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Towards understanding neurocognitive mechanisms of parenting: Maternal behaviors and structural brain network organization in late childhood

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Abstract

A substantial body of knowledge suggests that exposure to adverse family environments - including violence and neglect - influences many aspects of brain development. Relatively less attention has been directed toward the influence of "normative" differences in parenting behaviors. Given the rapid brain reorganization during late childhood, parenting behaviors are particularly likely to impact the structure of the brain during this time. This study investigated associations between maternal parenting behaviors and the organization of structural brain networks in late childhood, as measured by structural covariance. One hundred and forty-five typically developing 8-year-olds and their mothers completed questionnaire measures and two observed interaction tasks; magnetic resonance imaging (MRI) scans were obtained from the children. Measures of maternal negative, positive, and communicative behavior were derived from the interaction tasks. Structural covariance networks based on partial correlations between cortical thickness estimates were constructed and estimates of modularity were obtained using graph theoretical analysis. High levels of negative maternal behavior were associated with low modularity. Minimal support was found for an association between positive maternal behaviors and modularity and between maternal communicative behaviors and modularity. Our findings suggest that variation in negative maternal behavior is associated with the structural organization of brain networks in children.

KEYWORDS

cortical thickness, environment, graph theory, magnetic resonance imaging, modularity, neurodevelopmental outcomes

Marc L. Seal and Sarah Whittle are senior authors.

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1 | INTRODUCTION

The influence of environmental factors on the developing brain is likely to be strongest during sensitive periods when neuronal properties are particularly receptive to acquiring certain kinds of information and susceptible to modification by experience (Hensch, 2004). Identifying environmental factors that impact on the brain during these sensitive periods is important in furthering our understanding of what drives neurodevelopmental trajectories to unfold in a particular way and may inform prevention/intervention strategies for optimal brain development (Lenroot et al., 2009). Parenting behaviors represent critical environmental inputs that shape behavioral and emotional development (Phua, Kee, & Meaney, 2019; Schwartz et al., 2016; Whittle, Yap, et al., 2011; Wu & Lee, 2020; Yap & Jorm, 2015) however the underlying mechanisms are unclear. Parenting behaviors are likely to be particularly important during late childhood (from approximately 8 years), as it is an important transition period with unique neurodevelopmental patterns associated with the development of internalizing and externalizing symptoms (Papachristou æ Flouri, 2019; Whittle, Vijayakumar, Simmons, & Allen, 2020) and this occurs prior to the adolescent period when the influence of peers becomes more prominent (Lamblin, Murawski, Whittle, & Fornito, 2017). Given that childhood is a time of intensive brain reorganization involving synaptic pruning, cortical thinning, and white matter organization (Petaniek et al., 2011; Vértes & Bullmore, 2015; Walhovd, Fjell, Giedd, Dale, & Brown, 2017), it is likely to be a sensitive period where brain development may be shaped by parenting behaviors.

Although the study of adverse family environments, generally involving child maltreatment, on brain development has amassed a substantial body of evidence, relatively little attention has been given to the influence of "normative" differences in parenting behaviors (Belsky & de Haan, 2011; Farber, Gee, & Hariri, 2020). Positive (warm and supportive) parenting has been found to enhance child functioning (Landry, Smith, Swank, Assel, & Vellet, 2001; Landry, Smith, Swank, & Guttentag, 2008; Phua et al., 2019) and has been associated with changes in brain structure, including accelerated cortical thinning in a number of regions (Luby, Belden, Harms, Tillman, & Barch, 2016; Whittle, Simmons, et al., 2014). Negative (e.g., controlling, aggressive, and hostile) parenting behavior, in contrast, has been prospectively linked to increased risk of psychopathology (Ong et al., 2018), and has been associated with attenuated cortical thinning (Schwartz et al., 2016).

Recent research has suggested that looking at patterns of connectivity may reveal additional insights into the relationship between the brain and family environments. Investigations have begun shifting from individual brain regions to whole brain networks, and evidence for associations between childhood maltreatment (evaluated retrospectively) and structural network architecture is emerging (Ohashi et al., 2017; Teicher, Anderson, Ohashi, & Polcari, 2014). Based on structural covariance analysis of cortical thickness (CT), which provides a complementary source of neurodevelopmental information compared to structural networks based on underlying fiber/tract connections (Gong, He, Chen, & Evans, 2012), our group recently demonstrated that high levels of negative affective maternal behaviors were associated with decreased local network efficiency in children, whereas high levels of positive affective maternal behaviors were associated with increased local efficiency (Richmond, Beare, et al., 2019). No associations were found with global efficiency. These findings suggest that parenting practices may impact the *integration* of brain network organization, influencing the ability of the brain to transmit information across distributed regions. Given that local efficiency has been shown to decrease during late childhood (prior to increasing in adolescence; Khundrakpam, Reid, et al., 2013), we speculated that reduced local efficiency associated with less optimal parenting may reflect an accelerated pattern of brain maturation, which may be an adaptive mechanism.

In the present study, we applied a network neuroscience perspective to investigate associations between parenting behaviors and *segregation* of structural covariance networks (which reflects the degree to which specialized processing occurs within densely interconnected groups of brain regions) in late childhood. Specifically, we build on our previous investigation into the efficiency of childhood structural brain networks (Richmond, Beare, et al., 2019) and a limited number of existing studies (Khundrakpam, Lewis, Jeon, et al., 2019; Khundrakpam, Lewis, Zhao, Chouinard-Decorte, & Evans, 2016), by focusing on identifying communities or modules within networks to complement and gain a fuller picture of parenting effects. Modularity was chosen because (a) it is biologically meaningful and may offer insight into the major building blocks of networks, and (b) has been investigated across development and has been demonstrated to change with age.

Modules may offer insight into the major building blocks of networks and increase our understanding of their functional role, and therefore the detection of distinct network modules is biologically relevant (Sporns, 2018; Sporns & Betzel, 2016). In brain networks, modules typically correspond to clusters of nodes that are densely interconnected and sparsely connected to the rest of the network (Sporns & Betzel, 2016). Modules are thought to have been shaped by evolutionary constraints to allow the brain to communicate efficiently with low wiring cost and enable functional specialization and complex brain dynamics (for review see Sporns & Betzel, 2016). Developmental studies that have investigated modularity of structural covariance networks suggest that non-random modular organization is detected from infancy to young-adulthood (Alexander-Bloch, Raznahan, Bullmore, & Giedd, 2013; Fan et al., 2011), and there is evidence that modularity changes with age, with a significant decrease in late childhood, similarly to local efficiency (8-11-year-olds; Khundrakpam, Reid, et al., 2013). More recently, higher modularity has been associated with higher verbal but not performance intelligence quotient in children and adolescents (Khundrakpam, Lewis, Reid, et al., 2016).

