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Differential Developmental Associations of Material Hardship Exposure and Adolescent Amygdala–Prefrontal Cortex White Matter Connectivity

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Abstract

Accumulating literature has linked poverty to brain structure and function, particularly in affective neural regions; however, few studies have examined associations with structural connections or the importance of developmental timing of exposure. Moreover, prior neuroimaging studies have not used a proximal measure of poverty (i.e., material hardship, which assesses food, housing, and medical insecurity) to capture the lived experience of growing up in harsh economic conditions. The present investigation addressed these gaps collectively by examining the associations between material hardship (ages 1, 3, 5, 9, and 15 years) and white matter connectivity of frontolimbic structures (age 15 years) in a low-income sample. We applied probabilistic tractography to diffusion imaging data collected from 194 adolescents. Results showed that material hardship related to amygdala–prefrontal, but not hippocampus–prefrontal or hippocampus–amygdala, white matter connectivity. Specifically, hardship during middle childhood (ages 5 and 9 years) was associated with greater connectivity between the amygdala and dorsomedial pFC, whereas hardship during adolescence (age 15 years) was related to reduced amygdala–orbitofrontal (OFC) and greater amygdala–subgenual ACC connectivity. Growth curve analyses showed that greater increases of hardship across time were associated with both greater (amygdala–subgenual ACC) and reduced (amygdala–OFC) white matter connectivity. Furthermore, these effects remained above and beyond other types of adversity, and greater hardship and decreased amygdala–OFC connectivity were related to increased anxiety and depressive symptoms. Results demonstrate that the associations between material hardship and white matter connections differ across

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key prefrontal regions and developmental periods, providing support for potential windows of plasticity for structural circuits that support emotion processing.

INTRODUCTION

In the United States, 16.2%, or approximately 11.9 million, children and adolescents live below the poverty line, and 32% are near poor (within 200% of the poverty line; Semega, Kollar, Creamer, & Mohanty, 2019). Children who grow up in poverty are at an increased risk for adverse outcomes, including behavioral problems, psychopathology, and delayed cognitive development (Brooks-Gunn & Duncan, 1997). Research has shown that neural regions involved in emotion processing such as the pFC, amygdala, and hippocampus are implicated in the effects of poverty and adverse developmental outcomes (Merz, Tottenham, & Noble, 2018; Kim et al., 2013; Luby et al., 2013; Noble, Houston, Kan, & Sowell, 2012; Hanson, Chandra, Wolfe, & Pollak, 2011). Despite evidence linking poverty to brain function (e.g., functional activation and connectivity) and structure (e.g., cortical volume and surface area; Farah, 2017; Johnson, Riis, & Noble, 2016; Brito & Noble, 2014), much less work has focused on its association with structural connectivity (i.e., white matter tracts that facilitate communication between distinct neural structures) in adolescence. Moreover, although the effects of economic hardship differ across development (Green, Stritzel, Smith, Popham, & Crosnoe, 2018; Duncan, Yeung, Brooks-Gunn, & Smith, 1998), most existing investigations on neural correlates of poverty have not considered the critical importance of developmental timing in the experience of economic hardship (Hyde et al., 2020). Thus, more research is needed to examine how economic hardship across development relates to adolescent white matter tracts that link emotion processing regions.

Although most studies examining frontolimbic white matter structures using diffusion MRI (dMRI) have focused on density of major white matter tracts (e.g., fractional anisotropy of the superior longitudinal fasciculus; Rosen, Sheridan, Sambrook, Meltzoff, & McLaughlin, 2018; Noble, Korgaonkar, Grieve, & Brickman, 2013), there has been no examination of white matter microstructures using probabilistic tractography method. As compared with conventional deterministic approaches to tracking white matter fibers, probabilistic tractography has been found to be more sensitive in modeling complex multiple-fiber crossings in diffusion-based data (Behrens, Berg, Jbabdi, Rushworth, & Woolrich, 2007). Using this method to estimate the maximum likelihood of white matter connectivity between seed and target ROIs (Greening & Mitchell, 2015; Behrens et al., 2007), our previous investigation (Goetschius et al., 2019) drew on evidence from tracer studies in nonhuman primates to identify the presence of white matter fibers connecting the amygdala to numerous pFC regions. Results demonstrated that white matter connectivity is particularly widespread from the amygdala to the subgenual ACC (sgACC) BA 25, orbitofrontal (OFC) BA 11 and BA 47, and dorsomedial pFC (dmPFC) BA 10 regions of the pFC—regions that are all implicated in emotion processing (Barbas, 2015; Ghashghaei, Hilgetag, & Barbas, 2007; Barbas, Saha, Rempel-Clower, & Ghashghaei, 2003). Studies have also identified structural connections between these prefrontal regions and the hippocampus (Barbas & Blatt, 1995) as well as robust white matter connections between the hippocampus and amygdala (Colnat-Coulbois et al., 2010). These findings indicate that further examination of

white matter connectivity within these frontolimbic circuits is necessary to better understand affective processes relating to socioeconomic hardship.

Though more research is needed to examine the potential impacts of poverty on white matter microstructure across the corticolimbic circuit, it is also important to consider the developmental timing of when children experienced poverty. Recent evidence suggests that activities in specific brain regions (e.g., amygdala) are more sensitive to disadvantaged environment during early childhood whereas others (e.g., pFC) during adolescence (Gard et al., 2021). Although the amygdala and hippocampus develop rapidly during early childhood, pFC undergoes an especially protracted development lasting through adolescence (Blakemore, 2012; Giedd & Rapoport, 2010; Lenroot & Giedd, 2006). Equally important, stress during later childhood and adolescence may also contribute to structural development of frontolimbic regions, especially considering that white matter proliferates in density and mass through adolescence and into adulthood (Giedd et al., 1999). Thus, it is important to consider the timing of exposure to poverty as both the brain and social ecology surrounding poverty are different during early childhood versus adolescence.

Moreover, though the timing of exposure to poverty may be important, the total exposure to poverty across time and how poverty changes over time may have distinct effects. A wealth of literature demonstrates that the cumulative effect and duration of exposure to poverty influences many child developmental outcomes (National Institute of Child Health and Human Development Early Child Care Research Network, 2005; Korenman, Miller, & Sjaastad, 1995). Furthermore, research has found that changes in poverty across time (i.e., trajectory) may be important consideration for its relation to mental health (McLeod & Shanahan, 1996). Thus, improvements or deepening of poverty over time produces qualitatively different experiences for children and are important considerations when examining poverty effects on neural development.

In addition to understanding the impact of timing and cumulative exposure to poverty, work on the neuroscience of poverty should consider measures that capture the lived experience of economic hardship. Measuring whether food, residential, and medical needs are being met (i.e., material hardship) characterizes the immediate challenges and impacts of poverty (Nelson, 2011; Gershoff, Aber, Raver, & Lennon, 2007). Moreover, the effect of family income on child development may vary by region (e.g., cost of living) and other attributes of the family (e.g., accumulated wealth, family support; Gershoff et al., 2007). Although material hardship has been linked to children's behavioral outcomes (Zilanawala & Pilkauskas, 2012; Gershoff et al., 2007), only one study thus far has examined the association between material hardship with white matter density in school-aged children (Lichtin et al., 2021), and no known study has examined developmental timing effects of material hardship, or how it may relate to frontolimbic white matter connections quantified using probabilistic tractography in adolescents.

This study addressed two aims using open-science preregistered analyses: (1) to examine the association between cumulative material hardship and frontolimbic white matter connectivity (amygdala–pFC, hippocampus–pFC, and hippocampus–amygdala) and (2) to assess longitudinal/developmentally specific associations of material hardship from

childhood to adolescence with adolescent white matter connectivity. We predicted that there would be negative associations between cumulative material hardship and white matter connectivity given the existing literature documenting decreased white matter density associated with poverty (Brito & Noble, 2014) and that the associations between material hardship and white matter would differ across developmental periods. Because few studies have examined the developmental timing of poverty with brain structures and both early and late childhood experiences are important for brain development, we did not have a directional hypothesis for timing effects. Finally, in an exploratory analysis, we examined links among material hardship, white matter connectivity, and internalizing symptoms to anchor our brain findings to adolescent affective functioning. Specifically, we assessed the associations between material hardship with symptoms of anxiety and depression in adolescence and the associations between white matter connectivity with anxiety and depressive symptoms.

METHODS

Sample

We addressed these aims in a sample of families with identities and experiences that have been underrepresented in neuroimaging research (e.g., marginalized or non-Western, educated, industrialized, rich, and democratic samples; Falk et al., 2013; Henrich, Heine, & Norenzayan, 2010). Participants were recruited from the Fragile Families and Child Wellbeing Study, a population-based sample of 4,898 children born in large U.S. cities (population over 200,000), with an oversampling of nonmarital births, which led to a high representation of low-income and minority families (Reichman, Teitler, Garfinkel, & McLanahan, 2001). Longitudinal data were collected when the child was 1, 3, 5, 9, and 15 years old through in-home visits and phone calls (Reichman et al., 2001). At 15 years of age, a subsample of families was invited to participate in the Study of Adolescent Neural Development. Out of 237 teens, 52.7% were female with an average age of 15.9 years old, 76% identified as Black, and more than 54% reported family income below $40,000. After exclusions because of missing data and poor imaging quality, this study included 189 participants with amygdala–seed and 191 participants with hippocampus–seed (see Table 1 for characteristics of full sample and included sample). Statistical tests comparing demographic characteristics (age, pubertal development, sex, race, and income) of included sample from the full sample showed no significant differences between the groups ($p > .5$).

Behavioral Measures

Material Hardship—Longitudinal measurements of material hardship were collected in five waves. During assessments of children ages 1, 3, 5, 9, and 15 years, caregivers indicated whether or not they experienced housing, utility, food, medical, and financial hardship within the past year. The primary caregiver reported yes (1) or no (0) on each of the following eight items: (1) received free meals, (2) did not pay full rent/mortgage, (3) evicted for not paying full rent/mortgage, (4) did not pay full gas/oil/electric bill, (5) borrowed money from family/friends to pay bills, (6) moved in with people because of financial problems, (7) stayed in place not meant for regular housing, and (8) did not receive medical care. These items were drawn from the 1996 Survey of Income and Program Participation;

the 1997 and 1999 New York City Social Indicators Survey; and the 1999 Study of Work, Welfare, and Family Well-Being of Iowa families on Iowa's assistance program and are comparable to past investigations on material hardship and poverty (Bauman, 1999; Mayer & Jencks, 1989). Responses were collected from the mother if the child lived with their mother at least half of the time. Cumulative material hardship score was measured by summing endorsed material hardship items across all waves. Material hardship during early (ages 1 and 3 years) and middle (ages 5 and 9 years) childhood were computed by averaging the age-specific material hardship scores within the developmental period (see Table 2 for zero-order correlations of all hardship variables).

