding and/or threatening stimuli; Uddin, 2016) and executive functioning (e.g. response inhibition; Marek and Dosenbach, 2018). Not surprisingly, these three networks contain many of the neural structures previously associated with youth AB, including the medial prefrontal cortex and precuneus (Dalwani *et al.*, 2014; Zhou *et al.*, 2016), the anterior insula and anterior cingulate cortex (White *et al.*, 2013, 2014) and the dorsolateral prefrontal cortex (Rubia *et al.*, 2009; Crowley *et al.*, 2010). However, to properly capture these complex neural networks, it is important to understand the overall organization and functioning of these networks as a whole, rather than simply examining specific structures or subsets of connections within these networks (Reijneveld *et al.*, 2007; Stam and Reijneveld, 2007; Bullmore and Sporns, 2009).

Graph analytic techniques provide a way to examine the higher-level organization of distributed neural networks. By computing the overall organization, or topology, of neural networks, graph analysis can provide quantifiable metrics for the 'optimality' (e.g. efficiency and robustness) of neural information processing throughout a neural network or the brain as a whole. For example, graph analysis can calculate the global efficiency of a network, delineating the resources necessary for information to be communicated and integrated throughout a network. Similarly, graph analysis can provide a clustering metric, which taps the degree to which the functioning of a neural network may be robust to disruptions (e.g. damage or overload; Reijneveld *et al.*, 2007; Stam and Reijneveld, 2007; Bullmore and Sporns, 2009). These neuro-topological features play critical roles in neurocognitive functioning, with more efficient and/or robust neural network topologies supporting positive cognitive outcomes (e.g. higher IQ; Langer *et al.*, 2012; Suprano *et al.*, 2019). Similarly, less efficient or robust network organization has been linked to psychopathology (e.g. schizophrenia, autism; Itahashi *et al.*, 2014; Yang *et al.*, 2020). However, prior research applying graph analytic methods in youth AB has been limited.

Youth AB and neural topology

Thus far, the few existing studies using graph analysis to explore functional neural topology in youth AB have yielded conflicting results. Two case–control studies of conduct disorder, a developmental disorder characterized by persistent engagement in AB during childhood or adolescence, found that conduct disorder was linked with less efficiently organized neural communication throughout the entire brain, but found no differences in global clustering (an indication of functional segregation and robustness; Jiang *et al.*, 2016, 2021). In contrast, a recent study found that, in a large representative cohort, conduct disorder symptomatology was related to enhanced global clustering but was not related to differences in global efficiency (Tillem *et al.*, 2021). Finally, a third case–control study found no relationship between conduct disorder and differences in either global efficiency or global clustering (Lu *et al.*, 2017).

Though these studies suggest that AB, at least as measured narrowly by conduct disorder symptoms, may be related to differences in global neural topology, they are limited in several ways. First, these studies targeted neural topology globally, that is, across all the networks in the brain, even though prior empirical and theoretical work suggest only certain networks are likely to be affected in youth AB. That is, no prior studies have examined the neural topology of the default, salience or frontoparietal networks despite their theoretical and empirical relevance to youth AB (Dalwani *et al.*, 2011, 2014; Cohn *et al.*, 2015; Zhou *et al.*, 2016; Sethi *et al.*, 2018; Waller *et al.*, 2020).

Second, with one exception (Tillem *et al.*, 2021), prior research has exclusively used case–control studies with small samples with conduct disorder. However, evidence continues to accumulate that AB occurs on a continuum ranging from relatively normative levels of rule breaking and defiance to more extreme behaviors such as violence and aggression (Patrick *et al.*, 2002; Krueger *et al.*, 2007). Accordingly, research using dimensional methods that capture the entire spectrum of youth AB is needed, particularly in well-sampled cohorts that have enrichment for risk for AB. These types of samples provide greater generalizability but still contain youth exhibiting a wide range of AB, including some who meet diagnostic criteria for conduct disorder or other AB diagnoses (e.g. ODD).

