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Family income buffers the relationship between childhood adverse experiences and putamen volume

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Abstract

Adverse experiences and family income in childhood have been associated with altered brain development. While there is a large body of research examining these associations, it has primarily used cross-sectional data sources and studied adverse experiences and family income in isolation. However, it is possible that low family income and adverse experiences represent dissociable and potentially interacting profiles of risk. To address this gap in the literature, we examined brain structure as a function of adverse experiences in childhood and family income in 158 youths with up to five waves of MRI data. Specifically, we assessed the interactive effect of these two risk factors on six regions of interest: hippocampus, putamen, amygdala, nucleus accumbens, caudate, and thalamus. Adverse experiences and family income interacted to predict putamen volume ($B = 0.086$, $p = 0.011$) but only in participants with family income one standard deviation below the mean (slope estimate $= -0.11$, $p = 0.03$). These results suggest that adverse experiences in childhood result in distinct patterns of brain development across the socioeconomic gradient. Given previous findings implicating the role of the putamen in psychopathology-related behaviors, these results emphasize the importance of considering life events and socioeconomic context when evaluating markers of risk. Future research should include interactive effects of environmental exposures and family income to better characterize risk for psychopathology in diverse samples.

KEYWORDS
adverse experiences, poverty, brain structure, psychopathology, early childhood

1 | INTRODUCTION

Adverse events in childhood are associated with alterations across a broad array of domains, including physical and behavioral health and brain development (Afifi et al., 2016; Lupien et al., 2009; Taillieu et al., 2016; Teicher et al., 2016). Unfortunately, experiencing adverse events, such as poverty, parental maladjustment, interpersonal loss, or maltreatment, is common in childhood. More than half of the children in the United States will experience an adverse event prior to adulthood and are more likely to go on to develop psychopathology as a result (J. G. Green et al., 2010; McLaughlin et al., 2012). In fact, it has been suggested that adverse experiences (AEs) in childhood may be responsible for nearly 30% of all psychopathology (Kessler et al., 2010). The mechanisms by which AEs in childhood lead to increased rates of

psychiatric illness have long been sought and a number of theories tested, including alterations to stress physiology, increased levels of inflammation, and modification of brain structure and function (Danese & McEwen, 2012). Of particular interest to this investigation, stressful experiences in early life have been consistently linked to altered brain structure years after the adverse event.

Research on the neurobiological effects of AEs has found global changes in brain development and more specific regional differences (Edmiston et al., 2011; Gold et al., 2016; Kelly et al., 2013; Luby et al., 2019; Teicher et al., 2016). Youth exposed to adversity have exhibited smaller whole brain volumes compared to control groups at 8–21 years of age, with gray matter variations reported in frontal, temporal, parietal, and occipital regions (De Brito et al., 2013; Gur et al., 2019; Kelly et al., 2015; Lim et al., 2014). These structural abnormalities extend to subcortical areas where AEs have been associated with morphological differences in limbic structures. Infants and children exposed to different forms of AEs (i.e., maltreatment, neglect, institutionalization) have exhibited smaller hippocampal volumes in late childhood, including specific reductions in stress sensitive CA1 and CA3 subfields, compared to children with no history of adversity (Dahmen et al., 2018; Hanson et al., 2015). Volumetric differences have also been reported in the amygdala following adversity, though the nature of these variations is inconsistent. For example, early childhood maltreatment and neglect has been linked to larger (Mehta et al., 2009; Tottenham et al., 2010), smaller (Edmiston et al., 2011), and equivalent amygdala volumes compared to youth without AEs (Carrion et al., 2001; McLaughlin et al., 2014; Sheridan et al., 2012). While existing studies have largely focused on the effects of adversity on the amygdala and hippocampus, recent reports have also associated AEs with smaller putamen, caudate, and thalamus volumes and stress hormones related to adverse experiences with smaller nucleus accumbens volumes, highlighting the need for further investigation into subcortical structures (Gehred et al., 2021; C. Green et al., 2021). Notably, early AEs have been linked to structural brain alterations in adulthood (Mackes et al., 2020; Tomalski & Johnson, 2010), suggesting that these early traumatic and stressful events have lasting effects on brain development.