Internalizing Symptoms—Anxiety symptoms were measured separately using parent-only and child-only report on the Screen for Child Anxiety Related Disorders (Birmaher et al., 1997) at child age of 15 years. Anxiety was measured using a 3-point scale (0 = *almost never*, 1 = *sometimes*, 2 = *often*), and scores range from 0 to 76 (α = .92). Depressive symptoms were measured separately using parent-only and child-only report on the Mood and Feelings Questionnaire (Angold, Costello, Messer, & Pickles, 1995) at child age of 15 years. Depression was measured using a 3-point scale (0 = *not true*, 1 = *sometimes*, 2 = *true*), and scores range from 0 to 68 (α = .91). Scores on both scales were computed by summing all responses endorsed, with higher scores indicating greater report of adolescent anxiety and depression symptoms.

Covariates—The following measures were added as covariates in subsequent sensitivity analyses: birth city, maternal education, child race, sex, pubertal age, and family structure. Birth city was added as a covariate to account for any confounding sampling differences and was based on the location where child was born and the first interview with mother was conducted. In this study, birth city was classified as either Detroit, MI; Toledo, OH; Chicago, IL; or other (i.e., Baltimore, Indianapolis, Jacksonville, Pittsburgh). Cities under "other" were categorized under one singular variable to minimize the number of variables, and three dummy-coded variables were created to reflect birth city, with Detroit set as the reference group. Maternal education at child's birth was measured using a 4-point scale (0 = *less than high school*, 1 = *high school or equivalent*, 2 = *some college/technical school*, 3 = *college or graduate school*) and was added as a covariate to account for any confounding differences stemming from parental education. Ethnoracial identity, a socially constructed category, was utilized as a covariate to adjust for the potential effects of structural or interpersonal racism and the various ways in which these experiences shape development (e.g., neighborhood residence, school access). Ethnoracial identity was measured using three dummy-coded variables to reflect Black (reference variable), White, Hispanic, and other/multiracial and was self-reported at 15 years of age. In the event of missing reports, parent-reported race/ethnicity was utilized (n = 8). Sex was measured through parent report at child age of 1 year using two categories: 1 = *male* and 0 = *female*. Pubertal age was measured via youth report at 15 years of age on the Pubertal Development Scale (Petersen, Crockett, Richards, & Boxer, 1988) that measured changes in child height, body hair, skin, facial hair and voice (male participants only), and breast development and menarche (female participants only). Responses were coded on a 4-point scale (1 = *no development* to 4 = *completed development*); score was a sum of all items endorsed. Pubertal development was treated

as a covariate to account for differences in neural structures stemming from differences in pubertal-related maturation (Giedd et al., 1999). Family structure was included as a covariate to consider household composition and differences in family function between 0 = single and 1 = two-parent families (McLanahan & Sandefur, 1994). A household was considered a two-parent household when the biological father was cohabitating with the biological mother and child at the time of the child's birth.

Additionally, to account for stress-related factors that may be related to white matter connectivity, as well as examine mechanisms isolated to material hardship above and beyond the effects of other measures of poverty, we adjusted for (1) childhood exposure to violence and social deprivation and (2) annual household income in subsequent sensitivity analyses. Violence exposure and social deprivation were measured using composite standardized scores of exposure at ages 3, 5, and 9 years (Hein et al., 2020). Violence exposure score was based on: (1) parent responses on child physical and emotional abuse items in the Parent–Child Conflict Tactics Scale (Straus, Hamby, Finkelhor, Moore, & Runyan, 1998), (2) child's exposure to or victimization of violence in the neighborhood report (Zhang & Anderson, 2010), and (3) maternal report of intimate partner violence (physical, emotional, or sexual) in the home (Hunt, Berger,& Slack, 2017). Social deprivation composite score was based on (1) parent responses on child physical and emotional neglect on the Parent–Child Conflict Tactics Scale (Straus et al., 1998) and (2) report of neighborhood social cohesion (Donnelly et al., 2016; Morenoff, Sampson, & Raudenbush, 2001). In both measures, observation was coded as missing if child did not live with parent for at least half the time at each wave. Scores on each measure were first standardized, and *z* scores across each dimension (violence exposure; social deprivation) across each wave (ages 3, 5, and 9 years) were then summed. Annual household income was reported by primary caregiver at each wave. In instances where annual household income was not reported, the variable was imputed using the partner's report (if cohabitating) or regression-based imputation if neither caregiver reported household income.

MR Measures

MRI Acquisition—As described in other related publications (Goetschius et al., 2019; 2020; Hein et al., 2018), MRI images were acquired using 3 T GE Discovery MR750 scanner with eight-channel head coil at University of Michigan Functional MRI Laboratory. Head movement was limited through the use of head paddings and detailed instructions provided to participants. T1-weighted gradient-echo images were taken before subsequent scans using same field of view (repetition time = 12 msec, echo time = 5 msec, inversion time = 500 msec, flip angle = 15°, field of view = 26 cm, slice thickness = 1.44 mm, 256 × 192 matrix, 110 slices).

dMRI Processing—dMRI images that were used to identify the microstructural properties of white matter tracts (Jones, Knösche, & Turner, 2013) were captured using spin-echo EPI diffusion sequence. Scan parameters used were as follows: repetition time = 7250 msec, minimum echo time, 128 × 128 acquisition matrix, field of view = 22 cm, 3-mm thick slices (no gap), 40 slices acquired using alternating–increasing order, *b* value = 1000 s/mm^2, 64 nonlinear directions; five *b* = 0 s/mm^2 T2 images (b0) acquired. dMRI data from this

sample were utilized in prior work (Goetschius et al., 2019, 2020; Hein et al., 2018), and we used the same processing procedures from previous publications (Goetschius et al., 2019, 2020). dMRI images were visually inspected for quality. Slices with an average intensity lower than 4 *SD*s or more, as predicted by *eddy's* Gaussian, were marked as outliers and replaced with model predictions (Andersson, Graham, Zsoldos, & Sotiropoulos, 2016). Participants were excluded if more than 5% of slices were replaced ($n = 1$). For included participants, 0–4.35% of slices (median of 0.43%) were replaced. Images for 10 participants with most replaced slices were further visually inspected to ensure there were no additional abnormal artifacts. White matter fibers were mapped using probabilistic tractography method. Probabilistic tractography utilizes a Bayesian algorithm to track pathways between neural regions by creating an individual-level density function representing the probability that specified seed region voxels will reach specified target region voxels (Greening & Mitchell, 2015; Behrens et al., 2007). Using this method, we quantified the likelihood of white matter connections between amygdala–pFC, hippocampus–pFC, and hippocampus–amygdala for each participant. Each lateral hemisphere of the amygdala and hippocampus was utilized as separate seed regions, and each lateral pFC region was utilized as separate targets. pFC targets were BA 9, BA 10, BA 11, BA 24, BA 25, BA 32, and BA 47, corresponding to medial and OFC regions that have been found to be particularly connected to the amygdala and hippocampus, consistent with previous Study of Adolescent Neural Development investigations (Goetschius et al., 2019) and nonhuman primates tracer studies (Barbas & Blatt, 1995).

Raw dMRI images in DICOM format were first converted to NIFTI format using MRIcron (dcm2niix – 2MAY2016) for off-line analysis using MRtrix (v.3.0.R3; Veraart et al., 2016) and FSL (v.5.0.9) FMRIB's Diffusion Toolbox (v.3.0; Jenkinson, Beckmann, Behrens, Woolrich, & Smith, 2012). The NIFTI diffusion files were corrected for motion, eddy current, and signal dropout with MRTrix *dwipreproc* using FSL *eddy* (v.5.0.11; Andersson & Sotiropoulos, 2016).

Bedpostx was performed using standard settings (number of fibers modeled per voxel = 2, multiplicative factor weight = 1, burn in = 1000; Hernández et al., 2013) using Markov chain Monte Carlo sampling at individual voxel (Behrens et al., 2007); FLIRT registration (Jenkinson, Bannister, Brady, & Smith, 2002; Jenkinson & Smith, 2001) was performed to allow linear mapping between diffusion, standard, and structural spaces; probtrackx2 (nsamples per voxel = 5000, nsteps per sample = 2000, step length = 0.5 mm, curvature threshold = 0.2, fibthresh = 0.01, distthresh = 0.1; Hernández-Fernández et al., 2016) then computed the estimated probability of white matter connectivity between target and seed regions (Eickhoff et al., 2010; Behrens et al., 2003, 2007; Johansen-Berg et al., 2004). Individual masks for target and seed regions were then created using WFU Pick Atlas (v.3.0.5b; Maldjian, Laurienti, Kraft, & Burdette, 2003). After individual-level probabilistic tractography mapping was completed, resulting processed images were transformed to Montreal Neurological Institute space for group-level analysis. Each image was divided by the number of samples per voxel (5000), and an average seed image was created. Additionally, peak voxel representing the maximum probability of white matter connectivity between seed and target regions was identified using fslmaths (FMRIB). Amygdala–pFC

white matter tracts were analyzed using FSL Version 5.9, whereas hippocampus–pFC and hippocampus–amygdala tracts were analyzed using identical scripts on updated FSL 6.0.

Processed dMRI yielded a likelihood coefficient that seed and target regions were connected. Higher coefficient indicated greater connectivity between seed and target regions. The five connections (within each pair) with the highest white matter connectivity coefficient values were included in subsequent analyses to simplify our model estimates (see Table 3 for zero-order correlations of all white matter connectivity metrics). Amygdala–pFC pairs that were included in our analyses are as follows: left amygdala–BA 25, right amygdala–BA 10, right amygdala–BA 11, right amygdala–BA 25, and right amygdala–BA 47 (Figure 1). The highest five hippocampus–pFC connections that were included in subsequent analyses are as follows: left hippocampus–BA 10, right hippocampus–BA 10, right hippocampus–BA 11, right hippocampus–BA 25, and right hippocampus–BA 47. Both right and left hippocampus–amygdala connectivity were included in our analyses.

Statistical Analysis

Preregistered Analyses—Study measures and statistical analyses were preregistered at the Open Science Framework (https://osf.io/c3tf8). Three series of multiple regression models were performed to address our study aims. First, a cumulative score of material hardship was used in three separate multiple regression models to predict amygdala–pFC, hippocampus–pFC, and hippocampus–amygdala white matter connectivity at age 15 years. Second, to examine differential developmental associations between material hardship and white matter connectivity, we performed five multiple regression analyses (one for each prefrontal region) using material hardship summed across developmental stages (early: ages 1 and 3 years; middle: ages 5 and 9 years; adolescent: age 15 years) as separate predictors within the same model. Third, material hardship at each developmental age (1, 3, 5, 9, and 15 years) was included as separate predictors within the same five regression models. To correct for multiple seed–target combinations and avoid false positives, false discovery rate (Benjamini & Hochberg, 1995) correction was performed across each type of analysis (cumulative, developmental stages, and individual ages) for each brain region (five for amygdala–seed analysis: left amydala–BA 25, right amygdala–BA 10, right amygdala–BA 11, right amygdala–BA 25, and right amygdala–BA 47; five for hippocampus–seed analysis: left hippocampus–BA 10, right hippocampus–BA 10, right hippocampus–BA 11, right hippocampus–BA 25, and right hippocampus–BA 47; two for hippocampus–amygdala analysis: left and right hemispheres). Following preregistration, multiple regression analyses with main predictors and outcome variables were performed to test for main effects. If main effects were found, covariates were then added as sensitivity analyses.

