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Parental psychopathology, family conflict, brain function, and child autistic-like traits in early adolescents

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Abstract

Background. Parental psychopathology is a known risk factor for child autistic-like traits. However, symptom-level associations and underlying mechanisms are poorly understood.
Methods. We utilized network analyses and cross-lagged panel models to investigate the specific parental psychopathology related to child autistic-like traits among 8,571 adolescents (mean age, 9.5 years at baseline), using baseline and 2-year follow-up data from the Adolescent Brain Cognitive Development study. Parental psychopathology was measured by the Adult Self Report, and child autistic-like traits were measured by three methods: the Kiddie Schedule for Affective Disorders and Schizophrenia for DSM-5 autism spectrum disorder (ASD) subscale, the Child Behavior Checklist ASD subscale, and the Social Responsiveness Scale. We also examined the mediating roles of family conflict and children's functional brain connectivity at baseline.
Results. Parental attention-deficit/hyperactivity problems were central symptoms and had a direct and the strongest link with child autistic-like traits in network models using baseline data. In longitudinal analyses, parental attention-deficit/hyperactivity problems at baseline were the only significant symptoms associated with child autistic-like traits at 2-year follow-up ($\beta = 0.014$, 95% confidence interval [0.010, 0.018], FDR $q = 0.005$), even accounting for children's comorbid behavioral problems. The observed association was significantly mediated by family conflict (proportion mediated = 11.5%, p for indirect effect <0.001) and functional connectivity between the default mode and dorsal attention networks (proportion mediated = 0.7%, p for indirect effect = 0.047).
Conclusions. Parental attention-deficit/hyperactivity problems were associated with elevated autistic-like traits in offspring during adolescence.

Introduction

Autism spectrum disorder (ASD) is a neurodevelopmental condition characterized by social impairments and restricted and repetitive behaviors or interests (Hirota & King, 2023). The Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) described that ASD symptoms are dimensional and represent an extreme end of autistic-like traits, in accordance with the neurodiversity perspective (American Psychiatric Association, 2013; J. N. Constantino & Todd, 2003; Gillberg, 1992). This dimension, acknowledged as autistic-like traits, refers to behavioral patterns that resemble characteristics commonly associated with ASD and on a continuum in the general population, typically including differences in social communication (e.g. interest in social interaction) (J.N. Constantino & Gruber, 2005; J. N. Constantino & Todd, 2003; Posserud, Lundervold, & Gillberg, 2006). The social communication abilities demonstrate substantial heritability (approximately 50%), comparable to ASD (J. N. Constantino & Todd, 2003). Meanwhile, these traits are also modulated by environmental factors, including family dynamics (Gerstein & Crnic, 2018; Martini et al., 2024). One longitudinal study has indicated that subclinical autistic social and communication traits may vary in their severity from childhood to adolescence (Pender, Fearon, Pourcain, Heron, & Mandy, 2023). Specifically, there were 7.3% of children undergoing a dramatic increase in autistic-like traits from 10 to 16 years old, and another 6.9% of children experienced a decrease in autistic-like traits over adolescence. The temporal variability might be contributed to environmental and genetic triggers starting at puberty, and non-autistic social communication difficulties secondary to depression and anxiety in adolescence. Elucidating the environmental risk factors associated with autistic-like traits during this critical developmental period would enhance our understanding of their etiology. However, empirical evidence is lacking on this topic.

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Parents of children with ASD frequently experience psychopathological problems, such as depression, anxiety, interpersonal sensitivity, paranoid ideation, obsessive–compulsive behaviors, and somatic complaints (Catalano, Holloway, & Mpofu, 2018; Hodge, Hoffman, & Sweeney, 2011). Also, maternal depressive symptoms, anxiety, obsessive–compulsive symptoms, emotional problems, and symptoms of attention-deficit/hyperactivity disorder (ADHD) during and after pregnancy were linked to more social communication problems and peer problems in childhood and adolescence, though the effect sizes were generally small to moderate (Amiri et al., 2020; Efron, Furley, Gulenc, & Sciberras, 2018; El Marroun et al., 2014; Goh et al., 2018; Kleine et al., 2020). Parental psychopathology has been identified as a risk factor for family conflict, which may in turn affect child autistic-like traits (Agha, Zammit, Thapar, & Langley, 2013; Zhang, Lee, White, & Qiu, 2020). Moreover, brain functions undergo significant developmental changes during early adolescence, with functional brain connectivity exhibiting high plasticity (Dahl, 2004). Previous studies have suggested neurobiological dysfunction as a potential mechanism of intergenerational transmission of psychopathologies such as depression and ADHD (Beardslee, Gladstone, & O'Connor, 2011; Epstein et al., 2007). Adverse family environments have been recognized to influence children's behavioral problems by modifying specific functional brain connectivity patterns (Zhi et al., 2024), which also underlie autistic-like traits (Nenadić et al., 2024; Tu et al., 2016). However, most existing studies examined isolated dimensions of parental psychopathology rather than employing a comprehensive assessment (Loeber, Hipwell, Battista, Sembower, & Stouthamer-Loeber, 2009). Given the well-documented high correlations between various psychopathologies (Guo et al., 2024; Hartman et al., 2023), the observed associations may be confounded by unmeasured comorbid conditions. Additionally, previous studies were mostly cross-sectional, limiting the ability to examine the directionality of this association.

