

Original Article

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Childhood environment clusters reveal heterogeneous associations between large-scale brain networks and youth mental health symptoms

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Abstract

Background. Youth mental health and brain development are profoundly shaped by highly heterogeneous childhood environments. However, research often operates under the assumption that neural networks linked to psychopathology function in the same way across different individuals, with limited consideration of how brain-behavior associations themselves may vary across environmental contexts. This poses challenges for identifying the precise neural correlates of risk or resilience to psychopathology.

Methods. In a large, longitudinal sample ($N = 8,078$), we examined differences in psychological symptoms and their associations with brain network functional connectivity across three clusters of youth identified by their home, school, and community environments.

Results. Child environment groups differed in mental health symptoms, as well as the links between large-scale functional network connectivity and symptoms. Youth exposed to high trauma and familial risk showed the highest symptom levels over time compared to those youth in low-risk or economically disadvantaged environments. Moreover, youth in the high trauma and familial risk group showed stronger functional connectivity between the salience and frontoparietal networks with increased symptoms, whereas youth in the high disadvantage group showed the opposite pattern. Notably, these brain and mental health associations were not observed when examined across the entire sample, and group differences were more pronounced in female and older youth.

Conclusions. The same neural patterns of functional network connectivity can have different implications for mental health depending on the environment. These findings highlight the importance of context-sensitive approaches for developing personalized interventions in supporting youth mental health.

Introduction

Children raised in similar environments can have vastly different mental health outcomes, underscoring the profound but complex influence of childhood environments on mental health and related neural processes (Bick & Nelson, 2016; Gee, 2021; Hosseini-Kamkar et al., 2023; McLaughlin et al., 2012; Rutter, 2005). These divergent outcomes arise in part because childhood environments are highly heterogeneous: no two children grow up in exactly the same circumstances, even within similar sociodemographic backgrounds (Abell et al., 1996; Green et al., 2010). The same experience can also have markedly different influences on brain and behavior, depending on individual context (Briggs, Amaya-Jackson, Putnam, & Putnam, 2021; Lacey & Minnis, 2020).

Adding to this complexity, brain development itself varies across individuals. Neural systems adapt to their specific environments (Greenough, Black, & Wallace, 2002; McEwen, 2012), shaping brain function and connectivity patterns to be highly specific to each person (Foulkes & Blakemore, 2018; Gordon et al., 2017). Consequently, neural patterns linked to risk for psychopathology in one context may serve a different function in another. Despite growing recognition of this environmental and neurobiological heterogeneity (Feczko et al., 2019; Gee, 2021), most research has sought to identify universal biomarkers of psychiatric risk (Briggs et al., 2021; Marek et al., 2022; Westlin et al., 2023), assuming that the same neural correlates of psychopathology generalize across individuals in heterogeneous environments, even though brain-psychopathology associations may be context-dependent. Developmental and transactional systems theories (Sameroff, 2009) emphasize that development reflects ongoing interactions between the individual and environment, such that the influences of context on outcomes are dynamic and person-specific. This perspective shifts the question from whether neural processes serve as a pathway through which

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the environment shapes psychopathology to whether brain-behavior associations are themselves context-dependent across individuals.

To address this heterogeneity, research is needed to examine the neural correlates of psychiatric risk within more homogenous subgroups defined by shared environmental characteristics. This is consistent with a person-oriented, holistic-interactionist perspective, which conceptualizes development as an individualized process (Magnusson & Stattin, 2007; Molenaar, 2004), emerging from interacting systems (Magnusson, 1985). Thus, the same biological correlate may index risk in one context or resilience in another, motivating research that conceptualizes the environment as a person-centered profile (Bergman & El-Khoury, 2003; Howard & Hoffman, 2018). As children are typically exposed to co-occurring experiences across multiple environmental domains, identifying subgroups using information across multiple informants and types of data can capture a more holistic account of an individual's childhood environment. Subgrouping approaches can also isolate patterns that are otherwise obscured in full, broader samples (Fisher, Medaglia, & Jeronimus, 2018; Hardi, Beltz, et al., 2024; Molenaar, 2015), and have been applied in neuroscience and psychiatry to reveal profiles of clinical disorders with distinct neural signatures (Drysdale et al., 2017; Fair, Bathula, Nikolas, & Nigg, 2012). However, work examining how neural correlates of psychopathology vary by individual context is only emerging (Ellwood-Lowe, Whitfield-Gabrieli, & Bunge, 2021).

This question is especially relevant to large-scale resting-state brain networks, which have been linked to a wide range of psychiatric disorders (Woodward & Cascio, 2015; Xia et al., 2018). In particular, the well-established triple network model of psychopathology (Menon, 2011) proposes that risk for psychiatric disorders arises from aberrant communication among three core networks: the default mode network (DMN) (Raichle, 2015), salience network (SN) (Uddin, 2015), and frontoparietal network (FPN) (Zanto & Gazzaley, 2013). Empirical work in clinical samples has identified altered communication among these networks (Menon, 2020; Sheline, Price, Yan, & Mintun, 2010; Sylvester et al., 2012); however, whether the same patterns of connectivity vary across different environments remains unknown. Clarifying these distinctions is critical not only for identifying which functional neural correlates may confer vulnerability or adaptation, but also for clarifying the conditions under which they do so.

In this study, we leveraged a large, longitudinal sample ($N = 8,078$) of youth (ages 9 to 15) to address two questions: (1) Do the trajectories of internalizing and externalizing symptoms during the transition to adolescence differ across environmental contexts? (2) Do the associations between functional connectivity of large-scale networks (DMN, SN, FPN) and symptoms vary by these contexts? We adopted a multisystemic approach as children's environments are shaped by multiple, interacting ecological systems (Bronfenbrenner, 1977), ranging from proximal contexts such as parent and family relationships to other influences within school, peer networks, and the community more broadly. We integrated measures of the environment by clustering individuals using multi-informant (parent and child) and multi-modal (self-report and administrative) data spanning six ecological systems: child, parent, family, school, community, and neighborhood, and then examining the trajectories of internalizing and externalizing symptoms as well as the associations between symptoms and functional connectivity within and between the DMN, SN, and FPN across the environmental clusters.