Finally, there is growing evidence of the importance of callous-unemotional (CU) traits in understanding the etiology of youth AB. CU traits are defined by low empathy and guilt, as well as low or manipulative interpersonal emotions (Frick *et al.*, 2014) and are a specifier for the diagnosis of conduct disorder in the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) (American Psychiatric Association, 2013). Youth high on AB and CU traits engage in significantly more varied and violent AB (Enebrink *et al.*, 2005; Pardini and Fite, 2010), are more likely to continue engaging in AB into adulthood (McMahon *et al.*, 2010; Kahn *et al.*, 2013) and are likely at increased risk to develop psychopathy in adulthood (Frick *et al.*, 2014; Frogner *et al.*, 2016; Viding and McCrory, 2018). Critically, CU traits appear to moderate some neurocognitive correlates of youth AB, including the associations among AB, emotion regulation and amygdala reactivity during socioemotional processing (Viding *et al.*, 2012a, 2012b; Dotterer *et al.*, 2020b). In fact, one of the few studies linking youth conduct disorder to global neural topology found that associations were specific to youth with conduct disorder *and* CU traits (Jiang *et al.*, 2021). Thus, it is important to examine whether any AB-related neural effects are unique to youth AB, CU traits and/or their interaction.

Current study

To address these gaps in the literature, we examined whether dimensional measures of AB, CU traits or their interaction significantly related to differences in the topology of neural communication within the default, salience and/or frontoparietal networks by completing three unweighted, undirected graph analyses, using a proportional thresholding approach. We examined these questions using resting-state functional magnetic resonance imaging (rs-fMRI) data collected from a birth cohort that was over-sampled for low-income, urban families with nonmarital births. This sampling frame increased risk for poverty, which is, unfortunately, a robust risk factor for the development of youth AB (see Bradley and Corwyn, 2002 for review).

Given differences in socioemotional processing, reward and threat processing and executive functioning associated with youth AB, we expected youth AB to be associated with less optimal network topologies for each of the three networks. Specifically, given prior research linking differences in global efficiency and global clustering to neurocognitive functioning, in general (Langer *et al.*, 2012; Suprano *et al.*, 2019), and to youth AB, in particular (Jiang *et al.*, 2016, 2021; Tillem *et al.*, 2021), we hypothesized that youth AB would be associated with lower global efficiency and clustering in all three networks. Though our central focus of this study was on neural correlates of AB, given that CU traits have been associated with abnormal connectivity within the default network (Cohn *et al.*, 2015), we hypothesized that CU traits also would be associated with lower global efficiency and clustering in the default network.

Finally, given the evidence suggesting that the presence of CU traits moderates the impact of youth AB on socioemotional processing and threat detection (Blair *et al.*, 2014), we hypothesized that CU traits would moderate the association between AB and differences in default and salience network topologies. In contrast, since deficits in response inhibition and decision-making may be associated with AB more generally, independent of levels of CU traits (Iacono *et al.*, 2008; Blair *et al.*, 2014), we hypothesized that CU traits would not moderate the impact of youth AB on frontoparietal network topology.

Methods

Participants

The study sample was drawn from 183 adolescents from Detroit, Toledo or Chicago who were part of the Study of Adolescent Neural Development (SAND; Hein *et al.*, 2018; Goetschius *et al.*, 2019; Dotterer *et al.*, 2020b; Goetschius *et al.*, 2020), a substudy of the Future of Families and Child Wellbeing Study (FFCWS; Reichman *et al.*, 2001), which contains multiple measures of context, psychopathology, brain function and biology. The FFCWS is a longitudinal cohort of 4898 (52.4% boys) children sampled from births in 20 large US cities from 1998 to 2000 (Reichman *et al.*, 2001) with an over-sample for nonmarital births (~3:1). Families living in Detroit, Toledo and Chicago were invited to take part in additional data collection at the University of Michigan as part of the SAND when the focused child was 15 years old. The complete list of measures and data for this project is publicly available from the National Institute of Mental Health data archive (https://nda.nih.gov/). The University of Michigan Medical School Institutional Review Board approved this study (UM IRBMED: HUM00074392). All adolescent participants provided written informed assent, and their primary caregivers provided written consent for both themselves and their adolescent children. Of the 183 adolescents in the study sample, 22 participants were excluded due to issues in MRI data quality and/or missing behavioral or demographic data (see Supplementary Table S1), resulting in a final sample of 161 adolescents with complete, available, and high-quaility rs-fMRI data. Of the 161 adolescents included in the final sample, 59% were female, 75.2% were identified as Black/African American, 11.2% were identified as White/European American; 46.6% of families reported annual income below $25 000 (see Supplementary Table S2 for additional demographics).