Exploratory Analyses—To confirm the results of our preregistered multiple regression analyses, we tested the associations between material hardship and white matter connectivity using structural equation modeling framework. All material hardship predictors at each time point, amygdala–pFC white matter connectivity at each region, and all covariates were examined in the same model using MPlus (Muthén & Muthén, 1998–2011). Furthermore, to examine the trajectory of material hardship exposure over time and its relation to white matter connectivity during adolescence, we performed a latent growth curve analysis using

MPlus. Following the approach used by Gard et al. (2021), linear and quadratic trajectories were fit separately on material hardship data over five waves (ages 1, 3, 5, 9, and 15 years), and model fit indices were examined based on standard threshold (Hu & Bentler, 1999). Material hardship at age 1 year was fixed with loading 0 to indicate initial point of trajectory (i.e., the intercept). Loadings for subsequent time points were fixed at 2, 4, 8, and 14, which account for unequal time intervals in between observations (Duncan & Duncan, 2004). Estimated latent slope (change over time) and intercept (initial point) of material hardship were examined before paths were added from latent slope and intercept predicting white matter connectivity metrics. Model fit indices were then reexamined and path coefficients were reported if model fit was deemed adequate. Finally, in an exploratory analysis to anchor findings with affective-related behavioral outcomes, associations between material hardship and white matter connectivity with adolescent anxiety and depression were tested. Results from the regression analyses were utilized to inform these two exploratory analyses, and only regions that were found to have significant associations in previous analyses were examined. Furthermore, to examine any associations via white matter connectivity, indirect effects between hardship and adolescent anxiety and depression symptoms through white matter connectivity were additionally tested using path modeling in MPlus.

RESULTS

Preregistered Analyses

Null Cumulative Effect of Material Hardship—There were no significant relations between cumulative material hardship with amygdala–prefrontal and hippocampus–prefrontal white matter connectivity (Table 4). There was a significant positive relation between cumulative material hardship and left hippocampus–amygdala, but it did not survive correction for multiple comparisons ($r = .144$, $p = .047$, $p_{adjust} = .094$).

Differential Relations between Material Hardship at Specific Developmental Stages and Specific Amygdala–pFC White Matter Connectivity—Material hardship experienced at specific developmental stages differentially related to white matter connectivity between amygdala and pFC regions (Table 5; Figure 2). Specifically, controlling for other developmental periods, hardship experienced in middle childhood (ages 5 and 9 years) related to increased right amygdala–BA 10 (dmPFC; $\beta = .27$, $p = .009$, $p_{adjust} = .045$) white matter connectivity. Additionally, material hardship during adolescence (age 15 years) was related to increased left amygdala–BA 25 (sgACC; $\beta = .24$, $p = .009$, $p_{adjust} = .040$) and decreased right amygdala–BA 11 (OFC; $\beta = -.22$, $p = .016$, $p_{adjust} = .040$) white matter connectivity, when accounting for other developmental periods. These relations remained significant after false discovery rate correction (Table 5 and adjusted p values reported above) and sensitivity analyses including all covariates (see Table 7). There were significant associations between material hardship at adolescence (age 15 years) and left hippocampus–BA 10, but this association did not survive adjustment for multiple comparison (dmPFC; $\beta = .19$, $p = .038$, $p_{adjust} = .190$). There were no association between material hardship and hippocampus–amygdala white matter connectivity.

Examination of material hardship at individual ages and amygdala–pFC white matter connectivity reflected similar patterns (Table 6). Material hardship at age 5 years was related to increased right amygdala–BA 10 (dmPFC; β = .33, p = .001, p_{adjust} = .005) and material hardship at adolescence (age 15 years) was related to increased left amygdala–BA 25 (sgACC; β = .24, p = .010, p_{adjust} = .049), when controlling for other age time points. There was also a trending negative association between material hardship and amygdala–BA 11 (OFC; β = −.20, p = .031, p_{adjust} = .078) and left hippocampus–BA 10 (dmPFC; β = .20, p = .034, p_{adjust} = .170), but results did not survive correction for multiple comparisons. Furthermore, positive associations between hardship at age 5 years and amgydala–BA 10 (dmPFC) as well as hardship at age 15 years and amygdala–BA 25 (sgACC) remained when accounting for all covariates (Table 7).

Exploratory Analyses

Differential Associations between Material Hardship at Specific Ages Were Also Reflected Using Structural Equation Modeling Framework—Structural equation model testing the associations between material hardship at all ages and amygdala–pFC white matter connectivity showed consistent findings with multiple regression results. There were positive associations between material hardship at age 5 years with amygdala–BA 10 (dmPFC; β = .37, p < .001) white matter connectivity and material hardship at age 15 years with amygdala–BA 25 (sgACC; β = .21, p = .010; Figure 3). Furthermore, there were trending negative associations between material hardship at 9 and 15 years with amygdala–BA 11 (OFC) white matter connectivity (age 9 years: β = −.15, p = .081; age 15 years: β = −.14, p = .096). All covariates were included in the model, and the model had excellent fit, $\chi^2(45) = 47.931$, p = .355 (root mean square error of approximation [RMSEA] = .019 [.000, .053], comparative fit index [CFI] = .980, Tucker–Lewis index [TLI] = .965, standardized root mean square residual [SRMR] = .047).

Change of Material Hardship Over Time Differentially Related to Specific Amygdala–pFC White Matter Connectivity—We utilized latent growth curve modeling to examine early childhood levels (intercept) and change over time (slope) of material hardship and how they related to amygdala–pFC white matter connectivity. Model fit indices indicated that the linear trajectory of material hardship had moderate fit (Hu & Bentler, 1999), $\chi^2(10) = 21.577$, p = .017 (RMSEA = .070 [.028, .111], CFI = .934, TLI = .934, SRMR = .064). Estimated mean for initial levels of material hardship (i.e., intercept) was positive (mean [*SE*] = 1.01 [0.077], p < .001; variance [*SE*] = 0.91 [0.166], p < .001), and there was an increasing trajectory of material hardship across time (estimated slope mean [*SE*] = 0.02 [0.008], p = .009; variance [*SE*] = 0.01 [0.002], p = .004). Intercept and slope were negatively related to each other (β = −.40, p < .001; Figure 4). A quadratic curve model did not converge, indicating poor model fit of nonlinear trajectory of material hardship.

Next, covariates and additional paths from slope and intercept to select amygdala–pFC white matter connectivity regions were added to the base growth curve model. There were significant associations between change in material hardship over time (slope) and two separate amygdala–pFC targets (left BA 25 sgACC and right BA 11 OFC; Figure 5).

Specifically, faster increases in material hardship over time were associated with greater left amygdala–sgACC (β = .23, p = .007) and less right amygdala–OFC (β = −.21, p = .017) white matter connectivity (Figure 5). The model also demonstrated moderate but not strong fit: $\chi^2(74) = 102.37$, p = .016; RMSEA = .045 [.020, .065], CFI = .920, TLI = .892, SRMR = .062.

Increased Material Hardship Was Related to Increased Anxiety and Depression, and Decreased Amygdala–OFC White Matter Connectivity Was Related to Increased Anxiety and Depression—Material hardship (cumulative, ages 3, 5, 9, and 15 years) was positively related to anxiety and depressive symptoms (Table 8). Moreover, decreased right amygdala–OFC was related to both increased anxiety and depression (anxiety, r = −.16, p = .027; depression, r = −.16, p = .032). There were no significant indirect effects in any models testing white matter connectivity as mediators (Figure 6). Furthermore, these results were observed using parent-reported symptoms, but not youth-reported symptoms (Table 9).

DISCUSSION

This study examined the association between material hardship across developmental stages and emotion-related white matter connectivity in adolescence. Our results indicate that material hardship in middle childhood (particularly at age 5 years) was related to increased amygdala–dmPFC white matter connectivity, whereas material hardship experienced during adolescence (age 15 years) was related to increased amygdala–sgACC and decreased amygdala–OFC white matter connectivity. When examined across development using growth curve modeling, a greater rate of increase in material hardship over time was related to increased amygdala–sgACC white matter connectivity and decreased amygdala–OFC white matter connectivity. Furthermore, in an exploratory analysis, material hardship was related to greater adolescent anxiety and depression, and decreased amygdala–OFC white matter connectivity was also related to increased anxiety and depression. Taken together, these findings demonstrate how the associations between hardship and corticolimbic white matter connectivity, which were developmental timing-specific and were compounded by deepening exposure to hardship across time, are important considerations for adolescent mental health.

These associations between material hardship and white matter connectivity clarify prior inconsistent findings that examined associations of socioeconomic hardship and white matter structures. Although several studies found that greater financial hardship was associated with reduced white matter density (Rosen et al., 2018; Dufford & Kim, 2017; Gianaros, Marsland, Sheu, Erickson, & Verstynen, 2013), one study found a positive association (Simon et al., 2021) and another found no associations between socioeconomic hardship and white matter density (Chiang et al., 2011). Here, utilizing probabilistic tractography in a longitudinal sample, this study suggests that the adversity–brain associations may depend on when hardship was experienced and where in the brain it occurred. In particular, exposure to more material hardship during middle childhood was positively related to white matter connectivity between the amygdala and a prefrontal region associated with higher level social cognition (i.e., dmPFC BA 10; Roca et al., 2011),

whereas hardship during adolescence was related to structural connections between the amygdala and prefrontal regions linked to emotion processes (i.e., OFC BA 11; Ueda, Fujimoto, Ubukata, & Murai, 2017) and psychopathology (i.e., sgACC BA 25; Mayberg et al., 2005). Moreover, the present findings echo research on the associations between poverty and reduced white matter density (Brito & Noble, 2014), as well as support potential accelerated maturation relating to adverse experiences (Callaghan & Tottenham, 2016; Gee et al., 2013) that are specific to certain neural regions (e.g., ACC; Thijssen, Collins, & Luciana, 2020). Collectively, these results suggest that certain brain regions may have greater sensitivity for adversity experienced at particular developmental ages. Findings that are specific to both brain regions and developmental timing in this study may have been afforded by the spatial precision of probabilistic tractography as well as the longitudinal approach.