Methods and materials

Setting and participants

A sample of 8,078 individuals from the Adolescent Brain Cognitive Development (ABCD) Study (Casey et al., 2018; Karcher & Barch, 2021) was included in the study. Data were collected at multiple waves and accessed from the National Institutes of Mental Health Data Archives 5.0 release. Participants were included in the present study if they had data on all environmental measures. All participants provided informed consent or assent at all visits (Clark et al., 2018).

Measures

Childhood environment

Thirty variables contributing to mental health were included from data across baseline, one-year, and two-year follow-up: child (negative life events, discrimination, traumatic events), parent (parental acceptance, parental monitoring, parent psychopathology, secondary parent psychopathology), family (family conflict, family cohesion, family expressiveness, family intellectual and cultural engagement, family organization, familism), peers and school (prosocial peers, rule-breaking peers, protective peer network, protective school environment, peer aggression victimization), community (community collective efficacy, community resources, parks, safety, disadvantage), and neighborhood (urbanicity, lead risk, air pollution (PM 2.5), area deprivation index, child opportunity index (education; health; social/economic)). More information is in the [Supplement](#) and [Supplemental Table 1](#).

Mental health symptoms

Internalizing and externalizing symptoms were measured by the youth-report on the ASEBA Brief Problem Monitor (BPM) questionnaire (Achenbach, McConaughy, Ivanova, & Rescorla, 2011; Karcher & Barch, 2021). Youth responses on the BPM were collected across 6 timepoints: T1 = Mage 10.4 years ($SD = 0.63$), T2 = 10.9 (0.64), T3 = 11.4 (0.64), T4 = 12.0 (0.66), T5 = 12.4 (0.64), T6 = 12.9 (0.64). The mean interval was 0.5 years (0.12).

Functional magnetic resonance imaging (fMRI)

Resting-state functional connectivity data from the baseline and two-year follow-up were included. Imaging acquisition and processing were described previously (Casey et al., 2018; Hagler et al., 2019) and are summarized in the [Supplement](#). Anatomical and resting-state fMRI data were collected from youth and pre-processed by the ABCD consortium's Data Analysis and Informatics Core (Hagler et al., 2019). Functional connectivity was computed using a seed-based correlation approach, and within- and between-network functional connectivity (DMN, SN, FPN) were examined. To improve coherence with other functional atlases, the SN in this study included all regions of interest labeled as either Salience or Cingulo-Opercular in the Gordon atlas (Uddin et al., 2023; Uddin, Yeo, & Spreng, 2019).

Statistical analysis

Childhood environment groups

Groups were derived using the hierarchical agglomerative clustering algorithm with Manhattan distance and the Ward D2 linkage method. Visual inspection of the resulting cluster dendrogram suggested five plausible clusters ([Supplemental Figure 1](#)), which

were then evaluated using 28 fit indices determining the optimal solution (Charrad, Ghazzali, Boiteau, & Niknafs, 2014). Based on majority rule, a three-cluster solution was selected (see [Supplemental Table 2](#) for index comparisons). Robustness checks using alternative distance and linkage specifications showed similar support for a three-cluster solution ([Supplemental Table 3](#)), and subsampling-based stability analyses across 500 iterations of 80% subsamples showed moderate stability of the primary clustering solution (median ARI = 0.52) ([Supplemental Method](#)). Analysis of variance was conducted to compare mean levels of environmental measures across the resulting groups. Post-hoc pairwise comparisons were then performed and adjusted using False Discovery Rate (FDR) (Benjamini & Hochberg, 1995) to account for multiple testing (denoted by q).

Group differences in trajectories of mental health symptoms

Differences in trajectories of mental health (internalizing, externalizing) symptoms across child environment groups and sex were examined using mixed-effects models using all available six waves of data. Given the significance of differences in age and sex in emotional development, symptoms were modeled separately as a nonlinear function of age, with interactions between cluster, age, and sex to allow trajectories to vary across clusters and sex. To account for repeated measures, models included subject-level and site-level random intercepts. Pubertal development at baseline and the interval between waves were included as covariates, and sampling weights were applied to account for population-level sampling variation (Gard et al., 2023). Post-hoc pairwise contrasts were FDR-corrected. Sex at birth was parent-reported at baseline. Pubertal development was parent-reported at baseline using the Pubertal Development Scale (Petersen et al., 1988).

Group differences in brain-behavior coupling across time

Differences in brain-behavior coupling across child environment groups were tested using mixed-effects models using baseline and 2-year follow-up data. Internalizing and externalizing symptoms were tested separately as a function of each brain network connectivity, including the interactions among network connectivity, cluster membership, age, and sex, to evaluate context-, development-, and sex-dependent coupling. Similar to the mental health trajectory models, brain-behavior models included subject-level and site-level random intercepts, as well as pubertal development, interval between waves, and sampling weights. Additionally, these models included neuroimaging-specific covariates: head motion and scanner manufacturer. Primary analyses focused on cluster differences in brain-symptom coupling, evaluated using post-hoc pairwise contrasts of cluster-specific slopes (simple effects of connectivity on symptoms) evaluated at the mean age and within sex. FDR correction was applied within each outcome across network measures and pairwise tests.

To further evaluate temporal changes, a change-on-change model was tested using similar model specifications. Within-person change in symptoms (difference between follow-up and baseline) was modeled as a function of within-person change in network connectivity, including interactions with cluster and sex, to test whether coupling change differed across groups and covariates (puberty, baseline age, interval between waves, motion, scanner manufacturer, site-level random intercept), with sampling weights applied.