The aim of this study was to investigate the relationship between parenting behavior and the organization of structural brain networks, constructed by structural covariance of cortical thickness in late childhood. Based on our previous findings, we might anticipate that, similarly to local efficiency, more optimal parenting behaviors would be linked to reduced modularity. However, given modularity enables networks to communicate efficiently and enables functional specialization, it is possible that parenting may have divergent associations with network integration versus specialization, and as such we broadly hypothesized that positive parenting behaviors would be associated with increased modularity and negative parenting behaviors with decreased modularity.

2 | MATERIALS AND METHOD

2.1 | Participants and recruitment

The data included in this study came the Families and Childhood Transitions Study (FACTS) conducted at The University of Melbourne, Australia. The Human Research Ethics Committee at The University of Melbourne approved the research. Written informed consent was obtained from each child and a parent/guardian. Abbreviated summaries relevant to the measures used in this analysis are provided below and were previously described in Richmond, Beare, et al. (2019). Further details can be sourced in the study protocol (Simmons et al., 2017). Participating dyads comprised 145, 8-year-old typicallydeveloping children and their mothers (Table 1).

Although not applicable to all families, socioeconomic disadvantage has been associated with suboptimal parenting practices (Newland, Crnic, Cox, & Mills-Koonce, 2013; Pereira, Negrão, Soares, & Mesman, 2013). To avoid recruiting a sample biased for high socioeconomic advantage and subsequent low variation in negative and positive parenting behaviors, participant recruitment focused on suburbs of Melbourne that scored within the lowest tertile on the

TABLE 1 Participant Demographics (N = 145)

Characteristic	M (SD) or n (%)
Child age, M(SD), years	8.42 (0.33)
Males, No. (%)	68 (46.90)
CDI-2, M(SD) ^a	8.32 (6.07) T-Score 55, "Average or Lower"
SCAS, M(SD) ^b	26.27 (13.07) T-Score 52, "Normal"
LITE, M(SD) ^c	3.82 (2.33)
Child ethnicity	
Caucasian, No. (%)	102 (71.03)
Other, No. (%)	30 (20.70)
Maternal age, M(SD), years	40.25 (5.5)
Maternal Occupational Status, M(SD) ^d	62.38 (19.94)

^aImputed data, The Children's Depression Inventory 2, maximum T-Score for boys and girls 7–12 years (Kovacs, 2011).

^bImputed data, The Spence Children's Anxiety Scale maximum T-Score for boy and girls aged 8–11 years (Spence, 1998).

^cLifetime Incidence Traumatic Events, *n* = 143 (Greenwald & Rubin, 1999). ^dSocioeconomic Index 2006 (AUSEI06), *n* = 138 (McMillan, Beavis, & Jones, 2009). Socioeconomic Indexes for Areas scale of advantage and disadvantage (Australian Bureau of Statistics, 2013).

Eight-year-old typically developing children and their mothers were invited to participate in the study. Participation was not restricted to families with biological mothers and one mother not biologically related to her child participated (0.69% of sample). Families who indicated they wished to participate were contacted for a brief telephone interview to assess the exclusion criteria, which included significant motor or sensory impairments, and criteria related to having a Magnetic Resonance Imaging (MRI) scan (Simmons et al., 2017).

2.2 | Procedure

Children and their mothers completed an assessment and videotaped family interactions. Children only completed an MRI scan. Mothers completed an interview comprising questions about the children's demographics, health, and developmental histories.

2.3 | Questionnaire Measures

The Children's Depression Inventory 2 (CDI-2; Kovacs, 2011) is a 28 item self-report measure, based upon the previous two weeks, of cognitive, affective, and behavioral signs of depression in children and adolescents aged 7–17 years. The CDI-2 yields a Total score, two scale scores (Emotional Problems, Functional Problems) and four sub-scale scores (Negative Mood/Physical Symptoms, Negative Self-Esteem, Interpersonal Problems, Ineffectiveness). Total and scale scores were examined. The CDI-2 has normative data in the relevant age range and reliability and validity evidence across community and clinical populations (Kovacs, 2011).

The Spence Children's Anxiety Scale (SCAS; Spence, 1998) is a 44 item, self-report measure of anxiety symptoms for children aged from 8 to 15 years. Participants rate the degree to which they have experienced an event, on a four-point scale, ranging from *never* to *always*. The SCAS yields a Total score and six sub-scale scores: Obsessive-compulsive Problems, Separation Anxiety, Social Phobia, Panic/Agoraphobia, Generalized Anxiety Symptoms, and Concerns of Physical injury (Spence, 1998). Total score was examined. The SCAS has been identified as a reliable and valid measure across diverse childhood populations (Essau, Muris, & Ederer, 2002; Holly, Little, Pina, & Caterino, 2014).

The Lifetime Incidence of Traumatic Events (LITE; Greenwald & Rubin, 1999) is a 16-item parent-report screening instrument which assesses the type of loss or trauma a child has experienced. The LITE has good reliability and adequate validity (Greenwald & Rubin, 1999). At the request of The University of Melbourne Ethics Committee, two items on sexual abuse were removed and items covering mother-child separations and domestic relocation were added. The LITE parent report has no standardized scoring system and was scored by summing the number of endorsed items (Greenwald & Rubin, 1999).

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The Australian Socioeconomic Index 2006 (AUSEI06) was used to assess maternal occupational status (McMillan et al., 2009). The AUSEI06 is based upon 2006 Australian Census data and is a continuous measure of occupational status, ranging from 0, low status, to 100, high status (McMillan et al., 2009).

2.4 | Family interaction assessment and measures

Mother-child dyads completed two 15-min laboratory-based interaction tasks, which were video recorded for subsequent coding using a modified version of The Family Interaction Macro-coding system (FIMS, see supplemental information; Holmbeck, Zebracki, Johnson, Belvedere, & Hommeyer, 2007). First, dyads were asked to plan enjoyable activities together, such as vacations or birthday parties (event-planning interaction [EPI]; MacPhillamy & Lewinsohn, 1982). The EPI was followed by a problem-solving interaction (PSI), where the dyads were asked to discuss and try to resolve areas of conflict chosen (Gilboa & Revelle, 1994; Prinz, Foster, Kent, & O'Leary, 1979). The EPI and PSI tasks were intended to differentially elicit positive and negative behaviors, respectively.