Similar gray matter changes have been reported in children experiencing poverty, an environmental risk factor for adversity. Low family income-to-needs ratio (INR), or poverty, in childhood has been associated with reduced gray matter volume (Dufford et al., 2020). Consistent with AE findings, associations with poverty are often observed at the whole brain level, but it is also observed within subcortical regions, particularly the hippocampus (Brody et al., 2017; Hair et al., 2015; Jednoróg et al., 2012; McDermott et al., 2019; Noble et al., 2015; Staff et al., 2012). Recently, work from the same

sample used in the present study reported smaller cortical and subcortical gray matter volumes in children and youth with lower family INR (Barch et al., 2021). Among the subcortical regions implicated in this research, the hippocampus, caudate, and thalamus showed specific associations with family INR. Further, subcortical gray matter volume mediated the association between childhood INR and cognition and high-risk behaviors in adolescence, highlighting the importance of these alterations in brain development on adaptive outcome following AEs. The few other longitudinal studies that have been completed suggest that low INR affects brain volumes as early as the first year of life (Lawson et al., 2013), may contribute to slower gray matter growth (Hanson et al., 2013), and exerts effects from childhood into young adulthood (McDermott et al., 2019). Other work has begun to investigate potential mediators of the relationship between poverty and neural outcomes, including stress, caregiving, or environmental toxin exposure (Avants et al., 2015; Luby et al., 2013). Critically, however, there is still relatively little longitudinal work investigating how INR impacts the development of specific subcortical structures and how it interacts with other environmental factors, like AEs, to elucidate differences in brain development.

Whereas both childhood poverty and experiencing AEs have been consistently associated with alterations in brain structure, the relationship between these constructs, including whether they interact to predict unique associations with brain volume, is not yet known. Making the relationship between poverty and adversity more complicated, it is widely understood that there is an association between lower family income and experiencing more AEs in childhood. Data from a recent nationally representative sample indicate that children who live at or below the poverty line are three times more likely to have experienced 2+ adverse events relative to children living at or above 400% of the poverty line, and five times as likely to have experienced 4+ adverse events (Halfon et al., 2017). Nonetheless, AEs are not unique to children living below the poverty threshold but are distributed across income strata, though some types of AEs may be more common in lower-income contexts (e.g., exposure to neighborhood violence) while others are experienced more equally (e.g., death of a loved one). This distribution of AEs implies that higher income may not necessarily buffer against the deleterious health outcomes associated with experiencing adverse events in childhood (Halfon et al., 2017). Despite clear links between poverty and AEs, and the key role that both appear to play in brain development and health outcomes, research on these constructs is not well integrated (see Walsh et al., 2019 for a review). Further, findings that relationships between early AEs and negative health outcomes, for example, persist after adjusting for socioeconomic characteristics suggest that these constructs may capture dissociable aspects of risk (e.g., Kelly-Irving et al., 2013).

The possibility that low family income and AEs represent unique profiles of risk is especially important when identifying targets for intervention and prevention efforts related to the development of psychopathology. Investigations focused on associations between family income or AEs and total brain volume in isolation need more specificity to further inform clinical translation. Recent theoretical work in developmental psychopathology suggests that, just as pathways to depression and behavior problems are diverse, the interventions that will be most effective for individuals need to recognize diverse developmental pathways to risk (Doom & Cicchetti, 2020). Interventions specific enough to address heterogenous pathways to risk may have the added benefit of interrupting developmental cascades that can lead to amplified negative outcomes (Masten & Cicchetti, 2010). Approaches that focus on interactive effects are consistent with this idea, as they better characterize individual experiences and may provide multifaceted risk profiles better suited to effective intervention and prevention. Further, targeting more specific brain regions of interest impacted by family income and AEs may shed light on the kinds of behavioral outcomes associated with their combined impact. Consistent with this idea, meta-analytic work has suggested that the inclusion of neuroimaging data can improve accuracy in studies predicting clinical outcomes better than behavioral data alone (Jollans & Whelan, 2016).

In this study, we extended the prior literature by investigating family income-to-needs ratio as a moderator of the relationship between AEs in childhood and subcortical brain structures. Specifically, we assessed the interactive effect of AEs and family INR on six subcortical regions of interest: the hippocampus, amygdala, nucleus accumbens, caudate, putamen, and thalamus. These regions of interest were chosen due to prior research demonstrating their association with socioeconomic status and AEs. We completed these analyses using data from a large longitudinal study spanning nearly two decades, which included five waves of neuroimaging assessment spanning childhood through late adolescence. To leverage the longitudinal data available and test for developmental timing effects, separate models were run using AEs experienced in the preschool, school-age, and early adolescent periods. Given the paucity of research regarding the interaction of family INR and AEs in childhood, we made no specific predictions regarding the direction of effects prior to beginning the analyses.