AB and CU traits

Latent factors for both AB and CU traits were previously generated for the SAND sample using a multi-informant, multimethod approach, allowing us to mitigate reporter-specific and/or method-specific sources of error (e.g. informant bias; see Dotterer *et al.*, 2020b for details). For AB, the latent factor was generated combining indicators from the following measures: (a) parent-reported rule breaking and (b) aggression from the Child Behavior Checklist (Achenbach, 1994), (c) the total score (excluding substance use items) of the youth-reported Self-Report of Delinquency (Elliott *et al.*, 1985) and (d) combined lifetime symptom count (i.e. past/lifetime and present subclinical and clinical threshold symptoms) of the DSM-5 for conduct disorder and ODD (American Psychiatric Association, 2013) on the basis of clinician ratings assessed via a modified version of the Kiddie Schedule for Affective Disorders and Schizophrenia (K-SADS; Kaufman *et al.*, 1997). Thus, the latent factor captured AB as a dimension including broad behaviors from minor rule breaking and defiance to more serious aggression to full clinical symptoms of conduct disorder and ODD. For CU traits, the latent factor was generated combining the following measures: total scores for (a) parent-reported and (b) youth-reported Inventory of Callous-Unemotional Traits (ICU) (Frick *et al.*, 2000; consistent with prior studies, two items were excluded from the total score based on an examination of polychoric inter-item correlations; Waller *et al.*, 2015) and (c) clinician ratings of total lifetime symptom counts (i.e. past/lifetime and present subclinical and clinical threshold symptoms) using the Michigan Addendum to the K-SADS (Walker *et al.*, 2021), which consists of items that are meant to overlap with the recently developed DSM-5 'limited prosocial emotions' specifier (American Psychiatric Association, 2013) derived from the Clinical Assessment of Prosocial Emotions (Frick, 2013) and embedded into the K-SADS interview. Both latent factors were calculated using Confirmatory Factor Analysis in Mplus (Version 7.3; Muthén and Muthén, 2015), with maximum likelihood estimation with robust standard errors (to account for skew and zero-inflation). See Tables 1 and 2 for factor loadings and model fit statistics.

While the current study was designed to examine AB and CU traits dimensionally across a broad continuum of behaviors, it is important to note that participants reported a range of AB and CU traits scores from normative to clinical level. For AB, several participants met diagnostic criteria for conduct disorder (past diagnosis: $n = 13$, 8.1%; current diagnosis: $n = 5$, 3.1%; any CD

Table 1. Antisocial behavior factor loadings and model fit

Variable	Loadings
Factor loadings	
CBCL aggression	0.88
CBCL rule breaking	0.93
SRD total score	0.39
K-SADS ODD/CD symptoms	0.69
Model fit statistics	
Chi-square test of model fit	2.67, $df = 1$, $P = 0.10$
CFI	0.99
TLI	0.96
RMSEA	0.08
SRMR	0.01

Factor loadings and model fit statistics for the antisocial behavior factor previously generated by Dotterer *et al.* (2020b) using a confirmatory factor analysis with a maximum likelihood estimation approach and robust standard errors. CBCL = Child Behavior Checklist; SRD = Self-Report of Delinquency; K-SADS = Kiddie Schedule for Affective Disorders and Schizophrenia; ODD = oppositional defiant disorder; CD = conduct disorder; *df* = degrees of freedom; TLI = Tucker–Lewis index; CFI = comparative fit index; RMSEA = root mean square error of approximation; SRMR = standardized root mean residual.