To identify different aspects of maternal behavior, an exploratory principal components analysis was conducted using the FIMS mother-child data (Richmond, Schwartz, et al., 2018). The PCA was run for 155 mother-child dvads (10 of whom did not have MRI data) to obtain composite maternal parenting behavior scores. A four-factor solution explained a total of 56.76% of the variance, with components comprising (1) Negativity EPI - negative maternal behaviors during the EPI, such as negative and aggressive affect; (2) Warmth - codes related to positive affect, such as humor and warmth: (3) Negativity PSI - negative maternal behavior during the PSI, such as negative and aggressive affect; and (4) Communication - codes related to listening, structuring dialogue, and clarity of thought (see Tables S1 and S2). Participant scores for each parenting component were estimated (Harman, 1976) and divided into tertiles: low-, moderate-, and high-. Structural covariance analysis is based on correlations and therefore requires groups for analysis. That is, networks are produced for a group of participants (rather than for each individual).

2.5 | MRI acquisition and processing

Before the MRI procedure, children completed a mock scan in a replica MRI to minimize the likelihood of movement artefact and participant anxiety. Neuroimaging data were acquired on the 3T Siemens TIM Trio scanner at the Murdoch Children's Research Institute, Royal Children's Hospital, Melbourne. Participants were positioned supine with their head supported in a 32-channel head coil. T_1 - weighted images were acquired during a 5:19 min sequence (MPRAGE: repetition time = 2,530 ms; multiple echo times = 1.74; 3.6; 5.5; 7.3 ms; flip angle = 7°, field of view = 256 × 256 mm²) and produced 176 contiguous 1.0 mm thick slices (voxel dimensions = 1.0 mm³). A radiographer inspected image quality at the time of acquisition and if movement artefact was detected the sequence was repeated with the participant's consent.

2.6 | Structural image processing

Cortical surfaces and thickness were generated by FreeSurfer (Version 5.3; Fischl, 2012) from the T_1 -weighted images. The processing steps have been described in detail elsewhere (Dale, Fischl, & Sereno, 1999; Fischl, Sereno, & Dale, 1999). All T_1 -weighted images were subject to a manual quality assessment procedure which involved visual inspection of all image slices per participant. Manual edits were made where cortical surfaces were under- or overestimated on four or more image slices. In short, editing comprised correcting errors related to the standard FreeSurfer pipeline: (1) skull stripping; (2) white matter intensity normalization; and (3) topological errors, for example, white matter and pial (Waters, Mace, Sawyer, & Gansler, 2019). Of the 153 acquired scans, manual edits were made to 55 and one was excluded due to excessive motion.

2.7 | Missing data

There was no missing family interaction data. For CDI-2 and SCAS data, 19% and 18% of participants, respectively, had missing data on at least one item. The mechanism for missing data was investigated for both questionnaire measures (CDI-2 and SCAS; see Appendix S1 for details). To predict missing values, multiple imputation was carried out at the item level; five imputed data sets were generated, and pooled results were reported (Enders. 2010: van Buuren & Groothuis-Oudshoorn, 2011). Each imputed data set was visually inspected per CDI-2 and SCAS variable for errors related to the imputation model, no errors were found (e.g., extreme outliers, refer to Figures S1 and S2; White, Royston, & Wood, 2011). For SCAS data, one participant did not complete any items and was removed. For the LITE data, two participants did not complete any items and were removed. Missing items on the LITE were not imputed as the measure is a screen only, missing items were assumed as not endorsed. Similarly, missing maternal occupational data (n = 7) was not imputed.

2.8 | Structural covariance network: Node and edge definition

The structural covariance networks, previously described in Richmond, Beare, et al. (2019), were generated as follows. Network nodes (74 per hemisphere) were defined by the FreeSurfer parcellation of the cortical gray matter into regions in accordance with the Destrieux atlas (Destrieux, Fischl, Dale, & Halgren, 2010). Network edges were defined by partial correlations of cortical thickness between pairs of nodes (Alexander-Bloch, Raznahan, et al., 2013). A sparse partial correlation estimation procedure was applied to identify significant, nonzero partial correlations (Lasso, least absolute shrinkage and selection

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operator; Tibshirani, 1996, see Appendix S1 for details). Sparse inverse covariance estimates have been applied recently to characterize functional and structural networks in clinical populations and to identify statistically significant group differences (Lefort-Besnard et al., 2018). A regularization parameter for the sparse estimation procedure was selected by cross-validation based on the data for all participants and applied to all groups. All networks were analyzed as binary and undirected, which assumes the edges have no orientation (Sporns, 2012).

2.9 | Structural covariance network analysis

2.9.1 | Parenting component characteristics

To establish whether there were any variables confounding associations between parenting and brain network parameters, we investigated between-group differences for the low-, moderate, and highgroups for each of the four parenting components for the following six variables using ANOVA: child age, sex, incidence of traumatic events, and maternal occupational status. We applied an FDR (5%) to adjust for the multiple comparisons across the four parenting components (i.e., 24 in total).

2.9.2 | Network parameters

The binarized graphs were used to calculate modularity and related parameters (all modularity related findings are unique). As the equations for these graph metrics are defined elsewhere only brief definitions are provided.

2.9.3 | Modularity

Modules were detected using modularity maximization, which partitions a network's nodes into communities to maximize the modularity function, *Q*, using the Louvain heuristic (Betzel & Bassett, 2016; Blondel, Guillaume, Lambiotte, & Lefebvre, 2008). Modularity maximization is subject to biases and limitations which we endeavored to address by considering the resolution limit and applying consensus clustering (Sporns & Betzel, 2016).

2.9.4 | Resolution Limit

Under certain conditions modularity maximization is unable to detect modules below a certain size even if those communities are otherwise well-defined: this problem is known as the resolution limit of modularity (Fortunato & Hric, 2016). In a practical sense, this implies that a network that is maximized for *Q* could potentially be divided further, into smaller, better-defined communities (Sporns & Betzel, 2016). To address the resolution limit, we used the Louvain algorithm with a multiresolution technique incorporated into the measure (Reichardt & Bornholdt, 2006).

To investigate the influence of the resolution parameter, it was varied from 0.5 to 1.5, in increments of 0.05. We obtained the mean modularity for the empirical and randomized networks, $Q_{empirical}$ and Q_{random} . For the modularity analysis, we chose the scale at which the quality of the empirical partitions exceeded that of the random partitions by the greatest amount (Sporns & Betzel, 2016). The comparison was carried out for the network of the whole group and 20 random networks, matched on the degree of the empirical network.

2.9.5 | Consensus clustering

To improve the stability of the assignment of nodes to communities, we ran the Louvain algorithm (at the scale selected for the resolution limit) 100 times to identify average or consensus communities for each network (Blondel et al., 2008; Sporns & Betzel, 2016).

