Associations among Household and Neighborhood Socioeconomic Disadvantages, Resting-state Frontoamygdala Connectivity, and Internalizing Symptoms in Youth

Ka I. Ip[1], Lucinda M. Sisk[1], Corey Horien[2], May I. Conley[1], Kristina M. Rapuano[1], Monica D. Rosenberg[3], Abigail S. Greene[2], Dustin Scheinost[2], R. Todd Constable[2], BJ Casey[1], Arielle Baskin-Sommers[1], and Dylan G. Gee[1]

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Abstract

■ Exposure to socioeconomic disadvantages (SED) can have negative impacts on mental health, yet SED are a multifaceted construct and the precise processes by which SED confer deleterious effects are less clear. Using a large and diverse sample of preadolescents (ages 9–10 years at baseline, n = 4038, 49% female) from the Adolescent Brain Cognitive Development Study, we examined associations among SED at both household (i.e., income–needs and material hardship) and neighborhood (i.e., area deprivation and neighborhood unsafety) levels, frontoamygdala resting-state functional connectivity, and internalizing symptoms at baseline and 1-year follow-up. SED were positively associated with internalizing symptoms at baseline and indirectly predicted symptoms 1 year later through elevated symptoms at baseline. At the household level, youth in households characterized by higher disadvantage (i.e., lower income-to-needs ratio) exhibited more strongly negative frontoamygdala coupling, particularly between the bilateral amygdala and medial OFC (mOFC) regions within the frontoparietal network. Although more strongly positive amygdala–mOFC coupling was associated with higher levels of internalizing symptoms at baseline and 1-year follow-up, it did not mediate the association between income-to-needs ratio and internalizing symptoms. However, at the neighborhood level, amygdala–mOFC functional coupling moderated the effect of neighborhood deprivation on internalizing symptoms. Specifically, higher neighborhood deprivation was associated with higher internalizing symptoms for youth with more strongly positive connectivity, but not for youth with more strongly negative connectivity, suggesting a potential buffering effect. Findings highlight the importance of capturing multilevel socioecological contexts in which youth develop to identify youth who are most likely to benefit from early interventions. ■

INTRODUCTION

Socioeconomic disadvantages (SED), operationalized here as lower household and neighborhood socioeconomic resources and neighborhood unsafety, are social determinants of health embedded in the living conditions of individuals that can result in chronic activation of the stress response (Amaro, Sanchez, Bautista, & Cox, 2021; Braveman, Egerter, & Williams, 2011). Although extensive research has demonstrated that exposure to SED early in life can have profound and lasting impact on mental health (Baranyi, Di Marco, Russ, Dibben, & Pearce, 2021; Peverill et al., 2021; Farah, 2017; Goldman-Mellor, Margerison-Zilko, Allen, & Cerda, 2016; Johnson, Riis, & Noble, 2016; Reiss, 2013; Hackman, Betancourt, Brodsky, Hurt, & Farah, 2012), including elevated internalizing problems (e.g., Demidenko et al., 2021), the precise processes by which SED confer deleterious effects are less clear (Peverill et al., 2021; Farah, 2017). The overall goal of this study is to examine the direct, indirect, and moderating associations among household-related (i.e., income-to-needs ratio and material hardship) and neighborhood-related (i.e., neighborhood unsafety and area deprivation) SED, frontoamygdala resting-state functional connectivity (rs-FC), and internalizing symptoms during preadolescence.

Alterations in frontoamygdala circuitry are one potential factor linking SED with the development of internalizing psychopathology. Throughout development, dynamic interactions between the amygdala and various regions of the PFC play a central role in down-regulating negative affect (Silvers et al., 2017; Tottenham & Galván, 2016; Silvers, Shu, Hubbard, Weber, & Ochsner, 2015; Kim et al., 2011) and fear extinction (Gold et al., 2020; Gee et al., 2018; Milad & Quirk, 2012). Although the directionality of findings is sometimes inconsistent, alterations in functional connectivity between the amygdala and the

This article is part of a Special Focus entitled Finances and Feelings: The Affective Neuroscience of Poverty; deriving from a symposium at the 2020 Annual Meeting of the Cognitive Neuroscience Society.

[1]Yale University, New Haven, CT, [2]Yale School of Medicine, New Haven, CT, [3]University of Chicago

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ventral medial PFC (vmPFC) extending to the subgenual ACC (sgACC; BA 25; Marusak et al., 2016) at rest have been associated with heightened anxiety and depression symptoms among youth (Jalbrzikowski et al., 2017; Marusak et al., 2016; Pagliaccio et al., 2015; Connolly et al., 2013; Herringa et al., 2013; Burghy et al., 2012; Hahn et al., 2011). Developmentally, functional connections between the vmPFC and amygdala appear to undergo marked changes throughout childhood and adolescence (Brieant, Sisk, & Gee, 2021; Jalbrzikowski et al., 2017; Gabard-Durnam et al., 2014, 2016; Wu et al., 2016; Gee, Humphreys, et al., 2013; Perlman & Pelphrey, 2011). Although the precise nature of these changes and the directionality of connectivity remains unclear, some evidence suggests that a shift toward more strongly negative coupling within this circuitry may reflect neural maturation of top–down processes to regulate negative affect (Silvers et al., 2015, 2017; Wu et al., 2016; Gee, Humphreys, et al., 2013). For the sake of clarity, we use more strongly positive connectivity to refer to a stronger positive correlation between two nodes and more strongly negative connectivity to refer to a stronger negative correlation (i.e., anticorrelation) between two nodes.

Importantly, the vmPFC is a heterogeneous region encompassing different subregions, including BA 11, BA 25, and ventral parts of BA 24 and BA 32, that support heterogeneous functions (Delgado et al., 2016; Myers-Schulz & Koenigs, 2012) and likely reflect different connectivity networks (Shen, Tokoglu, Papademetris, & Constable, 2013). For example, the anterior part of medial OFC (mOFC; BA 11) is part of the frontoparietal network (FPN) implicated in cognitive control (Cole, Yarkoni, Repovš, Anticevic, & Braver, 2012), whereas the posterior part of mOFC, anterior (BA 10) and ventral medial part of PFC, and rostral part of the ACC (BA 32) are part of the default mode network (DMN) implicated in internal mental-state processes, such as self-referential processing, autobiographic memory retrieval, or future thinking (Raichle, 2015). Given that the FPN is implicated in cognitive control (Cole et al., 2012), amygdala connectivity with vmPFC subregions of the FPN may be more strongly linked with internalizing symptoms than amygdala connectivity with vmPFC subregions of the DMN. Furthermore, it is unclear whether amygdala connectivity with vmPFC subregions that are functionally heterogeneous and/or do not belong to the same functional connectivity networks (i.e., FPN vs. DMN) may differentially relate to SED and internalizing symptoms during preadolescence.

Three additional gaps hinder our understanding of the links between SED, frontoamygdala development, and internalizing symptoms. First, although SED are one of the most common chronic stressors across diverse populations and geographical locations, the generalizability of prior findings is often limited by reliance on samples with limited ethnic–racial, socioeconomic, and geographic variability.

Second, SED are often indexed using a single factor (e.g., income-to-needs ratio) that does not capture the multilevel ecological contexts in which children develop (Hyde et al., 2020; Sameroff, 2010). For example, among youth, lower household income was associated with more negative amygdala–vmPFC resting-state coupling (Hanson et al., 2019) and reduced amygdala connectivity with multiple regions (e.g., superior frontal cortex, lingual gyrus; Brody et al., 2019; Barch et al., 2016). Naturally, some SED (e.g., lower income-to-needs ratio and higher area deprivation and neighborhood unsafety) are more likely to co-occur, and children from more disadvantaged households also disproportionately live in neighborhoods with lower resources and safety (Wheaton & Clarke, 2003). It is, however, unclear how the cumulative effect of SED and SED at different ecological levels relate to resting-state frontoamygdala circuitry and internalizing symptoms among preadolescents, given that few studies have simultaneously examined these factors (Rakesh, Seguin, Zalesky, Cropley, & Whittle, 2021). Examining shared variance across distinct types of SED is consistent with prior evidence that cumulative risk is more strongly associated with internalizing problems than singular risk exposure (Evans, Li, & Whipple, 2013).

At the same time, SED are a multifaceted construct, and distinct types of SED may pose different environmental risks and compromise access to different types of resources (Farah, 2017). Household-level SED such as material hardship and lower income-to-needs ratio may be more closely related to risk for exposure to deprivation (e.g., lower cognitive stimulation and nutrition; Rosen et al., 2020; Sheridan & McLaughlin, 2014). By contrast, certain neighborhood-level SED such as unsafe neighborhoods may be characterized by greater threat (e.g., community violence; McLaughlin & Sheridan, 2016), and higher neighborhood/area deprivation may be associated with both deprivation and threat (e.g., higher neighborhood deprivation often involves higher densities of household-level SED, along with environmental toxins, violence, lower community cohesion, and resources; Browning & Cagney, 2003). Examining the ways in which heterogeneity in SED relates to frontoamygdala connectivity is important for two primary reasons.

Contemporary theories have proposed key dimensions of adversity exposure, such as threat and deprivation, that may differentially associate with neurodevelopment (see McLaughlin, Weissman, & Bitrán, 2019, for a review). Moreover, emerging evidence suggests that the effects of neighborhood-level disadvantages on corticolimbic development may be dissociated from household-level disadvantages (Gard et al., 2021; Rakesh et al., 2021; Ramphal et al., 2020; Tomlinson et al., 2020; Marshall et al., 2018). In a sample of participants aged 5–25 years with and without attention-deficit/hyperactivity disorder, Ramphal et al. (2020) found that amygdala–vmPFC connectivity moderated associations between neighborhood deprivation (as assessed by the area deprivation index [ADI]) and internalizing symptoms, but not with household socioeconomic status. However, whether increased or

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decreased amygdala–vmPFC connectivity was associated with higher internalizing symptoms depended on age and level of neighborhood deprivation, and the inconclusive findings may stem from the limited sample size, broad age range, and lack of differentiating amygdala connectivity with distinct vmPFC subregions. Collectively, these findings highlight the need to examine both the shared (i.e., cumulative) and distinct effects of household- and neighborhood-level SED on frontoamygdala circuitry and internalizing symptoms during preadolescence.

Third and finally, theories have postulated two plausible ways that SED may be associated with brain and behavioral functioning: (1) a mediation model by which brain function mediates the effects of SED on behavioral outcomes through biological alterations of brain function (e.g., glucocorticoid release, synapse formation, and pruning; Hackman, Farah, & Meaney, 2010) or (2) a moderation model by which brain function moderates the effects of SED on behavioral outcomes (Ramphal et al., 2020; Farah, 2017). To date, associations between SED and other forms of early adversity, brain function, and mental health outcomes have been primarily tested in a mediation framework (e.g., Barch et al., 2016; Herringa et al., 2013; Burghy et al., 2012). However, emerging studies have suggested that a moderation approach may be better suited (Ramphal et al., 2020). No study, thus far, has examined how these models may differentially fit in a large-scale, population-based data set.

The Current Study

Using a large and diverse sample of preadolescents from the longitudinal Adolescent Brain and Cognitive Development Study (ABCD Study), the current study aims to examine the direct, indirect, and moderating roles of household-related (i.e., income-to-needs ratio and material hardship) and neighborhood-related (i.e., neighborhood unsafety and area deprivation) SED and their associations with frontoamygdala rs-FC (i.e., amygdala–vmPFC coupling) and internalizing symptoms at baseline (9–10 years old) and 1-year follow-up. Given heightened risk for anxiety and depression during adolescence (Kessler et al., 2012; Merikangas et al., 2010), we focus on internalizing symptoms during this developmental period to identify youth who are at risk for the emergence of internalizing problems, which could foreshadow long-term negative outcomes in adulthood (Beesdo, Knappe, & Pine, 2009).

Our first aim was to examine the shared (i.e., cumulative) and distinct associations among various forms of SED (i.e., income-to-needs ratio, material hardship, neighborhood unsafety, neighborhood area deprivation), frontoamygdala rs-FC, and internalizing symptoms among preadolescents at baseline and 1-year follow-up using structural equation modeling and mediation analysis. We hypothesized that, first, higher levels of SED would be associated with more strongly negative frontoamygdala rs-FC, given a prior study showing that lower household income was associated with more negative amygdala–vmPFC resting-state coupling (Hanson et al., 2019). Second, we hypothesized that higher levels of SED would be associated with higher internalizing symptoms (direct effect) at baseline and 1-year follow-up. Third, we hypothesized that frontoamygdala rs-FC would be associated with internalizing symptoms. No directional hypothesis was made regarding whether positive or negative frontoamygdala rs-FC would be associated with higher internalizing symptoms given prior mixed findings (Hanson et al., 2019; Jalbrzikowski et al., 2017; Pagliaccio et al., 2015; Connolly et al., 2013; Herringa et al., 2013; Burghy et al., 2012; Hahn et al., 2011). Finally, we hypothesized that higher SED would be indirectly associated with elevated internalizing symptoms through frontoamygdala rs-FC among preadolescents.

Our second aim was to test an alternative moderation model in which frontoamygdala rs-FC moderates the effect of SED (both shared and distinct effects) on internalizing symptoms (no directional hypothesis given prior mixed findings; Ramphal et al., 2020). Given prior mixed findings on effects of amygdala–vmPFC resting-state connectivity (Hanson et al., 2019; Jalbrzikowski et al., 2017; Pagliaccio et al., 2015; Connolly et al., 2013; Herringa et al., 2013; Burghy et al., 2012; Hahn et al., 2011), we also explored effects of amygdala functional coupling with distinct subregions of the vmPFC for Aims 1 (mediation model) and 2 (moderation model). Although we aimed to examine both shared and distinct effects of SED, we did not make specific predictions about the shared versus distinct effects of SED given insufficient evidence in the existing literature and the exploratory nature of this approach.

METHODS

Participants

Participants in the current study are drawn from the ongoing ABCD Study—a 10-year longitudinal study of over 11,000 youth ages 9 and 10 years at baseline across 21 sites (Garavan et al., 2018; Volkow et al., 2018). A subset of 5772 participants with available resting-state data downloaded from ABCD Fast Track (April 2018) were included in this study (see additional imaging exclusion criteria below). The ABCD Study's primary motivation was to characterize brain and behavioral development throughout adolescence development (Volkow et al., 2018) from a diverse community sample of preadolescents (Lisdahl et al., 2018). To approximate the diversity of the United States on sex, ethnicity/race, and urbanicity and to minimize systematic bias in sampling, preadolescents (ages 9–10 years) were recruited using a stratified probability sampling of elementary schools at the 21 recruitment sites across the United States (https://abcdstudy.org/study-sites/). A detailed description of the motivation for the ABCD Study (Volkow et al., 2018), the recruitment and

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sampling procedures (Garavan et al., 2018), and the cultural/environmental variables (Zucker et al., 2018) is described elsewhere.

Parental consent and child assent were obtained from all participants and approved by institutional review boards at each data collection site. Study-wide exclusionary criteria were as follows: child not fluent in English; major neurological disorder or certain seizure disorders (Lennox–Gastaut syndrome, Dravet syndrome, and Landau Kleffner syndrome); an intellectual disability; gestational age of less than 28 weeks or birth weight of less than 1200 g; history of traumatic brain injury; MRI contraindication (e.g., irremovable ferromagnetic implants or dental appliances, claustrophobia, and pregnant); or have a current diagnosis of schizophrenia, moderate-to-severe autism spectrum disorder, or alcohol/substance use disorder. The current study includes baseline (9–10 years old) and 1-year follow-up assessment time points of the ABCD Study. Behavioral data are drawn from Release 3.0 (DOI 10.15154/1519007; https://nda.nih.gov/study.html?id=901) of the ABCD Study, and raw dicom images were downloaded via ABCD Fast Track (April 2018; see additional imaging exclusion criteria below). The final *n* of the current study is 4038 participants (49% female; 61.3% White, 8.2% Black, 18.4% Latinx, 2.2% Asian, and 9.9% from other ethnic–racial backgrounds including Native Hawaiian, Pacific Islander, Alaskan Native, American Indian, and multiracial). Most parents (73.2%) reported being married, with 1% widowed, 9.2% divorced, 3.2% separated, 8.1% never married, 4.9% living with a partner, and 0.4% not reported.