Table 2. Callous-unemotional traits factor loadings and model fit

Variable	Loadings
Factor loadings	
ICU parent-report total score	0.40
ICU self-report total score	0.33
CAPE/K-SADS limited prosocial emotions symptoms	0.87
Model fit statistics	
Chi-square test of model fit	0, $df = 0$, $P < 0.001$
CFI	1.00
TLI	1.00
RMSEA	0.00
SRMR	0.00

Factor loadings for the callous-unemotional traits factor previously generated by Dotterer *et al.* (2020b) using a confirmatory factor analysis with a maximum likelihood estimation approach and robust standard errors. The model is saturated. ICU = Inventory of Callous-Unemotional Traits; K-SADS = Kiddie Schedule for Affective Disorders and Schizophrenia; CAPE = Clinical Assessment of Prosocial Emotions; df = degrees of freedom; TLI = Tucker–Lewis index; CFI = comparative fit index; RMSEA = root mean square error of approximation; SRMR = standardized root mean residual.

diagnosis: $n = 13$, 8.1%) and ODD [past diagnosis: $n = 12$, 7.5%; current diagnosis: $n = 8$, 5.1%; any ODD diagnosis: $n = 13$, 8.1% ($n = 8$ participants who met criteria for ODD also met criteria for past or present CD)]. For CU traits, some participants did meet diagnostic criteria for the 'with limited prosocial emotion' specifier (past diagnosis: $n = 6$, 3.7%; current diagnosis: $n = 5$, 3.1%; any diagnosis: $n = 6$, 3.7%). Similarly, ICU total scores for several participants fell within the 'clinical' ($n = 9$, 5.6%) and 'at-risk' ($n = 10$, 6.2%) score ranges for community samples (https://faculty.lsu.edu/pfricklab/icu.php).

Imaging procedures and processing

MRI acquisition and preprocessing

MRI image data for the SAND were acquired on a GE Discovery MR750 3 T MRI scanner with an 8-channel head coil. Data acquisition included a T1-weighted structural scan and an 8 min rs-fMRI scan obtained using functional T2*-weighted BOLD images with a gradient echo spiral sequence (TR = 2000 ms, TE = 30 ms, 40 contiguous 3 mm axial slices, flip angle = 90°, FOV = 22 cm, voxel size = 3.44 mm × 3.44 mm × 3 mm) aligned with the AC-PC plane. Resting-state functional images were collected while participants were awake, passively viewing a fixation cross at the end of the scanning session. Slices were acquired contiguously. Images were reconstructed offline using processing steps to remove distortions caused by magnetic field inhomogeneity and other sources of misalignment to the structural data. Standard preprocessing, slice timing, realignment and coregistration to the structural scans, and normalization to MNI 152 space, and a spatial smoothing using a Gaussian kernel (6 mm) was completed in SPM12 using defaults. The top five white matter components were regressed out as well. All brain activity was filtered through a bandpass filter between 0.01 and 0.1 Hz.

Motion correction and denoising

A conservative, multistep procedure was used to correct for motion artifacts combining multiple correction strategies (Parkes *et al.*, 2018). First, 8 min scans were motion scrubbed to identify and remove motion artifacts from the fMRI time series, using a mean frame displacement cutoff value of 0.5 mm (Power *et al.*, 2012). Second, independent component analysis-based Automatic Removal of Motion Artifacts was applied to data at the subject level to remove motion-related artifacts (Pruim *et al.*, 2015a, 2015b).

Brain connectome generation

To produce a whole-brain resting functional connectome, we placed 264 ROIs following the Power *et al.* (2011) atlas. Each ROI consisted of a 3.2 voxel center-to-voxel center radius pseudosphere. Connection strength, measured as the strength of BOLD signal correlation between each of the ROIs, was then calculated for connectome generation. Following the connectome generation, connectivity matrices for each network of interest (i.e. default, salience and frontoparietal networks) were extracted from the whole-brain connectome.