2 | METHODS

2.1 | Participants

Participants were 158 youth that took part in the larger preschool depression study, a 17-year longitudinal study that began when participants were 3–5 years of age and includes five waves of brain imaging data. Youth and their primary caregivers were recruited from the St. Louis metropolitan area and oversampled for depression symptoms using the Preschool Feelings Checklist (Luby et al., 2004). When the original participants were 7–12 years of age, those who were either healthy or had any depression history were invited to participate in brain imaging. An additional 42 new healthy children were also recruited to participate in neuroimaging ($N = 210$ at scan wave 1). Children were excluded at study entry for head injury with loss of consciousness for 5+ minutes, diagnosis of an autism spectrum disorder, neurological illness, treatment for lead poisoning, or contraindications for MRI scanning (scan wave 1 specific). All participants included in this analysis provided complete data for baseline INR, usable imaging data from at least one scan wave, and AE data in each developmental period of interest. See Table 1 for demographic information. Caregivers and participants provided informed consent and assent, respectively. All methods were approved by the Washington University Institutional Review Board (IRB #201502094).

2.2 | Income-to-need ratio

Family INR was defined as total family income at baseline (T1) divided by the federal poverty level associated with the appropriate family size such that an INR of 1 is equal to the poverty line (McLoyd, 1998). For the additional 42 participants recruited at scan wave 1, INR was calculated using parent retrospective report of family income in 2003 (the first year of the study).

2.3 | Adverse experiences

AE scores were created using parent-reported variables from the life events section of the preschool age psychiatric assessment (PAPA, when participants were 3–7 years; Egger, 2009; Egger et al., 2006) or child and adolescent psychiatric assessment (CAPA, 8 years and older; Angold & Costello, 2000), and family interview for genetic studies (for parental psychopathology; Maxwell, 1992). The number of AEs endorsed on each assessment were summed to create a single score at each wave and standardized within that wave. AE scores were then created for each developmental period by averaging sum scores for each assessment that fell within the age range for each period: preschool age (3;0–5;11 years), school-age (6;0–9;11 years), early adolescence (10;0-14;11 years). See Supporting Information Appendix 1 for variables included in the AE scores and methods used to create these scores.

TABLE 1 Participant demographics

	Overall (N = 158)
Sex	
Male	83 (52.5%)
Female	75 (47.5%)
Race/Ethnicity	
White	87 (55.1%)
Black	54 (34.2%)
Other	17 (10.8%)
Baseline INR	
Mean (SD)	2.05 (1.16)
Median [Min, Max]	2.32 [0, 4.17]
Age at Scan 1	
Mean (SD)	10.3 (1.27)
Median [Min, Max]	10.4 [6.97, 12.8]
Missing	14 (8.9%)
Age at Scan 2	
Mean (SD)	11.9 (1.16)
Median [Min, Max]	11.9 [9.32, 14.9]
Missing	28 (17.7%)
Age at Scan 3	
Mean (SD)	13.1 (1.07)
Median [Min, Max]	13.1 [10.6, 15.7]
Missing	44 (27.8%)
Age at Scan 4	
Mean (SD)	16.5 (1.01)
Median [Min, Max]	16.5 [14.2, 19.4]
Missing	43 (27.2%)
Age at Scan 5	
Mean (SD)	18.7 (1.04)
Median [Min, Max]	18.8 [16.1, 21.0]
Missing	53 (33.5%)
Putamen Volume at Scan 1	
Mean (SD)	6.31 (0.597)
Median [Min, Max]	6.27 [4.38, 8.47]
Missing	14 (8.9%)
Putamen Volume at Scan 2	
Mean (SD)	6.30 (0.589)
Median [Min, Max]	6.26 [4.53, 8.47]
Missing	28 (17.7%)
Putamen Volume at Scan 3	
Mean (SD)	6.21 (0.596)
Median [Min, Max]	6.15 [4.52, 7.41]
Missing	44 (27.8%)
Putamen Volume at Scan 4	
Mean (SD)	6.71 (0.682)
Median [Min, Max]	6.70 [4.85, 8.64]

(Continues)

TABLE 1 (Continued)