Sociodemographic and Behavioral Measures

SED at Baseline

Income-to-needs ratio. The income-to-needs ratio is a commonly used measure of household socioeconomic status (Rosen, Sheridan, Sambrook, Meltzoff, & McLaughlin, 2018) that captures the amount of annual household income relative to the federally defined poverty threshold for a given family size. Parents reported on combined household income (measured in deciles) in the past 12 months by selecting an income category ranging from (1) "less than $5000" to (10) "$200,000 and greater" at baseline. The median of the income category was used for Categories 2–9. For members in a household, parents self-reported the number of individuals living or staying at their address for more than 2 months (excluding visiting college students or Armed Forces on deployment). Income-to-needs ratio was calculated by dividing the total household income by the 2017 U.S. Census Bureau guideline of poverty threshold for a family of that size, with a value of 1 or lower indicating income below the poverty line.

Material hardship. Material hardship is based on parental responses of yes (1) or no (0) to the following seven questions in the ABCD Longitudinal Parent Demographic Survey at baseline: (1) "needed food but couldn't afford to buy it or couldn't afford to go out to get it?"; (2) "were without telephone service because you could not afford it?"; (3) "didn't pay the full amount of the rent or mortgage because you could not afford it?"; (4) "were evicted from your home for not paying the rent or mortgage?"; (5) "had services turned off by the gas or electric company, or the oil company wouldn't deliver oil because payments were not made?"; (6) "had someone who needed to see a doctor or go to the hospital but didn't go because you could not afford it?"; and (7) "had someone who needed a dentist but couldn't go because you could not afford it?". A sum score of the seven items was used, and higher scores indicate greater hardship. This measure was used in recent investigations with the ABCD sample (Karcher, Shiffman, & Barch, 2021; Taylor, Cooper, Jackson, & Barch, 2020) and is comparable to the established construct of material hardship that captures food, medical, or residential insecurity.

ADI. The Area Community Service ADI uses census block group-level data to contextualize neighborhood-level SED (Kind & Buckingham, 2018) at baseline. Index scores are derived from 17 measures related to education (e.g., percentage of population >25 years old with <9 years of education), employment (e.g., percentage of employed persons >16 years old in white collar occupations), housing equality (e.g., median home value, percentage of owner-occupied housing units), income (e.g., percentage of families below the poverty level), single-parent households, and living conditions (e.g., percentage of occupied housing units without a motor vehicle, percentage of occupied housing units with >1 person per room) within a 5-year period. The scaled weighted sum scale based on Kind and Buckingham (2018) was reported, where higher scores indicate more disadvantage.

Neighborhood unsafety. A PhenX Toolkit measure (three items) derived from the Neighborhood and Crime Safety Scale (Echeverria, Diez-Roux, & Link, 2004) assesses parental self-report of neighborhood safety at baseline. Parents rated statements such as "My neighborhood is safe from crime" on a 5-point Likert scale ranging from (1) *strongly disagree* to (5) *strongly agree*. Items were averaged and the score was reverse-coded, such that higher scores indicated higher perception of neighborhood unsafety.

Child Internalizing Symptoms

Parental report on the Child Behavior Checklist (Achenbach & Rescorla, 2001) was used to assess children's internalizing symptoms using the broadband internalizing subscale (33 items) at baseline and 1-year follow-up. Parents rated their child's withdrawn, somatic, anxious, and depressive symptoms on a 3-point Likert-type scale from

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(0) *never* to (2) *often*. Parents' ratings were used to create an internalizing problem summary score. Raw scores at each wave were used in all analyses. Higher scores indicated higher levels of internalizing symptoms.

Covariates

Child age (in months), sex, and scanner type (three scanner types coded as two dummy variables) were included as covariates in all analyses.

MRI and Resting-state fMRI Data Acquisition

MRI acquisition. For additional information regarding MRI and resting-state data acquisition in the ABCD Study, see Casey et al. (2018). Scans were performed on different 3-T scanners from Siemens (Prisma VE11B-C, Siemens Medical Systems), Philips (Achieva dStream, Ingenia, Philips Medical Systems), or General Electric (MR750, DV25-26, General Electric) with 32-channel head coils. At each site, children were acclimated to the scanner environment through either a mock scanner or a play tunnel that was the size of the scanner bore. To encourage motion compliance in the scanner, behavioral shaping was used to monitor head motion (Epstein et al., 2007) and provide feedback to the child. Additionally, to minimize head motion in the scanner, the head was stabilized with foam padding.

The scan session consisted of a fixed order beginning with a localizer, acquisition of 3-D T-weighted images, two runs of resting-state fMRI (rs-fMRI), T2-weighted images, one to two runs of rs-fMRI, and task-based fMRI. A T1-weighted (T1w) anatomical scan was acquired using the following parameters: Siemens: repetition time (TR) = 2500 msec, echo time (TE) = 2.88 msec, inversion time (TI) = 1060 msec, flip angle = 8°, 176 transverse slices; Philips: TR = 6.3 msec, TE = 2.9 msec, TI = 1060 msec, flip angle = 8°, 225 transverse slices; General Electric: TR = 2500 msec, TE = 2 msec, TI = 1060 msec, flip angle = 8°, 208 transverse slices. Voxel size was 1 × 1 × 1 mm for all three scanner types. Functional images were collected through 60 slices in the axial plane using EPI sequence with the following parameters: TR = 800 msec, TE = 30 msec, flip angle = 52°, voxel size = 2.4 mm^3, multiband slice acceleration factor = 6.

Participants completed up to four runs of 5-min rs-fMRI. We included one resting-state run for each participant in the analyses. Mean framewise displacement was computed for each resting-state run, and participants with at least one resting-state scan with mean framewise displacement under a motion threshold of 0.15 mm were included in the analysis to maximize sample size (Rapuano et al., 2020; Horien, Shen, Scheinost, & Constable, 2019; Greene, Gao, Scheinost, & Constable, 2018).[1] For participants who had more than one run with mean framewise displacement under the 0.15-mm threshold, we selected the run with the lowest mean framewise displacement for use in the analyses (see below). Additionally, to reduce head motion artifact, ABCD sites with Siemens scanners used Framewise Integrated Real-time MRI Monitoring (Dosenbach et al., 2017) to detect head motion in real time.

Resting-state Preprocessing

Rs-fMRI data from 5772 participants were downloaded as raw dicom images via ABCD Fast Track (April 2018; see Sisk et al., 2022; Rapuano et al., 2020) and preprocessed using BioImage Suite (Joshi et al., 2011) following the processing steps described in detail elsewhere (Horien et al., 2019; Greene et al., 2018). T1-weighted anatomical images were skull stripped using optiBET (Lutkenhoff et al., 2014)—a modified version of FMRIB Software Library's brain extraction tool (Smith, 2002) and nonlinearly registered to Montreal Neurological Institute (MNI) stereotaxic space using B-spline free form deformation. Functional images were realigned to correct for motion, nonlinearly registered to MNI space, and anatomically parcellated using a 368-node whole-brain atlas, which was created by combining different delineations of brain regions (see Horien et al., 2019, for a detailed description). For the cortex, a group-wise parcellation algorithm (Shen, Papademetris, & Constable, 2010) was applied and obtained 164 and 163 nodes in the left and right hemispheres, respectively. This parcellation approach is based on correlated BOLD signals (rather than cytoarchitecture or anatomic distinction) across time courses to define and extract nodes to ensure functional homogeneity within each node and that node definitions are consistent across participants (Shen et al., 2010).

For the subcortical area (i.e., amygdala), anatomic definitions of subcortical structures were used (Lacadie, Fulbright, Arora, Constable, & Papademetris, 2008). Covariates of no interest were regressed from the data, including linear, quadratic, and cubic drifts, 24 motion parameters (Satterthwaite et al., 2013), mean cerebral–spinal fluid signal, mean white matter signal, and overall global signal. Data were temporally smoothed with a Gaussian filter, $\sigma = 1.95$ (approximate cutoff frequency of 0.12 Hz). Pearson correlation coefficients between time courses for every pair of nodes were computed, followed by Fisher z transformation, resulting in a 368 × 368 functional connectivity matrix for each participant.

A total of 4163 participants had at least one rs-fMRI run with mean framewise displacement of <0.15 mm. After additional exclusion of participants who did not pass the quality check of anatomical images by FreeSurfer (ABCD NDA name: *fsqc_qc*, $n = 124$) or had missing data for covariates ($n = 1$), the final sample included 4038 participants with usable data.

We reported results from available data ($n = 4038$) to maximize power; however, to ensure that our models and results were not driven by artifacts in the neural data, we conducted additional sensitivity analyses with a stricter

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inclusion criterion following the recommendations from the ABCD Consortium with a subset of participants (n = 3864). The results were similar when applying these stricter inclusion/exclusion criteria. Hence, we only reported these results in Appendices M–X.

Functional Connectivity Node Extractions

Our goal was to examine associations between SED, frontoamygdala rs-FC, and internalizing symptoms in preadolescents and to examine differential relations with connectivity in vmPFC subcomponents within different connectivity networks (i.e., frontoparietal, default mode, and limbic networks). From the 368 × 368 functional connectivity matrix, we extracted frontoamygdala rs-FC values from nine different nodes within bilateral vmPFC/sgACC regions (see Table 1 and Figure 1) based on (a) prior findings that these areas are linked to internalizing symptoms and/or early adversity (Marusak et al., 2016; Thomason et al., 2015; Burghy et al., 2012; Hahn et al., 2011; Kim et al., 2011; Table 1) and (b) these areas are all located within the vmPFC mask as defined in the Harvard-Oxford atlas (Makris et al., 2006). PCA with nonorthogonal promax rotation was first performed to examine whether edges between each of the vmPFC nodes and the amygdala could be loaded onto different components. PCA indicated a three-factor solution (see Table 1 and Figure 1). The first factor consisted of amygdala–mPFC/ACC, amygdala–sgACC, and amygdala–posterior mPFC connectivity nodes in the right hemisphere, which we labeled as "amy-mPFC/ACC–R rs-FC." The vmPFC nodes in this component belong to the DMN. The second factor consisted of amygdala connectivity with similar regions, which we labeled as "amy-mPFC/ACC–L rs-FC." The vmPFC nodes in this component belong to the DMN. The third factor consisted of bilateral amygdala–anterior mOFC connectivity nodes, which we labeled as "amy-mOFC rs-FC." The vmPFC nodes in this component belong to the FPN. Each component was extracted as a factor score and used in subsequent analyses (see below).

Data Analytical Plan

Before the primary analyses, Pearson correlation and descriptive statistics were first conducted. To examine the shared effect of SED at both household and neighborhood levels, confirmatory factor analysis (CFA) of income-to-needs ratio, material hardship, ADI, and neighborhood unsafety was first conducted to examine whether various SED factors could be loaded onto a latent construct using Mplus 7.2 (Muthén & Muthén, 2012). Following Kline's (2015) suggestion, maximum likelihood estimation with robust standard errors (MLR) that is robust to nonnormality was used to handle missing data given that some variables were skewed (i.e., material hardship > 3). Multiple-fit statistics were reported and interpreted as outlined by Kline (2015): (a) Pearson χ^2, for which nonsignificant values signify good fit and a χ^2/df ratio of <3 is acceptable; (b) comparative fit index (CFI); (c) Tucker–Lewis index (TLI), for which a value of >.90 is considered a good fit; (d) root-mean-square error of approximation (RMSEA), for which a value of <.08 is considered acceptable and <.05 is considered good; and (e) standardized root mean squared residual (SRMR) of <.08. To account for the sampling effect of siblings within a family cluster,

Table 1. PCA Examining Factor Loadings of Amygdala rs-FC with vmPFC Functional Subunits

		MNI						*PCA*		
vmPFC Subunits	*H*	*x*	*y*	*z*	*BA*	*Network*	*Node*	*amy-mPFC/ ACC–R*	*amy-mPFC/ ACC–L*	*amy-mOFC*
1. mPFC/ventral ACC	R	3.2	27.07	−9.86	11	DMN	306	.779		
2. Anterior mPFC/ACC	R	5.92	42.93	−6.2	11	DMN	175	.747		
3. Posterior mOFC	R	4.2	35.58	−20.4	10	DMN	235	.702		
4. mPFC/sgACC	R	14.05	24.31	−20.09	25/11	Limbic	261	.653		
5. mPFC/ACC	L	−6.5	38.3	−4.78	11	DMN	60		.762	
6. Posterior mOFC	L	−6.12	33.13	−18.42	11	DMN	109		.758	
7. mPFC/sgACC	L	−6.5	38.3	−4.78	32	Limbic	39		.757	
8. Anterior mOFC	R	11.12	55.52	−19.09	11	FPN	215			.793
9. Anterior mOFC	L	−10.23	57.23	−17.65	11	FPN	73			.739

Coordinates are presented in MNI space. Node numbers are based on the Shen 368 atlas parcellation, which can be found in BioImage Suite Connectivity Viewer, https://bioimagesuiteweb.github.io/unstableapp/connviewer.html#. Networks are based on network definitions from Noble et al. (2017). PCA identified a three-factor solution explaining 77% of the total variance. amy = amygdala; H = hemisphere; BA = Brodmann's area.

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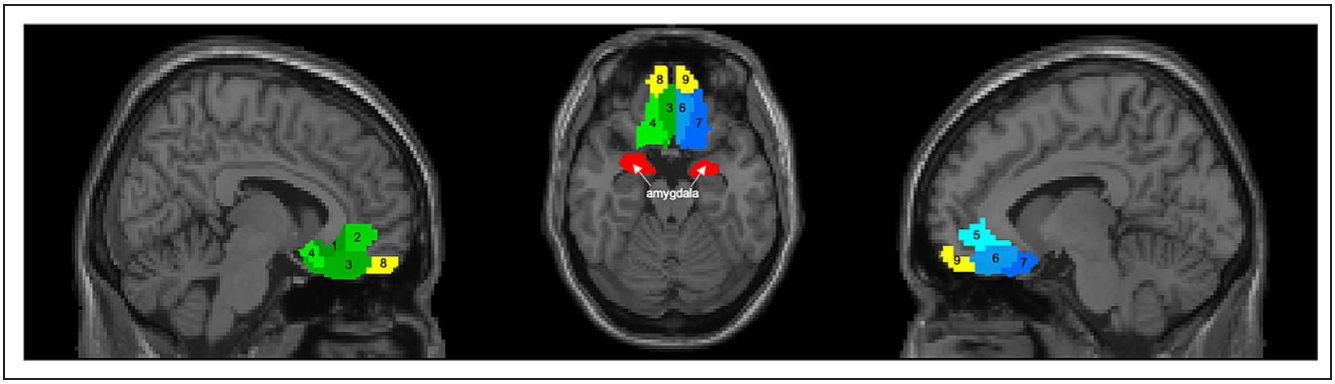

Figure 1. Right-hemisphere sagittal, axial (center), and left-hemisphere sagittal views of ROIs for amygdala–vmPFC rs-FC. The vmPFC parcellations are based on the Shen 368 atlas. The numbers refer to the corresponding vmPFC subunits referenced in Table 1. Regions in shades of green depict Component 1 (amy-mPFC/ACC–R rs-FC). Regions in shades of blue depict Component 2 (amy-mPFC/ACC–L rs-FC). Regions in shades of yellow depict Component 3 (amy-mOFC rs-FC).

all CFA and subsequent structural equation models (SEMs; see below) were estimated using Taylor series linearization using *Type = Complex* in Mplus. To account for the multisite design of the ABCD Study, we specified *Stratification = ABCD site* (to adjust for standard errors and that the chi-square test of model takes into account nonindependent observations due to cluster sampling of study variables) for all models in Mplus.

Our first aim was to examine the role of frontoamygdala rs-FC in a mediation analysis by which SED have a direct effect on frontoamygdala rs-FC and internalizing symptoms, and frontoamygdala rs-FC mediates the effect of SED on internalizing symptoms. Three separate SEM (one for each amygdala–vmPFC rs-FC subcomponent) were conducted to examine the associations between the CFA-identified SED latent construct, frontoamygdala rs-FC, and internalizing symptoms at baseline and 1-year follow-up using Mplus. Mediation/indirect effects were tested using the MODEL INDIRECT command in Mplus. Covariates of child age (in months), sex, and scanner type (coded as two dummy variables based on $k - 1$) were included in all SEMs. Multiple-fit statistics (i.e., χ^2, CFI, TLI, RMSEA, SRMR) were reported and interpreted as outlined by Kline (2015) as described above. Akaike information criterion (AIC), Bayesian information criterion (BIC), and sample size-adjusted BIC (SSABIC) of the models were also reported.