Graph analysis

All graph analyses were completed in Matlab (version 2018b) using a combination of the Brain Connectivity Toolbox (Rubinov and Sporns, 2010) and the MIT graph toolbox (http://strategic.mit.edu/downloads.php?page=matlab_networks. To ensure all graphs were fully connected, a minimum spanning tree analysis using the Kruskal algorithm (Kruskal, 1956) was implemented to generate an initial fully connected subgraph for each network, for each participant. These subgraphs acted as an initial skeletal structure for the main, proportional thresholded graph analysis.

Following this initial subgraph generation, connections were added to each subgraph at proportional thresholds of 0.01 to 0.35 at 0.01 step intervals to generate 35 unweighted, undirected graphs of differing levels of sparsity per network, per participant. Our two graph metrics of interest, efficiency and clustering, were then extracted from each of these thresholded graphs for each participant. To help ensure that our graph metrics accurately reflected neural organization across different levels of sparsity, the area under the curve (AUC) was calculated for each graph metric across sparsity levels (Ginestet *et al.*, 2011; Hosseini *et al.*, 2012), producing one AUC value, per metric, per network, per participant. All AUC graph metrics were winsorized to limit the leverage of outliers on the subsequent regression analyses.

Graph metrics

Efficiency

Efficiency was calculated as the inverse average shortest path length across the graphs. Accordingly, within graphs with higher efficiency, information theoretically travels through fewer connections to get from any node to any other node in the network, allowing for more efficient neural communication and information integration (i.e. communication/integration requiring less time or neural resources; Bullmore and Sporns, 2009).

Clustering

Clustering was calculated as the global fraction of nodes in a graph, which form triangular connections (i.e. the fraction of nodes in a graph whose neighbors are also interconnected with each other). Graphs with higher clustering tend to exhibit higher degrees of functional segregation and may be more robust to disruptions or damage (Bullmore and Sporns, 2009).

Data analysis

Separate linear regression models were run for each of the graph metrics of interest in each of the three network analyses. In each of these regression models, the latent AB factor, the latent CU traits factor and the AB × CU interaction were entered as

simultaneous predictors of interest. Additionally, consistent with prior studies examining these factors in this sample (see also Dotterer et al., 2020b), self-reported gender (dichotomously coded, female *vs* male), self-reported race (a social construct included to control for differences in exposure to discrimination and structural racism and inequality in opportunity; two simultaneous, dichotomously coded variables: Black *vs* Non-Black and White *vs* Non-White), familial income (z-scored) and pubertal development score (z-scored; as measured by the Pubertal Development Scale; Petersen et al., 1988) were included as nuisance regressors in the models (see Supplementary Table S3 for zero-order correlations).

All β-values were Bonferroni corrected within each network analysis separately to control for multiple comparisons. *P*-values reported in the results section reflect the Bonferroni-corrected *P*-values. Any significant moderation effects were decomposed and graphed using the online utility by Preacher et al. (2006) to assess simple slopes and regions of significance. While all results were assessed via Bonferroni-corrected *P*-values, we also generated supplementary Bayes Factors (BF) for any null findings to provide additional information on the strength and confidence of any null results (see Supplementary Materials).

Supplementary analyses

Since prior work in this field has examined the relationship between youth AB at different levels of analysis (e.g. whole-brain graph theory metrics; Jiang et al., 2016, 2021; Tillem et al., 2021), we also ran supplementary, exploratory analyses at the whole-brain and node levels. These analyses can be found in the Supplementary Materials to aide future research and were not part of our focal analyses.