	Overall (N = 158)
Missing	43 (27.2%)
Putamen Volume at Scan 5	
Mean (SD)	6.63 (0.676)
Median [Min, Max]	6.60 [4.64, 8.59]
Missing	53 (33.5%)
AE Sum Score During Preschool	
Mean (SD)	0.107 (0.975)
Median [Min, Max]	0.0117 [−1.42, 3.09]
AE Sum Score During School−Age	
Mean (SD)	0.0907 (0.954)
Median [Min, Max]	−0.0826 [−1.23, 3.20]
AE Sum Score During Early Adolescence	
Mean (SD)	0.119 (0.938)
Median [Min, Max]	0.0226 [−1.57, 3.34]

2.4 | Structural MRI acquisition and processing

Scan waves 1–3 were collected using a 3.0T Siemens Trio whole-body scanner with a 12-channel head coil. Scan waves 4–5 were collected using a 3.0 T Siemens Prisma whole-body scanner with a 32-channel head coil. The FreeSurfer Longitudinal processing stream was used to process the structural imaging data (v 5.3 http://surfer.nmr.mgh.harvard.edu; Reuter et al., 2012). Specific MRI acquisition parameters and data processing information can be found in the appendix. Scanning sessions also included task-based and resting-state scans that are not considered here.

2.5 | Statistical analysis

The volumes of the subcortical regions of interest, averaged across hemispheres at each timepoint, were investigated using multilevel modeling as implemented by the "lme4" package (Bates et al., 2015) in R (version 4.0.2; R Core Team, 2020). Volumes of the subcortical regions of interest in cubic centimeters were used as the dependent variable and the interaction of AEs and baseline INR was the predictor of interest. Covariates included sex at birth, age, the quadratic effect of age, the AE sum scores from the other developmental periods, an age by AE score interaction, and intracranial volume. Additional model information is provided in the Supporting Information Appendix. Separate models were run for each subcortical region using AE sum scores from each of three developmental periods: preschool age, school-age, and early adolescence. Missing brain volume data was handled via list wise deletion in each multi-level regression model.

TABLE 2 Means, standard deviations, and correlations with confidence intervals for the demographic and behavioral measures used in this study

Variable	M	SD	1	2	3	4
1. Sex	1.49	0.50				
2. Family INR	2.03	1.15	−.02			
			[−.17, .13]			
3. AEs During Preschool Period	0.11	0.96	.09	−.26**		
			[−.06, .24]	[−.40, −.11]		
4. AEs During School Age Period	0.12	0.99	.07	−.35**	.61**	
			[−.08, .22]	[−.47, −.21]	[.50, .70]	
5. AEs During Early Adolescence	0.13	0.96	.11	−.41**	.56**	.78**
			[−.04, .26]	[−.53, −.28]	[.45, .66]	[.71, .83]

Note. M and SD are used to represent mean and standard deviation, respectively. Values in square brackets indicate the 95% confidence interval for each correlation. The confidence interval is a plausible range of population correlations that could have caused the sample correlation (Cumming, 2014)., * indicates $p < .05$. ** indicates $p < .01$. INR = Income-to-Need Ratio.

A Bonferroni-adjusted alpha of $\alpha = 0.017$ was used to correct for multiple comparisons (three models for each dependent variable of interest, representing the three developmental periods investigated) in the primary analyses. Follow-up analyses, which investigated whether the effects of AEs were due to different types of exposures in high- versus low-income contexts used an exploratory, uncorrected alpha of $\alpha = 0.05$.

2.6 | Model interpretation

Only the interactive effects were interpreted as they were the predictors of interest. The main effects of baseline INR in this sample have been reported previously (Barch et al., 2021), and the main effects of baseline INR are concordant with previous research (e.g., Hanson et al., 2011, 2015; Jednoróg et al., 2012). For completeness, the main effects of the AE sum scores are reported in the Supporting Information Appendix (see Tables A1–A6).

3 | RESULTS

The means, standard deviations, and correlations among the main environmental and demographic variables are presented in Table 2. Additional tables reporting the correlation of the environmental and demographic variables with each brain region of interest can be found in the Supporting Information Appendix (Tables A7 – A12).