To explore unique and independent effects of each type of household- and neighborhood-level SED (i.e., income-to-needs ratio, material hardship, ADI, neighborhood unsafety), three additional mediation analyses (one for each amygdala–vmPFC rs-FC subcomponent) were conducted to allow each type of SED to covary, and we examined their independent associations with frontoamygdala rs-FC and internalizing symptoms at baseline and at 1-year follow-up. The same covariates and multiple-fit statistics described above were reported.

Our second aim was to examine an alternative moderation model by which frontoamygdala rs-FC moderates the association between SED and internalizing symptoms. Three separate SEM (one for each amygdala–vmPFC rs-FC subcomponent) were performed. An interaction term of SED × frontoamygdala rs-FC was constructed using the XWITH command (for creating interaction terms of latent constructs) with *Type = Random* in Mplus. The same covariates described above were included in the models. Mplus does not provide model fit statistics when using *Type = Random*; therefore, only AIC, BIC, and SSABIC were reported.

To explore unique and independent effects of each type of household- and neighborhood-level SED, three additional moderation models (one for each amygdala–vmPFC rs-FC subcomponent) were conducted to examine the interactive effect of each type of SED × frontoamygdala rs-FC on internalizing symptoms. To prevent multicollinearity, nonsignificant interaction terms were dropped in the final model (to be conservative, we also examined each SED × frontoamygdala rs-FC term in separate models, and results were the same). Significant interaction terms were probed using simple slopes analysis, and partial residuals were plotted using the Visreg package in R to visualize the simple slopes analysis. The same covariates and multiple-fit statistics described above were reported. AIC, BIC, and SSABIC were also reported.

Finally, we conducted sensitivity analyses with a subset ($n = 3864$) of participants who met all inclusion criteria based on recommendations from the ABCD Consortium (see Appendices M–X) with similar models and procedures.

RESULTS

Descriptive statistics and Pearson correlations for primary study variables are reported in Table 2. Lower income-to-needs ratio, higher material hardship, ADI, and neighborhood unsafety were positively associated with each other. All SED variables at baseline were associated with higher levels of internalizing symptoms at baseline and 1-year

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Table 2. Pearson Correlations for Primary Study Variables

	1	*2*	*3*	*4*	*5*	*6*	*7*	*8*
1. Income-to-needs ratio	–							
2. Material hardship	−.346***	–						
3. Neighborhood unsafety	−.258***	.211***	–					
4. ADI	−.420***	.197***	.158***	–				
5. Amy-mOFC rs-FC	.028	.014	.019	.004	–			
6. Amy-mPFC/ACC–L rs-FC	.003	−.013	−.006	−.001	.345***	–		
7. Amy-mPFC/ACC–R rs-FC	−.003	.006	−.001	.029	.357***	.470***	–	
8. INT sx at baseline	−.103***	.175***	.106***	.072***	.029	−.010	.001	–
9. INT sx at T1	−.070***	.122***	.100***	.068***	.049**	.005	.020	.692***

Amy = amygdala; INT sx = internalizing symptoms; T1 = 1-year follow-up.

* $p < .05$.

** $p < .01$.

*** $p < .001$.

follow-up. Moreover, more strongly positive bilateral rs-FC between the amygdala and mOFC within the FPN at baseline was marginally associated with higher levels of internalizing symptoms at baseline ($r = .03$, $p = .066$) and significantly associated with higher internalizing symptoms at 1-year follow-up ($r = .05$, $p = .003$), whereas rs-FC between the amygdala and mPFC/ACC within the DMN in both hemispheres at baseline was not associated with internalizing symptoms at baseline or 1-year follow-up. There were no significant correlations between SED variables and frontoamygdala (in all three vmPFC subcomponents) rs-FC.

CFA: SED

The results of the CFA showed that income-to-needs ratio (factor loading: $\beta = -.81$, $p < .001$), material hardship (factor loading: $\beta = .43$, $p < .001$), neighborhood unsafety (factor loading: $\beta = .33$, $p < .001$), and ADI (factor loading: $\beta = .51$, $p < .001$) can be loaded onto a single latent construct, albeit neighborhood unsafety and material hardship had low to moderate factor loadings. Thus, exploratory analyses were also conducted to examine the independent effects of each type of SED on frontoamygdala rs-FC and internalizing symptoms (see below). Model fit indices indicated adequate fit in the model: $\chi^2(2) = 27$, $p < .001$, RMSEA = .056, CFI = .977, TLI = .931, SRMR = .02.

SEMs

Hypothesis 1a: Higher levels of SED will be associated with more negative frontoamygdala rs-FC and higher internalizing symptoms

The SEM results demonstrated that higher levels of the SED latent construct at baseline were significantly associated with higher levels of internalizing symptoms at baseline. Internalizing symptoms at baseline mediated the positive association between the SED latent construct and internalizing symptoms at 1-year follow-up (indirect effect = 0.17, $p < .001$; Figure 2). However,

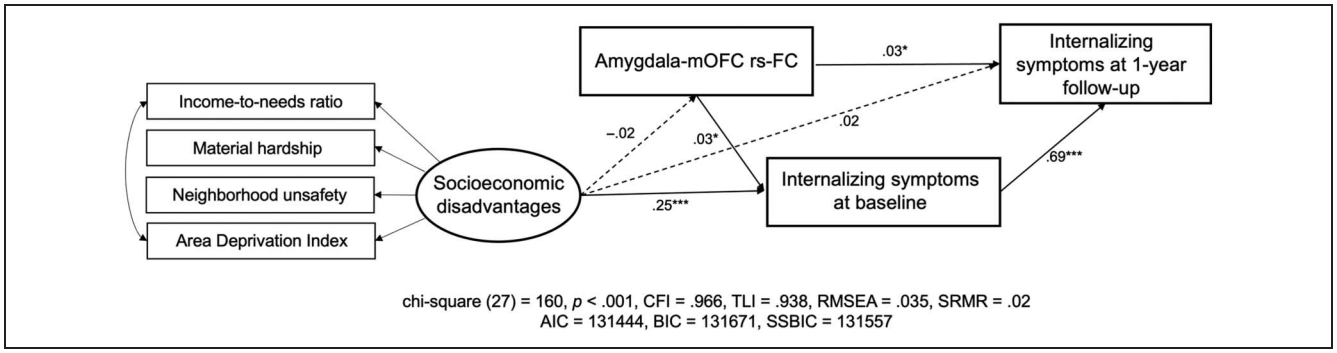

Figure 2. SEM depicting the associations between SED, amygdala–mOFC rs-FC, and internalizing symptoms among preadolescents. Standardized beta coefficients are reported. *$p < .05$, **$p < .01$, ***$p < .001$.

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the SED latent construct was not associated with rs-FC between the amygdala and mOFC, nor rs-FC between the amygdala and mPFC/ACC (Figure 2; Appendices D and E).

Additional SEMs (Figure 3) that examined the distinct effects of specific types of SED (i.e., income-to-needs ratio, material hardship, ADI, and neighborhood unsafety) at baseline revealed that higher material hardship and neighborhood unsafety (but not income-to-needs ratio or ADI) were significantly associated with higher internalizing symptoms at baseline and indirectly associated with higher internalizing symptoms at 1-year follow-up through elevated internalizing symptoms at baseline.

Hypothesis 1b: Frontoamygdala rs-FC will be associated with internalizing symptoms

More strongly positive rs-FC between the bilateral amygdala and mOFC was associated with higher levels of internalizing symptoms at 1-year follow-up, even accounting for internalizing symptoms at baseline (Figure 2). However, functional connectivity between the bilateral amygdala and mPFC/ACC was not associated with internalizing symptoms at baseline or 1-year follow-up (Appendices D and E).

Hypothesis 1c: SED will be indirectly associated with elevated internalizing symptoms through frontoamygdala rs-FC

The SED latent construct was not associated with frontoamygdala rs-FC, and there was no significant indirect effect linking the SED latent construct with internalizing symptoms through frontoamygdala rs-FC.

SEMs examining the distinct roles of various types of SED indicated that only income-to-needs ratio at baseline was associated with more strongly positive rs-FC between the bilateral amygdala and mOFC. More strongly positive amygdala–mOFC rs-FC was associated with higher levels of internalizing symptoms at 1-year follow-up (Figure 3), even after accounting for other types of SED in the model. However, there was no indirect effect linking income-to-needs ratio to internalizing symptoms at 1-year follow-up via amygdala–mOFC rs-FC (indirect effect = 0.001, p = .09). There were no associations between specific types of SED and bilateral amygdala–mPFC/ACC rs-FC nor associations between bilateral amygdala–mPFC/ACC rs-FC and internalizing symptoms at baseline or 1-year follow-up (Appendices F and G). Model fit indices indicated adequate fit in the models.

Hypothesis 2: Frontoamygdala rs-FC will moderate the association between SED and internalizing symptoms at 1-year follow-up

There was no significant interaction between frontoamygdala rs-FC and the SED latent construct on internalizing symptoms for any of the three vmPFC subcomponents (Appendices H–J).

When examining the moderating role of frontoamygdala rs-FC on associations between household- and neighborhood-level SED on internalizing symptoms, there was a significant interactive effect of ADI × amygdala–mOFC rs-FC on internalizing symptoms at 1-year follow-up (Figure 4B), even after accounting for effects of each type of SED. The partial residual plot shown in Figure 4C indicated that a more disadvantaged neighborhood environment (as indexed by higher levels of ADI) at baseline predicted higher levels of internalizing symptoms at 1-year follow-up only when preadolescents exhibited more strongly positive rs-FC between bilateral amygdala and mOFC (simple slope = 0.01, p = .01). No such association was found for those with more strongly negative amygdala–mOFC rs-FC (simple slope = 0, p = .50). Model fit indices indicated adequate fit in all the model. No other significant interactions were found among other types of SED (income-to-needs ratio, material hardship, and neighborhood unsafety) and amygdala–mOFC rs-FC nor rs-FC between bilateral amygdala and mPFC/ACC on internalizing symptoms (see Appendices K and L). Model fit indices indicated adequate fit in the models.

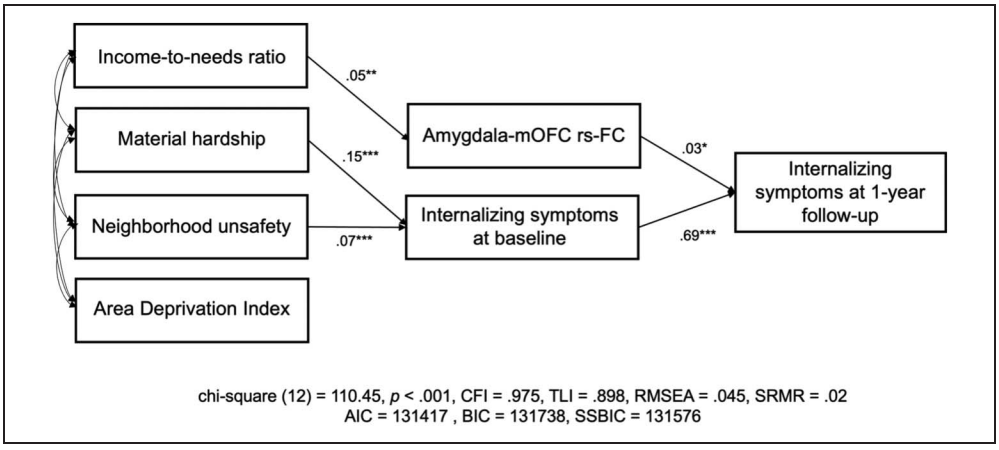

Figure 3. SEM depicting the associations between various types of SED, amygdala–mOFC rs-FC, and internalizing symptoms among preadolescents. Standardized beta coefficients are reported. Only significant paths are shown. *p < .05, **p < .01, ***p < .001.

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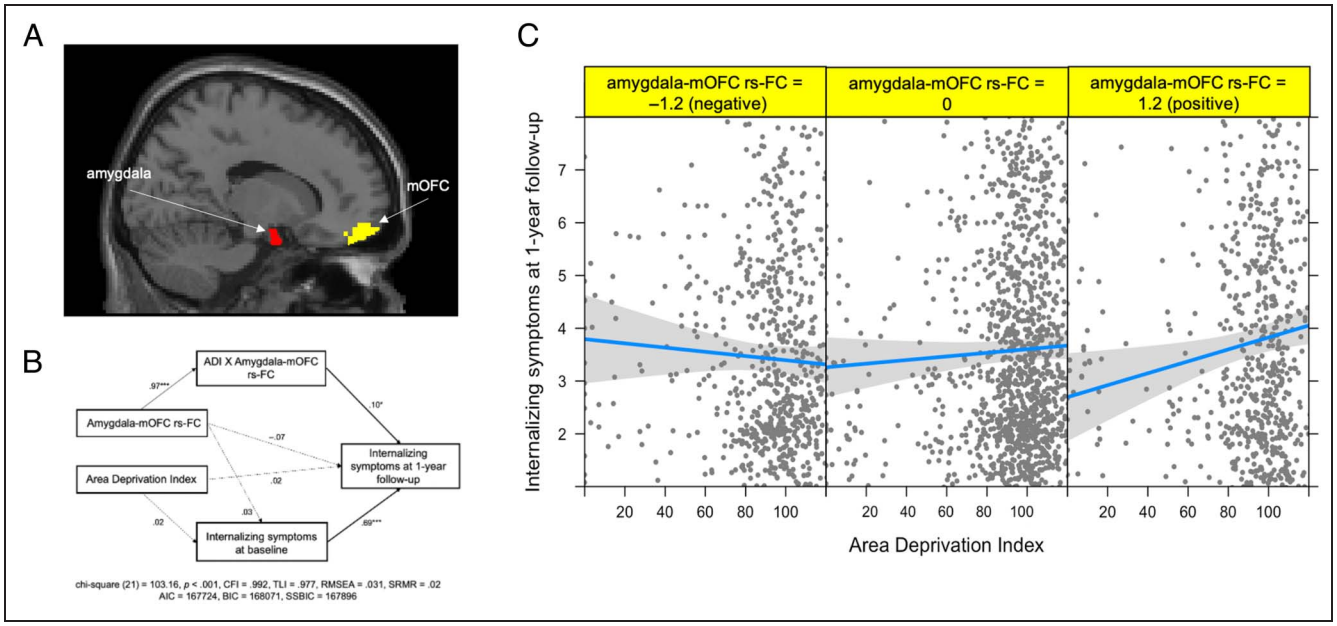

Figure 4. (A) Right-hemisphere sagittal view depicting the location of amygdala and mOFC. (B) SEM depicting the interactive effect between ADI and bilateral amygdala–mOFC rs-FC on internalizing symptoms among preadolescents. Standardized beta coefficients are reported. (C) Partial residual plot depicting the simple slopes of the interaction. Higher neighborhood deprivation (as indexed by ADI) was associated with higher internalizing symptoms at 1-year follow-up only for youth who had more positive amygdala–mOFC rs-FC. $*p < .05$, $**p < .01$, $***p < .001$.

DISCUSSION

The overall goal of this study was to examine the complex associations among various types of SED (i.e., income–needs, material hardship, area deprivation, and neighborhood unsafety), resting-state frontoamygdala connectivity, and internalizing symptoms. Investigation in a large, ethnically/racially, geographically, and socioeconomically diverse sample of preadolescents yielded four key findings. First, all indicators of SED at household and neighborhood levels were associated with increased risk for internalizing symptoms. Second, more strongly positive frontoamygdala coupling was associated with increased risk for internalizing symptoms, but only for coupling between the bilateral amygdala and mOFC regions within the FPN. Third, lower income-to-needs ratio was associated with more strongly negative connectivity between the bilateral amygdala and mOFC regions, though frontoamygdala coupling (regardless of vmPFC subcomponents) did not mediate the association between SED and internalizing symptoms. Fourth, instead, we found that frontoamygdala circuitry moderated associations between SED and internalizing symptoms. Specifically, higher neighborhood deprivation (as indexed by the ADI) was associated with higher internalizing symptoms at 1-year follow-up for youth who had more positive amygdala–mOFC coupling, but not for youth with more negative coupling, suggesting a buffering effect. Collectively, these findings highlight (a) the importance of resting-state frontoamygdala circuitry, particularly between the amygdala and mOFC, for understanding risk and resilience in the context of internalizing psychopathology and (b) potentially distinct roles of SED at the household and neighborhood levels: Household-related SED may have a more direct association with brain function, whereas neighborhood-related SED may interact with brain function to predict developmental outcomes.