Results

AB was significantly related to lower efficiency in the frontoparietal network (β = –0.25, *P* = 0.024; Table 3). However, the effect of AB was qualified by a significant AB × CU traits interaction (β = 0.28, *P* = 0.028). A region of significance analysis for this moderation effect revealed that the association between lower frontoparietal efficiency and AB was only significant at low or average levels of CU traits (CU traits ≤0.23 s.d. above the mean; see Figure 1 for simple slopes). In contrast, at extremely high levels of CU traits (CU traits ≥3.42 s.d. above the mean), AB was associated with higher efficiency in the frontoparietal network. However, this frontoparietal hyperefficiency should be interpreted with caution as, in the current sample, only three participants had CU trait scores within this region of significance (i.e. CU trait scores ≥3.42 SD above the mean).

In contrast, the main effect of CU traits on efficiency within the frontoparietal network was not significant (β = –0.08, *P* = 0.964). Similarly, neither the main effect of AB (β = 0.15, *P* = 0.222), the main effect of CU traits (β = 0.02, *P* = 1.000), nor the AB × CU interaction (β = –0.19, *P* = 0.186) was significant for clustering in this network.

For both the default and salience networks, there were no significant associations between AB, CU traits, nor their interaction and measures of efficiency or clustering (see Table 3 for full regression results). Moreover, based on the BF findings, the current study provided 'strong' evidence in favor of the null hypothesis for our model examining efficiency in the default network (BF = 0.07) and 'substantial' evidence for the null hypothesis for the remaining models examining the default and salience networks (BFs range from 0.012 to 0.20).

Table 3. Regression results

	β	*t*	*P*	$P_{corrected}$
Frontoparietal network				
Efficiency				
AB	–0.25*	–2.53	0.012	0.024
CU traits	–0.08	–0.71	0.482	0.964
AB × CU traits	0.28*	2.49	0.014	0.028
Clustering				
AB	0.15	1.60	0.111	0.222
CU traits	0.02	0.20	0.840	1.000
AB × CU traits	–0.19	–1.69	0.093	0.186
Default network				
Efficiency				
AB	0.04	0.39	0.701	1.000
CU traits	–0.06	–0.54	0.587	1.000
AB × CU traits	–0.03	–0.22	0.826	1.000
Clustering				
AB	–0.09	–0.97	0.331	0.662
CU traits	0.04	0.40	0.689	1.000
AB × CU traits	0.13	1.17	0.244	0.488
Salience network				
Efficiency				
AB	–0.07	0.68	0.499	0.998
CU traits	0.08	0.67	0.503	1.000
AB × CU traits	–0.08	–0.70	0.485	0.970
Clustering				
AB	–0.01	–0.06	0.954	1.000
CU traits	–0.05	–0.41	0.684	1.000
AB × CU traits	0.11	0.97	0.336	0.672

Results from six linear regression models examining the relationship between network-specific graph analysis metrics (i.e. global efficiency and global clustering) and youth AB, CU traits and the AB × CU traits interaction across three networks (the frontoparietal network, the default network and the salience network). Models controlled for self-reported gender (dichotomously coded, female *vs* male), self-reported race (two dichotomously coded variables: Black *vs* Non-Black and White *vs* Non-White), familial income (z-scored) and pubertal development score (z-scored). Given the correlations among youth AB, CU traits and pubertal status, all models were rerun excluding CU trait and puberty scores from the models to ensure that the current AB findings were not due to suppression effects. Excluding these variables from the models did not meaningfully change any findings..
*$P < 0.05$.

Supplementary results

Full results from the supplementary, exploratory analyses can be found in the Supplementary Materials; however, briefly, neither AB, CU nor their interaction were significantly related to differences in whole-brain efficiency, whole-brain clustering or inter-network communication. The exploratory hubness analysis, however, revealed that, within the default network, CU traits were related to increased hubness in the right-temporal pole (β = 0.491, $P < 0.001$). No other hubness effects were significant.

Discussion

By applying graph analytic techniques to rs-fMRI data collected from a well-sampled community cohort with increased exposure to poverty and thus risk for AB, we found that youth AB was associated with a less efficiently organized frontoparietal network at rest, but this was only true for youth with average or low levels of CU traits. In contrast, neither youth AB nor CU traits were significantly related to differences in the topology of the default or salience networks. Collectively, these findings highlight the specificity of neural network topology alterations to the frontoparietal network and to youth with AB but not high CU traits.