Differential effects of hardship across development are unsurprising when we consider the dissimilar social environment of younger children from their older counterparts. Compared with adolescents, younger children spend considerable time at home with their caregivers (National Institute of Child Health and Human Development Early Child Care Research Network, 2005; Hofferth & Sandberg, 2001). From age 6 years onward, children spend an increasing amount of time in school and outside-the-home activities (Hofferth & Sandberg, 2001). As they enter adolescence, children also become more self-sufficient and demonstrate increased autonomy from parents (Steinberg, 1988). Resources providing nutritional and intellectual enrichment previously available solely through primary caregivers are now accessible through the school system (Bradley, Convyn, Burchinal, McAdoo, & Coll, 2001). As such, exposure to poverty, particularly material hardship, which captures the lack of very specific provisions, may subsequently shape emotion-linked neural structures differentially. Here, our results show that these differences in the environment from early to later childhood and adolescence may also be observed in specific amygdala–pFC (dmPFC, sgACC, and OFC) white matter connectivity. It is noteworthy that we found no early childhood (i.e., ages 1 and 3 years) correlates of material hardship and white matter connectivity. This finding is consistent with research examining material hardship and child behavioral outcomes (internalizing, externalizing problems) in the Fragile Families and Child Wellbeing Study sample (Zilanawala & Pilkauskas, 2012), which, taken together with present findings, suggests that more proximal experiences may pose greater influence on white matter connectivity. Nevertheless, more research is needed to understand long-term consequences of hardship during early childhood on brain development.

Consistent with results showing associations between age-specific material hardship and white matter connectivity, results from the latent growth curve analysis showed that increased material hardship across time also related to white matter connectivity in the same regions (dmPFC, sgACC). These results suggest that worsening exposure to material hardship over time, but not its initial level, may play a role in white matter development. Consistent with prior research, deepening experience of poverty over time may lead to greater impact on children's health and wellbeing (McLeod & Shanahan, 1996). This pattern of increasing material hardship over time in the sample may be due to the Great Recession (Garfinkel, McLanahan, & Wimer, 2016), which coincided with data collection at age 9 years. Financial burden on families normally diminishes when children enter school age,

allowing parents to return to the workforce and bring more income for the family (Traub, Hiltonsmith, & Draut, 2016). However, in the present investigation, parents reported the highest level of material hardship at age 9 years, reflecting the notable impact of the recession on children's development. Here, our findings demonstrate that faster increases in material hardship with age exhibit greater amygdala–sgACC white matter connectivity and reduced amygdala–OFC white matter. If replicated, this pattern of amygdala–pFC white matter connectivity that arises from worsening economic conditions may reflect neural adaptation that could confer costs and advantages.

The present findings also provide further specificity on how associations between adverse experiences and adolescent white matter may differ across types of adversity. Our previous investigation found that childhood exposure to the combination of violence exposure and social deprivation at ages 3, 5, and 9 years related solely to decreased amygdala–OFC (BA 47) white matter connectivity (Goetschius et al., 2020). In contrast, here, material hardship related to amygdala–pFC white matter tracts in different regions (dmPFC BA 10, OFC BA 11, and sgACC BA 25), and the results remained when adjusting for childhood exposure to violence exposure and social deprivation. Although threat and deprivation are potential mediators through which distal experiences such as material hardship may influence brain development, we controlled for violence exposure and social deprivation in our models to demonstrate hardship associations that are separate from violence and deprivation exposure. Additionally, these material hardship associations with white matter connectivity remained after adjusting for household income, further demonstrating the unique impact of material hardship from other measures of adversity. Collectively, these findings suggest that specific types of adverse experiences have distinct associations with adolescent white matter microstructures and may help prompt the search for additional mechanisms that may connect material hardship to white matter (e.g., lack of specific provisions contributing to parental stress; Hyde et al., 2020).

Although poverty has been extensively linked to greater risk for emotional and behavioral problems in children (Brooks-Gunn & Duncan, 1997), investigations focused specifically on material hardship and internalizing symptoms are relatively limited. Consistent with existing literature, our results found that increased experiences of material hardship across childhood and adolescence were associated with increased adolescent anxiety and depression. Moreover, decreased amygdala–OFC white matter connectivity was related to greater anxiety and depression, suggesting that white matter connectivity is implicated in the potential consequences of material hardship on youth affective processes. Interestingly, anxiety and depression symptoms only related to white matter connectivity where material hardship was linked to less connectivity. This suggests that, although material hardship may lead to both increases and decreases in adolescent white matter connectivity, internalizing problems may be selectively associated with decreases in connectivity. It is notable, however, that we found these associations using only parent-reported and not youth-reported measures of symptoms and that these associations were not corrected for multiple comparisons. Although evidence suggests that both parent and child provide distinct information on adolescent symptoms that are equally meaningful (Bowers et al., 2020), these findings should be interpreted with caution. Furthermore, there were no significant indirect effects between hardship and anxiety or depressive symptoms through

any white matter connectivity; thus, more research is needed to further explain these intricate associations between hardship, white matter structures, and internalizing symptoms in adolescence.

Contrary to our hypothesis, our present results found no associations between material hardship and hippocampus–pFC or hippocampus–amygdala white matter connectivity. Despite evidence for direct projections between the hippocampus and prefrontal regions (Barbas & Blatt, 1995), it is possible that communication between pFC and the hippocampus is fully mediated through other neighboring structures, such as the amygdala, and that the hippocampus and the amygdala are too structurally interrelated for their white matter connectivity to be differentially affected by the environment. Given their importance in stress regulation and affective functioning, future examinations on how these structures specifically differ would further clarify underlying processes of poverty effects on the brain.

There are several limitations to this present investigation. First, no data were collected from families between the ages of 10–14 years; thus, we were not able to document material hardship experienced during those transitional years and our measurement of material hardship during adolescence is limited to a single time point. Second, it is not possible to determine the direction of white matter connections between the seed and target regions using our current white matter tract mapping methodology. Although conceptually pFC is commonly believed to be regulating subcortical limbic function in a top–down capacity, communication between amygdala and pFC is bidirectional (Crone & Dahl, 2012). Third, material hardship findings that were specific to ages 9 and 15 years overlap with the dMRI acquisition at 15 years of age; thus, it may not be possible to disentangle the recency effect from a possible sensitive period in our findings.

Conclusion

This study examined longitudinal associations between material hardship and white matter connectivity in adolescents underrepresented in neuroscience research. Although poverty has previously been linked to increased risk for child emotional problems and their neurodevelopmental correlates, our findings extended research on neuroscience of poverty to include measures of lived experience of poverty across a wide span of development. The developmental specific findings indicate that material hardship has potentially distinct associations with brain development depending on age. Furthermore, the increased trajectory of material hardship across development related to increased amygdala–prefrontal white matter connectivity in one region and decreased connectivity in another suggest a nonuniformity in the influence of poverty across emotion processing regions. These findings provide support for heterogeneity in the associations between environment and brain development, which can be explained by both timing and structural regions.

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Data Availability Statement

Open Science Framework preregistration can be viewed at https://osf.io/c3tf8. Data will be openly available at https://nda.nih.gov/edit_collection.html?id=2106.

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. The authors of this article report its proportions of citations by gender category to be as follows: M/M = .375; W/M = .232; M/W = .161; W/W = .232.

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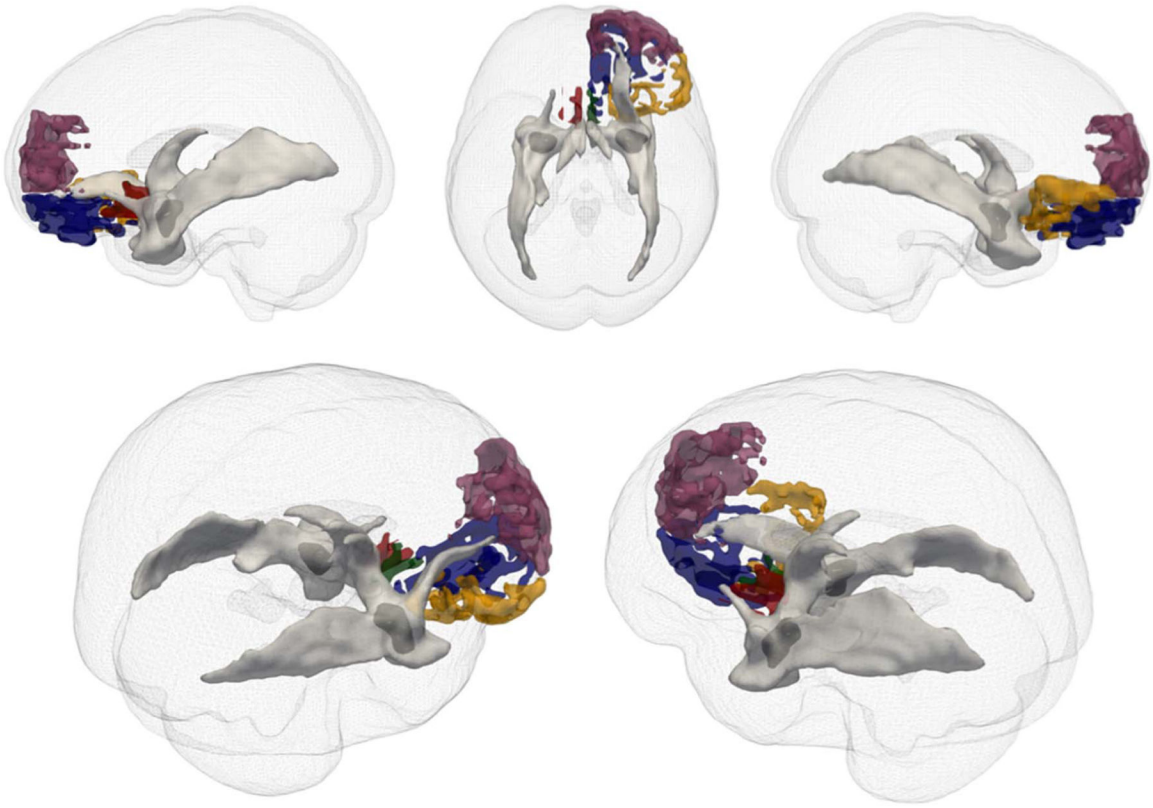

Figure 1.

3-D renderings of amygdala to specific pFC targets. Seed amygdala targets in dark gray. pFC targets: left sgACC BA 25 (red); right dmPFC BA 10 (plum); right OFC BA 11 (blue); right sgACC BA 25 (green); right OFC BA 47 (yellow). White matter is illustrated in light gray. White matter in figure depicts all tracts originating from seed amygdala region for illustration purposes.