2.9.6 | Differences in modular organization

To determine if differences in the modular organization existed between the three networks (low-, moderate -, and high-) for each parenting component, we calculated the normalized mutual information for the three pairwise comparisons (NMI; Alexander-Bloch, Lambiotte, et al., 2012; Kuncheva & Hadjitodorov, 2004). The NMI ranges from 0 to 1, when the NMI is close to zero, the two partitions (the assignments of nodes to modules) are independent and when it approaches one, the two partitions are identical. We applied an FDR of 5% to adjust for multiple comparisons.

2.9.7 | Differences in modular segregation

We applied a similar approach to quantifying segregation as outlined by Baum et al. (2017). Two measures were considered, the participation coefficient and the modularity quality index, *Q*. The participation coefficient, which quantifies the diversity of a brain's region's connection across modules and has been described as a measure of intermodular connectivity, was the primary measure (Guimerà & Amaral, 2005). A high participation coefficient indicates regions with strong connections to many modules, and conversely a low participation coefficient indicates regions with strong connections to few modules. Subsequently, networks with high participation coefficients tend to display less segregation between modules than networks with low participation coefficients (Baum et al., 2017).

We quantified the segregation of specific modules, by averaging Pi across brain regions (nodes) assigned to the same modules. Global network segregation was quantified by averaging Pi across all the nodes in the network.

The modularity quality index (*Q*) was also calculated for all networks as a secondary measure of segregation. The index *Q*, measures 1850 WILEY-

how well a given network partition maximizes the strength of withinmodule connections relative to a specified null model, and therefore, *Q*, increases in more segregated networks (Baum et al., 2017). We applied an FDR of 5% to adjust for multiple comparisons.

2.9.8 | Intra-modular connectivity

Intra-modular connectivity was assessed by the normalized intramodular degree (Z_i), which is a measure of the connectivity from a given vertex to other vertices in the same module/community (Guimerà & Amaral, 2005). We classified nodes with Z_i greater than 1 as hubs.

2.9.9 | Network parameter differences between parenting groups

To determine if differences in modularity existed between the three parenting Groups (low-, moderate-, high-) for each of the four components (Negativity EPI, Warmth, Negativity PSI, Communication) a nonparametric permutation test procedure was carried out (Bullmore, Suckling, et al., 1999; He, Chen, & Evans, 2007). First, for each parenting component, the networks properties were calculated for each group using the whole group regularization parameter. Next, to test the null hypothesis that differences between the low-, moderate-, and high- groups might occur by chance, participants were randomly allocated to one of three groups and networks were constructed per the sparse partial correlation estimation procedure detailed previously. Next, for each network property, the absolute minimum difference for the three pairwise comparisons was determined. The random allocation procedure was repeated 5,000 times (per network property) and the 95 percentile points for each distribution were used as the critical values for a two-tailed test of the null hypothesis with a probability of type 1 error of 0.05.

In addition, difference between the distribution of participation coefficients across the regions (nodes) was investigated using the Kolmogorov–Smirnov two-sample test, which tests the hypothesis that two independent samples have been drawn for the same sample (Scheff, 2016).

Key analysis resources are listed in Table S5. Raw data is available by contacting the corresponding author.

3 | RESULTS

3.1 | Parenting group characteristics

The descriptive statistics for the low-, moderate, and high- groups of each parenting component are listed in Table 2 and the distributions presented graphically in Figures S3–S6. Correlations between the parenting components are presented in Table S3. For each parenting component (Negativity PSI, Warmth, Negativity EPI, and Communication)

l.06 (1.21) Mean (SD) Range 5.76 Чах 5.61 -0.16 Ξi High-64 z -0.33 (0.09) Mean (SD) Range 0.31 -0.16 Jax -0.47 Ξi Moderate 48 z -0.64 (0.23) Mean (SD) Range 1.45-0.48 Max -1.93Ξi Lo < 49 z Kurtosis 7.73 2.44 Skew Parenting component Negativity EPI

Group information from family interaction macro-coding system principal components analysis (N = 145)

2

TABLE

1.03 (0.65) 1.20 (0.74)

2.79 3.00 1.32

3.13 3.38

0.34

48 48 48

-0.09 (0.23)

0.73 1.02 0.50

0.29 0.34 0.52

-0.44

48 48

-1.00 (0.55) -0.96 (0.14) -1.11 (0.99)

3.11 0.71 4.25

-0.45 -0.68 -0.01

-3.56 -1.39 -4.24

49 49 49

1.28

0.24 0.97

0.27 3.19

-1.59

Communication

PSI

Negativity

Warmth

-0.68

0.02

48

0.38

0.85 (0.29)

1.85

0.53

-0.24 (0.29) 0.31 (0.14) no significant between-group differences were found for the low-, moderate-, and high- groups for child age, sex, depression symptoms, anxiety symptoms, incidence of traumatic events, or maternal occupational status (FDR 5%; Table S4). As such, these variables were not incorporated into the analysis and partial correlations of cortical thickness were used to generate the networks. Ethnicity was not included as a covariate because the majority of children were Caucasian (Table 1).

3.2 | Resolution parameter

The empirical partitions exceeded the random partitions by the greatest amount for $\gamma = 1.0$ (see Figure S7), and therefore, community structure was reported for a resolution parameter of one. A resolution parameter of one indicates there is no failure of the modularity optimization to resolve small modules. It is plausible given the resolution limit is more problematic for very large networks, that the resolution parameter of one is due to the relatively small size of our networks which is related to the size of the Destrieux parcellation (148 nodes; Fortunato & Hric, 2016).

3.3 | Structural network modules for whole group

Prior to investigating group differences in modular structure, we constructed a structural covariance network containing all participants to provide a baseline. The density of the baseline network, calculated as the ratio of the number of edges to the number of possible edges was 0.123 (Bullmore & Sporns, 2009). Five modules were identified, from which hub nodes ($Z_i > 1$: defined above) were identified (see Figure 1 and Table 5 for details). Module 1 was approximately symmetrical with two hubs located in the left anterior transverse temporal gyrus and left pericallosal sulcus, participation coefficients ranged from .627 to .720. Module 2 was the largest module with 50 nodes and approximately symmetrical. Module 2 had eight hub nodes, located in the frontal gyrus and sulcus and anterior cingulate gyrus, participation coefficients ranged from .403 to .538. Module 3 had six nodes and did not contain any hubs. Module 4 had six hub nodes, five of which were located in the left hemisphere; all hubs were located around the temporal and parietal lobes and participation coefficients ranged from .496 to .631. Module 5 had seven hub nodes, the majority located within the occipital lobe, participation coefficients ranged from .349 to .650. The average participation coefficient for the baseline network was .561 (SD .135). For the hub nodes, the correlation between the participation coefficients and the intra-modular connectivity (normalized intra-modular degree, Z_i) was .26, p > .05.