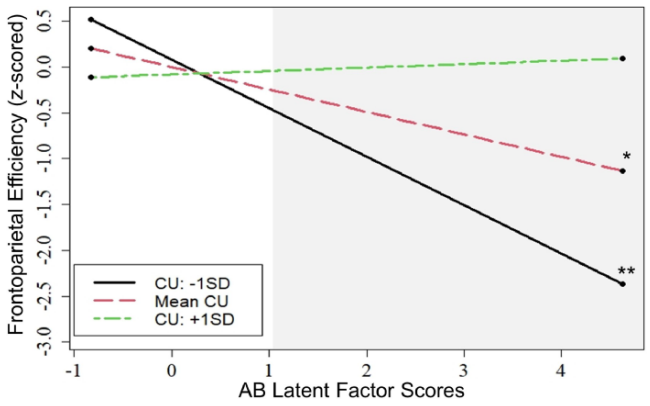

Fig. 1. Antisocial behavior × callous-unemotional traits interaction for frontoparietal efficiency. Simple slopes for the significant AB × CU traits interaction effect on frontoparietal efficiency, controlling for self-reported gender (dichotomously coded, female *vs* male), self-reported race (two dichotomously coded variables: Black *vs* Non-Black and White *vs* Non-White), familial income (z-scored) and pubertal development score (z-scored). The significance of the simple slopes was evaluated via a region of significance analysis. A secondary region of significance analysis was run to evaluate at what levels of AB the main effect of the moderator, CU traits, was significant. The secondary analysis revealed that the main effect of CU traits was significant in youth with AB scores ≥1.05 s.d. above the mean. The shaded region of the figure represents this region of significance. *$P < 0.05$, **$P < 0.01$.

Youth AB and the frontoparietal network

In line with our hypotheses, youth AB was associated with less efficient frontoparietal topology. These network findings are consistent with a prior resting-state connectivity study that linked youth AB to blunted connectivity in frontoparietal regions (Cohn *et al.*, 2015). Based on prior graph theory work in neurotypical populations, lower efficiency in a neural network may slow neural communication and information integration within the network, impeding rapid and cost-effective information processing and impairing associated neurocognitive functions (Reijneveld *et al.*, 2007; Stam and Reijneveld, 2007; Bullmore and Sporns, 2009). The frontoparietal network supports executive functioning, including response inhibition, sustained attention and decision-making (Marek and Dosenbach, 2018). Thus, these findings may help explain why youth who engage in AB have difficulties with inhibitory control and other executive functioning, particularly in time-limited and/or stressful contexts in which resources may be limited or rapid information processing may be critical (Fairchild *et al.*, 2009; Hobson *et al.*, 2011; Schoorl *et al.*, 2018).

Divergent neurocognitive profiles associated with youth AB at differing levels of CU traits

Contrary to our a priori hypotheses, the association between youth AB and lower frontoparietal efficiency was only present 'at low and average' (and not high) levels of CU traits, indicating that this neural profile is specific to youth engaged in AB who do not show relatively higher levels of CU traits. Moreover, in youth with extremely elevated levels of CU traits, AB actually was associated with greater efficiency in the frontoparietal network. Though this greater efficiency of the frontoparietal network should be interpreted with caution given the few participants in our sample at this level of CU traits, this finding, combined with the specificity of lower efficiency in the frontoparietal network for those with low or average levels of CU traits, is inconsistent with the idea that deficits in executive functioning, and the neural processes supporting them, are related to AB generally (i.e. regardless of the CU trait levels; Iacono *et al.*, 2008; Blair *et al.*, 2014).