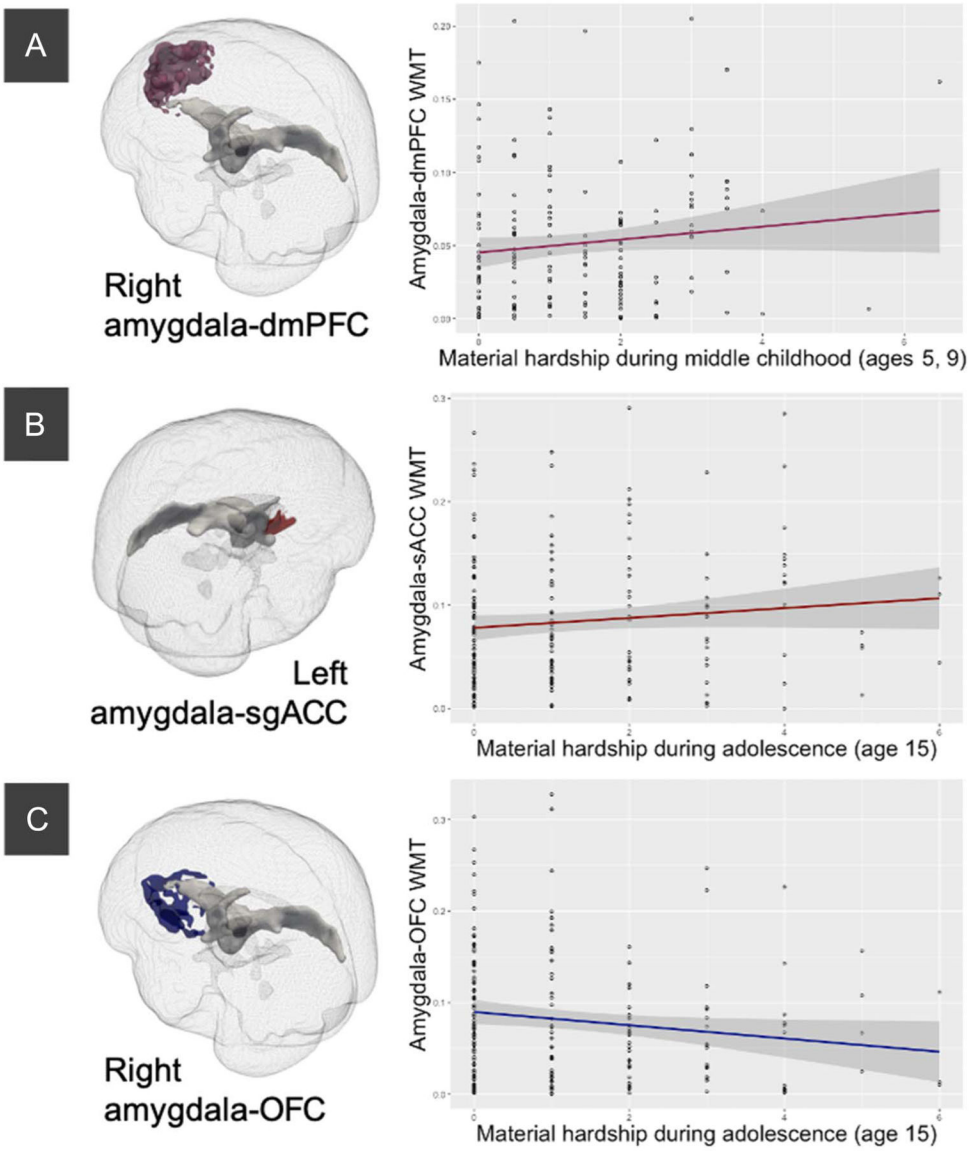

Figure 2.

Significant associations between material hardship at specific developmental stages and white matter connectivity after adjusting for other developmental stages. White matter connectivity and target on the left and zero-order plots on the right. (A) Increased material hardship at ages 5 and 9 years was related to increased right amygdala–dmPFC white matter connectivity, adjusting for other developmental stages. (B) Increased material hardship at age 15 years was related to increased left amygdala–sgACC white matter connectivity, adjusting for other developmental stages. (C) Increased material hardship at age 15 years was related to decreased right amygdala–OFC white matter connectivity, adjusting for other developmental stages.

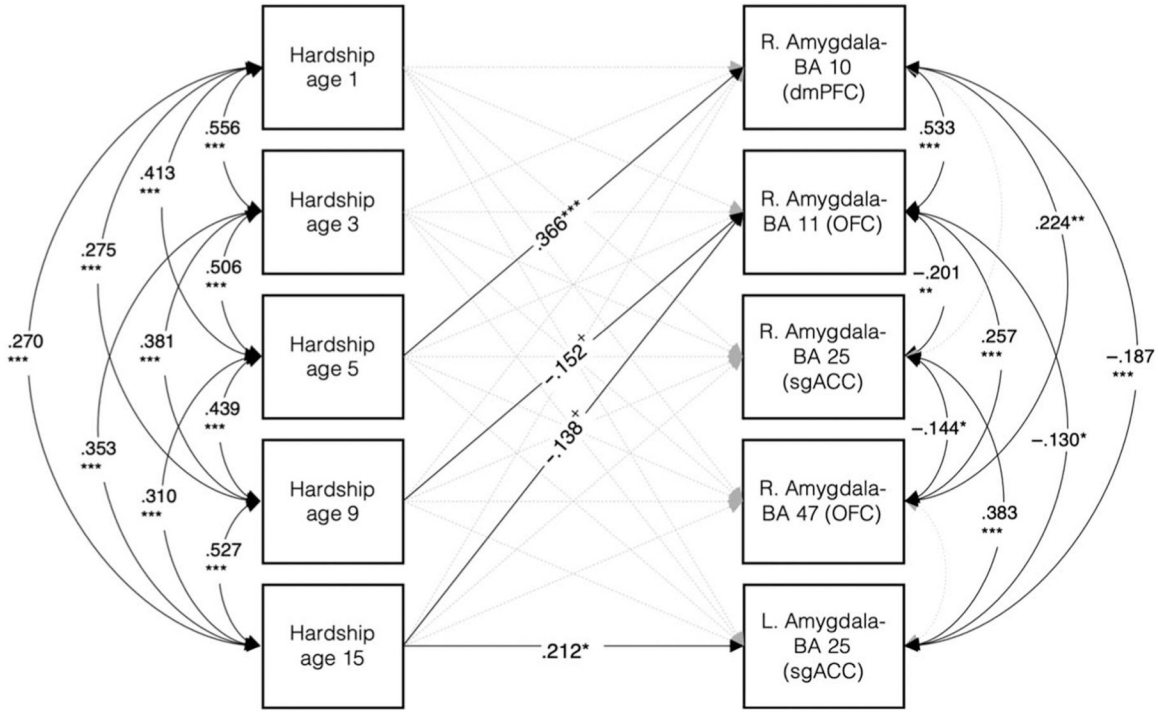

Figure 3.

Covariates-adjusted model using structural equation modeling including all material hardship predictors and white matter connectivity targets. Model fit statistics indicate excellent fit: $\chi^2[45] = 47.931$, $p = .355$; RMSEA = .019 [.000, .053], CFI = .980, TLI = .965, SRMR = .047. Consistent with regression results, there were positive associations between material hardship at age 5 years with right amygdala–BA 10 (dmPFC) and hardship at age 15 years with left amygdala–BA 25 (sgACC). Furthermore, there were trending negative associations between material hardship at ages 9 and 15 years with right amygdala–BA 11 (OFC) white matter connectivity. Covariates included in the model: ethnoracial identity, sex, pubertal age, birth city, maternal education, family structure, violence exposure, social deprivation, and annual household income at baseline. $^{+}\ p < .10$. * $p < .05$. ** $p < .01$. *** $p < .001$.

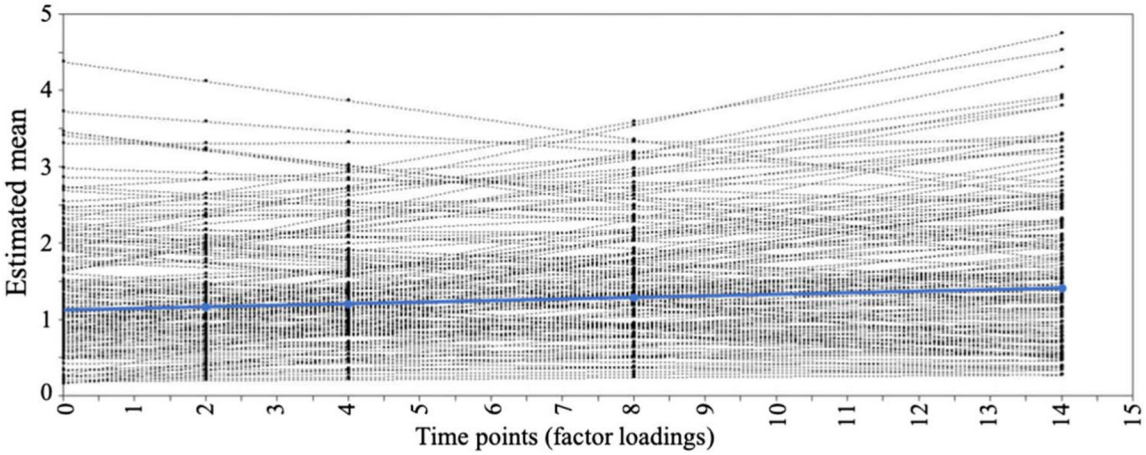

Figure 4.

Growth curve trajectory illustrating that material hardship increases with age. Figure shows linear estimated paths for each individual (black lines) as well as mean estimated group-level path trajectory (blue). Material hardship at age 1 year is set as point 0, where estimated mean [*SE*] for starting point (i.e., intercept) = 1.01 [.077], $p < .001$; variance [*SE*] = 0.91 [0.166], $p < .001$, and estimated mean [*SE*] change over time (slope) = 0.02 [0.008], $p = .009$; variance [*SE*] = 0.01 [0.002], $p = .004$.

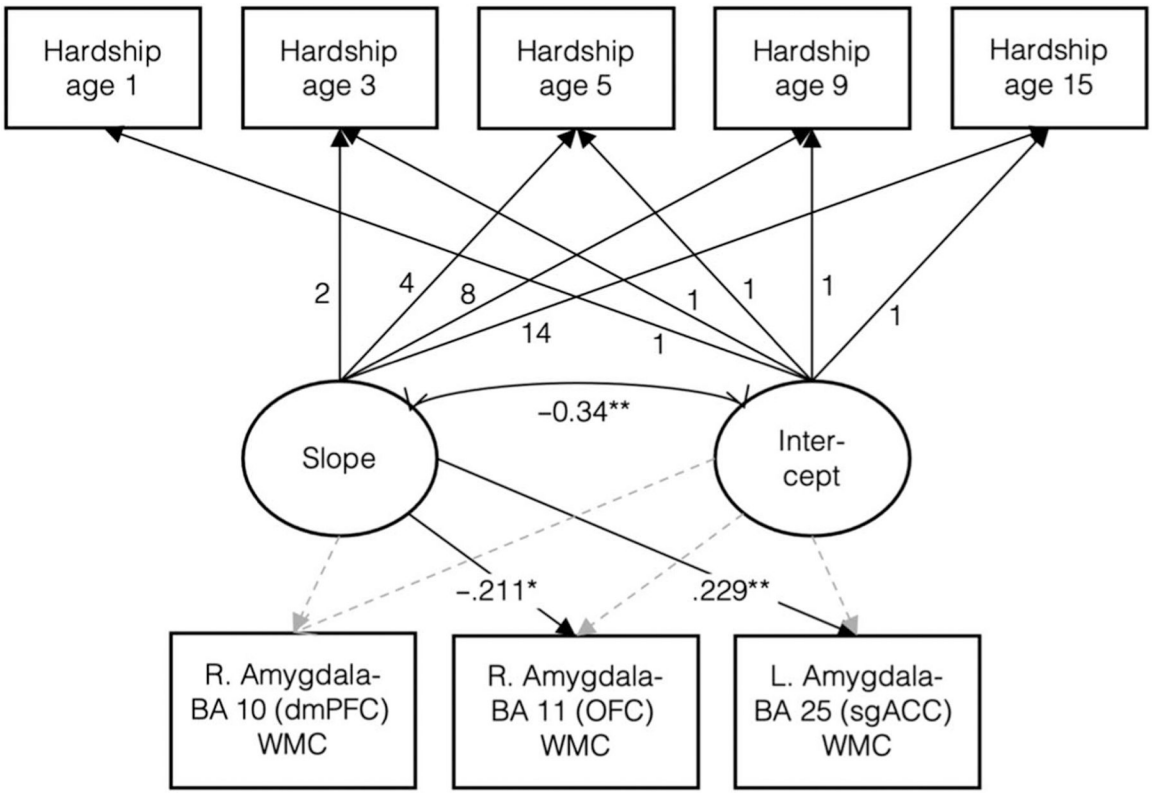

Figure 5.