SED and Internalizing Symptoms Among Preadolescents

The adverse effects of SED on psychological development (Peverill et al., 2021; Farah, 2017; Johnson et al., 2016; Reiss, 2013; Hackman et al., 2012), such as internalizing problems (Demidenko et al., 2021), are well documented. Consistent with this broader literature, in this large longitudinal sample of preadolescents, we observed an indirect association between SED measured at 9–10 years of age (baseline) and internalizing symptoms 1 year later, through elevated internalizing symptoms at baseline. Although we found distinct associations between various types of SED with frontoamygdala circuitry and internalizing symptoms, it is important to note that the shared (i.e., cumulative) effect of SED (as measured as a latent construct of household and neighborhood disadvantages) still had the largest effect size on internalizing symptoms, and this effect was greater than any individual type of SED. These findings are consistent with the perspective that cumulative exposure to adversity is more predictive of developmental outcomes than any singular exposure (Gach, Ip, Sameroff, & Olson, 2018; Evans et al., 2013) and highlight the importance of utilizing multivariate

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approaches to examine the influence of SED during development.

When examining various types of SED, only neighborhood unsafety and material hardship were uniquely associated with elevated internalizing symptoms at baseline. At the neighborhood level, chronic exposure to threat (e.g., gang violence and crimes) may result in altered hypothalamic–pituitary–adrenal axis function (Theall, Shirtcliff, Dismukes, Wallace, & Drury, 2017) and elevated internalizing symptoms. At the household level, material hardship may reflect proximal family environment (e.g., increasing family stress and conflict) more closely than income-to-needs ratio and thus better predict internalizing symptoms. Alternatively, it is possible that neighborhood unsafety and material hardship rated by parents are more closely related to perceived stress experienced by youth (Goldman-Mellor et al., 2016) and thus are more predictive of internalizing symptoms than indices of SED that may be more objective (i.e., income-to-needs ratio and ADI). Given that both neighborhood unsafety and material hardship were not associated with frontoamygdala rs-FC and we did not find support for a mediating effect of frontoamygdala rs-FC, future studies that incorporate other biological (e.g., cortisol: Theall et al., 2017; brain structures and volumes: Taylor et al., 2020; Johnson et al., 2016; Noble et al., 2015; connectivity within and between networks: Karcher, Michelini, Kotov, & Barch, 2021) or environmental factors (e.g., parenting sensitivity: Perry, Braren, Blair, & Family Life Project Key Investigators, 2018) are needed to elucidate the precise processes linking SED with internalizing symptoms at this stage of development. Moreover, findings on specific aspects of SED in the current study simultaneously highlight the importance of frameworks that emphasize distinctions in the types of adversity experienced (e.g., Cohodes, Kitt, Baskin-Sommers, & Gee, 2021; McLaughlin, Sheridan, & Lambert, 2014), as well as the importance of capturing experiences with SED that go beyond more objective measures (e.g., income–needs).

Frontoamygdala rs-FC and Internalizing Symptoms

More strongly positive connectivity between the bilateral amygdala and vmPFC, particularly mOFC regions within the FPN, was associated with preadolescents' internalizing symptoms at baseline and 1 year later. This finding is generally consistent with prior studies and the role of frontoamygdala connectivity in downregulating negative affect (Silvers et al., 2015, 2017; Milad & Quirk, 2012; Kim et al., 2011). Developmentally, some evidence has suggested that a shift toward more strongly negative rs-FC between the vmPFC and amygdala occurs with age (Brieant et al., 2021; Jalbrzikowski et al., 2017; Wu et al., 2016; Gee, Humphreys, et al., 2013). More strongly positive (or weaker negative) coupling between amygdala and vmPFC may reflect less top–down control over emotional reactivity (Silvers et al., 2015, 2017; Wu et al., 2016; Gee, Humphreys, et al., 2013). Indeed, only amygdala connectivity with vmPFC subregions within the FPN (implicated in cognitive control; Cole et al., 2012), but not DMN, was associated with internalizing symptoms. However, to avoid reverse inference and overinterpreting our results, future studies that incorporate behavioral or self-report measures of emotion regulation and examine amygdala connectivity with the broader FPN are needed to further test this idea.

Notably, although rodent and human studies have found that mOFC subregions within the vmPFC are important for fear extinction and emotion regulation (Hsieh & Chang, 2020; Stalnaker, Cooch, & Schoenbaum, 2015), vmPFC regions contributing to findings in our study were more anterior than in some prior human studies, which have generally found altered connectivity of limbic regions with the sgACC (Marusak et al., 2016) or mPFC (Thomason et al., 2015; Gee, Gabard-Durnam, et al., 2013; Burghy et al., 2012; Kim et al., 2011) in individuals with higher internalizing symptoms or following trauma exposure. This difference may stem from sample characteristics and methodological differences.

Whereas most studies have used anatomically defined atlases to extract nodes, we extracted cortex nodes from whole-brain parcellations based on groupwise graph theory (Shen et al., 2010) to ensure functional homogeneity within each subcomponent and that node definitions were consistent across subjects (Shen et al., 2013). Although it is beyond the scope of the current study, future investigations that compare different parcellation approaches (e.g., Bryce et al., 2021) will provide important insight and may help to explain inconsistency across findings. In addition, prior rs-FC studies with smaller sample sizes may artificially inflate effects (Marek et al., 2020) or be underpowered to detect small effects that we observed in the current study (Dick et al., 2021). Based on baseline data from the ABCD Study, estimates of associations between rs-FC and behavioral measures (e.g., psychopathology symptoms) stabilize and become more reproducible with sample sizes of $N \geq 2000$ (Marek et al., 2020). Notably, all brain–behavior effect sizes in the current study were small and fell around the median correlation values ($r = .03$) between questionnaires and task variables reported in the ABCD Study (Owens et al., 2021). Although future work will be important to explore the clinical significance of these findings, the current study adds to the growing body of literature suggesting that individuals' resting-state connectivity profiles, especially connectivity between the FPN and amygdala, can be reliably linked to individual differences in behavior (Seitzman et al., 2019; Finn et al., 2015). Moreover, the small effect size of the observed brain–behavior associations may reflect the equifinality of internalizing symptoms and that alterations in neural processes may manifest through different phenotypes across individuals. Our finding that resting-state frontoamygdala connectivity with the FPN was associated with internalizing symptoms has implications for identifying youth at heightened risk of psychopathology who

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could benefit from mechanistically driven intervention (Fitzgerald, Schroder, & Marsh, 2021).

Mediating and Moderating Roles of Household Versus Neighborhood Disadvantages

Among various types of SED, lower income-to-needs ratio was associated with more strongly negative coupling between the amygdala and mOFC, similar to a prior study of adolescents (Hanson et al., 2019). These results may align with the stress acceleration hypothesis (Belsky, 2019; Callaghan & Tottenham, 2016) that early adversity "accelerates" maturation as a context-specific adaptation (Herzberg et al., 2021; Miller et al., 2020; Silvers et al., 2016; Gee, Gabard-Durnam, et al., 2013). Interestingly, amygdala–mOFC coupling moderated the effect of neighborhood deprivation on internalizing symptoms, consistent with a prior study (Ramphal et al., 2020). Specifically, higher neighborhood deprivation was associated with higher internalizing symptoms for youth with more strongly positive connectivity, but not for youth with more strongly negative connectivity, suggesting a potential buffer against the impact of a disadvantaged neighborhood environment. Taken together, it is possible that preadolescents living in households with fewer resources show a more mature pattern of amygdala connectivity with prefrontal regions in the FPN (implicated in better emotion regulation; Silvers et al., 2015, 2017). More strongly negative frontoamygdala connectivity may facilitate youth living in a more deprived neighborhood adapting to their environmental conditions, consistent with the idea that alterations in frontoamygdala circuitry during preadolescence may reflect an adaptive response to harsh or unpredictable contexts (e.g., Ellis, Bianchi, Griskevicius, & Frankenhuis, 2017). However, this potential interpretation awaits further replication and corroboration with longitudinal studies of frontoamygdala circuitry and internalizing symptoms across development, which can be tested with future waves of ABCD data. Moreover, the long-term implications of this context-specific adaptation remain unclear, as do the reasons why other forms of SED (i.e., material hardship and neighborhood unsafety) did not show similar effects. A recent study with the ABCD sample found that connectivity of the FPN with other networks (i.e., auditory and sensorimotor networks) was associated with income-to-needs ratio but not neighborhood deprivation (Rakesh et al., 2021), suggesting that FPN connectivity may be more sensitive to household disadvantages.

Our study adds to the emerging literature highlighting the importance of delineating associations with neighborhood and household SED as related yet dissociated factors (Gard et al., 2021; Rakesh et al., 2021; Hyde et al., 2020; Ramphal et al., 2020; Tomlinson et al., 2020). Our findings also extend this research by showing that household-related SED may have a more proximal association with brain function, whereas neighborhood-related SED may interact with brain function to confer risk or resilience, highlighting the possibility that specific types of SED relate to behavior through distinct pathways. Theories have postulated distinct ways (e.g., mediation and moderation) that SED may be associated with neural and mental health outcomes (Farah, 2017). However, our current understanding of how SED may "get under the skin" to confer risk for psychopathology during development has primarily been studied via a model in which increased risk for mental health problems among children living in socioeconomically disadvantaged environments is explained by alterations in brain function due to adversity exposure. However, a more nuanced understanding of associations between SED, brain function, and behavior is important to avoid biological determinism and the tendency to examine poverty within deficit models that pathologize children living in disadvantaged environments (Simmons et al., 2021; Ellis et al., 2017; Frankenhuis & Ellis, 2017). Our study is among the first to empirically test both mediating and moderating roles of frontoamygdala circuitry linking SED and mental health outcomes and to delineate how corresponding models may fit with data from a large and diverse sample of preadolescents. We found that neighborhood deprivation was associated with higher levels of internalizing symptoms only among youth with more strongly positive (but not negative) frontoamygdala connectivity. This finding is consistent with the idea that brain function can foster resilience by buffering against the impact of an adverse environment and may inform how and which specific brain networks are enhanced through exposure to early adversity as a context-specific adaptation (Ellis et al., 2017; Frankenhuis & Ellis, 2017).

Strengths and Limitations

Novel findings from this study provide insight into the relations between various types of SED, frontoamygdala circuitry, and internalizing symptoms during development. The major strengths of this study include the use of a large sample from ethnically/racially, socioeconomically, and geographically diverse backgrounds, which increases the generalizability of results. We employed structural equation modeling to delineate direct, indirect, and moderating effects of multiple SED and brain–behavior associations and provided model fit indices (e.g., CFI or RMSEA) that allow us to test a priori hypotheses and inspect model differences in fit. Several key strengths of focusing on rs-FC include its (a) stability across time and states (Gratton et al., 2018; Shehzad et al., 2009), (b) reliable associations with trait-like individual differences (Seitzman et al., 2019; Finn et al., 2015), and (c) high reproducibility across samples (Marek et al., 2020).

Several limitations, however, must also be acknowledged. First, our study included only two time points assessed at a specific stage of development. Thus, future work is essential to understand long-term effects of SED

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on frontoamygdala circuitry and internalizing symptoms and how these relations vary across development. Second, our findings are based on observational data and cannot inform causality; therefore, results should be interpreted with appropriate caution. Third, we did not include behavioral measures of processes such as emotion regulation that will be important to better understand the links between SED and frontoamygdala alterations related to internalizing psychopathology. Fourth, studies that examine more proximal factors (e.g., stress and parenting) may better elucidate mechanisms linking SED to the development of psychopathology. Fifth, although our study sample was large and diverse, it is not guaranteed to match national estimates (Compton, Dowling, & Garavan, 2019; Garavan et al., 2018) and findings may not generalize to youth from cultural groups that are not adequately sampled in the ABCD Study (Simmons et al., 2021). Finally, although our goal was to better understand the complex associations between SED, frontoamygdala circuitry, and internalizing symptoms and how our a priori hypotheses of mediation and moderation pathways fit a large and diverse sample of preadolescents, multiple comparisons across models and examining connectivity with multiple nodes increase the potential for Type I error. Thus, future studies could also benefit from structural equation modeling in a Bayesian framework (Gelman, Hill, & Yajima, 2012).

Conclusions

Effects of social determinants of health on developmental outcomes cannot be explained by observing a single level of socioecological context (e.g., household or neighborhood level), highlighting the importance of capturing multifaceted ecological contexts in which children develop and examining unique effects of SED at distinct ecological levels to understand how SED affects brain and behavioral development. This study is among the few to delineate plausible pathways linking SED at both the household and neighborhood levels with frontoamygdala circuitry and internalizing problems among preadolescents. Among our findings, youth in households characterized by greater disadvantages (i.e., lower income-to-needs ratio) had more negative frontoamygdala connectivity. At the neighborhood level, findings suggested that more negative frontoamygdala connectivity may confer resilience against internalizing psychopathology in the context of neighborhood deprivation. Moreover, SED indirectly predicted internalizing symptoms 1 year later through baseline internalizing symptoms, suggesting that early intervention efforts may be important for youth living in disadvantaged households and neighborhoods. These findings add to a growing literature that can inform policy and efforts to identify and support youth who are most likely to benefit from early interventions.

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APPENDIX A
Inclusion Criteria for Sensitivity Analysis

Following the recommendations from the ABCD Consortium, we included participants with resting-state data that met eight additional inclusion criteria in the sensitivity analyses: (a) rsfMRI series passed rawQC (icq_rsfmri_ok_ser > 0), (b) T1 series passed rawQC (iqc_t1_ok_ser > 0), (c) rsfMRI number of frames > 375 (rsfmri_c_ngd_ntpoints > 375), (d) fMRI B0 Unwarp available (apqc_fmri_bounwarp_flag = 1), (e) fMRI manual postprocessing QC not failed (fmri_postqc_qc ~ = 0), (f) fMRI registration to T1w < 19 (apqc_fmri_regt1_rigit < 19), (g) fMRI maximum dorsal cutoff score of <65 (apqc_fmri_fov_cutoff_dorsal < 65), and (h) fMRI maximum ventral cutoff score of <60 (apqc_fmri_fov_cutoff_ventral < 60). Those who met all these criteria are indicated by the recommended rs-fMRI inclusion variable (imgincl_rsfmri_include = 1) provided in Release 3.0 of the ABCD Study. A subset of 3864 participants who met all inclusion criteria were included in the sensitivity analysis. Sensitivity analysis results are reported in Appendices M–X below.

Table A1. Descriptive Statistics for Primary Study Variables

	Income-to-Needs Ratio	*Material Hardship*	*Neighborhood Unsafety*	*ADI*	*Amy-mOFC rs-FC*	*Amy-mPFC/ACC–L rs-FC*	*Amy-mPFC/ACC–R rs-FC*	*INT sx baseline*	*INT sx T1*
Valid	3724	4006	4036	3927	4038	4038	4038	4038	3829
Missing	314	32	2	111	0	0	0	0	209
Mean	3.90	0.38	−4.00	90.84	0	0	0	5.02	4.95
SD	2.41	1	0.90	24.94	1	1	1	5.46	5.29
Skewness	0.33	3.21	0.97	−1.93	0.04	0.14	0.20	1.89	1.80
Kurtosis	−0.82	11.17	0.75	3.94	0.95	1.37	1.54	4.87	4.32
Min	0.10	0	−5	0	−4.52	−4.74	−5.08	0	0
Max	10.25	7	−1	124.50	4.80	5.75	6.89	49	38

Amy = amygdala; INT sx = internalizing symptoms; T1 = 1-year follow-up.