This pattern of results, however, may not be overly surprising. Recent studies have reported similar interactions between youth AB, CU traits and executive functioning. For example, consistent with the current findings, Dotterer *et al.* (2021) reported that, at low levels of CU traits, youth AB was associated with sustained attention deficits, whereas at higher levels of CU traits, youth AB was associated with improved sustained attention. Similarly, Graziano *et al.* (2019) found that youth with elevated AB and CU traits performed better on standardized measures of executive functioning than youth with AB without CU traits. Although not conclusive, these prior studies, combined with the current findings, suggest that the presence of CU traits may moderate both behavioral and neural executive functioning deficits in youth AB.

Youth AB, the default network and the salience network

Counter to our a priori hypotheses, we did not find any evidence that youth AB was related to altered network topology in either the default or salience networks. Though some studies have linked youth AB to differences in functioning or connectivity in regions within these networks (Dalwani *et al.*, 2014; Zhou *et al.*, 2016; Sethi *et al.*, 2018), it may be that AB-related differences are related to activation in or connectivity between very specific regions, and not to the broader pattern of connectivity within these networks. Alternatively, the current null findings may be due to the study's sample size; however, the BF findings suggest that we have relatively substantial evidence supporting our null results. Similarly, it is possible that the specific level of analysis we examined (i.e. the network-level) simply may not capture the types of disruptions that are present in these networks (e.g. differences in node-level hubness, Jiang *et al.*, 2016; or inter-network communication, Dotterer *et al.*, 2020a); however, our exploratory node-level and whole-brain analyses found no evidence that youth AB was linked with differences at these different levels of analysis (see Supplementary Materials).

Limitations

While the current findings provide evidence that youth AB is associated with altered frontoparietal topology dimensionally in a unique, enriched community sample, they must be considered in light of limitations. First, the current study was limited to examining resting-state data and AB; therefore, any theorized links between frontoparietal efficiency and executive functioning remain speculative. Although there is increasing evidence that neural topology at rest relates to various aspects of psychopathology, neurocognitive functioning and behavior (Kong *et al.*, 2018; Tillem *et al.*, 2021; Chan *et al.*, 2022), additional research is needed to link frontoparietal network topology at rest to behavioral measures of executive functioning in youth AB directly. Second, while the cohort who engaged in this study were at higher risk for AB based on their families SES at birth and associated increased risk for exposure to adversity, and we did have several cases that met diagnosable levels of AB ($n = 14$) and CU traits ($n = 6$), most of the sample did not meet diagnostic criteria, making it impossible to do a case–control comparison. Thus, our findings may not generalize to the more extreme levels of AB and CU traits found in clinical or forensic samples. Third, while the sample size of the current study is larger than previous case–control studies examining youth AB and neural topology, it is currently underpowered to explore more complex models with additional moderators (e.g.

examining three-way interactions between AB × CU traits × gender). Fourth, the current study is cross-sectional, limiting our ability to examine whether these neural correlates are causes or consequences of AB. Finally, future directions in this dataset and others are needed to identify the complex etiology of individual differences in network topology that may give rise to AB (e.g. examining experiences that may impact these circuits during development).

Conclusions

Youth AB was associated with alterations in the topology of the frontoparietal network; however, those alterations are dependent upon an individual's level of CU traits. Specifically, in youth with low or average levels of CU traits, youth AB was associated with reduced efficiency in the topology of the frontoparietal, but not the salience or default, network at rest. The specificity of these findings (1) suggest that differences in the frontoparietal network are related to AB but not CU traits, (2) indicate that network topology differences in youth AB may be specific to the frontoparietal network, which supports executive functioning and (3) add to a growing body of literature showing that the presence of CU traits may moderate executive functioning deficits in youth AB.

Supplementary data

Supplementary data is available at *SCAN* online.

Data availability

Data from the Study of Adolescent Neurodevelopment (https://nda.nih.gov/edit_collection.html?id=2106) and the Fragile Families and Child Wellbeing Study (https://opr.princeton.edu/archive/) are publicly available. The fMRI data are not publicly posted as this was not supported by the National Institutes of Health at the time of data collection.

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Conflict of interest

The authors declared that they had no conflict of interest with respect to their authorship or the publication of this article.

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