This study aims to fill these research gaps using a large cohort of adolescents in the United States. First, we hypothesized that specific parental psychopathology could influence child autistic-like traits in early adolescence. We applied a network approach to identify the most important parental psychopathology linked to child autistic-like traits. We then implemented two-wave cross-lagged panel models (CLPMs) to investigate the directionality of longitudinal associations. Second, we hypothesized that at the behavioral level, family conflict might mediate the association between parental psychopathology and autistic-like traits; at the neurobiological level, brain functional connectivity might mediate the association. Mediation analyses were applied. The findings are expected to inform clinical interventions aimed at promoting both parental and adolescent mental health.

Methods

Participants

Our study was comprised of participants from the Adolescent Brain Cognitive Development (ABCD) study (release 5.0; https://abcdstudy.org/), which recruited over 11,000 children aged 9–11 years from 21 centers across the United States (Garavan et al., 2018). Parents' written informed consent and children's assent were obtained at recruitment (Clark et al., 2018). Of the children with complete parental psychopathology and child autistic-like traits information (N = 10,313), we randomly sampled one child from each family to account for the family-clustered structure (N = 8,571).

Measures

Parental psychopathology was assessed using the Adult Self Report (ASR) from the Achenbach System of Empirically Based Assessment at baseline and 2-year follow-up (Achenbach, 2009). The ASR consists of 120 items and scores on six DSM-oriented dimensions: depressive, anxiety, somatic, avoidant personality, attention-deficit/hyperactivity, and antisocial personality problems. In addition, we computed 8 dimension-based composite scores of the syndrome scales utilizing an alternative established algorithm (i.e. anxious/depressed, withdrawn, somatic complaints, thought problems, attention problems, aggressive behavior, rule-breaking behavior, and Intrusive) (Achenbach, Bernstein, & Dumenci, 2005). Parents rated each item on a 3-point Likert scale ranging from 0 (not true) to 2 (very true or often true), reflecting their experiences in the past 6 months. The ASR has shown good reliability and validity (Achenbach & Rescorla, 2003). We computed composite scores for each dimension of psychopathology, with higher scores reflecting greater severity of psychopathology.

Previous studies have reported different measures of autistic-like traits, including: the Kiddie Schedule for Affective Disorders and Schizophrenia for DSM-5 (KSADS-5, ASD subscale), Child Behavior Checklist (CBCL, ASD subscale), and Social Responsiveness Scale (SRS), respectively. In the ABCD study, these three measures showed acceptable validity for identifying parent-reported ASD (area under the curve, 0.80 for KSADS-5, 0.81 for CBCL, and 0.90 for SRS). Meanwhile, the correlations between these measures were weak to moderate (Spearman's ρ, 0.38–0.52; Table S1). Thus, we performed analyses on all three measures for comparison.

The KSADS-5, a widely-used screening tool for mental disorders, contains three parent-reported and autism-related questions (i.e. unusual body movements, strict routines, and poor eye contact during the past 2 weeks) which showed acceptable reliability and validity for screening ASD (Barch et al., 2018; Townsend et al., 2020; Ünal et al., 2019). Each item score ranges from 0 (not at all) to 4 (nearly every day); the total score ranges from 0 to 12. The CBCL contains 113 items that measure emotional and behavioral problems (Achenbach, 2009), 15 of which were used to measure child ASD-related behavioral and emotional problems across the past 6 months (Offermans et al., 2023). Parents rated each item on a 3-point Likert scale ranging from 0 (not true) to 2 (very true or often true), and the total score ranges from 0 to 30. Parents also completed the 11-item abridged version of SRS, which was also applied in screening for ASD (Reiersen, Constantino, Grimmer, Martin, & Todd, 2008). Each item was rated using a 4-point Likert scale ranging from 0 (not true) to 3 (almost always true); the total score ranges from 0 to 33.

For all three measures, higher scores represent more evident autistic-like traits. To be noted, the KSADS-5 and CBCL data were available at both baseline and 2-year follow-up, while the SRS was only available at 1-year follow-up.

Covariates

Potential confounders included child age, sex (male, or female), race/ethnicity (white, black, Hispanic, or others), family annual income level ($34,999 and less, $35,000 through $74,999, $75,000 through $99,999, or $100,000 and higher), and recruitment site. The highest missing proportion was 8.1% for the family annual income level. Missing data were imputed using the multivariate imputation by chained equations (van Buuren & Groothuis-Oudshoorn, 2011).

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Previous studies suggested that autistic-like traits and behavioral problems are distinct but correlated constructs (Lundström et al., 2011). Thus, we additionally adjusted for the CBCL internalizing and externalizing problem scores of children at baseline to specifically investigate autistic-like traits. ASD and autistic-like traits have high heritability (Serdarevic et al., 2020). To investigate to what extent the observed associations could be explained by genetics, in an additional analysis, we further adjusted for the polygenic risk score (PRS) for ASD. We were also interested in the mediating role of family conflict and resting-state functional magnetic resonance imaging (fMRI) connectivity in the observed associations between parental psychopathology and child autistic-like traits.

PRS for ASD

The genome-wide association study of autism was from the latest release of the iPSYCH cohort, involving 18,382 ASD and 27,969 controls of European ancestry (Grove et al., 2019). PRS scores were generated for ASD using polygenic risk scoring with continuous shrinkage (Ge, Chen, Ni, Feng, & Smoller, 2019). Quality control was performed by PLINK v1.90, and missing data was imputed using the Michigan Imputation Server, 40 with the 1,000 Genomes Project EUR (Phase 3, hg19) reference panel and Eagle v2.4 phasing (Warrier et al., 2022; Yang et al., 2022). Finally, 3,548 white individuals with complete parental psychopathology and child autistic-like traits data were included in the analyses involving PRS.