APPENDIX B

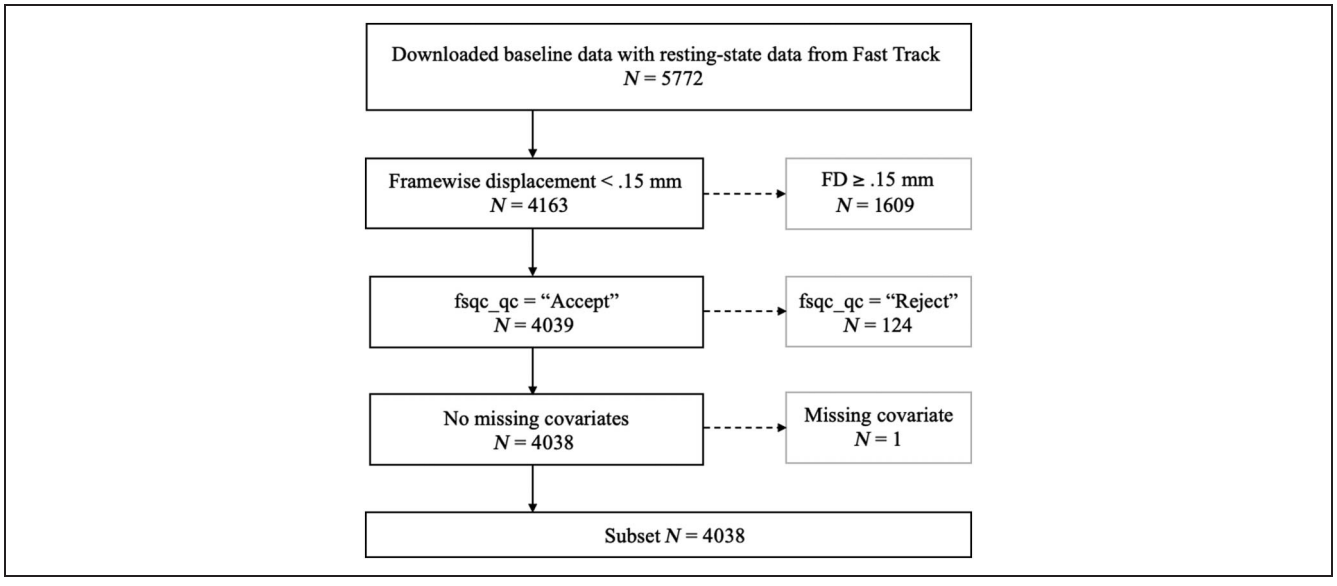

Figure B1. Inclusion flow chart.

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APPENDIX C

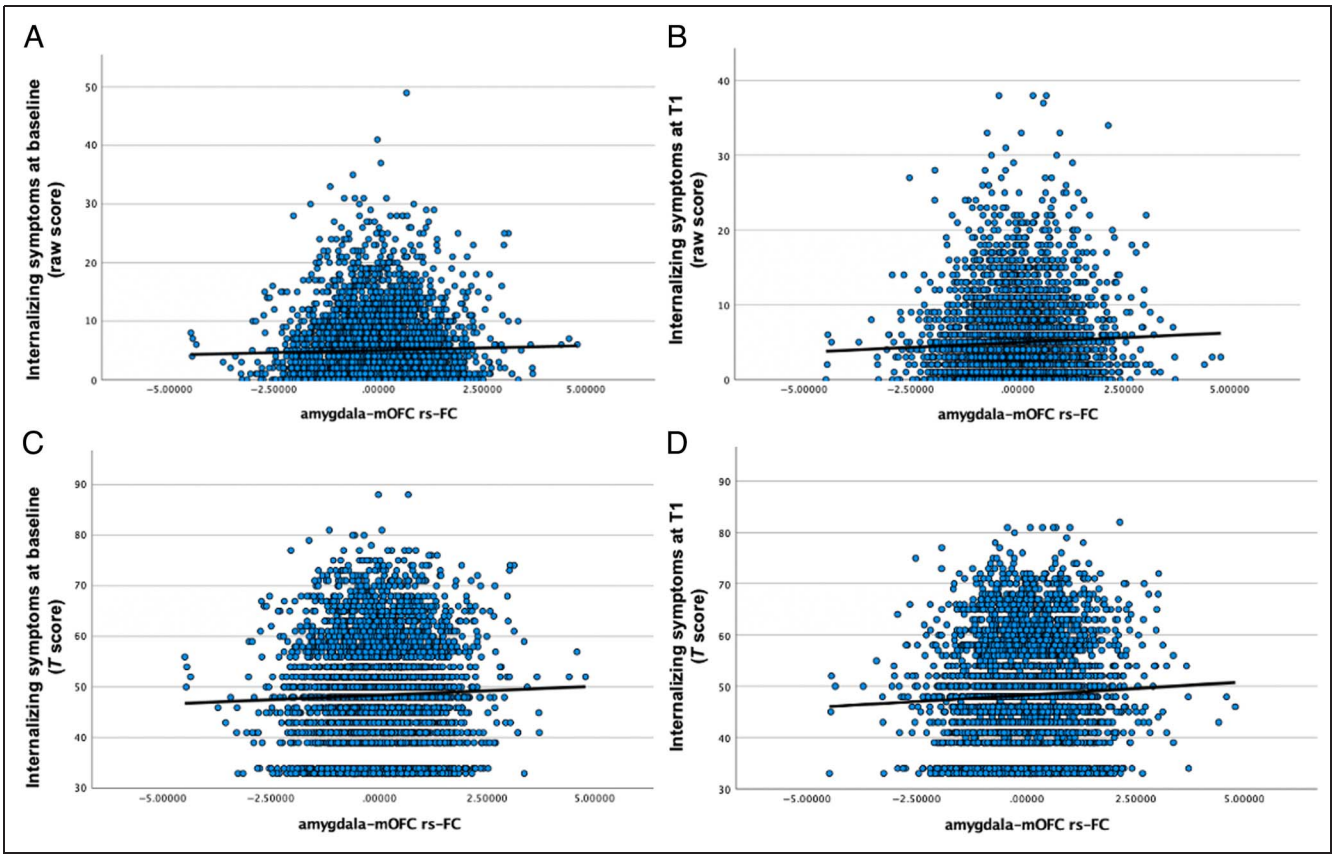

Figure C1. Scatter plots depicting the associations between amygdala–mOFC rs-FC and (A) internalizing symptoms (raw score) at baseline, (B) internalizing symptoms (raw score) at 1-year follow-up (T1), (C) internalizing symptoms (*t* score) at baseline, and (D) internalizing symptoms (*t* score) at 1-year follow-up.

APPENDIX D

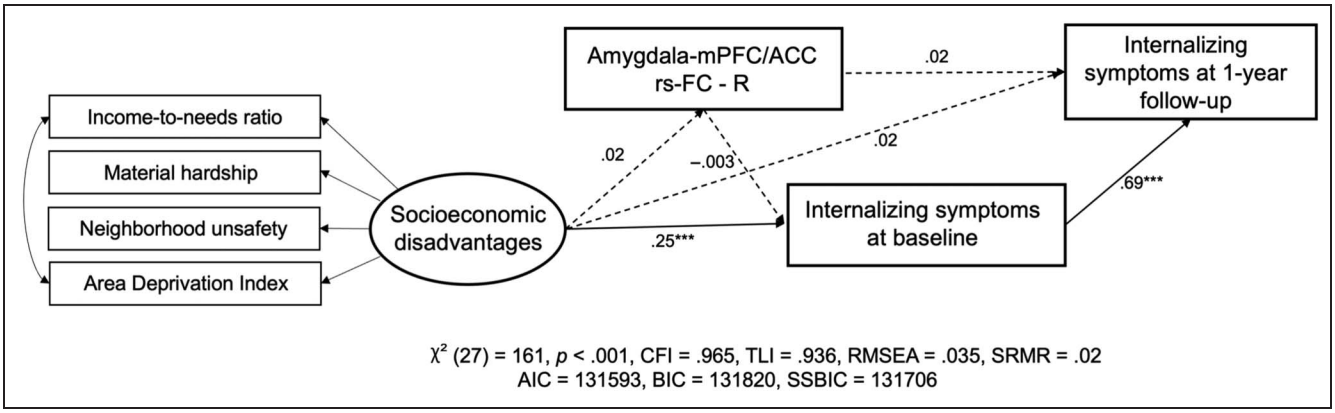

Figure D1. SEM depicting the associations between SED, right amygdala–mPFC/ACC rs-FC, and internalizing symptoms among preadolescents. Standardized beta coefficients are reported. *$p < .05$, **$p < .01$, ***$p < .001$.

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APPENDIX E

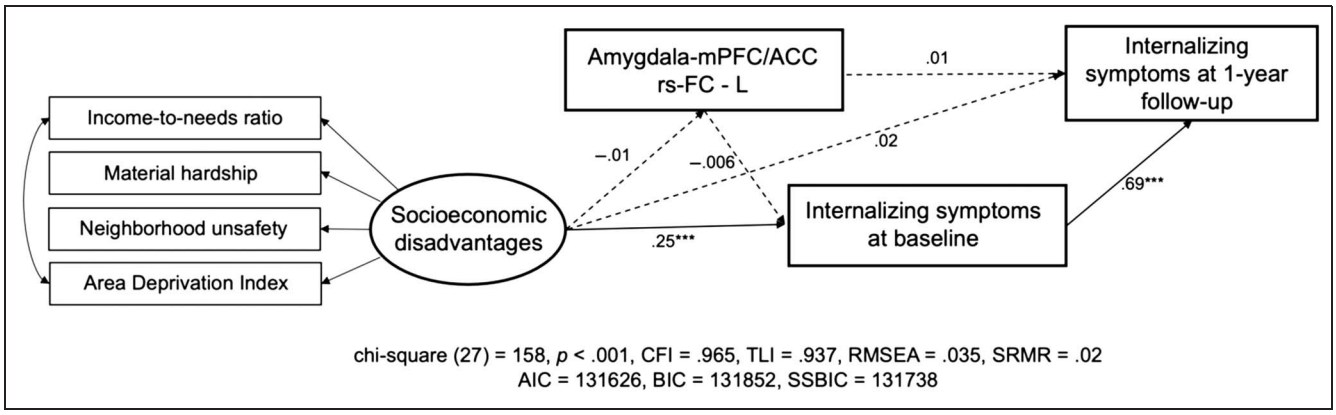

Figure E1. SEM depicting the associations between SED, left amygdala–mPFC/ACC rs-FC, and internalizing symptoms among preadolescents. Standardized beta coefficients are reported. *$p < .05$, **$p < .01$, ***$p < .001$.

APPENDIX F

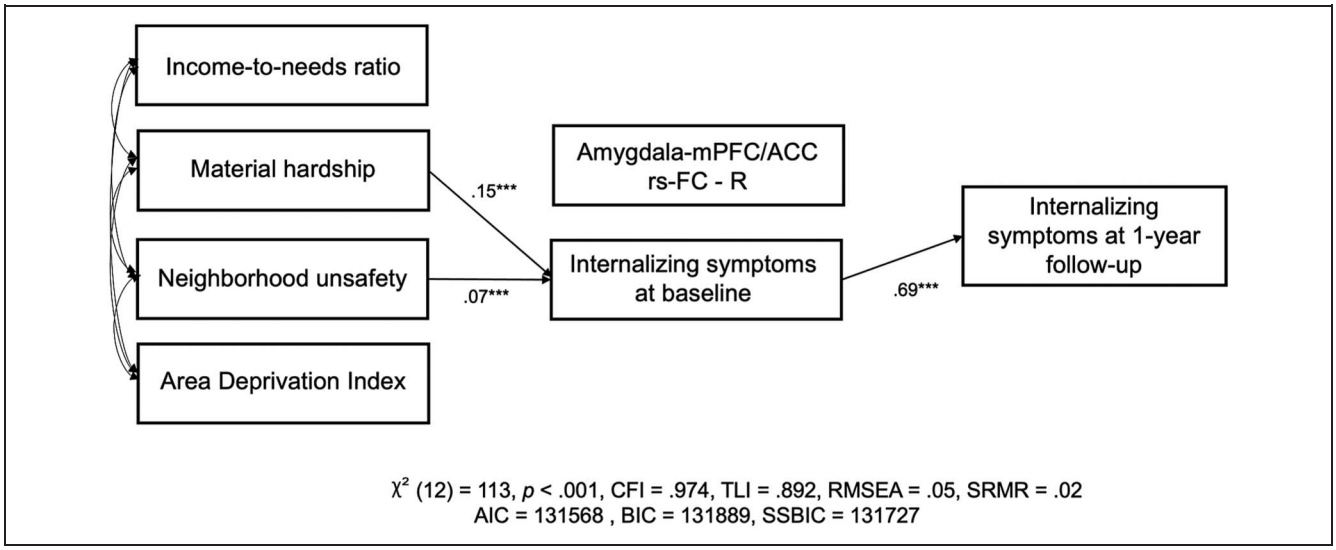

Figure F1. SEM depicting the associations between various types of SED, right amygdala–mPFC/ACC rs-FC, and internalizing symptoms among preadolescents. Standardized beta coefficients are reported. Only significant paths are shown. *$p < .05$, **$p < .01$, ***$p < .001$.

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APPENDIX G

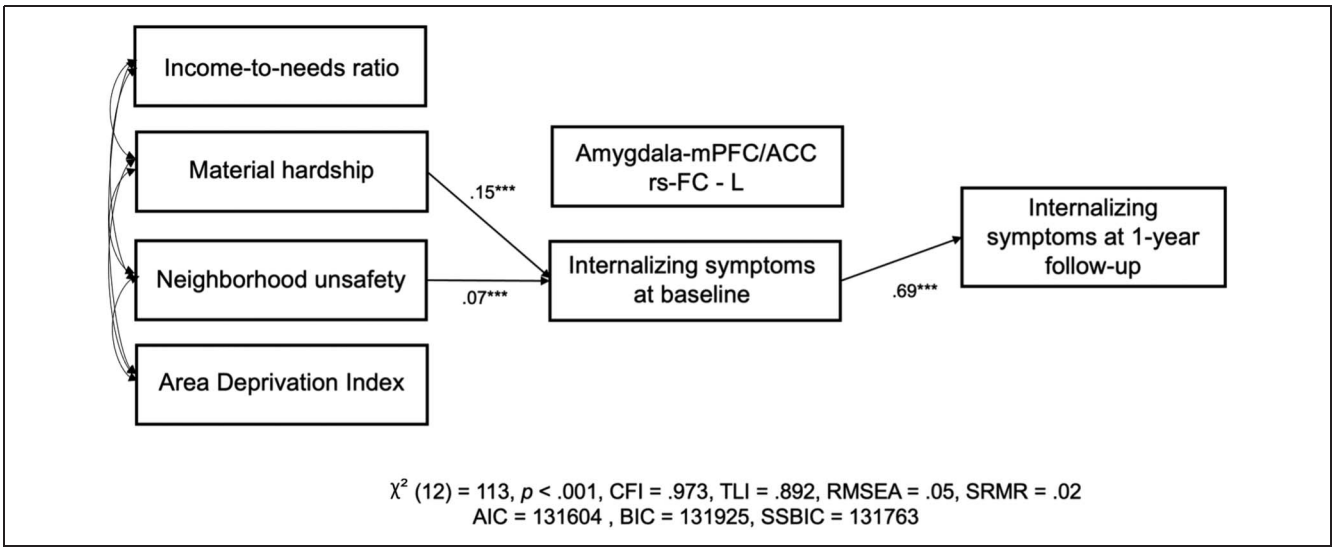
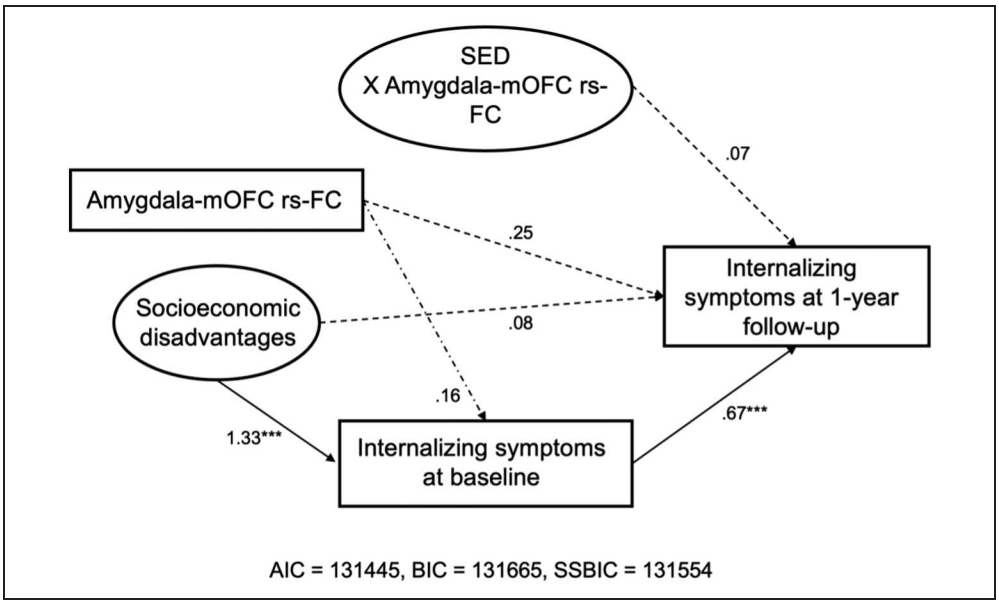


Figure G1. SEM depicting the associations between various types of SED, left amygdala–mPFC/ACC rs-FC, and internalizing symptoms among preadolescents. Standardized beta coefficients are reported. Only significant paths are shown. *$p < .05$, **$p < .01$, ***$p < .001$.