3.1 | Preschool AEs, INR, and subcortical volumes

The interaction of preschool AE sum scores with baseline INR was associated with putamen volume (see Figure 1
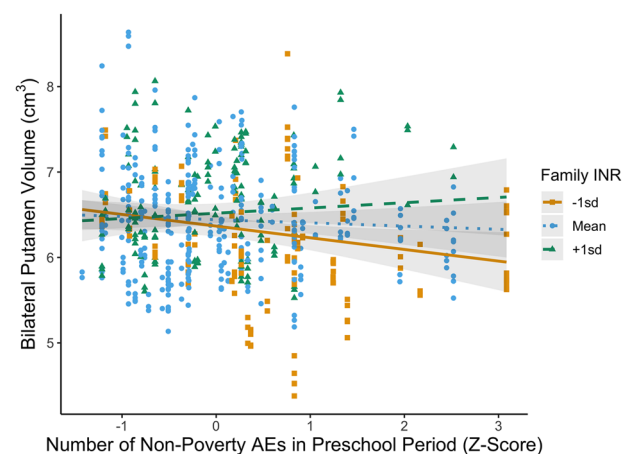
FIGURE 1 Model predicted bilateral putamen volume by the number of AEs during the preschool period at −1, 0, and +1 standard deviations from the INR mean. Error envelopes indicate 95% confidence interval for the predicted values. Raw data points are visualized in addition to the model predicted results

and Table 3). Simple slopes decomposition of the interaction effect indicated a negative relationship between AE sum scores and average putamen volume when INR was 1 standard deviation below the sample mean (slope estimate = −0.14, $p = 0.02$) but not at mean INR or 1 standard deviation above the sample INR mean (slope estimate = −0.04, $p = 0.48$ and slope estimate = 0.06, $p = 0.40$, respectively; see Figure A1). The growth curve of putamen volumes by family INR is available in the Supporting Information Appendix (Figure A2). To ensure this result was not driven by a single hemisphere, this model was also run with right and left putamen volumes separately. The hemisphere-specific results were nearly identical to those using bilateral putamen volume and can be found in the Supporting Information Appendix (Tables A13 – A15). No interaction effects were found in the

TABLE 3 Multi-level model predicting average putamen volume with AEs during the preschool period (N = 158)

DV: Bilateral Putamen Volume	Std. Beta	95% CI	t	df	p
Intercept	0.0284	[−0.0961, 0.1529]	6.7411	149.8234	<0.0001
Age	0.2460	[0.211, 0.281]	10.7506	209.1805	<0.0001
Age^2	0.0235	[−0.0058, 0.0528]	1.5715	437.3903	0.1168
Female Sex	−0.1177	[−0.2431, 0.0077]	−1.8396	146.4338	0.0679
INR	0.1323	[0.0041, 0.2605]	1.7390	145.9369	0.0842
Intracranial Volume (cm^3)	0.4426	[0.3136, 0.5715]	6.7257	150.1136	<0.0001
Preschool AE sum score	−0.0551	[−0.2086, 0.0983]	−2.7235	150.0861	0.0072
School-age AE sum score	0.0049	[−0.1832, 0.1931]	0.0512	147.4796	0.9592
Early Adolescent AE sum score	0.0685	[−0.1143, 0.2512]	0.7343	145.7700	0.4639
Age x Preschool AE sum score	−0.0010	[−0.034, 0.0321]	−0.0565	125.4392	0.9550
INR x Preschool AE sum score	0.1453	[0.0312, 0.2594]	2.4956	152.3784	0.0136

TABLE 4 Multi-level model predicting average putamen volume with AEs during the school-age period (N = 158)

DV: Bilateral Putamen Volume	Std. Beta	95% CI	t	df	p
Intercept	0.0313	[−0.0972, 0.1598]	6.6235	149.6362	<0.0001
Age	0.2454	[0.2106, 0.2803]	10.8147	208.9942	<0.0001
Age^2	0.0243	[−0.005, 0.0537]	1.6266	437.9153	0.1045
Female Sex	−0.0979	[−0.2242, 0.0284]	−1.5198	145.9461	0.1307
INR	0.1449	[0.0167, 0.2731]	2.0059	145.8159	0.0467
Intracranial Volume (cm^3)	0.4443	[0.3145, 0.5741]	6.7095	149.9753	<0.0001
School-age AE sum score	0.0304	[−0.1709, 0.2316]	−1.5585	150.7202	0.1212
Preschool AE sum score	−0.0955	[−0.2438, 0.0528]	−1.2626	148.3412	0.2087
Early Adolescent AE sum score	0.0831	[−0.0996, 0.2658]	0.8914	145.9206	0.3742
Age x School-age AE sum score	−0.0102	[−0.044, 0.0235]	−0.5948	124.5605	0.5530
INR x School-age AE sum score	0.1308	[0.0019, 0.2597]	1.9882	149.4136	0.0486

other subcortical regions of interest. Main effects of INR were found, however, for both hippocampal (Table A1) and caudate volumes (A5) with higher INR associated with larger volumes.