Structural equation path model showing path estimates between material hardship predictors and right amygdala–BA 10, right amygdala–BA 11, and left amygdala–BA 25 white matter connectivity. Material hardship at 1, 3, 5, 9, and 15 loadings estimating latent slope were fixed at 0, 2, 4, 8, and 14, respectively. Covariates included in the model: ethnoracial identity, sex, pubertal age, birth city, maternal education, family structure, violence exposure, social deprivation, and annual household income at baseline. * $p < .05$. ** $p < .01$.

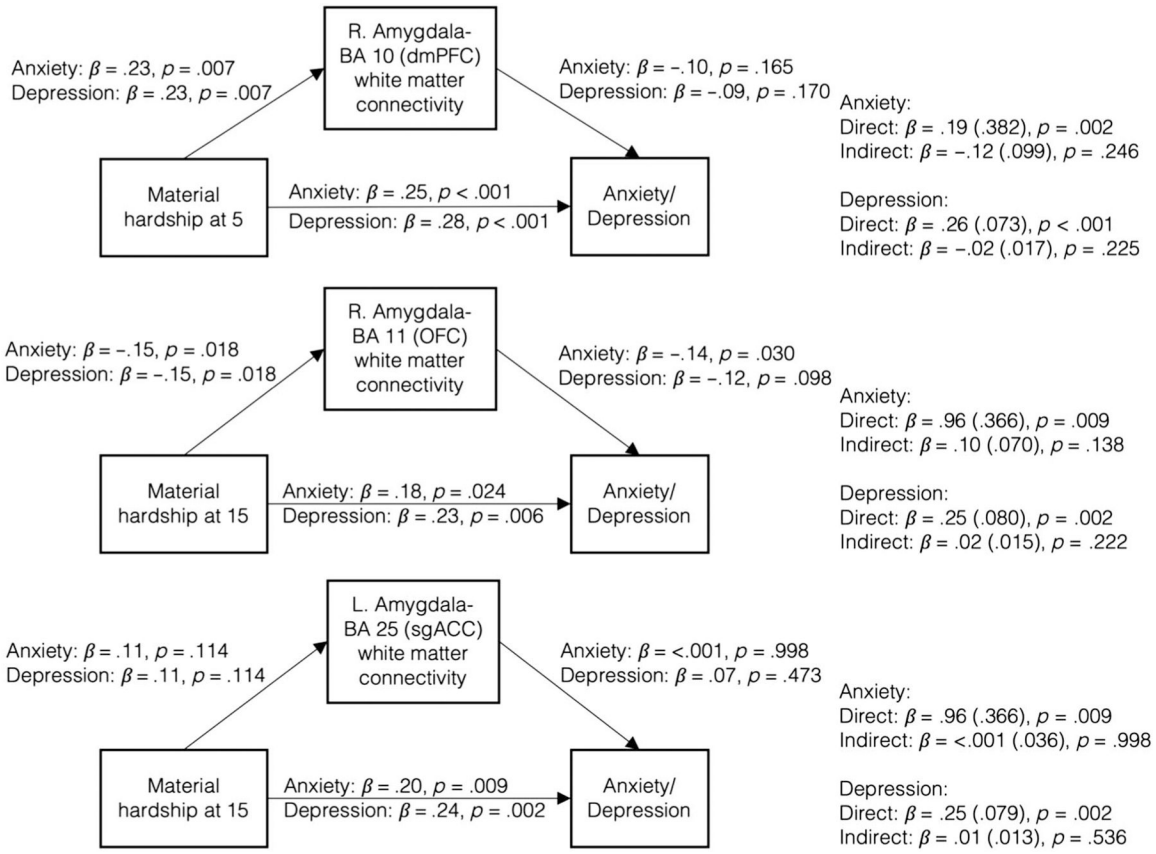

Figure 6.

Indirect effects of material hardship through white matter connectivity were nonsignificant for any regions with significant direct associations between material hardship and white matter connectivity.

Table 1.

Sample Exclusions and Comparisons

Reason for Exclusion	Excluded (Amygdala–Seed)	Excluded (Hippocampus–Seed)
Refused MRI	16	16
Exceeded MRI table weight limit	3	3
Medical restriction	1	1
Braces or other metal in body	7	7
Risk of pregnancy	1	1
Excluded for autism spectrum disorder diagnosis	2	2
Did not complete dMRI scan	11	11
Preprocessing outliers >5% in diffusion data[a]	1	3
No probabilistic tractography model convergence[b]	5	2
Less than 70% of voxels in pFC masks	1	0

Include vs. Full Sample Comparisons[c]

	Full Sample (n = 237)	Included Sample (Amygdala–Seed; n = 189)	Included Sample (Hippocampus–Seed; n = 191)
Age	*M* = 15.88 years	*M* = 15.84 years	*M* = 15.85 years
	SD = 0.54 years	*SD* = 0.52 years	*SD* = 0.53 years
Pubertal stage	*M* = 3.24	*M* = 3.26	*M* = 3.24
	SD = 0.59 years	*SD* = 0.58 years	*SD* = 0.59 years
Sex	F = 125 (52.7%)	F = 102 (54.0%)	F = 102 (53.4%)
	M = 112 (47.3%)	M = 87 (46.0%)	M = 89 (46.6%)
Race	Black = 181 (76.4%)	Black = 146 (77.2%)	Black = 148 (77.5%)
	White = 32 (13.5%)	White = 22 (11.6%)	White = 21 (11.0%)
	Hispanic/Latino = 13 (5.5%)	Hispanic/Latino = 12 (6.3%)	Hispanic/Latino = 12 (6.3%)
	Other/multiracial = 11 (4.6%)	Other/multiracial = 9 (4.8%)	Other/multiracial = 10 (5.2%)
Annual income	<$15,000 = 57 (24.1%)	<$15,000 = 48 (25.4%)	<$15,000 = 47 (24.6%)
	$15,000–39,999 = 70 (29.5%)	$15,000–39,999 = 57 (30.2%)	$15,000–39,999 = 58 (30.4%)
	$40,000–69,999 = 58 (24.5%)	$40,000–69,999 = 43 (22.8%)	$40,000–69,999 = 44 (23.0%)
	>$70,000 = 51 (21.5%)	>$70,000 = 40 (21.2%)	>$70,000 = 41 (21.5%)
	Missing/not reported = 1 (0.4%)	Missing/not reported = 1 (0.5%)	Missing/not reported = 1 (0.5%)

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[a] Outliers detected using dMRI method from Mrtrix (v.3.0.R3). Slices with average intensity 4 *SD*s or more lower than predicted by *eddy's* Gaussian process model were marked as outliers and replaced with model predictions. One participant was excluded from sample due to having more than 5% of total slices replaced.

[b] Zero value connectivity coefficient for at least one seed–target pair.

[c] Demographic characteristics did not significantly differ across samples: age, $F(2, 614) = 0.13$, $p = .878$; pubertal stage, $F(2, 614) = 0.08$, $p = .923$; sex, $F(2, 614) = 0.06$, $p = .941$; race, $F(2, 614) = 0.277$, $p = .758$; annual income, $F(2, 611) = 0.064$, $p = .938$.

Table 2.

Zero-Order Correlations of Material Hardship Predictors

Variable	M	SD	1	2	3	4
1. Hardship at age 1	1.02	1.30				
2. Hardship at age 3	1.06	1.33	.56**			
3. Hardship at age 5	1.27	1.40	.43**	.51**		
4. Hardship at age 9	1.55	1.46	.31**	.40**	.44**	
5. Hardship at age 15	1.30	1.50	.27**	.36**	.30**	.53**

M and *SD* are used to represent mean and standard deviation, respectively.

** $p < .01$.

Table 3.

Zero-Order Correlations of White Matter Connectivity (Amygdala–Seed)

Variable	M	SD	1	2	3	4
1. Right amygdala–BA 10	0.05	0.05				
2. Right amygdala–BA 11	0.08	0.07	.51**			
3. Right amygdala–BA 25	0.09	0.07	−.11	−.24**		
4. Right amygdala–BA 47	0.10	0.08	.23**	.27**	.13	
5. Left amygdala–BA 25	0.08	0.06	−.18*	−.17*	.42**	.04

M and *SD* are used to represent mean and standard deviation, respectively.

* $p < .05$.

** $p < .01$.

Table 4.

Zero-Order Correlations between Cumulative Material Hardship with Seed–Target (Amygdala–pFC, Hippocampus–pFC, Amygdala–Hippocampus) White Matter Connectivity

Model: Seed–Target White Matter Connectivity ~ Cumulative Material Hardship			
Seed–Target White Matter	**r**	**p**	**p_{adjust}**
Right amygdala–BA 10 (dmPFC)	.073	.317	.396
Right amygdala–BA 11 (OFC)	−.114	.116	.396
Right amygdala–BA 25 (sgACC)	−.081	.270	.396
Right amygdala–BA 47 (OFC)	−.077	.291	.396
Left amygdala–BA 25 (sgACC)	.011	.881	.881
Right hippocampus–BA 10 (dmPFC)	.002	.979	.979
Right hippocampus–BA 11 (OFC)	−.069	.346	.741
Right hippocampus–BA 25 (sgACC)	.039	.593	.741
Right hippocampus–BA 47 (OFC)	−.114	.116	.580
Left hippocampus–BA 10 (dmPFC)	.054	.456	.741
Right hippocampus–amygdala	−.042	.564	.564
Left hippocampus–amygdala	.144	.047	.094

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Table 5.