APPENDIX H

Figure H1. SEM depicting the nonsignificant interactive effect between SED latent construct and bilateral amygdala–mOFC rs-FC on internalizing symptoms among preadolescents. Unstandardized beta coefficients are reported. *$p < .05$, **$p < .01$, ***$p < .001$.



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APPENDIX I

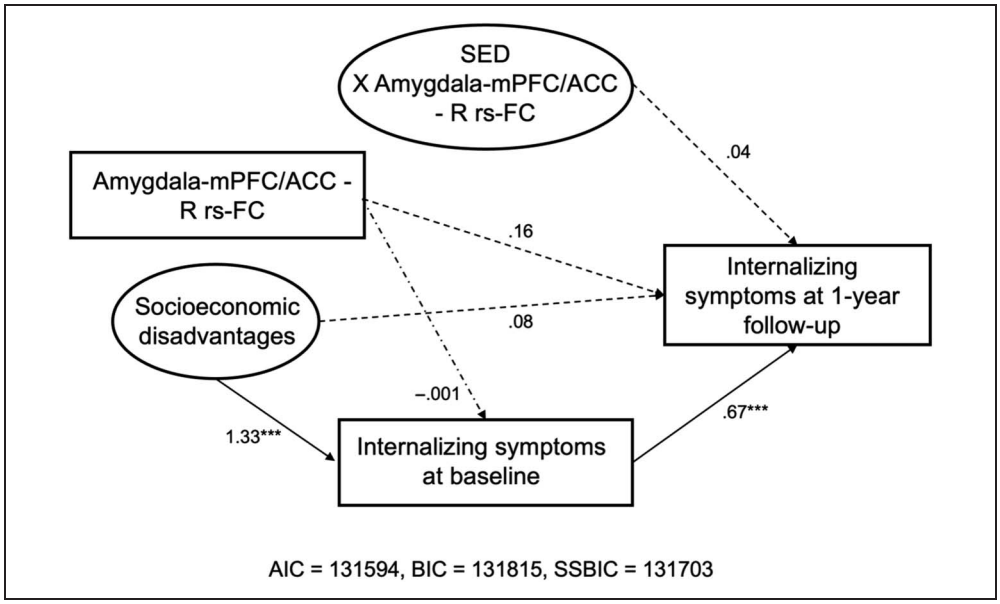

Figure I1. SEM depicting the nonsignificant interactive effect between SED latent construct and right amygdala–mPFC/ACC rs-FC on internalizing symptoms among preadolescents. Unstandardized beta coefficients are reported. *$p < .05$, **$p < .01$, ***$p < .001$.

APPENDIX J

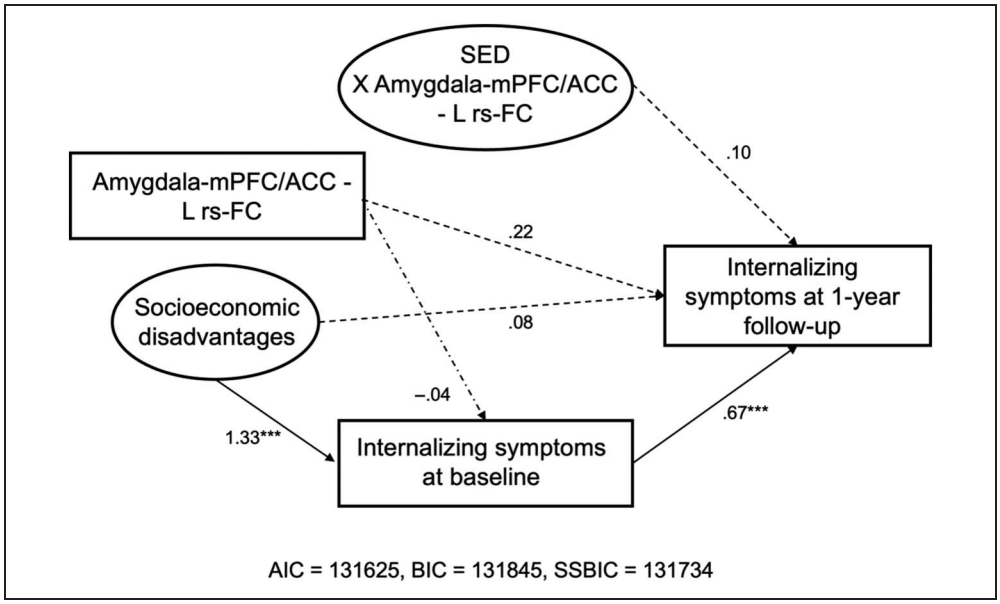

Figure J1. SEM depicting the nonsignificant interactive effect between SED latent construct and left amygdala–mPFC/ACC rs-FC on internalizing symptoms among preadolescents. Unstandardized beta coefficients are reported. *$p < .05$, **$p < .01$, ***$p < .001$.

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APPENDIX K

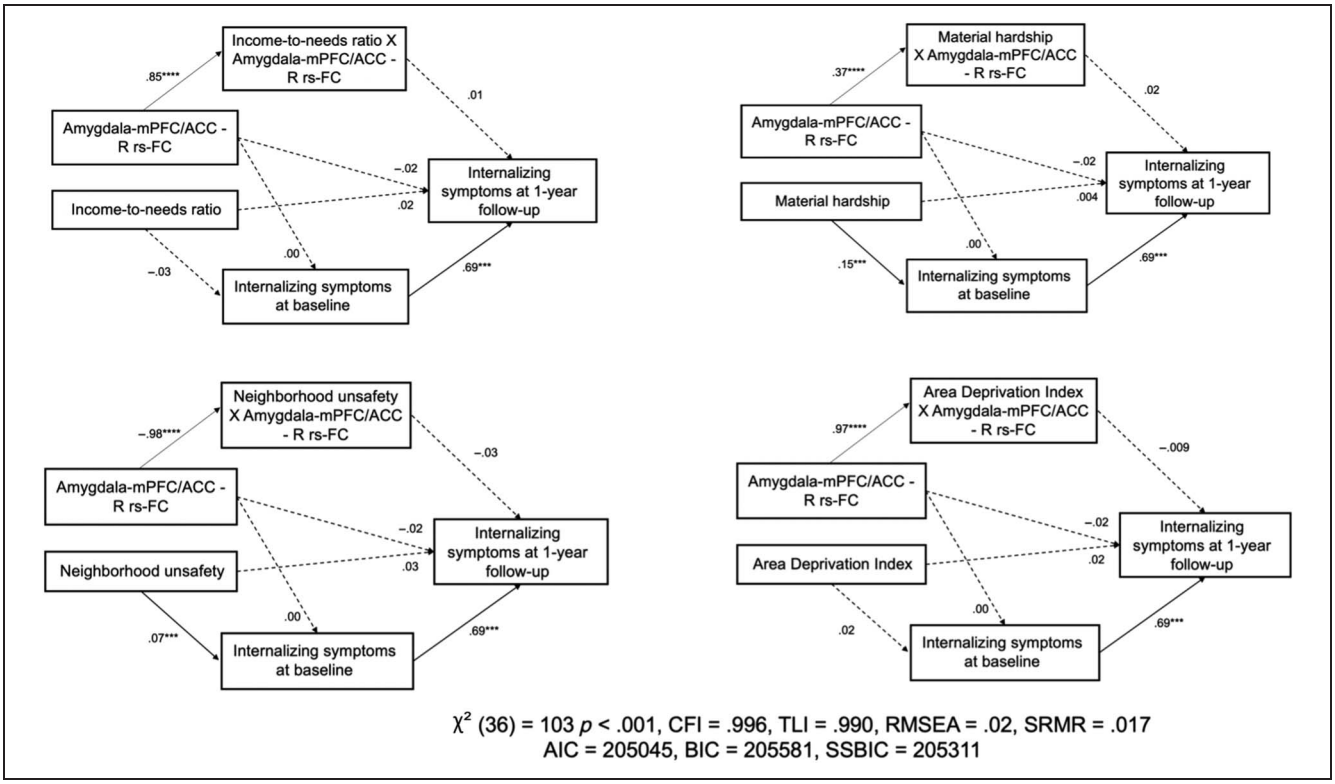

Figure K1. SEM depicting the moderating role of right amygdala–mPFC/ACC rs-FC between various types of SED and internalizing symptoms among preadolescents. Standardized beta coefficients are reported. *$p < .05$, **$p < .01$, ***$p < .001$.

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APPENDIX L

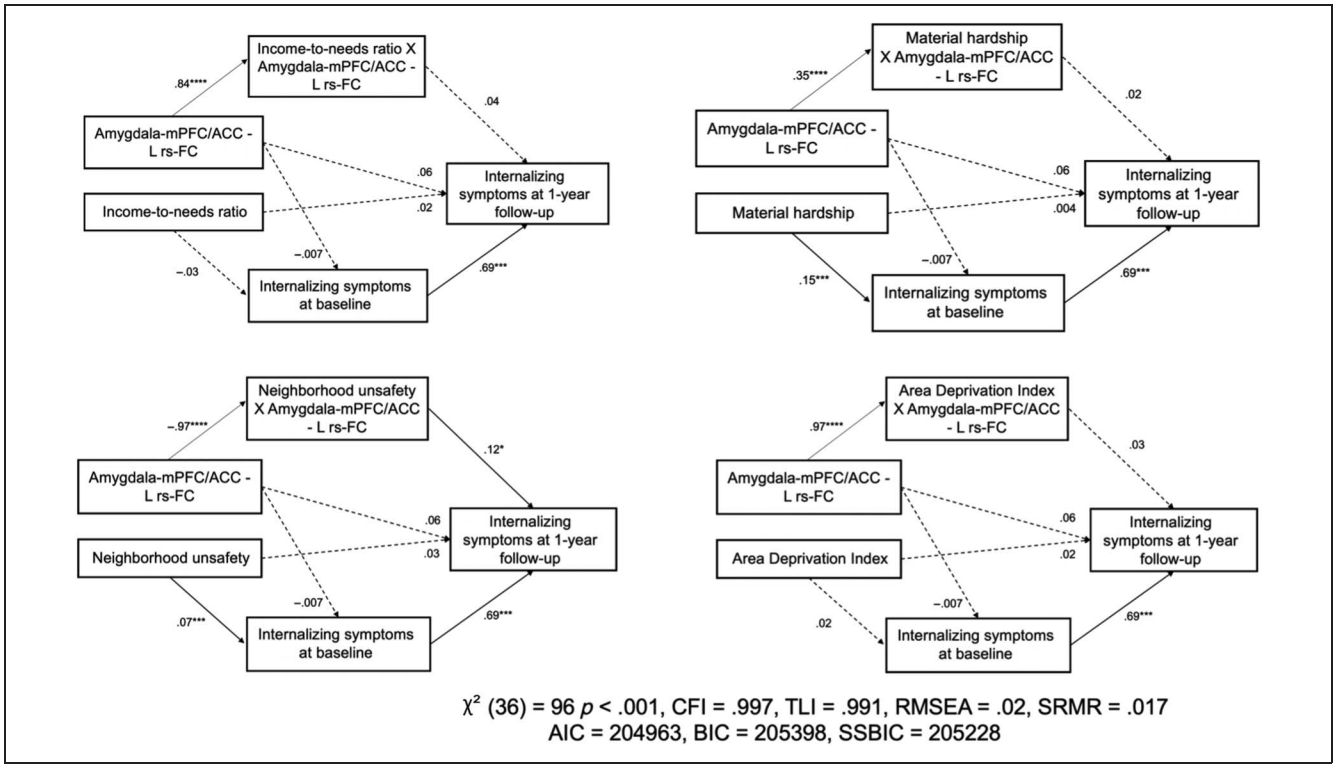

Figure L1. SEM depicting the moderating role of left amygdala–mPFC/ACC rs-FC between various types of SED and internalizing symptoms among preadolescents. Standardized beta coefficients are reported. *$p < .05$, **$p < .01$, ***$p < .001$. The SED model indicated that there was a significant interaction effect between left amygdala–mPFC/ACC rs-FC and neighborhood unsafety on internalizing symptoms at T1. Simple slopes analyses indicated that higher neighborhood unsafety was associated with higher internalizing symptoms for youth with more positive connectivity between left amygdala and left mPFC/ACC (simple slope = 0.03, $p < .001$), but not for youth with more negative connectivity (simple slope = 0.00, $p = .99$), suggesting a potential buffering effect. However, this significant interaction effect did not survive in subsequent sensitivity analysis with stricter inclusion/exclusion criteria following the recommendations from the ABCD Consortium with a subset of participants ($n = 3864$; see below for inclusion criteria), suggesting that the effect could be driven by artifacts in the neural data. To be conservative, we did not interpret this finding.

APPENDIX M

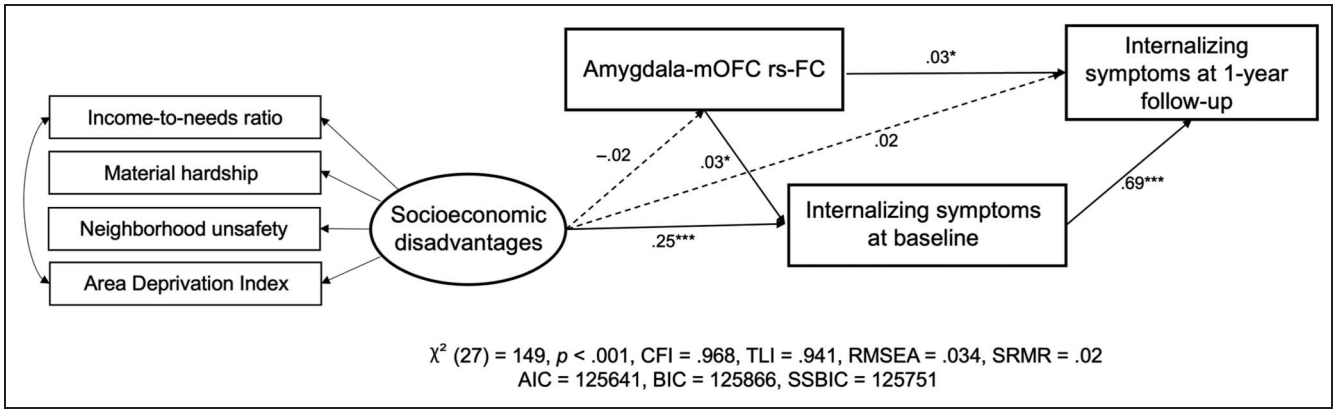

Figure M1. SEM depicting the associations between SED, amygdala–mOFC rs-FC, and internalizing symptoms among a subset ($n = 3864$) of participants who met all inclusion criteria for the sensitivity analysis. Standardized beta coefficients are reported. *$p < .05$, **$p < .01$, ***$p < .001$.