Family conflict

Family conflict was measured by the Family Conflict subscale of the Family Environment Scale (FES) at baseline (Moos & Moos, 1994). This is a 9-item dichotomous questionnaire reported by parents (Zucker et al., 2018). The scale ranges from 0 to 9, and higher scores indicate a more conflictual family environment.

Resting-state fMRI connectivity

Imaging acquisition, scanning parameters, and preprocessing procedures are described in detail by the ABCD team (Casey et al., 2018; Hagler et al., 2019). fMRI data were collected across sites with harmonized protocols on 3T scanner platforms, including Siemens Magnetom Prisma, General Electric Discovery MR750, and Philips Achieva scanners. Participants completed four 5-minute resting-state scans with eyes open to ensure at least 8 minutes of relatively low-motion data. Intra- and inter-network-level resting-state functional connectivity (rsFC; Pearson correlation) were calculated based on the Gordon parcellation scheme to group cortical-surface regions into 12 predefined resting-state networks (Gordon et al., 2016). Of these 12 networks, we specifically examined 8 networks that were reported to be associated with ASD, including auditory network (AN), cingulo-opercular network (CON), default mode network (DMN), dorsal attention network (DAN), fronto-parietal network (FPN), salience network (SN), ventral attention network (VAN), and visual network (VN) (Bi, Zhao, Xu, Sun, & Wang, 2018; Shan et al., 2023; Sun et al., 2021). They were Fischer z transformed, resulting in 36 network-level rsFC correlation averages (8 intra- and 28 inter-circuits).

Statistical analysis

All analyses were performed using R version 4.3.1.

Cross-sectional network analyses

A Graphical Gaussian Model (GGM) of network analyses provides a novel approach to visualizing the complex interaction among multiple symptoms based on correlations between each pair of symptoms (Epskamp, Waldorp, Mõttus, & Borsboom, 2018). We estimated GGMs to investigate the relationship among parental psychopathologies (ASR at baseline) and child autistic-like traits (KSADS-5 and CBCL at baseline, and SRS at 1-year follow-up). In this graph, each node corresponds to a symptom (parental dimensional psychopathologies and child autistic-like traits), and the edge weight indicates the strength of the partial correlation between the two symptoms. An optimal unregularized GGM with the lowest Extended Bayesian Information Criterion was selected for further analyses, which balanced model parsimony (number of edges) against model fit. Edges contributing minimally to log-likelihood were eliminated (Chen & Chen, 2008; Foygel & Drton, 2010). We applied flow diagrams with the package 'qgraph' (version 1.9.5) to visualize the network structure (Epskamp, Cramer, Waldorp, Schmittmann, & Borsboom, 2012). This study mainly focused on edge weights between child autistic-like traits and six parental dimensional psychopathologies. We estimated node centrality, which measured the relative importance of a node within a network, including strength, closeness, betweenness, and expected influence (definitions detailed in Table S2) (McNally, 2016). Network analyses were also performed using the 8 dimension-based composite scores from the syndrome scales, for comparison.

The network stability was examined using bootstrap methods with the package 'bootnet' (version 1.5.3) in following steps: (a) implementing case-dropping bootstrap to investigate the stability of centrality indices; (b) implementing nonparametric bootstrap to draw confidence intervals (CIs) of edge weights and test for significant differences between edge weights and centrality indices (Epskamp, Borsboom, & Fried, 2018; Martin, Maughan, Konac, & Barker, 2023). The correlation stability coefficient refers to the maximum proportion of cases that can be dropped, such that with 95% certainty, the correlation between original centrality indices and centrality of networks based on subsets is at least 0.7. It was applied to quantify the case-dropping bootstrap and should not be below 0.25.

We investigated the child sex difference of the network by permutation-based tests to examine differences between networks based on global strength invariance (i.e. the absolute sum of edge weights) and network structure invariance (i.e. distributions of edges) (van Borkulo et al., 2023). The analysis was performed using the package 'NetworkComparisonTest' (version 2.2.1). We performed the following sensitivity analyses to test the robustness of the network. First, we added children's behavioral problems to the network model and identified specific parental psychopathology that directly correlated with child autistic-like traits. Second, we re-estimated network models, additionally involving potential confounders in the network nodes, including child age, sex, race/ethnicity, family annual income level, and recruitment site. To be noted, adding children's behavioral problems to the network led to the elimination of almost all other edge weights in the unregularized GGM, due to the strong correlations between behavioral problems and autistic-like traits. Thirdly, we identified the eldest child from each family (N = 8,571) and re-estimated network models for comparison.

Longitudinal data analyses

Two-wave CLPMs based on structural equation modelling were implemented to investigate the longitudinal association between parental psychopathology and child autistic-like traits (KSADS-5 and CBCL) from baseline to 2-year follow-up with package 'lavaan' (version 0.6.15) (Shen et al., 2020). We adjusted for child age, sex,

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race/ethnicity, family annual income level, internalizing symptom score, externalizing symptom score, and recruitment site. A comparative fit index (CFI) >0.90 or a standardized root mean square residual (SRMR) <0.08 suggests adequate model fit (Hu & Bentler, 1999). A false discovery rate (FDR) correction using the Benjamini–Hochberg method was conducted for all 12 cross-lagged paths (6 parental dimensional psychopathologies × 2 directions). An FDR < 0.05 indicated statistical significance. We performed sensitivity analyses by the regression-with-residuals method to adjust for child internalizing and externalizing symptom scores at both baseline and 2-year follow-up, along with all other covariates from the main analysis. We also investigated the potential confounding role of genetic predisposition to ASD by additionally adjusting for PRS for ASD in a subsample of white children (N = 3,548).