Multiple Regression Results Examining Material Hardship Effect at Specific Developmental Stages

Developmental Stage Specific Material Hardship and Amygdala–Seed White Matter Connectivity						
	B	**β**	**SEB**	**t**	**p**	**p_{adjust}**
Model: Right amygdala–BA 10 (dmPFC) ~ developmental stage specific hardship predictors						
Hardship at ages 1, 3	−0.005	−.138	0.004	−1.428	.155	.258
Hardship at ages 5, 9	0.010	.267	0.004	2.637	.009	.045
Hardship at age 15	−0.005	−.149	0.003	−1.647	.102	.170
Model: Right amygdala–BA 11 (OFC) ~ developmental stage specific hardship predictors						
Hardship at ages 1, 3	0.006	.090	0.006	0.934	.352	.440
Hardship at ages 5, 9	−0.005	−.088	0.006	−0.876	.382	.856
Hardship at age 15	−0.011	−.220	0.005	−2.441	.016	.040
Model: Right amygdala–BA 25 (sgACC) ~ developmental stage specific hardship predictors						
Hardship at ages 1, 3	−0.013	−.207	0.006	−2.133	.035	.175
Hardship at ages 5, 9	0.000	.002	0.006	0.017	.987	.987
Hardship at age 15	0.006	.128	0.005	1.410	.161	.201
Model: Right amygdala–BA 47 (OFC) ~ developmental stage specific hardship predictors						
Hardship at ages 1, 3	0.001	.010	0.007	0.097	.923	.923
Hardship at ages 5, 9	−0.004	−.056	0.007	−0.541	.589	.856
Hardship at age 15	−0.005	−.093	0.005	−1.009	.314	.314
Model: Left amygdala–BA 25 (sgACC) ~ developmental stage specific hardship predictors						
Hardship at ages 1, 3	−0.009	−.154	0.005	−1.599	.112	.258
Hardship at ages 5, 9	−0.002	−.041	0.005	−0.407	.685	.856
Hardship at age 15	0.011	.239	0.004	2.641	.009	.040
Model: Right hippocampus–BA 10 (dmPFC) ~ developmental stage specific hardship predictors						
Hardship at ages 1, 3	−0.005	−.114	0.004	−1.155	.250	.556
Hardship at ages 5, 9	0.004	.094	0.004	0.922	.358	.569
Hardship at age 15	−0.001	−.039	0.003	−0.427	.670	.670
Model: Right hippocampus–BA 11 (OFC) ~ developmental stage specific hardship predictors						
Hardship at ages 1, 3	0.004	.126	0.003	1.288	.200	.556
Hardship at ages 5, 9	−0.004	−.123	0.003	−1.221	.224	.569

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Developmental Stage Specific Material Hardship and Amygdala–Seed White Matter Connectivity

	B	β	SEB	t	p	p_{adjust}
Hardship at age 15	−0.003	−.137	0.002	−1.505	.134	.223
Model: Right hippocampus–BA 25 (sgACC) ~ developmental stage specific hardship predictors						
Hardship at ages 1, 3	0.002	.058	0.004	0.590	.556	.556
Hardship at ages 5, 9	−0.004	−.111	0.004	−1.091	.277	.569
Hardship at age 15	0.003	.105	0.003	1.138	.257	.321
Model: Right hippocampus–BA 47 (OFC) ~ developmental stage specific hardship predictors						
Hardship at ages 1, 3	0.002	.064	0.003	0.656	.513	.556
Hardship at ages 5, 9	−0.002	−.073	0.003	−0.722	.471	.569
Hardship at age 15	−0.003	−.139	0.002	−1.517	.131	.223
Model: Left hippocampus–BA 10 (dmPFC) ~ developmental stage specific hardship predictors						
Hardship at ages 1, 3	−0.002	−.080	0.003	−0.817	.415	.556
Hardship at ages 5, 9	−0.002	−.058	0.003	−0.571	.569	.569
Hardship at age 15	0.004	.191	0.002	2.097	.038	.190
Model: Right hippocampus–amygdala ~ developmental stage specific hardship predictors						
Hardship at ages 1, 3	0.004	.097	0.004	0.987	.325	.325
Hardship at ages 5, 9	−0.006	−.155	0.004	−1.521	.130	.260
Hardship at age 15	−0.001	−.018	0.003	−0.193	.847	.847
Model: Left hippocampus–amygdala ~ developmental stage specific hardship predictors						
Hardship at ages 1, 3	0.006	.111	0.006	1.127	.262	.325
Hardship at ages 5, 9	0.001	.019	0.005	0.190	.850	.850
Hardship at age 15	0.001	.024	0.004	0.259	.796	.847

Table 6.

Multiple Regression Results Examining Material Hardship Effect at Individual Time Points

Age-Specific Material Hardship with Amygdala-Seed White Matter Connectivity						
	B	**β**	**SEB**	**t**	**p**	**p_{adjust}**
Model: Right amygdala–BA 10 (dmPFC) ~ age-specific material hardship predictors						
Hardship at age 1	−0.003	−.097	0.003	−1.019	.310	.673
Hardship at age 3	−0.003	−.101	0.004	−0.961	.338	.504
Hardship at age 5	0.010	.330	0.003	3.325	.001	.005
Hardship at age 9	<−0.001	−.016	0.003	−0.169	.866	.866
Hardship at age 15	−0.003	−.113	0.003	−1.233	.219	.334
Model: Right amygdala–BA 11 (OFC) ~ age-specific material hardship predictors						
Hardship at age 1	−0.002	−.032	0.005	−0.333	.740	.740
Hardship at age 3	0.006	.116	0.006	1.103	.272	.504
Hardship at age 5	0.003	.053	0.005	0.535	.594	.944
Hardship at age 9	−0.009	−.170	0.005	−1.739	.084	.420
Hardship at age 15	−0.010	−.200	0.005	−2.174	.031	.078
Model: Right amygdala–BA 25 (sgACC) ~ age-specific material hardship predictors						
Hardship at age 1	−0.003	−.060	0.005	−0.625	.533	.673
Hardship at age 3	−0.008	−.150	0.006	−1.403	.162	.504
Hardship at age 5	−0.006	−.123	0.005	−1.223	.223	.558
Hardship at age 9	0.007	.132	0.005	1.338	.183	.458
Hardship at age 15	0.005	.103	0.005	1.114	.267	.334
Model: Right amygdala–BA 47 (OFC) ~ age-specific material hardship predictors						
Hardship at age 1	0.004	.061	0.006	0.617	.538	.673
Hardship at age 3	−0.004	−.059	0.007	−0.542	.589	.589
Hardship at age 5	−0.001	−.022	0.006	−0.212	.832	.944
Hardship at age 9	−0.002	−.029	0.006	−0.293	.770	.866
Hardship at age 15	−0.005	−.090	0.005	−0.957	.340	.340
Model: Left amygdala–BA 25 (sgACC) ~ age-specific material hardship predictors						
Hardship at age 1	−0.004	−.090	0.005	−0.924	.357	.673
Hardship at age 3	−0.004	−.090	0.005	−0.838	.403	.504

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Hardship at age 5	<−0.001	−.010	0.004	−0.070	.944	.944
Hardship at age 9	−0.002	−.040	0.004	−0.410	.683	.866
Hardship at age 15	0.011	.243	0.004	2.618	.010	.049

Age-Specific Material Hardship and Hippocampus–Seed White Matter Connectivity

	B	β	SEB	t	p	p_{adjust}
Model: Right hippocampus–BA 10 (dmPFC) ~ age-specific material hardship predictors						
Hardship at age 1	−0.004	−.115	0.004	−1.162	.247	.612
Hardship at age 3	0.000	−.012	0.004	−0.106	.916	.916
Hardship at age 5	0.002	.066	0.003	0.639	.523	.813
Hardship at age 9	0.001	.030	0.003	0.300	.765	.765
Hardship at age 15	−0.001	−.037	0.003	−0.400	.689	.689
Model: Right hippocampus–BA 11 (OFC) ~ age-specific material hardship predictors						
Hardship at age 1	−0.001	−.020	0.003	−0.210	.834	.834
Hardship at age 3	0.005	.166	0.003	1.529	.128	.448
Hardship at age 5	−0.001	−.047	0.003	−0.454	.650	.813
Hardship at age 9	−0.003	−.117	0.003	−1.193	.235	.765
Hardship at age 15	−0.003	−.133	0.002	−1.443	.151	.252
Model: Right hippocampus–BA 25 (sgACC) ~ age-specific material hardship predictors						
Hardship at age 1	0.004	.122	0.004	1.236	.218	.618
Hardship at age 3	−0.002	−.066	0.004	−0.604	.546	.863
Hardship at age 5	−0.002	−.064	0.003	−0.622	.535	.813
Hardship at age 9	−0.001	−.040	0.003	−0.408	.684	.765
Hardship at age 15	0.003	.105	0.003	1.124	.263	.329
Model: Right hippocampus–BA 47 (OFC) ~ age-specific material hardship predictors						
Hardship at age 1	−0.001	−.053	0.003	−0.546	.586	.733
Hardship at age 3	0.004	.147	0.003	1.351	.179	.448
Hardship at age 5	−0.002	−.080	0.002	−0.780	.436	.813
Hardship at age 9	−0.001	−.031	0.002	−0.319	.751	.765
Hardship at age 15	−0.003	−.145	0.002	−1.565	.120	.252
Model: Left hippocampus–BA 10 (dmPFC) ~ age-specific material hardship predictors						
Hardship at age 1	−0.002	−.058	0.003	−0.588	.558	.733
Hardship at age 3	−0.001	−.044	0.003	−0.399	.690	.863

Hardship at age 5	0.000	.010	0.002	0.094	.926	.926	
Hardship at age 9	−0.002	−.075	0.002	−0.765	.445	.765	
Hardship at age 15	0.005	.198	0.002	2.138	.034	.170	
Model: Right hippocampus–amygdala ~ age-specific material hardship predictors							
Hardship at age 1	0.006	.176	0.004	1.806	.073	.073	
Hardship at age 3	−0.004	−.103	0.004	−0.951	.343	.343	
Hardship at age 5	0.000	−.010	0.003	−0.095	.924	.924	
Hardship at age 9	−0.004	−.129	0.003	−1.309	.192	.311	
Hardship at age 15	0.000	−.005	0.003	−0.050	.960	.960	
Model: Left hippocampus–amygdala ~ age-specific material hardship predictors							
Hardship at age 1	0.009	.178	0.005	1.842	.067	.073	
Hardship at age 3	−0.005	−.111	0.005	−1.031	.304	.343	
Hardship at age 5	0.008	.168	0.005	1.647	.102	.204	
Hardship at age 9	−0.005	−.099	0.004	−1.017	.311	.311	
Hardship at age 15	0.002	.049	0.004	0.536	.593	.960	

Table 7.