3.4 | Network parameter differences between parenting groups

Comparative analyses (nonparametric permutation tests) of modular organization (NMI) and segregation (participation coefficient and

modularity quality index) were performed for each of the four parenting components (Negativity EPI, Warmth, Negativity PSI, and Communication) between the three groups (low-, moderate-, and high-; Bullmore & Sporns, 2009; He, Chen, & Evans, 2008).

3.4.1 | Parenting and modular organization

Given that an NMI of 1.0 indicates identical modular structure, the NMI comparisons within each parenting component (maximum NMI .161, see Table 3) suggest the nodal compositions of the modules for the low-, moderate- and high- groups were dissimilar. The nonparametric permutation tests revealed that for four of the comparisons, for example the modules for the Low Warmth group compared to the High Warmth group were more similar than expected if random groups were used; eight comparisons were comparable to the result for random groups (see Table 3). These results were further investigated through a visual inspection check of the nodal composition. refer to the Appendix S1, Figure S8 for further details. As an example, the NMI comparisons for the low-, moderate- and high-Negativity EPI groups were all low, that is, less than or equal to 0.10. The inspection of the modules for the groups demonstrated a limited number of common nodes across modules (e.g., for the "frontal" module the following nodes were common: the left cuneus, the right anterior part of the cingulate gyrus and sulcus [ACC], the right triangular part of the inferior frontal gyrus, and the right Superior frontal gyrus), however often instead of containing the same nodes, the modules contained different but spatially adjacent nodes. To illustrate the low- Negativity EPI module contained node 5 (Transverse frontopolar gyri and sulci), the moderate-Negativity EPI module contained node 1 (Fronto-marginal gyrus [of Wernicke] and sulcus), and the high- Negativity EPI contain nodes 1 and 5 where the parcellations for nodes 1 and 5 are adjacent to each other (Destrieux et al., 2010).

3.4.2 | Parenting and global participation coefficient

Group comparisons for the participation coefficient are discussed below (see Table 4 for complete listing). For Negativity EPI, the moderate group had increased mean PC compared to the low group. For Negativity PSI, the moderate- group had increased mean PC compared to the low group and the high group also had increased mean PC compared to the low group. For Warmth and Communication there were no significant group comparisons (see Figure 2).

3.4.3 | Parenting and modularity quality index

Group comparisons for the modularity quality index, *Q*, are discussed below (see Table 4). For the four parenting components there were no significant group comparisons (see Figure 2).



FIGURE 1 Modular organization for whole group (*N* = 145)

TABLE 3Normalized mutualinformation and Kolmogorov-Smirnovtest for equality of local participationcoefficient distributions

Parenting Component	Low - Moderate	Low - High	Moderate – High
Normalized mutual information			
Negativity EPI	.070	.081	.100
Warmth	.110	.145*	.097
Negativity PSI	.110	.161**	.140*
Communication	.152**	.102	.111
Kolmogorov-Smirnov test			
Negativity EPI	.277***	.142	.196*
Warmth	.189*	.101	.142
Negativity PSI	.514***	.595***	.162
Communication	182 [*]	135	074

Note: FDR (5%) adjusted p-values.

Abbreviations: EPI, event-planning interaction; PSI, problem-solving interaction.

*p < .05;

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<sup>**</sup>p < .01;
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^{***}p < .001.

TABLE 4 Global participation index, Modularity quality index (Q), and absolute group differences

Parenting Component	Low	Moderate	High	Low - Moderate	Low - High	Moderate – High
Global participation index						
Negativity EPI	.700	.744	.723	.044*	.023	.021
Warmth	.740	.730	.729	.010	.011	.001
Negativity PSI	.642	.730	.750	.088****	.108***	.020
Communication	.737	.718	.721	.019	.017	.002
Modularity quality index (Q)						
Negativity EPI	0.242	0.227	0.229	.015	.013	.002
Warmth	0.230	0.223	0.233	.007	.003	.010
Negativity PSI	0.231	0.223	0.222	.007	.008	.001
Communication	0.227	0.230	0.221	.003	.006	.009

Note: FDR (5%) adjusted p-values.

Abbreviations: EPI, event-planning interaction; PSI, problem-solving interaction.

^{*}p < .05.

^{****}p < .001.

3.4.4 | Parenting and local participation coefficient per region (node)

The results for global participation coefficient were based on network averages. Given the significant differences identified (three comparisons; Table 4) for the global participation coefficient between low-, moderate, and high- groups of two negative parenting components, we investigated participation coefficients for the regions (nodes). The Kolmogorov–Smirnov test identified six comparisons (e.g., low-Warmth compared to moderate-Warmth) where there was a significant difference between the pairs (Table 3, FDR 5%). The group comparisons identified using the Kolmogorov– Smirnov test were the same as the permutation based approach with three additional comparisons: Moderate to High for Negativity EPI, Low – Moderate Warmth, and Low – Moderate Communication (Table 3).

For the six comparisons, the regional (nodal) participation coefficients were further investigated (FDR 5%). For Negativity EPI, comparisons indicated differences in the cingulate gyrus, frontal gyrus, insular gyri, straight gyrus/subcallosal gyrus, anterior occipital sulcus and preoccipital notch, and a parietal region incorporating the angular gyrus, supramarginal gyrus, sulcus intermedius primus, and intraparietal sulcus. For Warmth, differences were indicated in the cingulate gyrus and sulcus (middle-anterior and middle-posterior), subcallosal gyrus, superior frontal sulcus, intraparietal sulcus, and a region in the temporal lobe comprising the temporal pole, temporal sulcus and gyrus. For Negativity PSI, differences were indicated in the cingulate gyrus and a parietal region comprising the angular gyrus and superior parietal lobule. For Communication, differences were indicted in the cingulate gyrus (middle-posterior and posterior-dorsal), cuneus, parieto-occipital sulcus, a parietal region comprising the supramarginal gyrus, superior parietal lobule, and intraparietal sulcus, and