Sensitivity analyses

Several sensitivity analyses were conducted to evaluate robustness and specificity. First, to test whether group differences are robust to

shared environmental influences, group differences analyses were repeated after randomly sampling one sibling per family. Second, to probe the selectivity of group differences in brain-behavior relationships to the examined measures of global internalizing and externalizing behaviors, we examined group differences in the brain-behavior relationships to other symptom dimensions (i.e., attentional problems measured using the BPM). Next, to test whether the broad-based psychopathology findings map onto specific symptom domains, we examined whether the pattern of group differences was similar across other psychopathology measures (i.e., DSM-5 anxiety and depression scales) captured using the parent-reported Child Behavioral Checklist (Achenbach, Ivanova, & Rescorla, 2017). Finally, associations between network connectivity and behavior were examined using conceptually-related behavioral indices: Emotion regulation, measured using the youth-reported Emotion Regulation Questionnaire at 3-year follow-up (Gullone & Taffe, 2012); and executive functioning, measured using performance on the NIH Toolbox Flanker and Dimensional Card Sorting tasks at baseline (Zelazo et al., 2013).

Results

Youth characteristics differ across identified childhood environment groups

Three clusters were identified based on 30 multi-level ecological factors across child, parental, family, school, community, and neighborhood ([Figure 1](#)). Youth in the first group ($N = 5,329$; 66%) were relatively exposed to the lowest levels of risk factors and had the highest access to most economic and social resources. Youth in the second group ($N = 1,279$; 16%) experienced relatively more traumatic and negative life events, as well as more family-level risk factors (e.g., parent psychopathology, family conflict). Youth in the third group ($N = 1,470$; 18%) were more economically disadvantaged, but had higher levels of access to specific cultural and social resources (e.g. familism, access to social and community services). Given these characteristics, these groups will be described as 'low risk', 'high trauma and familial risk', and 'high disadvantage'. All group and pairwise comparisons are in [Supplemental Table 4](#).

There were no differences in age and sex between clusters, but they differed in other sociodemographic characteristics ([Table 1](#)). The low-risk group had the greatest proportion of White youth (70%) with more educated parents in higher income brackets (parental education: M (SD) = 17.6 years (2.10); median annual household income = \$100,000 through \$199,999). Comparatively, the high trauma and familial risk had a lower proportion of White (62%) and Asian youth (1% vs 3%), but a greater representation of Hispanic youth (18% vs 14%). The high disadvantage group had the highest proportion of Black (29%) and Hispanic (34%) youth, and the lowest income bracket (median annual household income = \$35,000 through \$49,999) and parental education (M (SD) = 15.2 years (3.01)).

Mental health trajectories differ across childhood environment groups

Accounting for age and sex, symptoms differed across environment groups (group main effect, internalizing: $F(2,16580) = 93.85, p < .001$; externalizing: $F(2,16150) = 174.89, p < .001$; [Supplemental Tables 5 and 6](#); [Figure 2](#)), with the high trauma and familial risk group showing the highest levels of symptoms (M internalizing = 2.13,