3.2 | School-age AEs, INR, and subcortical volumes

Conversely, the interaction of AE sum scores during the school-age period with baseline INR was not associated with putamen volume at the alpha level used for the ROI analysis (see Table 4). Simple slopes decomposition of the interaction effect likewise showed no relationship between AE sum scores and average putamen volume at 1 standard deviation below the sample INR mean (slope estimate = −0.07, $p <$ 0.32) nor at or above the INR mean (slope estimate = 0.02, $p =$ 0.77 and slope estimate = 0.11, $p = 0.25$, respectively). There were no interaction effects found in the other subcortical regions of interest.

3.3 | Early adolescent AEs, INR, and subcortical volumes

No interaction effects were found for AE sum scores during the early adolescent period in the putamen or the other regions of interest. As some of the MRI data was collected prior to the early adolescent period, the analysis was re-run with only the final two waves of MRI data. Once again, no interaction effects were found for AE sum scores during the adolescent period in any of the regions of interest.

3.4 | Differences in AEs exposure by income level

To evaluate whether the observed interaction between AE sum scores during the preschool period and baseline INR predicting putamen volume was attributable to differences in stress exposures, we compared the rates of each AE between higher and lower income groups, using a median split of INR in the sample (see Table A16). There were 10 AE items that were

TABLE 5 Multi-level model predicting average putamen volume with AEs that differ between INR groups during the preschool period (N = 156)

DV: Bilateral Putamen Volume	Std. Beta	95% CI	t	df	p
Intercept	0.0357	[−0.0952, 0.1667]	6.9353	148.1681	<0.0001
Age	0.2420	[0.2085, 0.2754]	10.9568	206.0925	<0.0001
Age^2	0.0251	[−0.0036, 0.0539]	1.7146	417.3482	0.0872
Female Sex	−0.1403	[−0.2679, −0.0127]	−2.1554	143.2283	0.0328
INR	0.1522	[0.0241, 0.2804]	2.2680	143.1018	0.0248
Intracranial Volume (cm^3)	0.4308	[0.3008, 0.5608]	6.4960	148.5122	<0.0001
Preschool different AE sum score	0.0458	[−0.1346, 0.2262]	−1.8140	148.8743	0.0717
School-age different AE sum score	0.0444	[−0.1118, 0.2007]	0.5573	145.9661	0.5782
Early adolescent different AE sum score	0.0306	[−0.0675, 0.1288]	0.6118	150.8857	0.5416
Age x Preschool different AE sum score	−0.0022	[−0.0354, 0.031]	−0.1308	146.8829	0.8961
INR x Preschool different AE sum score	0.1353	[0.0114, 0.2592]	2.1396	148.3669	0.0340

TABLE 6 Multi-level model predicting average putamen volume with AEs that are equivalent between INR groups during the preschool period (N = 156)

DV: Bilateral Putamen Volume	Std. Beta	95% CI	t	df	p
Intercept	−0.0169	[−0.1388, 0.1049]	6.8314	148.0165	<0.0001
Age	0.2411	[0.2076, 0.2747]	10.6768	212.1356	<0.0001
Age^2	0.0253	[−0.0037, 0.0542]	1.7123	421.8106	0.0876
Female Sex	−0.1346	[−0.2617, −0.0076]	−2.0775	143.9016	0.0395
INR	0.1475	[0.0263, 0.2686]	2.1242	145.4352	0.0353
Intracranial Volume (cm^3)	0.4343	[0.3034, 0.5653]	6.5012	148.2758	<0.0001
Preschool equivalent AE sum score	−0.0218	[−0.1456, 0.1021]	−2.2317	144.0640	0.0272
School-age equivalent AE sum score	−0.0538	[−0.1767, 0.0692]	−0.8569	145.2011	0.3929
Early adolescent equivalent AE sum score	0.0302	[−0.0653, 0.1258]	0.6201	151.2177	0.5361
Age x Preschool equivalent AE sum score	0.0106	[−0.0211, 0.0423]	0.6548	113.3990	0.5139
INR x Preschool equivalent AE sum score	0.1456	[0.0245, 0.2667]	2.3560	143.0709	0.0198

more common in the lower INR group than the higher INR group, particularly items related to intentional violence or harm in the child's environment. To address these differences in AE exposure, follow-up models assessing the interaction of AE sum scores during the preschool period with INR were run using only the AEs that were different across groups in one model (AE_INR_Diff) and equivalent across groups in a second model (AE_INR_SAME). The effect size of the interaction between AE sum scores that differ between income groups and INR was similar to those of the interaction that include only AE sum scores that are equivalent between the groups (see Tables 5–6). Both of the follow-up models were significant at the $p < 0.05$ level, see Figures A3–A5.