Mediation analyses

As the longitudinal association of parental attention-deficit/hyperactivity problems with child autistic-like traits was determined by CLPMs, we further examined the mediation effect of family conflict and functional brain connectivity on this association at baseline (Kerr-German, White, Santosa, Buss, & Doucet, 2022; Lau et al., 2018; Modabbernia, Janiri, Doucet, Reichenberg, & Frangou, 2021; Zhang et al., 2020). The mediation analyses applied a standard 3-variable path model with adjustment for child age, sex, race/ethnicity, family annual income level, and recruitment site (Baron & Kenny, 1986). The significance of the mediation was estimated by the bias-corrected bootstrap approach (with 1,000 random samplings). In this analysis aiming to investigate the potential mechanisms, we could not rule out that behavioral problems lay on the pathway between parental psychopathology and child autistic-like traits. Thus, in the main analyses, we did not adjust for behavioral problems to avoid overadjustment. In an additional analysis, we further adjusted for externalizing and internalizing symptom scores for comparison.

Results

Table 1 shows the demographic characteristics of the children involved in this study (N = 8,571; mean age, 9.5 [SD, 0.5] years; 53% boys; 53% white; 42.5% with family annual income > $100,000).

Cross-sectional network analyses

Figure 1 presents the network structure between parental dimensional psychopathology and child autistic-like traits, measured by three different methods. Each network had a network density of 0.9 and a mean edge weight of 0.1. Parental attention-deficit/hyperactivity problems had a direct and the strongest connection with child autistic-like traits, irrespective of the measurement methods of the traits. Within each network, the centrality values of parental attention-deficit/hyperactivity problems ranked second, following parental depressive problems. Meanwhile, the connection between parental depressive problems and child autistic-like traits was weak, irrespective of the measurement methods (ranked 5th to 6th out of 6 dimensional psychopathologies). Additional network analyses employing an 8-factor structure of the ASR scales revealed inconsistent results in the analyses utilizing different measures of autistic-like traits (Figure S1). The inconsistency limits further interpretation and investigation utilizing this approach.

Table 1. Demographic characteristics of the eligible children from the Adolescent Brain Cognitive Development study

Variable (N = 8,571, baseline)	Missing	Mean (SD)
Parental characteristics		
Psychopathology (baseline/two-year follow-up)[a]	–	
Depressive problems		3.97 (3.64)/4.03 (3.68)
Anxiety problems		3.75 (2.55)/3.66 (2.52)
Somatic problems		1.92 (2.36)/1.93 (2.36)
Avoidant personality problems		2.02 (2.15)/1.99 (2.12)
Attention-deficit/hyperactivity problems		3.77 (3.60)/3.68 (3.54)
Antisocial personality problems		2.08 (2.45)/2.01 (2.35)
Child characteristics		
Male, no. (%)	–	4540 (53.0)
Age, year	–	9.47 (0.54)
Race/ethnicity, no. (%)	34	
White		4522 (53.0)
Black		1235 (14.5)
Hispanic		1723 (20.2)
Others		1057 (12.4)
Family annual income, no. (%)	692	
$34,999 and less		1597 (20.3)
$35,000 through $74,999		1748 (22.2)
$75,000 through $99,999		1184 (15.0)
$100,000 and higher		3350 (42.5)
Family conflict score[b]	–	2.51 (1.94)
Co-occurring behavioral problems[c]	1	
Internalizing problem score		5.12 (5.51)
Externalizing problem score		4.40 (5.75)
Autistic-like traits[d]		
KSADS–5 score (baseline/two-year follow-up)	–	1.04 (1.81)/0.84 (1.57)
CBCL score (baseline/two-year follow-up)	2	2.34 (3.01)/2.31 (3.04)
SRS score (one-year follow-up)	211	14.37 (4.03)

Abbreviations: KSADS-5, Kiddie Schedule for Affective Disorders and Schizophrenia for DSM-5; CBCL, Child Behavior Checklist; SRS, Social Responsiveness Scale; SD, standard deviation.
[a]Parental psychopathology was measured by self-reported Adult Self Report.
[b]Family conflict was measured by parent-reported Family Conflict subscale of Family Environment Scale at baseline.
[c]Child co-occurring behavioral problem was measured by parent-reported Child Behavior Checklist at baseline.
[d]These three scales were all parent-reported.

To test the robustness of the network, we observed that the centrality indices were stable in each network (correlation stability coefficient = 0.75, indicating that 75% of the sample could be dropped while the network structure did not change significantly; Figure S2). The nonparametric bootstrap tests revealed that the edge weights and centrality indices differed significantly for most comparisons (Figures S3 and S4).

The distribution plots of network comparison tests (Figure S5) illustrated that the global strength and network structure did not show