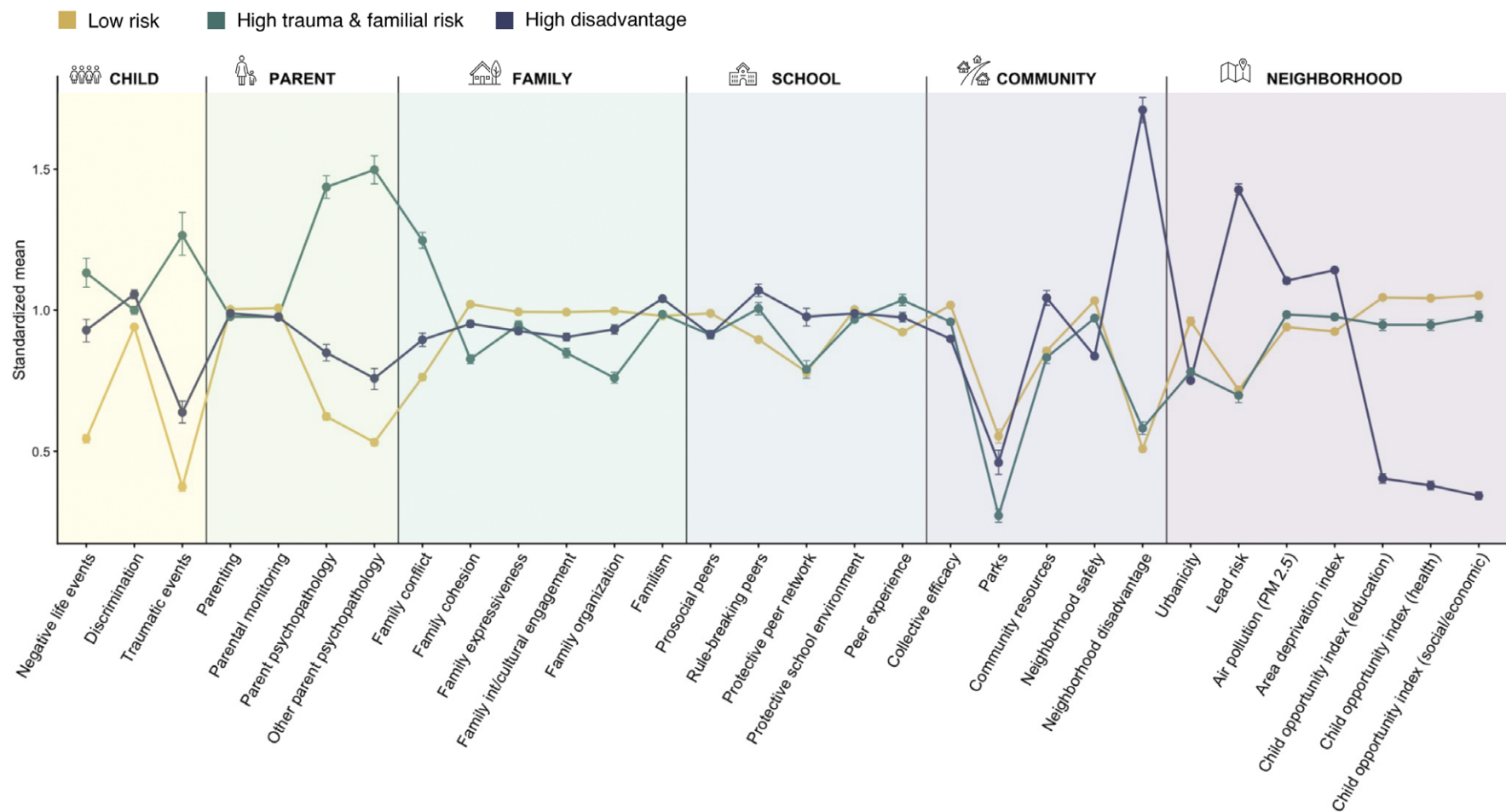


Figure 1. Childhood environment groups based on multi-level ecological measures. *Note:* Three groups were identified across six ecological domains: child (yellow), parent (green), family (cyan), school (blue), community (indigo), neighborhood (violet). The three groups were (1) Low risk, high resource (dark yellow; $N = 5,329$); (2) High trauma and familial risk, moderate economic resource (dark green; $N = 1,279$); and (3) High disadvantage, high social resource (dark purple; $N = 1,470$). Youth in the first group were exposed to relatively low risk levels and high resources across all domains. Youth in the second group experienced high levels of individual-level (i.e. negative life and traumatic events) and familial risks (i.e. family psychopathology and conflict), but moderate economic resources. Youth in the third group were exposed to high discrimination, disadvantaged neighborhoods and low opportunities, but high social and cultural resources. Each bar represents standardized values of each construct. Pairwise group comparisons are in [Supplemental Table 3](#).

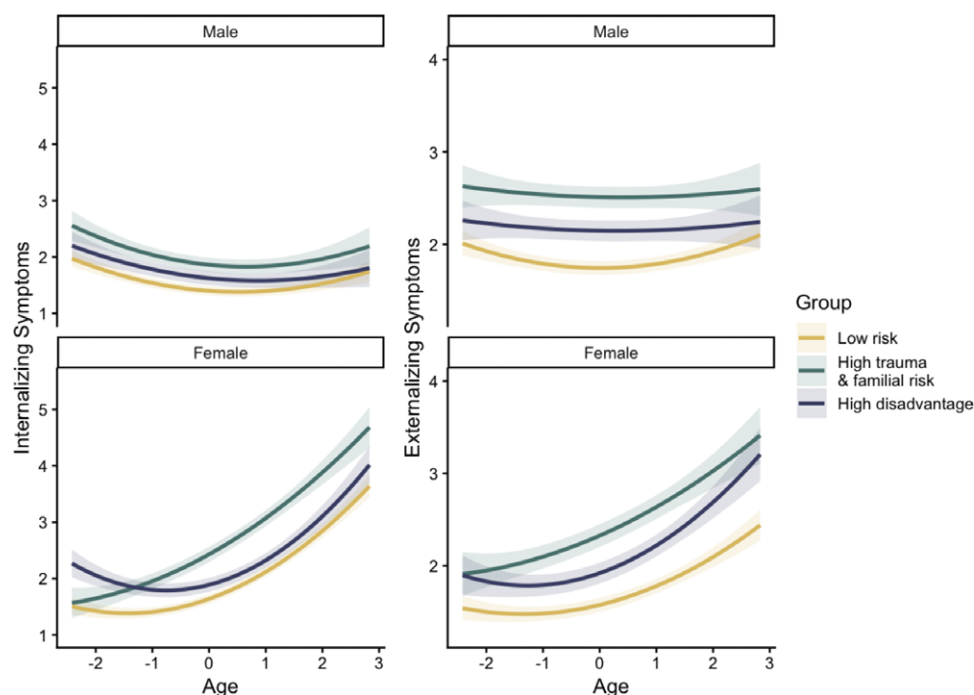


Figure 2. Trajectories of internalizing and externalizing symptoms, by childhood environment group. Internalizing symptoms were the most elevated for youth in the high trauma and familial risk group (in green), and the lowest for the low risk group (in yellow), both for internalizing and externalizing symptoms. Group differences were more pronounced in female youth (below) relative to male youth (top), where the high trauma and familial risk group showed the highest levels and change in symptoms over time compared to all other groups.