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APPENDIX N

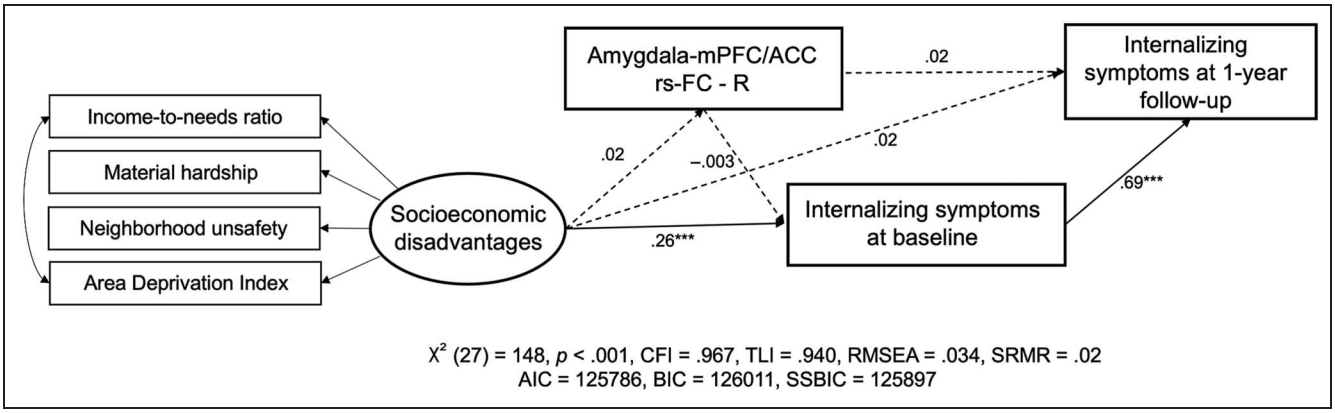

Figure N1. SEM depicting the associations between SED, right amygdala–mPFC/ACC rs-FC, and internalizing symptoms among a subset (n = 3864) of participants who met all inclusion criteria for the sensitivity analysis. Standardized beta coefficients are reported. *p < .05, **p < .01, ***p < .001.

APPENDIX O

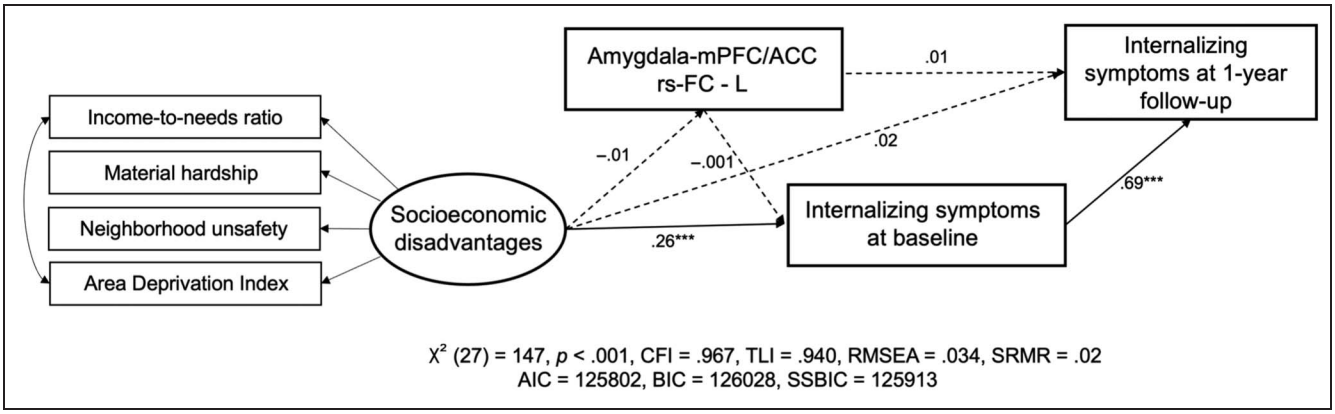

Figure O1. SEM depicting the associations between SED, left amygdala–mPFC/ACC rs-FC, and internalizing symptoms among a subset (n = 3864) of participants who met all inclusion criteria for the sensitivity analysis. Standardized beta coefficients are reported. *p < .05, **p < .01, ***p < .001.

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APPENDIX P

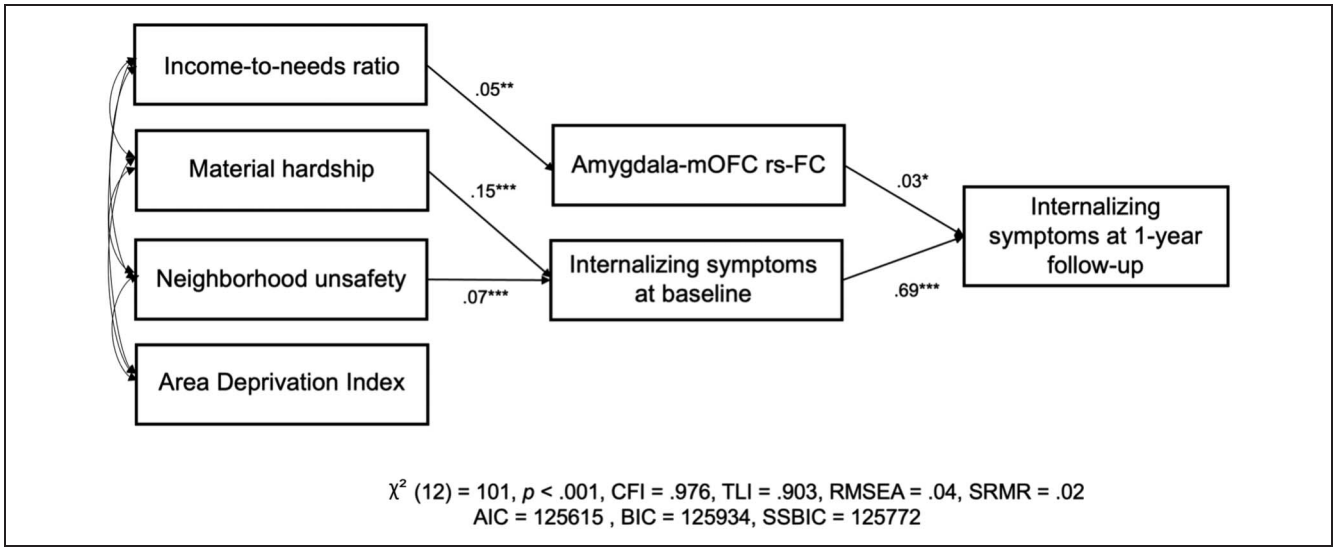

Figure P1. SEM depicting the associations between various types of SED, amygdala–mOFC rs-FC, and internalizing symptoms among a subset (n = 3864) of participants who met all inclusion criteria for the sensitivity analysis. Standardized beta coefficients are reported. Only significant paths are shown. *p < .05, **p < .01, ***p < .001.

APPENDIX Q

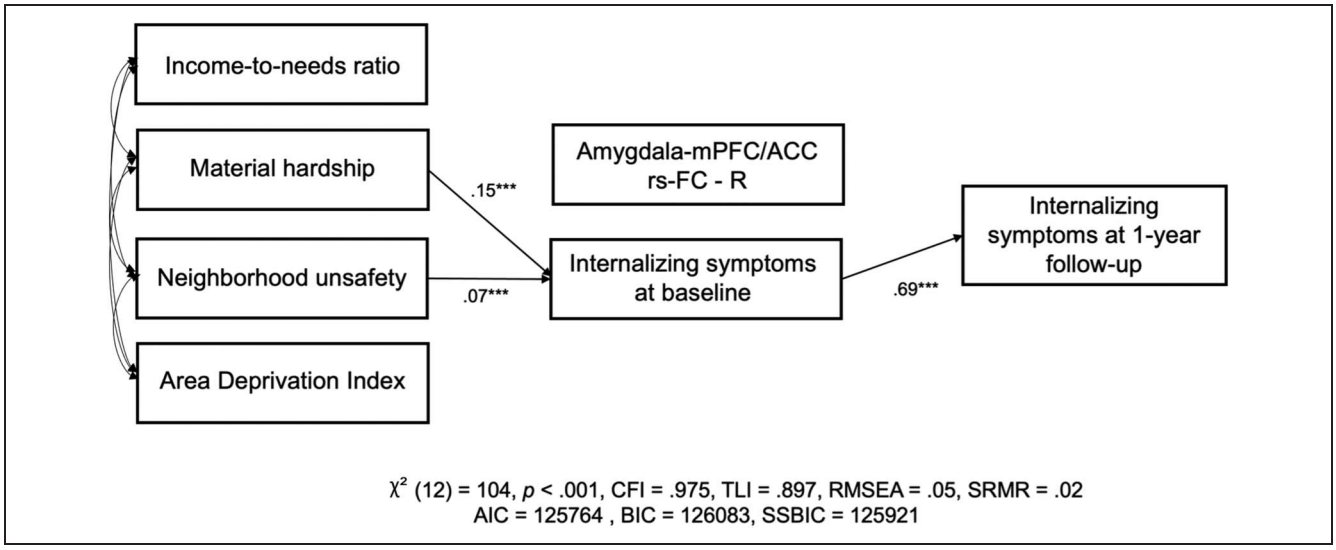

Figure Q1. SEM depicting the associations between various types of SED, right amygdala–mPFC/ACC rs-FC, and internalizing symptoms among a subset (n = 3864) of participants who met all inclusion criteria for the sensitivity analysis. Standardized beta coefficients are reported. Only significant paths are shown. *p < .05, **p < .01, ***p < .001.

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APPENDIX R

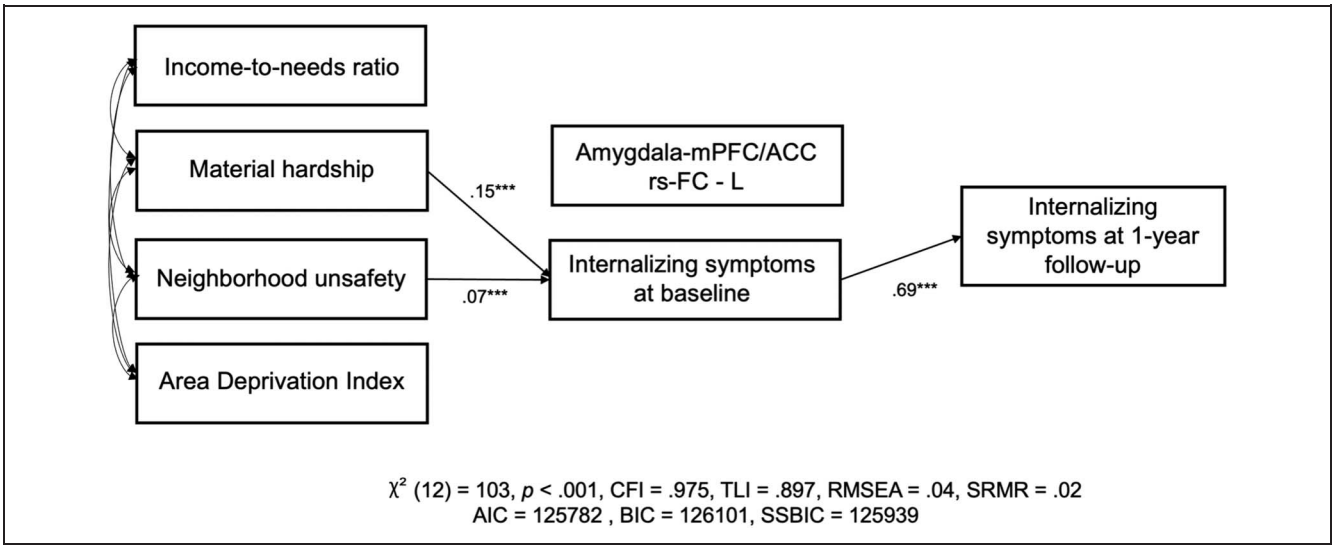
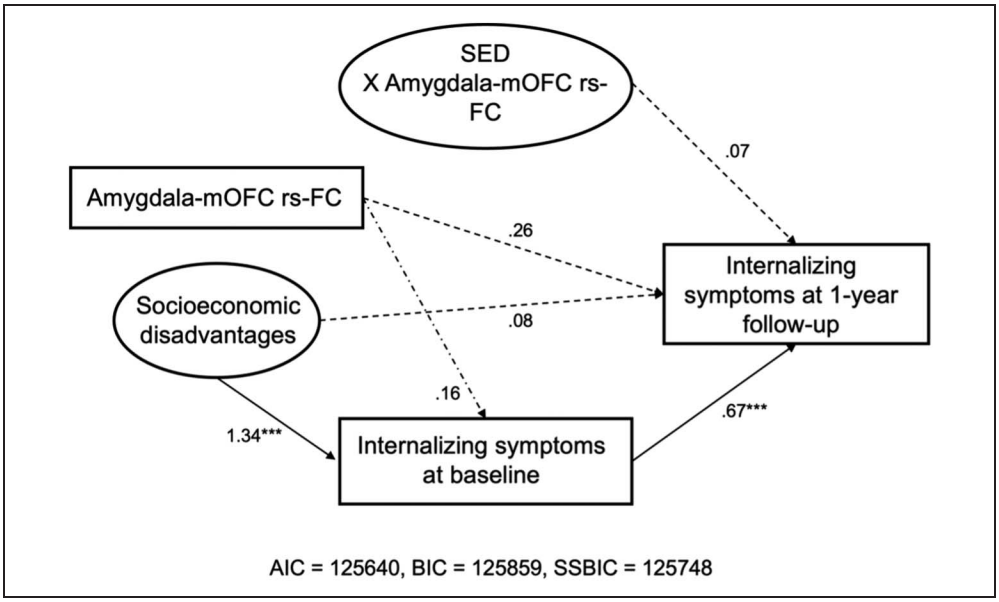


Figure R1. SEM depicting the associations between various types of SED, left amygdala–mPFC/ACC rs-FC, and internalizing symptoms among a subset (n = 3864) of participants who met all inclusion criteria for the sensitivity analysis. Standardized beta coefficients are reported. Only significant paths are shown. *p < .05, **p < .01, ***p < .001.

APPENDIX S

Figure S1. SEM depicting the nonsignificant interactive effect between SED latent construct and bilateral amygdala–mOFC rs-FC on internalizing symptoms among a subset (n = 3864) of participants who met all inclusion criteria for the sensitivity analysis. Unstandardized beta coefficients are reported. *p < .05, **p < .01, ***p < .001.



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APPENDIX T

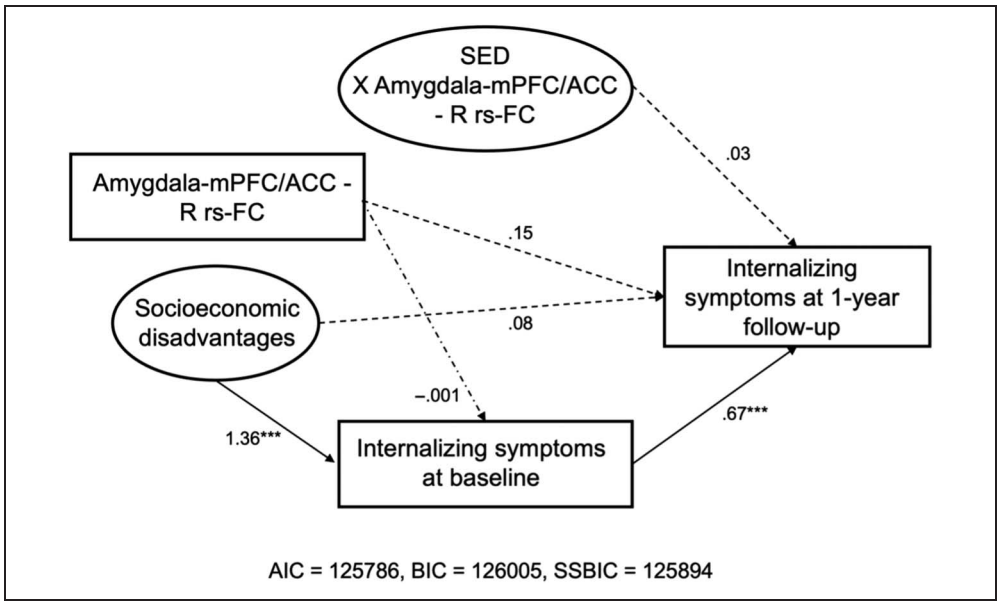

Figure T1. SEM depicting the non-significant interactive effect between SED latent construct and right amygdala–mPFC/ACC rs-FC on internalizing symptoms among a subset (n = 3864) of participants who met all inclusion criteria for the sensitivity analysis. Unstandardized beta coefficients are reported. *p < .05, **p < .01, ***p < .001.