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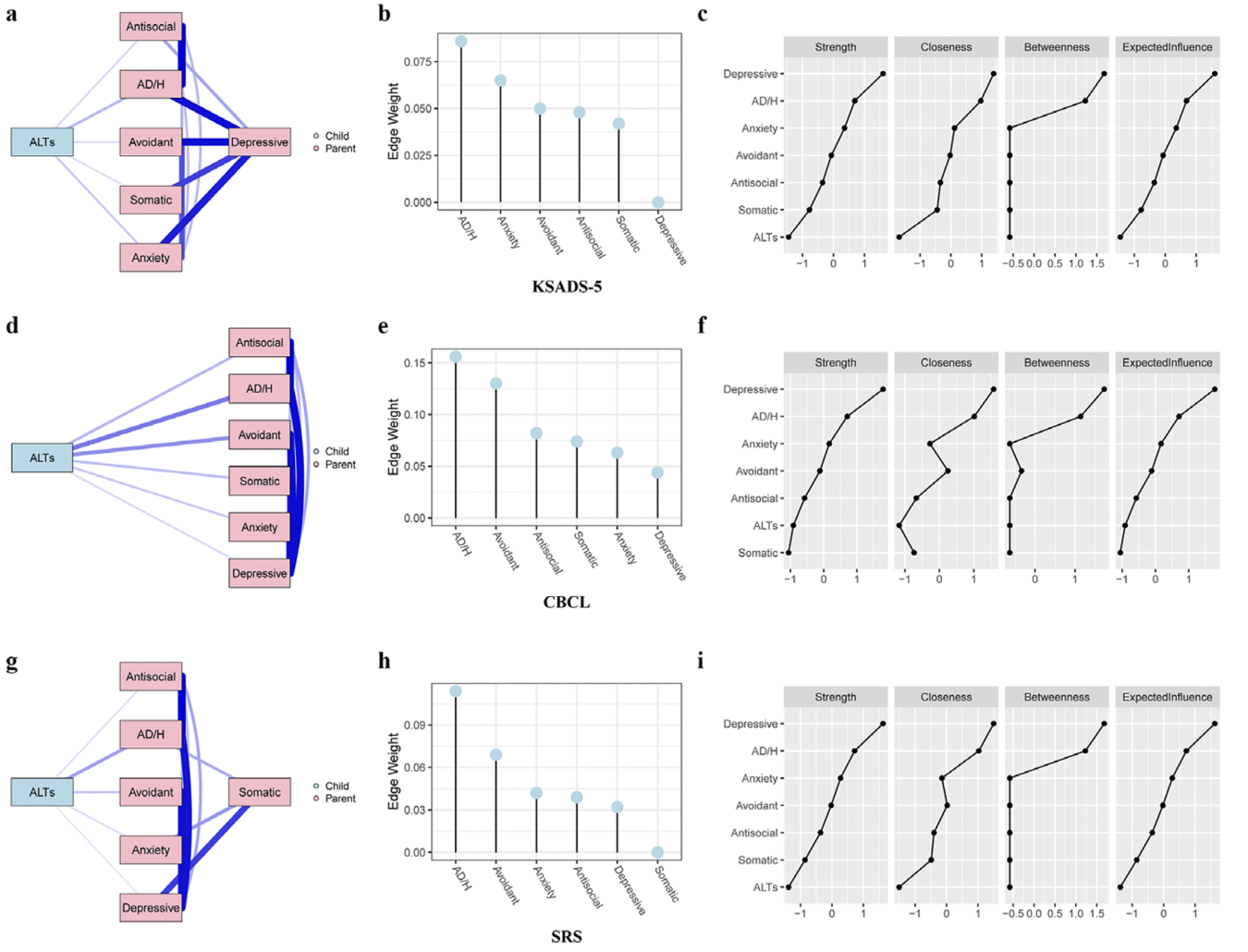

Figure 1. Network analyses of parental psychopathology and child autistic-like traits. Child autistic-like traits were assessed by (a–c) KSADS-5, (d–f) CBCL, and (g–i) SRS, respectively. (a, d, g) Network structure, (b, e, h) edge weight ranks between child autistic-like traits and six parental dimensional psychopathologies, and (c, f, i) node centrality measures are presented, respectively.

Abbreviations: KSADS-5, Kiddie Schedule for Affective Disorders and Schizophrenia for DSM-5; CBCL, Child Behavior Checklist; SRS, Social Responsiveness Scale; ALTs, autistic-like traits; AD/H, attention-deficit/hyperactivity.

significant sex differences in the three networks (e.g. KSADS-5-measured: $\text{strength}_{\text{diff}} < 0.001$, $p = 0.998$; $\text{edge}_{\text{maxdiff}} = 0.065$, $p = 0.125$). In the network models involving child internalizing and externalizing symptoms (Figure S6), parental attention-deficit/hyperactivity problem was the only parental psychopathology that showed consistent associations with children's autistic-like traits across multiple measures (CBCL and SRS, though not observed using the KSADS-5). Additionally, adding potential confounders into network models did not essentially alter the pattern and magnitude of associations (Tables S3 and S4). The sensitivity analyses using the eldest participant in each family of the ABCD study showed consistent results (Figure S7).

Longitudinal data analyses

We investigated longitudinal associations between parental psychopathology and child autistic-like traits at baseline and 2-year follow-up. After adjusting for demographic variables and internalizing and externalizing problems, we observed that the only significant cross-lagged association was between parental attention-deficit/hyperactivity problems at baseline and child autistic-like traits at 2-year follow-up, regardless of measurement methods (KSADS-5-measured: $\beta = 0.014$, 95% CI [0.010, 0.018], FDR $q = 0.005$; CBCL-measured: $\beta = 0.060$, 95% CI [0.052, 0.068], FDR $q < 0.001$; Figure 2 and Table S5). Additionally, adjusting for child internalizing and externalizing symptom scores at 2-year follow-up revealed similar results for CBCL-measured autistic-like traits, while the KSADS-5 measured result was not statistically significant (Table S6).

To account for the potential genetic factor underlying the observed associations, we calculated PRS for ASD in a subsample of white children ($N = 3{,}548$). Additionally adjusting for PRS for ASD did not essentially alter the effect size in CLPMs (Table S7 in the Supplementary Material).