Table 1. Sociodemographic information for each childhood environmental group

	Low risk (<i>N</i> = 5,329; 66%)	High trauma familial risk (<i>N</i> = 1,279; 16%)	High disadvantage (<i>N</i> = 1,470; 18%)	Statistical comparison
Sex assigned at birth				
Male	2,830 (53.1%)	705 (55.1%)	741 (50.4%)	χ^2 (4) = 7.05, <i>p</i> = .133
Female	2,497 (46.9%)	574 (44.9%)	728 (49.5%)	
Intersex-male	2 (0.0%)	0 (0%)	1 (0.1%)	
Age (years) ^a				
Mean (<i>SD</i>)	9.93 (0.63)	9.92 (0.63)	9.89 (0.61)	F (2, 8,075) = 2.24, <i>p</i> = .107
Race/ethnicity				
White	3,704 (69.5%)	787 (61.5%)	372 (25.3%)	χ^2 (8) = 1,481.5, <i>p</i> < .001
Black	224 (4.2%)	68 (5.3%)	419 (28.5%)	
Hispanic	740 (13.9%)	230 (18.0%)	505 (34.4%)	
Asian	161 (3.0%)	15 (1.2%)	16 (1.1%)	
Other	500 (9.4%)	179 (14.0%)	158 (10.7%)	
Annual household Income ^a				
<\$5,000	23 (0.4%)	14 (1.1%)	76 (5.2%)	χ^2 (18) = 1,580.9, <i>p</i> < .001
\$5,000 to \$11,999	39 (0.7%)	18 (1.4%)	94 (6.4%)	
\$12,000 to \$15,999	33 (0.6%)	24 (1.9%)	50 (3.4%)	
\$16,000 to \$24,999	71 (1.3%)	45 (3.5%)	125 (8.5%)	
\$25,000 to \$34,999	119 (2.2%)	78 (6.1%)	145 (9.9%)	
\$35,000 to \$49,999	249 (4.7%)	116 (9.1%)	184 (12.5%)	

(Continued)

Table 1. (Continued)

	Low risk (<i>N</i> = 5,329; 66%)	High trauma familial risk (<i>N</i> = 1,279; 16%)	High disadvantage (<i>N</i> = 1,470; 18%)	Statistical comparison
\$50,000 to \$74,999	606 (11.4%)	209 (16.3%)	244 (16.6%)	
\$75,000 to \$99,999	862 (16.2%)	199 (15.6%)	185 (12.6%)	
\$100,000 to \$199,999	2,168 (40.7%)	398 (31.1%)	160 (10.9%)	
\$200,000 and higher	877 (16.5%)	102 (8.0%)	24 (1.6%)	
Missing	282 (5.3%)	76 (5.9%)	183 (12.4%)	
Parent education (years) ^a				
Mean (SD)	17.6 (2.10)	16.9 (2.22)	15.2 (3.01)	$F(2, 8,065) = 627.9, p < .001$
Missing	5 (0.1%)	2 (0.2%)	3 (0.2%)	

^aAge, household income, and parent education measured at baseline.

externalizing = 2.42), followed by the high disadvantage (M internalizing = 1.74, externalizing = 2.03) and low-risk (M internalizing = 1.51, externalizing = 1.66) groups. There were also differences by age and sex (Supplemental Table 7). For internalizing, groups differed for female, but not male, youth at younger and mean age (all pairwise q s < 0.05), with the high trauma and familial risk group showing the steepest increase at mean age (slope = 0.56, q = < .001), followed by the low-risk (slope = 0.35, q = < .001) and high disadvantage (slope = 0.26, q = < .001) groups. For externalizing, age-related increases were also the largest for the high trauma and familial risk group in female youth (slope = 0.27, q = < .001 at mean age), though the group differences were more modest, with only select group differences in female youth reaching significance (Supplemental Table 8).

Associations between brain networks and internalizing symptoms were examined within childhood environment groups, but not in the full sample

There were notable differences among groups in the association between SN-FPN connectivity (Figure 3a) and internalizing symptoms (group $\times$ SN-FPN: $F(2, 11061) = 11.41, q < .001$). Controlling for age and sex, SN-FPN connectivity was positively associated with internalizing symptoms in the high trauma and familial risk group ($b = 4.90, q < .001$), whereas the association was negative in the high disadvantage group ($b = -2.47, q = .029$) and not significant in the low-risk group ($b = 0.87, q = .128$) (all pairwise contrasts $q < .01$; Supplemental Table 9; Figure 3c).

In addition to these overall differences between environmental groups in the relationship between SN-FPN connectivity and internalizing, these group differences varied by sex (group $\times$ SN-FPN $\times$ sex: $F(2, 11052) = 5.39, q = .015$) and age (group $\times$ SN-FPN $\times$ age: $F(2, 9850) = 7.60, q = .003$), but there were no significant group $\times$ SN-FPN $\times$ age $\times$ sex interaction ($F(2, 9852) = 1.64, q = .39$) (Figure 3d). In female youth, the high trauma and familial risk group showed consistently stronger positive coupling between the SN-FPN and internalizing than other groups at mean and older ages (all q s < .01, Supplemental Table 10), with the separation from the high disadvantage group increasing with age (slope difference increased from 3.47 (−1SD age) to 17.79 (+1SD age). Conversely, group differences were only evident at older ages in male youth.

No other two-way group-by-brain interactions survived correction; however, additional age- and/or sex-dependent group differences (three-way or four-way interactions) were observed for other

networks and symptoms (i.e., DMN-DMN and internalizing coupling specific to older male youth, and FPN-FPN and DMN-FPN with externalizing coupling in younger male youth, and SN-FPN with externalizing coupling specific to older female youth; Supplemental Table 11; Supplemental Figure 2). Given that the primary goal of the study was to examine how brain-symptom associations differ across contexts, we focus on the association between SN-FPN and internalizing, where between-cluster differences were most robust to age/sex.

We further validated these findings using change-on-change analyses, showing that within-person increases in SN-FPN connectivity were differentially associated with within-person changes in internalizing symptoms across child environment groups (group $\times$ SN-FPN change: $F(2, 4149) = 5.26, q = .032$) (Figure 4). Consistent with the mixed effects model, there was a similar higher slope in SN-FPN and internalizing symptoms coupling for female youth, specifically for the high trauma and familial risk group, compared to the high disadvantage group (group $\times$ SN-FPN change $\times$ sex: $F(2, 4144) = 5.56, q = .023$; slope difference 10.63, $SE = 3.56, q = .007$) (Supplemental Table 12). There were also other notable sex-specific group differences in change-on-change effects between SN and DMN-FPN functional connectivity with externalizing symptoms (Supplemental Table 13; Supplemental Figure 3). There were no other significant associations between change in network connectivity and change in symptoms after correction for multiple comparisons.