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ellow (5)	~		Inferior occipital gyrus (O3) and sulcus (2) PC = .531	Middle occipital gyrus (19) PC = .650	Superior occipital gyrus (20) PC = .395	Posterior transverse collateral sulcus (51) PC = .349	ght Inferior occipital gyrus (O3) and sulcus (76) PC = .483	Lateral occipito-temporal gyrus (95) PC = .463	Superior temporal sulcus (parallel sulcus, 147) PC = .527	Inferior occipital gyrus (O3) and sulcus (2)	Posterior-dorsal part of the cingulate gyrus (dPCC) (9)	Posterior-ventral part of the cingulate gyrus (vPCC, isthmus of the cingulate gyrus) (10)	Cuneus (11)	Middle occipital gyrus (O2, lateral occipital gyrus) (19	Superior occipital gyrus (O1) (20)	Lateral occipito-temporal gyrus (21)	Lingual gyrus, ligual part of the medial occipito-temporal gyrus, (O5) 22	Inferior temporal gyrus (37)	Occipital pole (42)
Magenta (4) Ye	37 38	6	L Precuneus (30) PC = .546 L	L Lateral aspect of the superior L temporal gyrus (34) PC = .542	L Postcentral sulcus (67) PC = .496 L	L Transverse temporal sulcus (74) PC = .558	R Superior parietal lobule (101) PC = .545	R Superior temporal sulcus (parallel R sulcus, 147) PC = .631	L Paracentral lobule and sulcus (3) R	L Subcentral gyrus (central operculum) L and sulci (4)	L Angular gyrus (25)	L Supramarginal gyrus (26)	L Superior parietal lobule (27)	L Postcentral gyrus (28)	L Anterior transverse temporal gyrus L (33)	L Planum temporale or temporal plane L of STG (36)	L Posterior ramus (or segment) of the L lateral sulcus (or fissure) (41)	L Temporal pole (43)	L Central sulcus (Rolando's fissure) (45) L
Blue (3)	6	0	L Precentral gyrus (29)	L Superior part of the precentral sulcus (69)	R Paracentral lobule and sulcus (77)	R Precentral gyrus (103)	R Sulcus intermedius primus (of Jensen) (129)	R Superior part of the precentral sulcus (143)											
Green (2)	50	ω	L Middle-anterior part of the cingulate gyrus and sulcus (aMCC, 7) PC = .454	L Middle frontal gyrus (15) PC = .513	L Superior frontal gyrus (16) PC = .538	L Orbital gyri (24) PC = .492	L Middle frontal sulcus (53) PC = .435	R Anterior part of the cingulate gyrus and sulcus (ACC, 80) PC = .427	R Triangular part of the inferior frontal gyrus (88) PC = .469	R Inferior frontal sulcus (126) PC = .403	L Fronto-marginal gyrus (of Wernicke) and sulcus (1)	L Transverse frontopolar gyri and sulci (5)	L Anterior part of the cingulate gyrus and sulcus (ACC) (6)	L Middle-posterior part of the cingulate gyrus and sulcus (pMCC) (8)	L Opercular part of the inferior frontal gyrus (12)	L Orbital part of the inferior frontal gyrus (13)	L Triangular part of the inferior frontal gyrus (14)	L Short insular gyri (18)	L Straight gyrus (31)
Red (1)	17	2	L Anterior transverse temporal gyrus of Heschl (33) PC = .720	L Pericallosal sulcus (S of corpus callosum (66) PC = .627	L Long insular gyrus and central sulcus of the insula (17)	L Parahippocampal gyrus of the medial occipito-temporal gyrus (23)	L Subcallosal area, subcallosal gyrus (32)	L Planum polare of the superior temporal gyrus (35)	L Temporal pole (43)	L Inferior segment of the circular sulcus of the insula (48)	R Posterior-dorsal part of the cingulate gyrus (dPCC) (83)	R Posterior-ventral part of the cingulate gyrus (vPCC, isthmus of the cingulate gyrus) (84)	R Long insular gyrus and central sulcus of the insula (91)	R Short insular gyri (92)	R Parahippocampal gyrus of the medial occipito-temporal gyrus (97)	R Subcallosal area, subcallosal gyrus (106)	R Planum polare of the superior temporal gyrus (109)	R Inferior segment of the circular sulcus of the insula (122)	R Pericallosal sulcus (S of corpus callosum) (140)
Module (see Figure 1)	No. Nodes	No. Hubs																	

TABLE 5 Modules for the whole group structural covariance network

(5)	arine sulcus (44)	rior transverse collateral sulcus	erior transverse collateral sulcus	lle occipital sulcus and lunatus us (57)	rior occipital sulcus and sverse occipital sulcus (58)	al occipito-temporal sulcus (60)	ial occipito-temporal sulcus ateral sulcus) and lingual sulcus	arietal sulcus (71)	ior occipital gyrus (O3) and Is (76)	(85) sue	łle occipital gyrus (O2, lateral bital gyrus) (93)	rior occipital gyrus (O1) (94)	al occipito-temporal gyrus (95)	ual gyrus, ligual part of the medial oito-temporal gyrus (96)	ior temporal gyrus R (111)	lle temporal gyrus (112)	pital pole (116)	
lagenta (4) Yellow	Marginal branch (or part) of the L Calcar cingulate sulcus (46)	Parieto-occipital sulcus (or fissure) L Anteri (65) (50)	Paracentral lobule and sulcus (77) L Poster (51)	Subcentral gyrus (central operculum) L Middl and sulci (78) sulcu	Angular gyrus (99) L Super trans	Supramarginal gyrus (100) L Latera	Postcentral gyrus (102) L Media (colla (61)	Precuneus (104) L Subpa	Lateral aspect of the superior R Inferi temporal gyrus (108) sulcu	Planum temporale or temporal plane R Cune of the superior temporal gyrus (110)	Middle temporal gyrus (T2) (112) R Middl occip	Posterior ramus (or segment) of the R Super lateral sulcus (or fissure) (115)	Central sulcus (Rolando's fissure) R Later (119)	Marginal branch (or part) of the R Lingu cingulate sulcus (120) occip	Sulcus intermedius primus (of Jensen) R Inferi (129)	Intraparietal sulcus (interparietal R Middl sulcus) and transverse parietal sulci (130)	Anterior occipital sulcus and R Occip preoccipital notch (temporo-occipital incisure) (133)	
Blue (3)	L ramus of the anterior bottom of the lateral sulcus (or 19) by the lateral sulcus (or 19) by the lateral sulcus (or 10) by the lateral substance of the lateral substance	mus of the anterior L of the lateral sulcus (or 00)	egment of the circular R the insula (47)	egment of the circular R the insula (49)	ontal sulcus (52) R	rontal sulcus (54) R	oital sulcus (62) R	oital sulcus (olfactory sulcus)	lci (64) R	art of the precentral sulcus	I sulcus (sulcus rostrales, tal sulcus) (70)	arginal gyrus (of Wernicke) s (75)	e frontopolar gyri and sulci	nterior part of the cingulate 1 sulcus (aMCC) (81)	ssterior part of the cingulate 1 sulcus (pMCC) (82)	part of the inferior frontal ()	rt of the inferior frontal)	
Red (1) Green (2)	L Horizonta segment fissure) (3	L Vertical ra segment fissure) (2	L Anterior s sulcus of	L Superior s sulcus of	L Inferior fr	L Superior f	L Lateral or	L Medial or (63)	L Orbital su	L Inferior p (68)	L Suborbita supraorbi	R Fronto-m and sulcu	R Transvers (79)	R Middle-ar gyrus and	R Middle-po gyrus and	R Opercular gyrus (86	R Orbital pa gyrus (87	
Module (see Figure 1) F																		