Mediation analyses

We examined the potential mediating role of family conflict and functional brain connectivity in the observed association between parental attention-deficit/hyperactivity problems and child autistic-like traits. Parental attention-deficit/hyperactivity problems were associated with a higher family conflict score ($\beta = 0.165$, 95% CI [0.159, 0.171], $p < 0.001$). Using three different measures for child autistic-like traits, we robustly observed that family conflict mediated the association between parental attention-deficit/hyperactivity problems and

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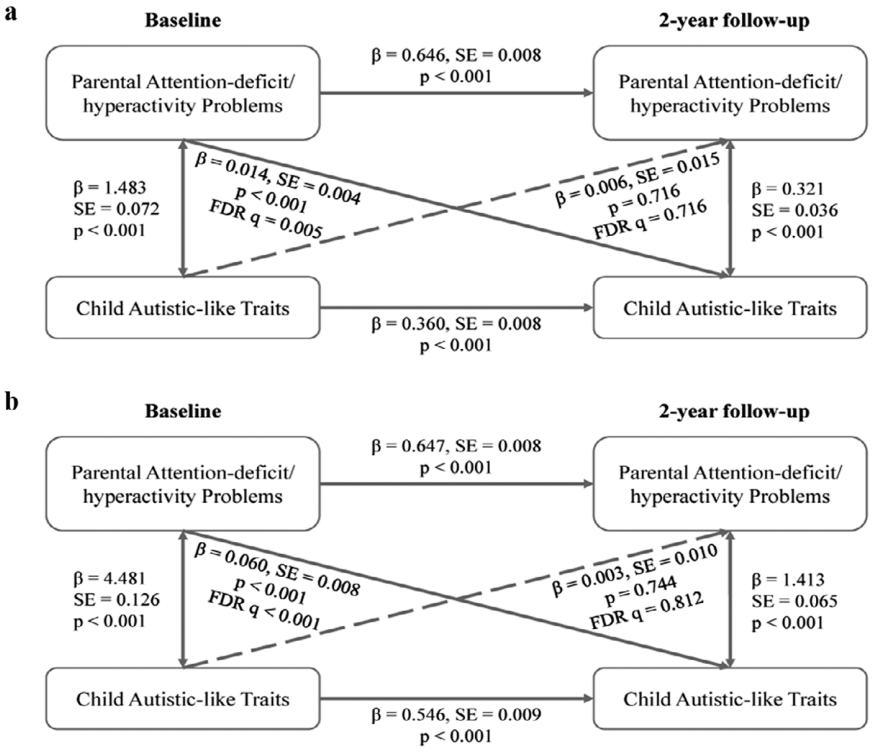

Figure 2. Two-wave cross-lagged panel models of parental attention-deficit/hyperactivity problems and child autistic-like traits. Child autistic-like traits were assessed by (a) KSADS-5 and (b) CBCL, respectively.
Abbreviations: KSADS-5, Kiddie Schedule for Affective Disorders and Schizophrenia for DSM-5; CBCL, Child Behavior Checklist; SE, standard error. *Note*: Solid lines represent statistical significance ($p < 0.05$), and dashed lines represent non-significance ($p > 0.05$).

child autistic-like traits (KSADS-5-measured: proportion mediated = 11.5%, p for indirect effect <0.001; CBCL-measured: proportion mediated = 8.0%, p for indirect effect <0.001; SRS-measured: proportion mediated = 6.8%, p for indirect effect <0.001; Figure 3a and Figure S8A,B).

We observed that parental attention-deficit/hyperactivity problems were associated with an increased DMN-DAN connectivity in children ($\beta = 0.011$, 95% CI [0.008, 0.114], $p < 0.001$, FDR $q = 0.029$; Table S8). DMN-DAN connectivity in children significantly mediated the observed association between parental attention-deficit/hyperactivity problems and child autistic-like traits (KSADS-5-measured: proportion mediated = 0.7%, p for indirect effect = 0.047; CBCL-measured: proportion mediated = 0.4%, p for indirect effect = 0.025; Figure 3b and Figure S8C). In the analysis on the SRS-measured autistic-like traits, we did not observe a statistically significant indirect effect of the DMN-DAN connectivity (p for indirect effect = 0.207; Figure S8D). In the additional analyses, further adjusted for externalizing and internalizing problems, the indirect effect of family conflict and DMN-DAN connectivity was not statistically significant (Figure S9).

Discussion

In a large cohort from the ABCD study, we first investigated how parental psychopathology influences autistic-like traits in early adolescents, then explored potential mediating mechanisms. We used a network approach and highlighted that parental attention-deficit/hyperactivity problems had the strongest link among psychopathologies that were directly associated with child autistic-like traits in early adolescence. In longitudinal analyses, we observed that parental attention-deficit/hyperactivity problems at baseline showed a robust link to child autistic-like traits at follow-up. Family conflict and DMN-DAN connectivity significantly mediate the association between parental attention-deficit/hyperactivity problems and child autistic-like traits.

Previous studies have reported that specific parental dimensional psychopathology was associated with child autistic-like traits. Goh et al. (2018) found that maternal depressive symptoms at 24 months were positively associated with toddler social-communicative autistic-like traits. Amiri et al. (2020) studied 3,942 children and found that maternal anxiety, obsessive–compulsive symptoms, and difficulties concentrating during prenatal and perinatal life were associated with later development of child autistic-like traits. Our network analyses, accounting for the high comorbidity of psychopathologies, corroborated previous findings and highlighted that parental attention-deficit/hyperactivity problems had a direct and the strongest link with child autistic-like traits. Importantly, in the longitudinal analyses accounting for the correlation between behavioral problems and autistic-like traits (Lundström et al., 2011; Simonoff et al., 2008), the only statistically significant cross-lagged association was between parental attention-deficit/hyperactivity problems at baseline and child autistic-like traits at 2-year follow-up. Our findings extend the current knowledge that parental attention-deficit/hyperactivity problems could be a risk factor for child elevated autistic-like traits during early adolescence.