APPENDIX U

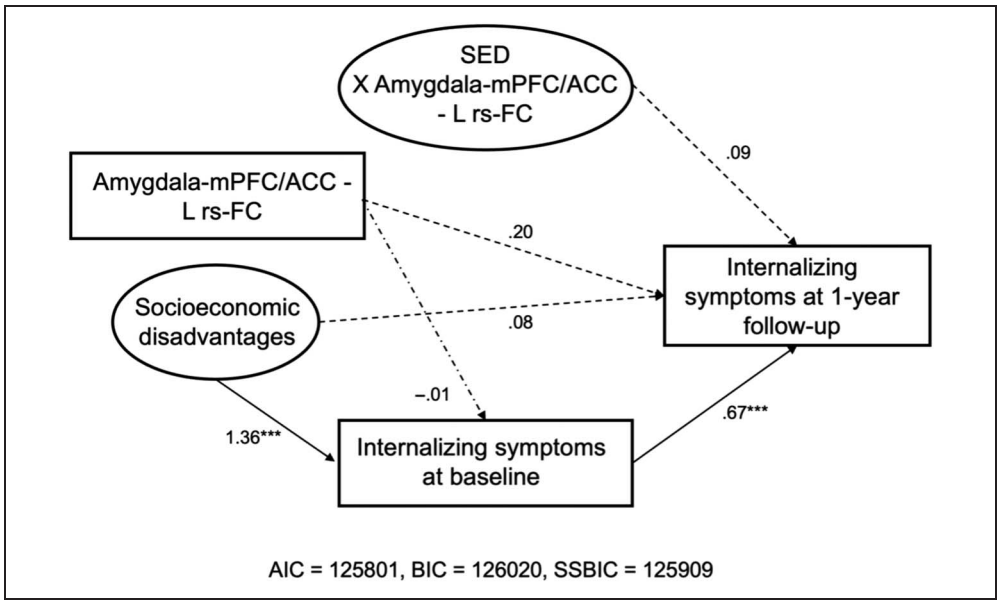

Figure U1. SEM depicting the nonsignificant interactive effect between SED latent construct and left amygdala–mPFC/ACC rs-FC on internalizing symptoms among a subset (n = 3864) of participants who met all inclusion criteria for the sensitivity analysis. Unstandardized beta coefficients are reported. *p < .05, **p < .01, ***p < .001.

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APPENDIX V

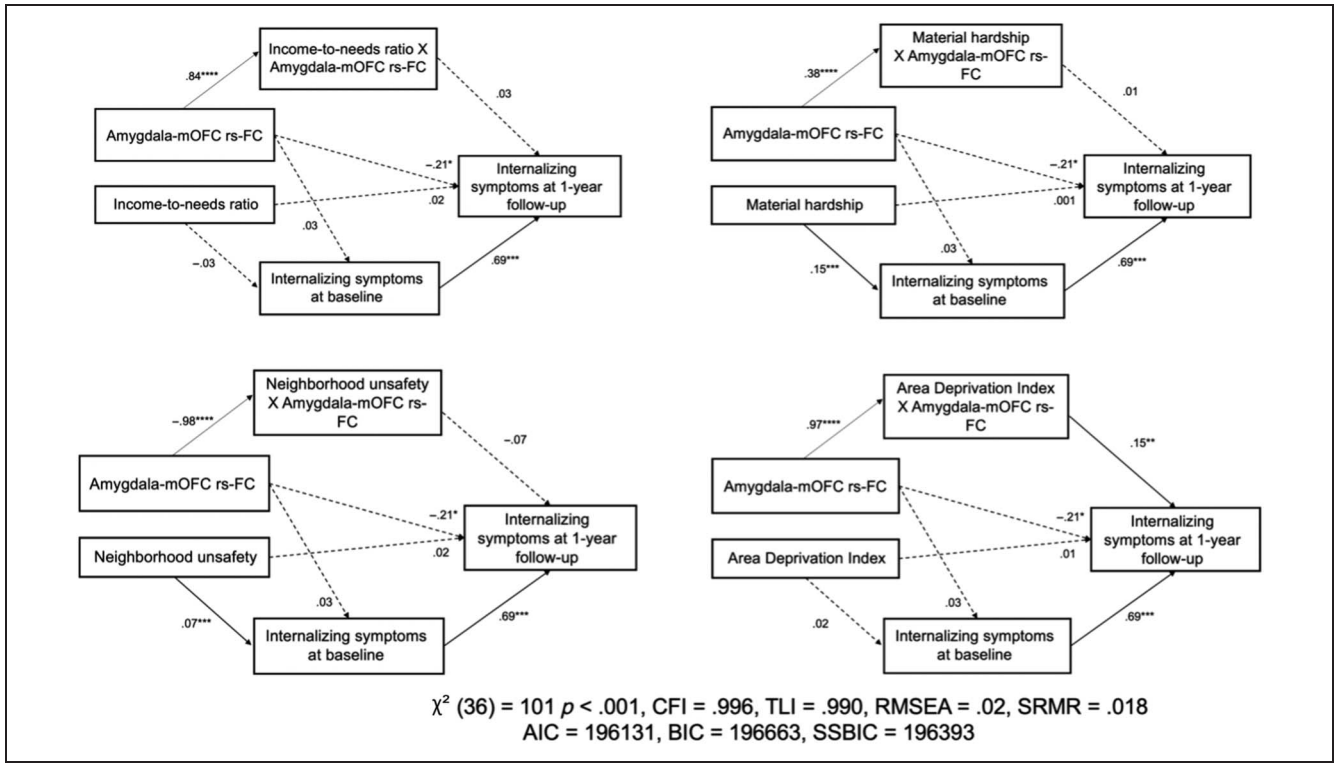

Figure V1. SEM depicting the moderating role of bilateral amygdala–mOFC rs-FC between various types of SED and internalizing symptoms among a subset (n = 3864) of participants who met all inclusion criteria for the sensitivity analysis. Standardized beta coefficients are reported. *p < .05, **p < .01, ***p < .001.

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APPENDIX W

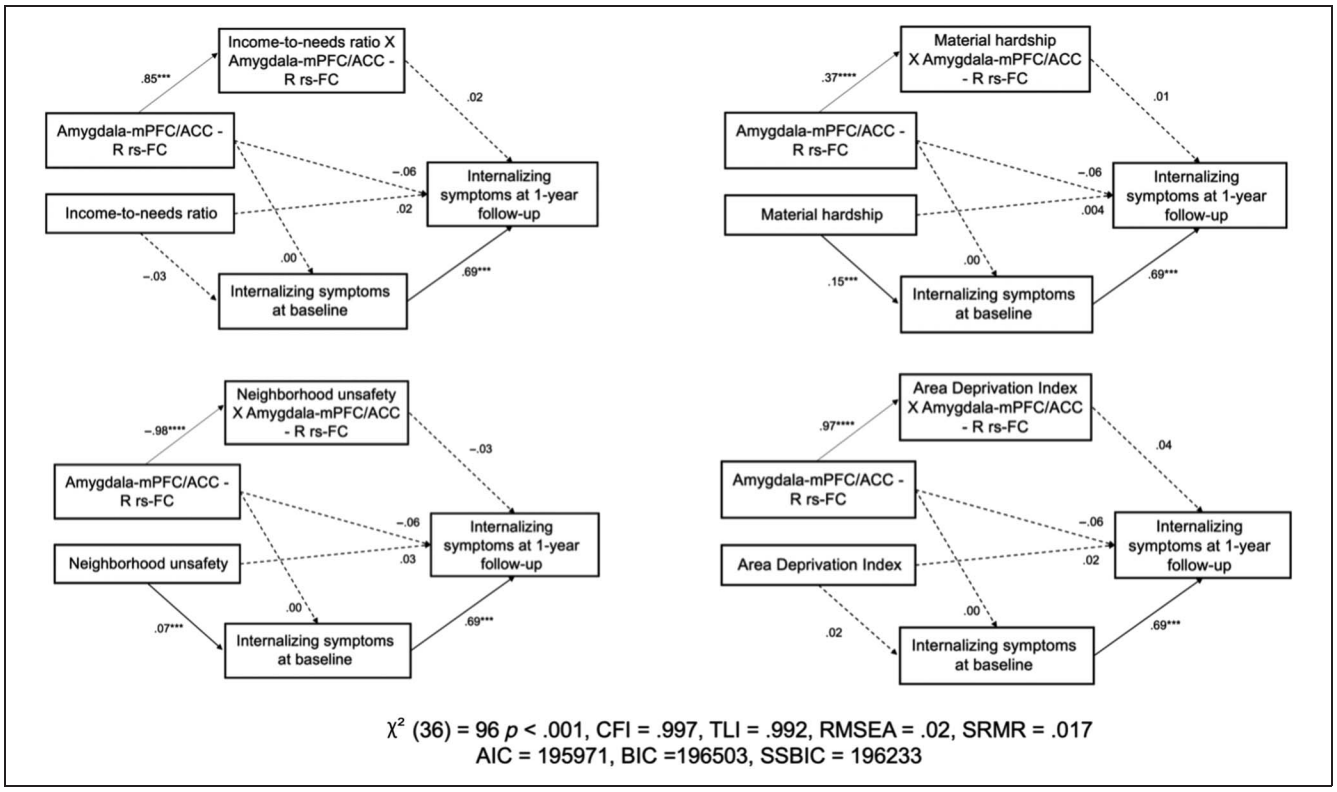

Figure W1. SEM depicting the nonsignificant moderating role of right amygdala–mPFC/ACC rs-FC between various types of SED and internalizing symptoms among a subset (n = 3864) of participants who met all inclusion criteria for the sensitivity analysis. Standardized beta coefficients are reported. *p < .05, **p < .01, ***p < .001.

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APPENDIX X

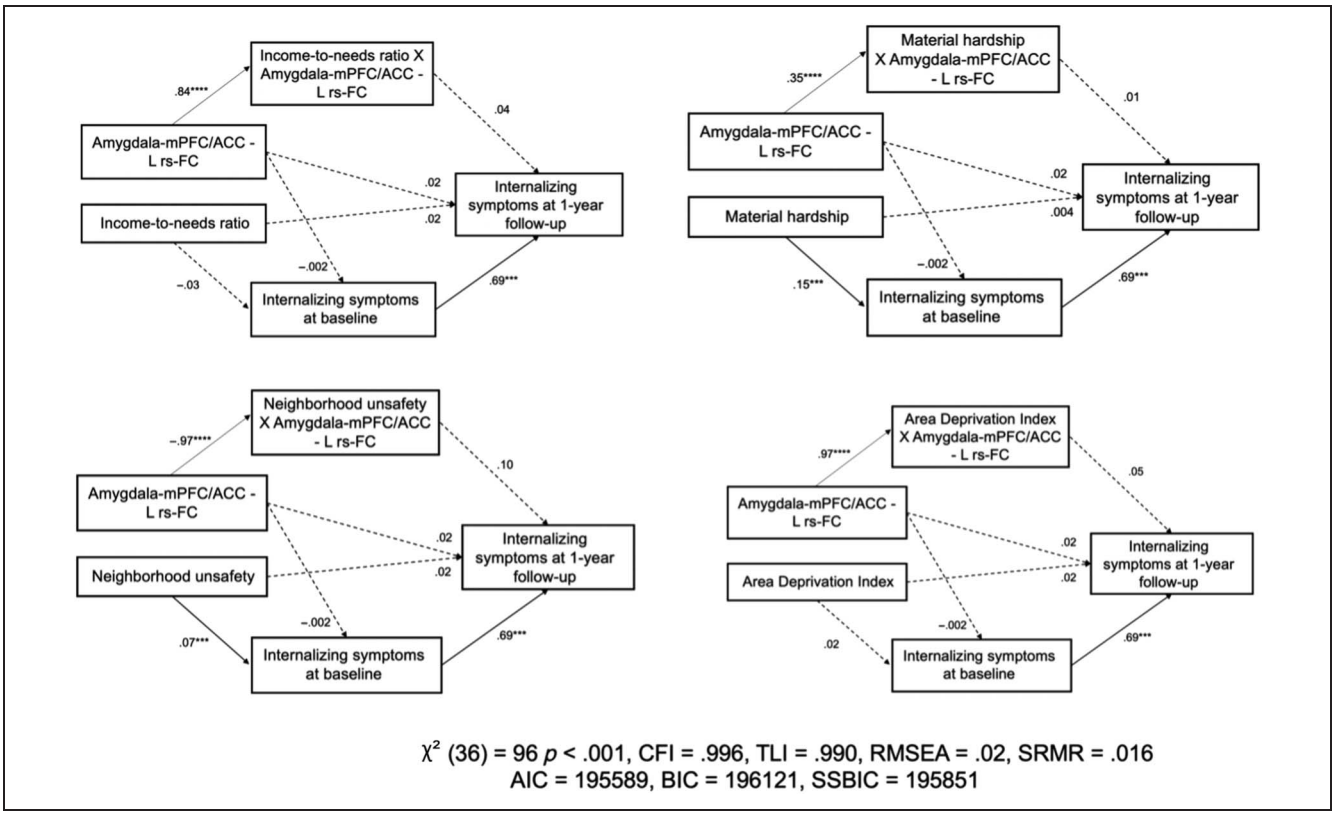

Figure X1. SEM depicting the nonsignificant moderating role of left amygdala–mPFC/ACC rs-FC between various types of SED and internalizing symptoms among a subset (n = 3864) of participants who met all inclusion criteria for the sensitivity analysis. Standardized beta coefficients are reported. *p < .05, **p < .01, ***p < .001.

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Data Availability Statement

Data used in the preparation of this article were obtained from the Adolescent Brain Cognitive DevelopmentSM (ABCD) Study (https://abcdstudy.org), held in the NIMH Data Archive (NDA). This is a multisite, longitudinal study designed to recruit more than 10,000 children age 9–10 and follow them over 10 years into early adulthood. The ABCD Study® is supported by the National Institutes of Health and additional federal partners under award numbers U01DA041048, U01DA050989, U01DA051016, U01DA041022, U01DA051018, U01DA051037, U01DA050987, U01DA041174, U01DA041106, U01DA041117, U01DA041028, U01DA041134, U01DA050988, U01DA051039, U01DA041156, U01DA041025, U01DA041120, U01DA051038, U01DA041148, U01DA041093, U01DA041089, U24DA041123, U24DA041147. A full list of supporters is available at https://abcdstudy.org/federal-partners.html. A listing of participating sites and a complete listing of the study investigators can be found at https://abcdstudy.org/consortium_members/. ABCD consortium investigators designed and implemented the study and/or provided data but did not necessarily participate in analysis or writing of this report. This manuscript reflects the views of the authors and may not reflect the opinions or views of the NIH or ABCD consortium investigators. The ABCD data repository grows and changes over time. The ABCD data used in this report came from NDA Release 3.0 (DOI 10.15154/1519007) and the ABCD Fast Track release (April 2018). The NDA Study for this project contains further details on the sample used and can be accessed at https://dx.doi.org/10.15154/1524642.

Funding Information

This work was supported by the Susan Nolen-Hoeksema Postdoctoral Fellowship to K. Ip; National Science Foundation Graduate Research Fellowship Program award (NSF DGE-1752134) to L. Sisk; Medical Scientist Training Program training grant (NIH/NIGMS T32GM007205) to C. Horien and A. Greene; National Institute of Mental Health R01MH121095 to D. Scheinost and R. Constable; NIH DP5OD021370, NARSAD Young Investigator Award from the Brain & Behavior Research Foundation, and Jacobs Foundation Early Career Research Fellowship to D. Gee; and NIH U01DA041174 to B. Casey, A. Baskin-Sommers, and D. Gee.

Diversity in Citation Practices

A retrospective analysis of the citations in every article published in this journal from 2010 to 2020 has revealed a persistent pattern of gender imbalance: Although the proportions of authorship teams (categorized by estimated gender identification of first author/last author) publishing in the *Journal of Cognitive Neuroscience* (*JoCN*) during this period were M(an)/M = .408, W(oman)/M = .335, M/W = .108, and W/W = .149, the comparable proportions for the articles that these authorship teams cited were M/M = .579, W/M = .243, M/W = .102, and W/W = .076 (Fulvio et al., *JoCN*, 33:1, pp. 3–7). Consequently, *JoCN* encourages all authors to consider gender balance explicitly when selecting which articles to cite and gives them the opportunity to report their article's gender citation balance.

Note

1. Although longer scan times are associated with increases in reliability of rs-FC estimates (e.g., Noble et al., 2017), we chose to use the run with the lowest mean framewise displacement to maximize power, given that our sample size would have been significantly reduced to 1603 participants if we imposed a low-motion threshold over the full course of the 20-min scan.

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