Notably, the associations between functional connectivity and internalizing were not evident when collapsing across the full sample (Figure 3b). When all groups were combined, neither SN-FPN connectivity ($b = 0.67, SE = 0.46, p = .146$) nor the change in SN-FPN ($b = 0.78, SE = 0.76, p = .310$) showed significant associations with internalizing symptoms and their change across time.

Results from sensitivity analyses

Sensitivity analyses showed that the group differences in mental health trajectories, SN-FPN coupling with internalizing symptoms, as well as change-on-change associations, were robust after random sampling of siblings (Supplemental Table 14).

To test whether group differences in SN-FPN coupling with symptoms were specific to internalizing behaviors, we examined group differences in the associations between SN-FPN connectivity and other behavioral domains, specifically attentional problems.

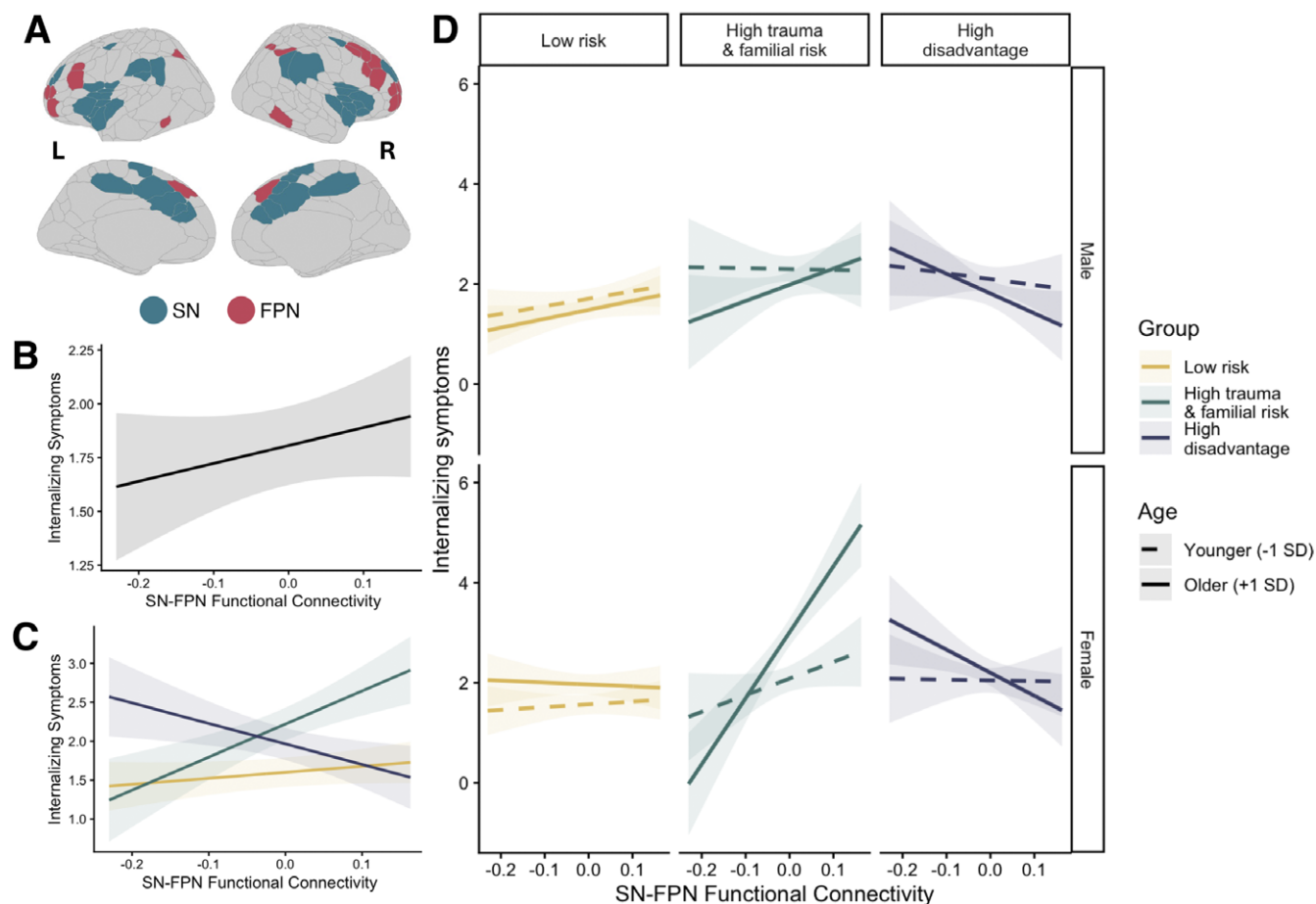


Figure 3. Associations between salience-frontoparietal network connectivity and internalizing symptoms differed across childhood environment groups. (a) Surface-based illustration of brain regions that were included in the salience network (SN) and frontoparietal Network (FPN). (b) The associations between SN-FPN functional connectivity and internalizing symptoms were not observed in the full sample, suggesting that aggregated data across all youth may obscure meaningful neural correlates of psychopathology. (c) The associations between SN-FPN functional connectivity and internalizing symptoms differed by child environment groups, suggesting that neural correlates of internalizing risk may vary as a function of the broader environment. (d) Group differences were larger in female youth and more pronounced in older individuals.

Results showed no significant group differences for SN-FPN coupling with attentional problems (group $\times$ SN-FPN: $F(2, 10,891) = 2.28, p = .10$) (Supplemental Table 15), suggesting that observed group differences may be relatively specific to internalizing symptoms. To further refine aspects of internalizing symptoms associated with SN-FPN connectivity, we examined anxiety and depressive symptoms. The coupling pattern for SN-FPN connectivity and broad internalizing symptoms was more similar to the pattern for depressive symptoms than for anxiety symptoms: correlations of model test statistics for SN-FPN-related terms showed a moderate link between the internalizing and depression models ($r = .52$), but little correspondence between the internalizing and anxiety models ($r = -.05$). These findings suggest that group differences in SN-FPN and internalizing coupling may be driven more strongly by depressive symptoms compared to anxiety symptoms in this sample.