Sensitivity Analyses Results with Covariates

Sensitivity Analyses Results of White Matter Connectivity Regions with Significant Main Effects with Developmental Stages Specific Material Hardship					
	B	**β**	**SEB**	**t**	**p**
Model: Right amygdala–BA 10 (dmPFC) white matter ~ predictors + covariates					
Hardship at ages 1, 3	−0.006	−.145	0.004	−1.396	.165
Hardship at ages 5, 9	0.008	.229	0.004	2.102	.037
Hardship at age 15	−0.004	−.136	0.003	−1.387	.168
Race/ethnicity (White)	−0.024	−.172	0.013	−1.858	.065
Race/ethnicity (Hispanic)	0.001	.003	0.015	0.035	.972
Race/ethnicity (other/multiracial)	−0.021	−.102	0.018	−1.152	.251
Sex	0.004	.043	0.009	0.422	.674
Pubertal age	0.007	.093	0.008	0.924	.357
Birth city (Chicago)	−0.001	−.009	0.011	−0.110	.913
Birth city (Toledo)	−0.003	−.024	0.011	−0.256	.799
Birth city (other)	−0.038	−.094	0.036	−1.054	.294
Maternal education	−0.004	−.105	0.004	−1.104	.272
Family structure	−0.001	−.007	0.008	−0.086	.931
Violence exposure	−0.001	−.075	0.001	−0.716	.475
Social deprivation	0.000	.042	0.001	0.421	.674
Household income at ages 5, 9	0.000	−.009	0.000	−0.084	.933
Model: Right amygdala–BA 11 (OFC) white matter ~ predictors + covariates					
Hardship at ages 1, 3	0.005	.084	0.007	0.814	.417
Hardship at ages 5, 9	−0.006	−.101	0.006	−0.945	.346
Hardship at age 15	−0.010	−.200	0.005	−2.068	.040
Race/ethnicity (White)	−0.029	−.128	0.021	−1.390	.167
Race/ethnicity (Hispanic)	−0.011	−.036	0.025	−0.437	.663
Race/ethnicity (other/multiracial)	0.005	.016	0.029	0.176	.860
Sex	0.001	.008	0.015	0.076	.939
Pubertal age	−0.010	−.082	0.012	−0.815	.416
Birth city (Chicago)	−0.015	−.069	0.018	−0.799	.425

Birth city (Toledo)	−0.021	−.112	0.018	−1.205	.230
Birth city (other)	−0.084	−.129	0.058	−1.460	.146
Maternal education	−0.006	−.091	0.006	−0.998	.320
Family structure	0.024	.160	0.013	1.856	.066
Violence exposure	−0.001	−.081	0.002	−0.785	.434
Social deprivation	0.001	.059	0.002	0.590	.556
Household income at age 15	0.000	.171	0.000	1.761	.080
Model: Left amygdala–BA 25 (sgACC) white matter ~ predictors + covariates					
Hardship at ages 1, 3	−0.007	−.129	0.006	−1.273	.205
Hardship at ages 5, 9	−0.002	−.038	0.005	−0.357	.722
Hardship at age 15	0.009	.209	0.004	2.199	.030
Race/ethnicity (White)	0.008	.038	0.018	0.420	.675
Race/ethnicity (Hispanic)	−0.017	−.064	0.021	−0.792	.430
Race/ethnicity (other/multiracial)	0.002	.008	0.025	0.090	.929
Sex	−0.017	−.133	0.013	−1.331	.185
Pubertal age	0.001	.013	0.011	0.136	.892
Birth city (Chicago)	−0.006	−.033	0.016	−0.393	.695
Birth city (Toledo)	0.018	.104	0.015	1.142	.255
Birth city (other)	0.098	.169	0.050	1.944	.054
Maternal education	0.009	.156	0.005	1.751	.082
Family structure	−0.024	−.179	0.011	−2.116	.036
Violence exposure	0.001	.043	0.002	0.421	.675
Social deprivation	−0.002	−.104	0.002	−1.062	.290
Household income at age 15	0.000	−.156	0.000	−1.640	.103

Sensitivity Analyses Results of White Matter Connectivity Regions with Significant Main Effects with Age-Specific Material Hardship

	B	β	SEB	t	p
Model: Right amygdala–BA 10 (dmPFC) white matter ~ predictors + covariates					
Hardship at age 1	−0.003	−.077	0.004	−0.758	.450
Hardship at age 3	−0.005	−.142	0.004	−1.267	.207
Hardship at age 5	0.010	.331	0.003	3.138	.002
Hardship at age 9	−0.001	−.044	0.003	−0.435	.664
Hardship at age 15	−0.003	−.090	0.003	−0.918	.360

Race/ethnicity (White)	−0.025	−.180	0.012	−2.003	.047
Race/ethnicity (Hispanic)	0.000	.001	0.015	0.017	.986
Race/ethnicity (other/multiracial)	−0.018	−.086	0.018	−0.981	.328
Sex	0.005	.057	0.009	0.563	.574
Pubertal age	0.007	.097	0.008	0.968	.335
Birth city (Chicago)	−0.001	−.008	0.011	−0.099	.921
Birth city (Toledo)	0.000	−.001	0.011	−0.015	.988
Birth city (other)	−0.049	−.121	0.036	−1.357	.177
Maternal education	−0.005	−.128	0.004	−1.391	.166
Family structure	0.000	.003	0.008	0.030	.976
Violence exposure	−0.001	−.049	0.001	−0.470	.639
Social deprivation	0.000	.011	0.001	0.110	.913
Household income at age 5	0.000	.016	0.000	0.161	.872
Model: Left amygdala–BA 25 (sgACC) white matter ~ predictors + covariates					
Hardship at age 1	−0.006	−.118	0.005	−1.174	.242
Hardship at age 3	−0.001	−.020	0.005	−0.178	.859
Hardship at age 5	−0.002	−.037	0.005	−0.353	.725
Hardship at age 9	−0.001	−.020	0.005	−0.202	.840
Hardship at age 15	0.009	.205	0.004	2.107	.037
Race/ethnicity (White)	0.008	.041	0.018	0.453	.651
Race/ethnicity (Hispanic)	−0.019	−.071	0.022	−0.864	.389
Race/ethnicity (other/multiracial)	0.001	.003	0.026	0.037	.971
Sex	−0.017	−.134	0.013	−1.333	.185
Pubertal age	0.001	.011	0.011	0.114	.910
Birth city (Toledo)	−0.006	−.033	0.016	−0.385	.701
Birth city (Chicago)	0.017	.100	0.016	1.087	.279
Birth city (other)	0.102	.176	0.051	1.983	.049
Maternal education	0.009	.157	0.005	1.745	.083
Family structure	−0.024	−.184	0.011	−2.151	.033
Violence exposure	0.001	.037	0.002	0.359	.720
Social deprivation	−0.001	−.095	0.002	−0.948	.345
Household income at age 15	0.000	−.159	0.000	−1.657	.100

Table 8.

Associations between Material Hardship and White Matter Connectivity with Child Anxiety and Depressive Symptoms Using Parent-Reported Measures

Zero-Order Correlations between Material Hardship and White Matter Connectivity with Parent-Reported Child Anxiety Symptoms at Age 15		
Predictors	**r**	**p**
Cumulative hardship	.218	.003
Hardship at age 1	.030	.698
Hardship at age 3	.196	.010
Hardship at age 5	.232	.002
Hardship at age 9	.170	.028
Hardship at age 15	.198	.008
Right amygdala–BA 10 (dmPFC)	−.042	.574
Right amygdala–BA 11 (OFC)	−.164	.027
Right amygdala–BA 25 (sgACC)	−.071	.339
Right amygdala–BA 47 (OFC)	−.108	.145
Left amygdala–BA 25 (sgACC)	.021	.774
Right hippocampus–BA 10 (dmPFC)	.005	.946
Right hippocampus–BA 11 (OFC)	−.017	.818
Right hippocampus–BA 25 (sgACC)	−.069	.342
Right hippocampus–BA 47 (OFC)	.118	.103
Left hippocampus–BA 10 (dmPFC)	.045	.538
Right hippocampus–amygdala	−.002	.982
Left hippocampus–amygdala	−.054	.457
Zero-Order Correlations between Material Hardship and White Matter Connectivity with Parent-Reported Child Depressive Symptoms at Age 15		
Predictors	**r**	**p**
Cumulative hardship	.255	<.001
Hardship at age 1	.103	.173
Hardship at age 3	.193	.010
Hardship at age 5	.259	<.001
Hardship at age 9	.226	.003
Hardship at age 15	.254	<.001
Right amygdala–BA 10 (dmPFC)	.006	.934

Right amygdala–BA 11 (OFC)	−.157	.032
Right amygdala–BA 25 (sgACC)	−.060	.418
Right amygdala–BA 47 (OFC)	−.102	.166
Left amygdala–BA 25 (sgACC)	.098	.185
Right hippocampus–BA 10 (dmPFC)	.107	.142
Right hippocampus–BA 11 (OFC)	.070	.333
Right hippocampus–BA 25 (sgACC)	−.119	.101
Right hippocampus–BA 47 (OFC)	.074	.310
Left hippocampus–BA 10 (dmPFC)	.068	.349
Right hippocampus–amygdala	.094	.194
Left hippocampus–amygdala	−.003	.964

Table 9.

Associations between Material Hardship and White Matter Connectivity with Child Anxiety and Depressive Symptoms using Child-Reported Measures

Zero-Order Correlations between Material Hardship and White Matter Connectivity with Child-Reported Child Anxiety Symptoms at Age 15		
Predictors	**r**	**p**
Cumulative hardship	.103	.170
Hardship at age 1	.035	.654
Hardship at age 3	.025	.743
Hardship at age 5	.081	.291
Hardship at age 9	.121	.123
Hardship at age 15	.060	.431
Right amygdala–BA 10	−.047	.529
Right amygdala–BA 11	−.023	.756
Right amygdala–BA 25	.095	.207
Right amygdala–BA 47	.044	.562
Left amygdala–BA 25	.058	.438
Right hippocampus–BA 10	−.053	.479
Right hippocampus–BA 11	−.005	.950
Right hippocampus–BA 25	−.051	.493
Right hippocampus–BA 47	−.046	.542
Left hippocampus–BA 10	−.058	.436
Right hippocampus–amygdala	−.072	.335
Left hippocampus–amygdala	−.024	.752
Zero-Order Correlations between Material Hardship and White Matter Connectivity with Child-Reported Child Depressive Symptoms at Age 15		
Predictors	**r**	**p**
Cumulative hardship	.101	.179
Hardship at age 1	.037	.630
Hardship at age 3	.021	.783
Hardship at age 5	.079	.300
Hardship at age 9	.122	.117
Hardship at age 15	.052	.493
Right amygdala–BA 10	−.047	.526
Right amygdala–BA 11	−.033	.660
Right amygdala–BA 25	.103	.168
Right amygdala–BA 47	.043	.569
Left amygdala–BA 25	.061	.418
Right hippocampus–BA 10	−.052	.488
Right hippocampus–BA 11	−.012	.873
Right hippocampus–BA 25	−.050	.502
Right hippocampus–BA 47	−.046	.541
Left hippocampus–BA 10	−.063	.400

Right hippocampus–amygdala	−.078	.297
Left hippocampus–amygdala	−.031	.681