TABLE 5 (Continued)

(Continues)

Yellow (5)	R Anterior transverse collateral sulcus (124)	R Posterior transverse collateral sulcus (125)	R Intraparietal sulcus (interparietal sulcus) and transverse parietal sulci (130)	R Middle occipital sulcus and lunatus sulcus (131)	R Superior occipital sulcus and transverse occipital sulcus (132)	R Anterior occipital sulcus and preoccipital notch (temporo-occipital incisure) (133)	R Lateral occipito-temporal sulcus (134)	R Medial occipito-temporal sulcus (collateral sulcus) and lingual sulcus (135)	R Subparietal sulcus (145)	R Superior temporal sulcus (147)						
Magenta (4)	R Postcentral sulcus (141)	R Inferior temporal sulcus (146)	R Transverse temporal sulcus (148)													
Blue (3)				iterior :us (113)	ior :us (114)	rcular	rcular		8)		tory		tral sulcus	ostrales,	46)	isson the second s
Green (2)	R Superior frontal gyrus (90)	R Orbital gyri (98)	R Straight gyrus (105)	R Horizontal ramus of the an segment of the lateral sulc	R Vertical ramus of the anter segment of the lateral sulc	R Anterior segment of the cir sulcus of the insula (121)	R Superior segment of the cir sulcus of the insula (123)	R Middle frontal sulcus (127)	R Superior frontal sulcus (128	R Lateral orbital sulcus (136)	R Medial orbital sulcus (olfac sulcus) (137)	R Orbital sulci (138)	R Inferior part of the precent (142)	R Suborbital sulcus (sulcus ro supraorbital sulcus) (144)	R Inferior temporal sulcus (14	Doctrioux of al. 2010) Doctrioux ID (E
Red (1)																and toot Earmant Destriction (
Module (see Figure 1)																Moto: Unbo in b

Note: Hubs in bold text. Format, Destrieux description (Destrieux et al., 2010), Destrieux ID (FreeSurfer description).

TABLE 5 (Continued)



FIGURE 2 Global participation coefficient (PC) and modularity (Q) for maternal parenting components. Abbreviations: EPI, event-planning interaction; PSI, problem-solving interaction

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a temporal region comprising the superior temporal gyrus, temporal pole, and temporal plane of the superior temporal gyrus.

4 | DISCUSSION

This study examined associations between variations in normative parenting behaviors and the organization of structural brain networks in late childhood. We chose to measure organization using a sophisticated estimate of brain network structure, modularity. We hypothesized that positive parenting behaviors would be associated with increased modularity and negative parenting behaviors would be associated with decreased modularity. We found that high levels of observed negative maternal behaviors were associated with low modularity. We found no relationship between positive maternal behaviors and modularity and maternal communication and modularity.

The findings, based on the global participation coefficient, display patterns of association between modularity and negative (affective) parenting. Overall, as levels of negative parenting behaviors increased the global participation coefficient also increased. Given a high participation coefficient indicates regions with strong connections to many modules, the networks for High- and Moderate Negativity PSI have less segregation between modules, that is, the networks are less modular than the network for Low- Negativity PSI.

There was however a distinction between negative parenting in a positive context compared to a negative context. For negative parenting in a negative context (Negativity PSI), as described above, the association with modularity was evident when comparing low levels to moderate and high. For negative parenting in a positive context (Negativity EPI), the negative association with modularity was only evident for the comparison between low and moderate levels - not high. Although the global participation coefficient was greater for high levels of negative parenting in a positive context compared to low levels this result was not significant. This finding was surprising given previous research has demonstrated that maternal behavior counter to interaction task demands (e.g., aggressive during a typically positive event-planning task) is linked with particularly poor child outcomes (Schwartz et al., 2016). Further, our previous work showed that negative maternal behavior counter to task demands was more consistently linked to decreased local efficiency of structural networks. Considering these findings, the stronger pattern of association was expected for negative parenting in a positive context, particularly when high levels of negative behaviors and incongruence are experienced by a child. The current results suggest that negative maternal behavior consistent with task demands may have a specific effect on modularity.

Although the primary measure for modular segregation was the global participation coefficient, the modularity quality index (*Q*) of network partitions was employed as an alternative. There were no associations demonstrated between modular segregation based on the quality index and the four parenting components. In contrast to the participation coefficient, the quality index increases in more segregated brain networks and therefore given the increase in participation

coefficient for negative parenting, the quality index was expected to decrease for these same parenting components. Although the quality index decreased as levels of negative parenting increased, these changes did not survive corrections for multiple comparisons.

In contrast to the findings for maternal negative behaviors, no support was found for an association between positive maternal behaviors or maternal communicative behaviors and modular segregation (for the participation coefficient and modularity quality index). Given positive (warm and supportive) parenting has been linked to structural brain changes (Luby et al., 2016; Whittle, Simmons, et al., 2014) this was also an unexpected finding. As the global participation coefficient and the modularity quality index provide measures for whole networks it is possible that differences were not evident due to "averaging." This idea is supported by the comparisons of the participation coefficients at the regional (or nodal) level where there was a difference between low to moderate levels for positive and communicative maternal behaviors. Differences in the cingulate gyrus (middle-anterior, middle-posterior, and posterior dorsal), the subcallosal area, the anterior and superior temporal gyrus, temporal pole, and intraparietal sulcus were found. Brain regions such as the cingulate gyrus and temporal regions have been implicated in social cognition and may be influenced by parenting (Pratt, Goldstein, & Feldman, 2018; Valadez, Tottenham, Tabachnick, & Dozier, 2020). Taken together, this suggests a potential association between positive and communicative maternal behaviors and modularity.

Five modules were identified for the whole group structural covariance network and, in general, for each module the associated hubs were co-located within a lobe (e.g., the hubs of module 5 were located within the occipital lobe). The findings suggest a correspondence between the structural modules and the functional roles of the brain lobes. Given that we did not make any assumptions about the composition of the SC networks (i.e., we did not generate networks from the seeds of functional networks), this finding adds to the validity of the structural covariance analytical approach.

We also explored the number of modules identified across the low-, moderate-, and high- groups, and observed fluctuations (typically +/-1 module) in the number of modules within all four parenting components. In general, changes in the number of modules for a group were also reflected in changes in modular segregation. For example, as levels of negative parenting in a negative context increase so also do the number of modules and modular segregation decreases. Given that networks appear to become more modular with age, it is possible that development may involve a reduction in the number of modules. However, diffusion data, also based on modularity maximization using the Louvain heuristic, has identified nine structural modules which were stable across childhood and adolescence (Baum et al., 2017). Furthermore, Baum et al. (2017) main finding of increased segregation with age was robust to the number of modules in the structural partitions. Potentially, in the current study, the number of modules is less salient than measures of modularity.