To better characterize how SN-FPN connectivity relates to affective and cognitive processes, we explored whether group differences in the relationship between SN-FPN connectivity and behavior were also observed for indices of emotion regulation and executive functioning. Results showed that higher SN-FPN connectivity was associated with greater emotional suppression ($b = 92.63, q < .001$) and lower performance on executive function tasks (inhibitory control: $b = -28.85, q = .003$; attention shifting:

$b = -30.44, q = .007$), specifically for individuals in the high disadvantage group (Supplemental Table 14).

Taken together, these findings further demonstrate the environmental-context specificity in brain-behavior link: higher SN-FPN connectivity was associated with higher internalizing symptoms driven by depressive symptoms in the trauma and familial risk group; in the high disadvantage group, higher SN-FPN connectivity was associated with lower overall internalizing symptoms, but greater emotional suppression and lower executive functioning.

Discussion

In a large longitudinal, nationally representative cohort of youth, we found that mental health trajectories and brain-symptom associations differed by person-centered environmental context. Youth with moderate resources but high trauma and familial risk showed the highest symptoms over time, especially in female youth. Moreover, the link between salience and frontoparietal network connectivity with internalizing symptoms varied across child environment groups, but was not observed in the entire sample. Results suggest that the roles of these brain networks may differ across individual contexts and may become obscured without parsing environmental heterogeneity. Collectively, these findings highlight the need to

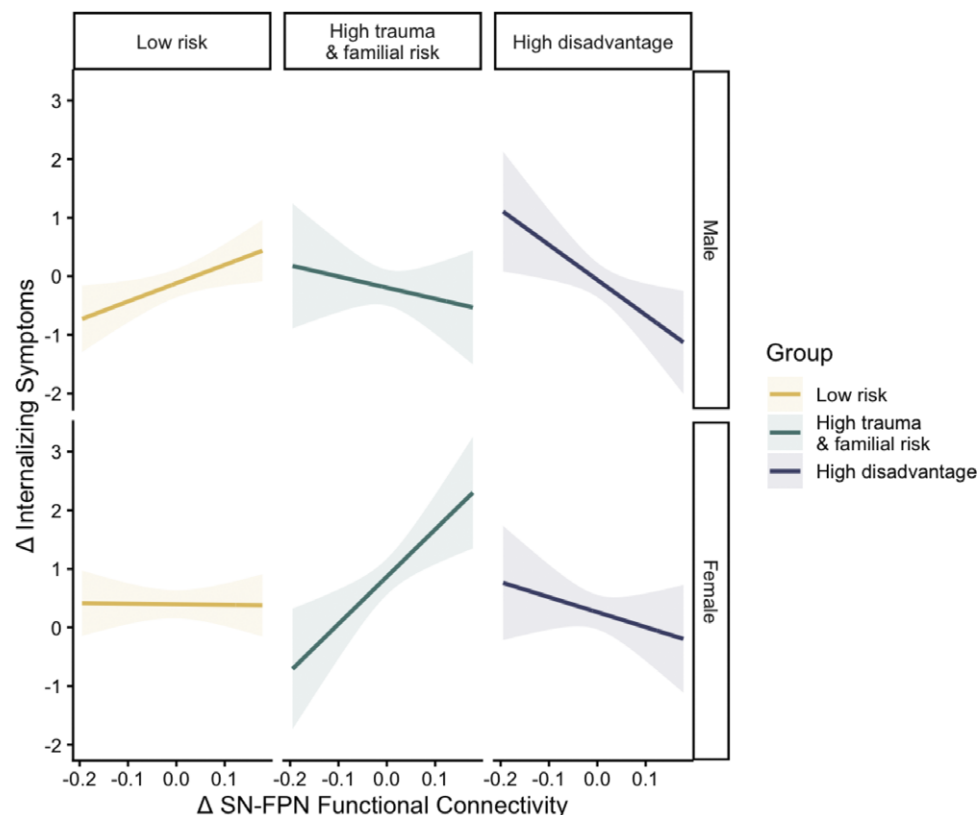


Figure 4. Associations between change in functional connectivity and change in internalizing symptoms across childhood environment groups. Within-person increases in SN-FPN connectivity were differentially associated with within-person changes in internalizing symptoms across groups. There was a positive association between change in SN-FPN and change in internalizing symptoms, particularly for female youth in the high trauma and familial risk group, and a negative association between change in SN-FPN and change in internalizing symptoms for male youth in the high disadvantage group.

consider environmental context when examining neural correlates of psychiatric risk.

The specificity of brain-symptom associations across childhood environment groups suggests that the same neural pattern may serve different functions across contexts. In particular, functional connectivity between the SN and FPN showed distinct associations with internalizing symptoms across identified groups. Aberrant communication among these networks has long been implicated in psychiatric risk (Menon, 2011), with the SN playing a key role in detecting and integrating salient cues and facilitating the switch between internally- and externally-oriented networks (Menon & Uddin, 2010), and the FPN supporting goal-directed control of cognition and emotion (Zanto & Gazzaley, 2013). Moreover, the SN-FPN connectivity and internalizing symptoms link appeared to align more closely with depressive phenotypes, consistent with studies implicating altered connectivity among SN and FPN with depression in clinical samples (Kaiser, Andrews-Hanna, Wager, & Pizzagalli, 2015; Lynch et al., 2024). Notably, while stronger SN-FPN connectivity was associated with greater internalizing symptoms in the high trauma and familial risk group, stronger SN-FPN connectivity was associated with lower internalizing symptoms but also with greater emotional suppression and lower executive functioning in the high disadvantage group. These findings suggest the possibility that stronger coupling of these networks may reflect regulatory processes that are contextually beneficial for some affective outcomes but cognitively costly (Richards & Gross, 2000), but only for youth in highly disadvantaged contexts. Collectively, these findings suggest that the same connectivity pattern

may index emotion and cognitive processes that are context-dependent and not uniformly adaptive. Future studies should examine whether this context specificity extends to neural pathways explaining environmental influences on psychopathology.