Multiple mechanisms have been identified concerning parent–child psychopathology transmission, including dysfunctional family environment, neurobiological dysfunction, and genetic predisposition (Beardslee et al., 2011; Ferreira et al., 2013; Gebru et al., 2023). Parental attention-deficit/hyperactivity symptoms have been identified as a risk factor for child social problems (Efron et al., 2018; Zhang et al., 2020). Compared with other parental psychopathological dimensions (Jones, Hall, & Kiel, 2021; Lovejoy, Graczyk, O'Hare, & Neuman, 2000), parental attention-deficit/hyperactivity

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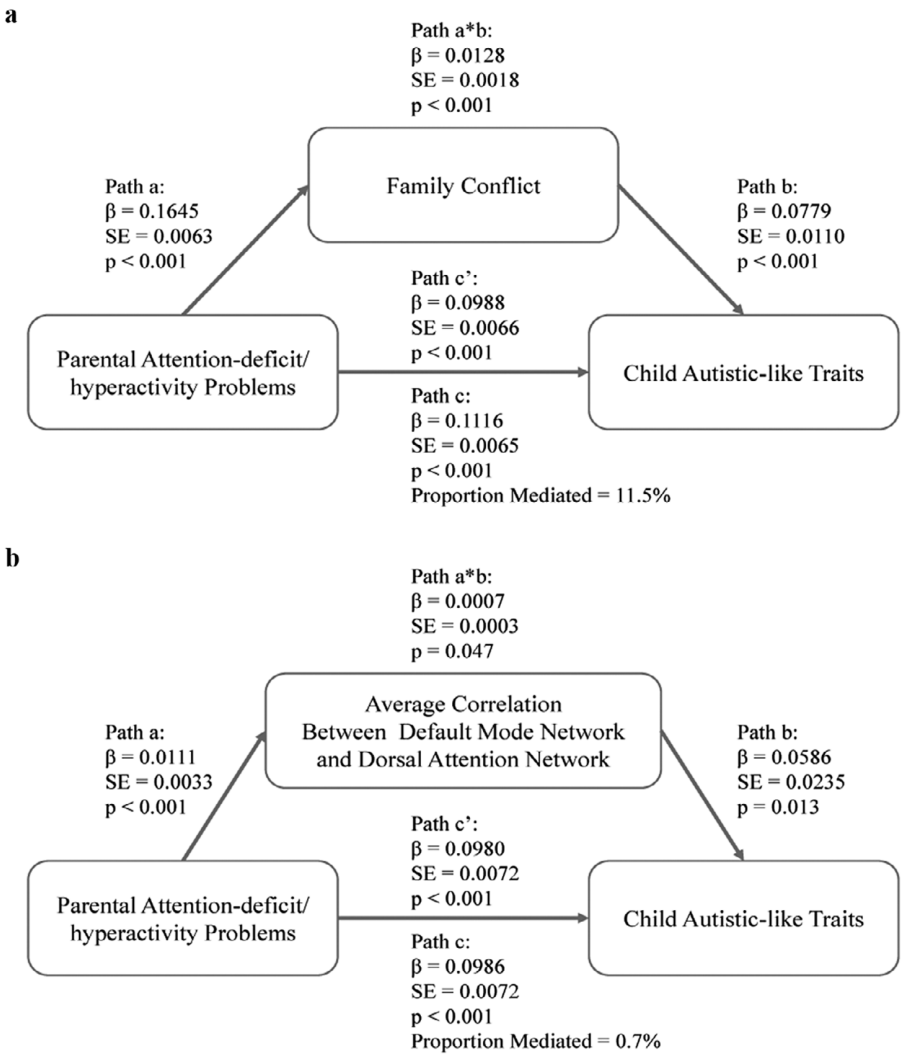

Figure 3. Mediation analyses of (a) family conflict and (b) average correlation between default mode network and dorsal attention network in the association between parental attention-deficit/hyperactivity problems and child autistic-like traits, measured by KSADS-5.
Abbreviations: KSADS-5, Kiddie Schedule for Affective Disorders and Schizophrenia for DSM-5; SE, standard error.

problems were more prominently associated with unfavorable parenting, such as hostility, criticism, physical or verbal aggression, inconsistent discipline, indulgent, neglectful, and lack of sensitivity and responsiveness to the child (Park, Hudec, & Johnston, 2017), which may in turn arouse family conflict and affect child social behavior (Agha et al., 2013). Consistently, we observed that family conflict significantly mediated the association between parental attention-deficit/hyperactivity problems and child autistic-like traits.

ASD and ADHD exhibited shared aberrant functional brain connectivity, other than depression and anxiety (Di Martino et al., 2013; Lukito et al., 2020). Specifically, DMN is an integrated system typically activated during self-related cognitive processes, including autobiographical memory, internal thoughts, and social functions (Menon, 2011), while DAN involves goal-directed top-down orienting of attention (Fox, Corbetta, Snyder, Vincent, & Raichle, 2006). The anticorrelation of these two networks is considered healthy functional brain connectivity (Fox et al., 2005), and this altered anticorrelation has been found to relate to ASD and ADHD (McKinnon et al., 2019; Strang et al., 2023; Tomasi & Volkow, 2012). Our findings were consistent with previous studies that supported the relationship between DMN-DAN connectivity and autistic-like traits (McKinnon et al., 2019; Strang et al., 2023), and our further mediation analysis showed that increased DMN-DAN connectivity in resting-state fMRI significantly mediated the association between parental attention-deficit/hyperactivity problems and child autistic-like traits. Interestingly, previous studies reported a generally decreased DMN-DAN connectivity as shared neurobiological dysfunction of ASD and ADHD in children at adolescence (Kernbach et al., 2018). This discrepancy in direction could be explained by the overall developmental shift from hyper-connectivity in childhood to hypo-connectivity in adolescence and adulthood (Padmanabhan, Lynch, Schaer, & Menon, 2017), considering that the children in this analysis were at a mean age of 9.5 years and at the transitional stage from childhood to adolescence. In addition, we observed that adjusting for behavioral problems would nullify the indirect effect through family conflict and DMN-DAN connectivity, indicating a common etiology of behavioral problems and autistic-like traits related to parental attention-deficit/hyperactivity problems.