Comparisons of the nodal compositions of the modules between the groups of the parenting components indicated very little similarity (as measured by NMI). This was confirmed by a visual inspection check for the Negativity EPI component, where a core group of nodes could not be identified across groups for either component. Furthermore, when we restricted the comparison to "hub" nodes, a consistent set of nodes could not be identified across the low-, medium-, and high- groups. This finding was surprising, given that for the community sample only modest changes in the nodes contained within each module were anticipated between low-, medium-, and highgroups. Further investigation, however, demonstrated that modules that did not contain exactly the same nodes, and hence the comparison had a low NMI, often contained nodes that were located in the same brain region (i.e., adjacent parcellations; Destrieux et al., 2010).

In the current study pairs of networks were compared (e.g., low-Negativity EPI and moderate-Negativity EPI) to determine a similarity score. Although many studies have compared modularity scores between groups at a global level (as has been done in the current study; Baum et al., 2017) fewer studies have explored these differences at the node and edge level particularly for structural covariance networks. In the current study, the normalized mutual information measure was used however other algorithms may have provided a different interpretation (e.g., hamming distance and the graph edit distance; Mheich, Wendling, & Hassan, 2020). Given the visual inspection discussed above, it is important to note that the normalized mutual information measure does not account for the spatial location of nodes, which can be key information when measuring similarity between networks (e.g., SimiNet algorithm: Mheich et al., 2020). In addition, the current study used sparse partial correlation estimation for the identification of network edges which will have influenced our findings. Overall, as the methodology for network similarity comparison is evolving these results should be interpreted with caution and it would be of interest for future work to replicate these finding using alternative algorithms and edge selection strategies (Mheich et al., 2020).

In many brain networks modularity and global network efficiency are inversely related, as a highly modular topology is likely to require long communication paths to integrate information across nodes (Baum et al., 2017). For the networks in the current study, we can compare the modularity findings to local and global efficiency values for the same networks detailed in Richmond, Beare, et al. (2019). Although there were no changes observed for the parenting components and global efficiency, high levels of negative affective maternal behaviors were associated with decreased local efficiency. Comparing across the two studies, the results suggest that high levels of negative affective behaviors are associated with less segregated and less locally efficient networks. While we previously interpreted our local efficiency findings as reflecting accelerated brain maturation in children experiencing poor parenting, combined with the current results, it is possible that these atypical network properties indicate disrupted neurodevelopment and may impact critical neural functions, such as emotion regulation and cognitive control thereby placing individuals at risk of mental disorders. Parenting behaviors may shape the neural underpinnings of emotion processing in children (Gee, 2016; Pozzi et al., 2019). The same parenting dimensions as used in the current article have been linked to alterations in the neural function underlying emotion regulation (Pozzi et al., 2019). These functional alterations may represent a possible mechanism between parenting and later depression symptoms or disorders in adolescents (Callaghan et al., 2017). Similar structural network alterations, that is, decreased local efficiency/clustering and atypical modularity, including decreased modularity based on the modularity index *Q*, have been demonstrated in adults with obsessive-compulsive disorder (Peng et al., 2014; Reess et al., 2018) and children with conduct disorder (Jiang et al., 2016). The observed network changes associated with parenting behaviors in the current study represent a potential framework for assessing the neurobiological mechanism which underlies the development of internalizing and externalizing problems in childhood and adolescence (Phua et al., 2019; Schwartz et al., 2016; Wu & Lee, 2020).

A limitation of this study was our inability to examine the relative contribution of fathers' behavior due to budget constraints. Fathers play a significant role in the emotional development of their children and the findings relating to mother's affective parental behaviors may not generalize to fathers (Cassano, Zeman, & Sanders, 2014; Schwartz et al., 2016). We did not investigate the bi-directional nature of the dyadic interactions and we cannot rule out that the association between parenting and structural brain networks in offspring is based on genetic factors. The study was also restricted to a between-subjects design and power may have been an issue. A power analysis would be difficult to conduct because, to the best of our knowledge, the available meta-analyses generally assess difference between groups with disease/disorder and healthy controls (e.g., loannidis, 2011 for brain volume abnormalities) and would be likely to overestimate effect size in typical development. In addition, given that covariance analysis does not provide network metrics for individuals it was not possible to calculate a standard measure of effect size. Estimating plausible effect sizes is important for reproducibility and is an area of exploration for future work. Finally, structural covariance in general, and more specifically the sparse partial correlation estimation applied in the current study, is a relatively new methodology for constructing structural brain networks and therefore the results should be interpreted with caution. Ultimately, exploring structural covariance using different methodologies is likely to increase our understanding but it is also important to consider underlying assumptions and limitations. We analyzed the data based on the Destrieux parcellation and it is possible that the results would be impacted if an alternative parcellation scheme was considered (Zalesky et al., 2010). Furthermore, we restricted the analysis to binary graphs and it is possible that the results would be impacted if weighted graphs, where the edges represent the strength of the partial correlation between regions, were applied (Fornito, Zalesky, & Bullmore, 2016). Future work in this area might include comparisons across multiple parcellation schemes and between binary and weighted graphs.

Previous work has typically accounted for the impact of multiple scanners, age, and sex though the use of linear regression with the resulting residuals then used to calculate the correlations between regions (Khundrakpam, Lewis, Zhao, et al., 2016; Teicher et al., 2014). In the current study, group differences on key participant variables were checked and given that there were none, we proceeded to use

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cal thickness after removing variance shared with other nodes; He et al., 2008; Teicher et al., 2014). In addition, age was tightly controlled and all scans were conducted on the same scanner, so neither of these variables warranted attention. It is likely though that sex may be related to structural covariance networks, given the findings for sex differences in brain structure (Cosgrove, Mazure, & Staley, 2007). Here the impact of sex was not investigated because under the current design, had we grouped by sex, the number of participants in the low-, moderate- and high- parenting groups would have been small (approximately 20 participants per group) and we were concerned the findings would have been under-powered. Future work in this area could evaluate the use of residual variances on our analytic approach, sparse partial correlation estimation.

This study is one of the first to explore the association between normative variations in parenting behavior and structural brain networks in childhood. The results provide preliminary evidence that variations in the emotional climate of typical family environments are associated with the modularity of structural covariance networks. It would be of interest for future research to explore whether these associations are related to the development of structural covariance networks.

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CONFLICT OF INTEREST

The authors declare no conflicts of interest..

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request due to privacy/ethical restrictions.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of this article.

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