Importantly, these brain/behavior associations were not observed when aggregating across the entire sample, suggesting that these network-symptoms associations were specific to youth with particular environmental characteristics. This pattern reflects how associations evident within subgroups can be obscured when data from heterogeneous populations are aggregated (Kievit, Frankenhuis, Waldorp, & Borsboom, 2013). More broadly, our results underscore the strength of data-driven subgrouping for parsing heterogeneity in the environment, particularly for revealing biologically meaningful associations that are masked in heterogeneous samples. Such masking may help explain inconsistencies in prior research that focused on examining biomarkers of psychopathology without explicitly modeling environmental context. This is especially relevant given the increasing availability of large datasets that allow for greater statistical power to identify biological correlates of functioning (Fair et al., 2021). Our findings demonstrate that without consideration of the environmental context, meaningful markers of psychiatric risk may remain elusive.

Findings of the present study also underscore the importance of developmental timing and sex. Adolescence is a period of rapid refinement of large-scale brain networks (Marek et al., 2015; Sherman et al., 2014), and their roles may vary by sex (Satterthwaite et al., 2015). Consistent with this, we found that differences in the associations of SN-FPN connectivity with internalizing in the high

trauma and familial risk versus the high disadvantage groups became more pronounced with age, with the most consistent group differences observed in female youth. These findings are consistent with studies showing that environmental influences accumulate over time (Hardi, Peckins, et al., 2024) and that vulnerability to trauma and familial risk may be salient to female youth in particular (Fergusson, Horwood, & Lynskey, 1995). Moreover, the additional group differences in select network and symptom associations specific to particular ages and/or one sex further indicate a complex variation by development and sex in the neural correlates of psychopathology.

Despite moderate economic resources, youth exposed to high trauma and familial risk showed the greatest risk for symptoms over time. Notably, these youth showed poorer mental health than more economically disadvantaged peers, suggesting that financial resources alone may not fully mitigate the impact of trauma and family stress. This aligns with prior research identifying trauma and familial risk as major contributors to child psychopathology (Hardi, Beltz, et al., 2024; Repetti, Taylor, & Seeman, 2002), and that economic hardship impacts children's development through downstream effects on parenting and family functioning (Masarik & Conger, 2017; McLoyd, 1998). These results underscore the importance of family-level interventions, especially in economically disadvantaged contexts (Lyon & Budd, 2010).

Youth in the high-disadvantage group, which included a higher proportion of youth from racial/ethnic minority backgrounds, reported relatively greater access to certain social resources (familism, positive peer networks, and community centers). These resources are examples of protective factors that can promote youth well-being, particularly for marginalized youth (Campos, Ullman, Aguilera, & Dunkel Schetter, 2014; Li, Nussbaum, & Richards, 2007). Social connections fostered through community engagement can reduce risks for maladaptive behaviors, particularly in contexts where parental support may be hindered due to financial constraints (Germán, Gonzales, & Dumka, 2009; Jose, Ryan, & Pryor, 2012; Richard et al., 2022). This may partly explain why youth in the highly disadvantaged group had lower symptoms than those with moderate resources but high trauma and familial risk. Presently, it remains unclear whether these protective factors were driving the observed differences or whether the lower trauma and familial risk underlie them. Likely, both risk and protective factors contribute to these divergent outcomes, highlighting the need for further research on their interplay.

It is important to consider this work in the context of several limitations. First, the ABCD Study does not include comprehensive information during early childhood (before age 9); thus, we are unable to fully account for individual differences in some early experiences. Second, although we used a multi-informant (parent, youth, census) approach, there was no information on environments from other sources, such as teachers or other caregivers (e.g., grandparents), who may provide other types of information about the school and family environments. Third, although resting-state functional connectivity is an established correlate of psychopathology (Woodward & Cascio, 2015; Xia et al., 2018) and intrinsic functional networks during tasks can be highly similar to resting-state architecture (Cole et al., 2014; Gratton et al., 2018), future studies should examine whether task-evoked brain-behavior coupling shows similar or distinct patterns across these groups. Fourth, although the effect sizes in the present study are comparable to the expected magnitude of brain-behavior associations (Dick et al., 2021; Marek et al., 2022), more research is needed in other samples, particularly to further examine the age- and sex-specific, as well as

potential longitudinal, effects. Lastly, there could be other experiences or environmental factors that may not be accounted for (e.g. other exposome measures capturing comprehensive exposures to pollutants) that may provide meaningful information about the child environment (Robinson et al., 2025). However, our expansive measures of 30 different variables were captured across multiple levels through different modalities (survey and administrative data) in a large nationally-representative sample.

Conclusions

Leveraging a person-centered approach, this study captures the complex and context-dependent nature of youth mental health and its neural correlates. By integrating multi-level environmental data with neuroimaging and mental health symptom trajectories, we identified groups of youth whose mental health and brain connectivity correlates differed as a function of environmental context. These findings demonstrate how the same neural features may confer risk versus resilience depending on context, underscoring the importance of personalized, context-sensitive approaches for developing interventions that address youth mental health.

Supplementary material. The supplementary material for this article can be found at <http://doi.org/10.1017/S0033291726104115>.

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