ADHD is among the most common psychiatric comorbidities in ASD (28% prevalence, compared to ~20% for anxiety and ~10% for depression) (Hirota & King, 2023), with substantial genetic risk overlap between the conditions (Stergiakouli et al., 2017; Wu et al.,

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2020). Considering the shared genetic predisposition of ADHD and ASD, we additionally adjusted for PRS for ASD in a subsample of white children, observing comparable risk estimates with the main analysis. Though this may indicate a limited role of genetic predisposition, it should also be noted that PRS for ASD could only explain less than 5% of the etiology of ASD, despite ASD's high heritability (>50%) (Grove et al., 2019). Therefore, we cannot rule out that shared genetic factors between ADHD and ASD may still partly explain the observed association between parental attention-deficit/hyperactivity problems and children's autistic-like traits.

Parental depression has been identified as a risk factor for child autistic-like traits (Avalos et al., 2023; Breider et al., 2022; Goh et al., 2018), and often coexists with other psychopathologies (Daga et al., 2011; Sandstrom, Perroud, Alda, Uher, & Pavlova, 2021; Tiller, 2013). Our network models revealed that parental depressive problems had the highest centrality index, while in two of the three networks, parental depressive problems had the lowest association with child autistic-like traits. As centrality index indicates relative importance of a node within a network (Robinaugh, Millner, & McNally, 2016), our results implied that parental depressive problems were most strongly related to other dimensional psychopathological symptoms, while they connected to offspring autistic traits mainly through other psychopathological symptoms.

The study has the following strengths. First, this study included a large sample with repeated measures data that permitted both cross-sectional network and longitudinal analyses. By combining the two methods, we provided evidence that may suggest the directionality of observed associations. Second, as the measure of autistic-like traits remains controversial, we used three measures of child autistic-like traits, and the results were consistent, indicating high validity of the findings. Third, the genetic, familial, and functional brain connectivity data enabled us to explore the possible effect-modifiers or mediators of the observed associations.

The study should be interpreted in the light of the following limitations. First, we lacked data on whether the psychopathology belonged to the mother or the father. A previous study suggested that the impact of maternal psychopathology on child development may differ from that of paternal psychopathology (Rogers et al., 2023). Second, the exposure and outcomes in this study were exclusively parent-reported, which may introduce measurement bias and inflate the strength of observed associations. While we attempted to mitigate this limitation by incorporating statistical adjustments for children's behavioral problems, the fundamental challenge of disentangling parental perceptions – potentially influenced by their own psychopathological traits – from children's actual behavioral manifestations remains. This methodological constraint underscores the need for future investigations to incorporate direct, objective assessments of autistic-like traits in children, such as tests of theory of mind, executive functions, and pragmatic language. Third, though we utilized three established instruments to assess autistic-like traits, studies should employ more comprehensive measurements, including Childhood Autism Rating Scale-Second Edition Questionnaire of Parent Concerns, the Autism Treatment Evaluation Checklist, Mental Synthesis Evaluation Checklist, and Behavior Rating Inventory of Executive Function. Fourth, the follow-up period was relatively short. Future research with extended follow-up data is required to better comprehend the variations in autistic traits in the crucial period from childhood to adolescence.

Conclusion

In summary, we observed a robust association between parental attention-deficit/hyperactivity problems at baseline and child autistic-like traits at follow-up during early adolescence. Family conflict and dysfunctional DMN-DAN connectivity significantly mediated the association.

Supplementary material. The supplementary material for this article can be found at http://doi.org/10.1017/S0033291725100779.

Data availability statement. The data that support the findings of this study are openly available in the ABCD Dataset Data Release 5.0 at https://nda.nih.gov/abcd. The scripts used for these analyses will be made available upon publication (doi: 10.15154/dkxz-0k07).

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Author contributions. MW, YQL, TLZ, QL, TR and FL designed the study. MW and TR conducted data analysis. MW, YQL, TLZ, and TR drafted the manuscript. MW, YQL, TLZ, RQH, LKH, LLZ, QLZ, YJS, WZ, YWP, JYC, HH, SSW, WRC, QLZ, QL, TR, and FL contributed to the interpretation of the data and critically revised the manuscript. QL, TR and FL contributed equally to the work as senior investigators. They designed the study, supervised the data analyses, and supervised the drafting and revising of the manuscript. The corresponding author attests that all the listed authors meet authorship criteria and that no others meeting the criteria have been omitted.

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Competing interests. The authors declare none.

Ethical standard. In the ABCD study, all procedures were approved by a central Institutional Review Board (IRB) at the University of California, San Diego, and in some cases by individual site IRBs (e.g. Washington University in St. Louis) (https://www.sciencedirect.com/science/article/pii/S1878929317300622). Parents or guardians provided written informed consent after the procedures had been fully explained and children assented before participation in the study.

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