4 | DISCUSSION

This study extended previous research investigating the impact of AEs on brain structure by examining the moder-ating role of family income. Our results indicated that low family income (i.e., at or below the poverty line) interacted with greater AEs in the preschool period to predict smaller putamen volume. The experiences of children and youth with lower income were also qualitatively different than those with higher income (i.e., higher frequency of intentional, inter-personal harm). However, when investigating models that included only AEs that were either equivalent or different between income levels, the results were largely consistent with those found when all AEs were considered together. That suggests that the interaction between income and AEs was not solely attributable to differences in the experiences reported in lower- versus higher-income youth. No interactive rela-tionships were found when AEs in the school-age or early adolescent periods were examined, suggesting that the impact of these events is particularly important during the preschool period.

Early periods of development, including the preschool period, are characterized by high levels of plasticity and serve

an important role in tuning neural systems to the kinds of environments the individual is likely to experience later in life (Lupien et al., 2009; Tottenham, 2014). In our data, only AEs reported during the preschool period interacted with family income to predict putamen volume, consistent with the importance of early experiences in development. This specificity may be further explained by the putamen's role in learning predictable environmental contingencies and updating behavior to optimize outcomes (Birn et al., 2017; Vidal-Ribas et al., 2019). Given that low income environments can be characterized by low levels of predictability (Blair & Raver, 2016; Ellis et al., 2017), it is possible that the observed differences in putamen volume are related to the demands of unpredictable low income environments in combination with AEs in early childhood. This possibility may further explain why the putamen was the only subcortical structure to exhibit this relationship: it may be that single exposure types, like low income or AEs, are sufficient to alter the structure of the amygdala and hippocampus (as has been shown in this sample and others; Barch et al., 2021; McLaughlin et al., 2016; Merz et al., 2018) but that putamen volume is affected only in the context of combined exposures. Alternatively, it could also be that the putamen is more vulnerable to early experiences than the amygdala and hippocampus, as suggested by studies of normative brain development and the effects of socioeconomic status on amygdala structure (Merz et al., 2018; Vlasova et al., 2017; Wierenga et al., 2014). Further, while the structure of caudate, thalamus, and nucleus accumbens have been associated with the effects of environmental stressors, including poverty and traumatic events (Barch et al., 2021; Gehred et al., 2021; C. Green et al., 2021), they were not predicted by the interaction of family income and AEs in any developmental stage. For the caudate, this may be due to the effects of family INR on caudate and thalamus volume in our sample while the effects of family income may have more impact on the connectivity of the nucleus accumbens (Marshall et al., 2018). Although these interpretations are speculative given the associative nature of the study design, they have important implications for translation. The structure and function of the putamen have been associated with depression in both adolescents and adults (Auerbach et al., 2017; Fischer et al., 2019; Sacchet et al., 2017), suggesting that prevention and/or intervention efforts aimed at decreasing risk for depression might focus on putamen-related learning mechanisms in individuals with complex early environmental exposures.

Our results also emphasize the importance of including contextual factors in studies of the effects of AEs on brain development. The inclusion of moderating variables—family INR in this case—is an important part of research aiming to improve the specificity of prevention and intervention efforts (Kraemer et al., 2008). Critically, this approach allows for investigation not only of the simple associations between AEs, brain structure, and clinically relevant behavior, but also *for whom* the association is impactful. The results reported here provide an example of the contribution of moderation effects to the interpretability and translational potential of developmental neuroscience research. Our findings suggest that experiencing AEs in the preschool period is negatively associated with putamen volume, but only for individuals with family income at or below the poverty line. Convergent with this finding, previous research has documented effects of socioeconomic status and stressful experiences on putamen volume individually. INR has been negatively associated with putamen volume in female adolescents and morphology differences in both males and females (Jenkins et al., 2020). Similarly, stressful experiences, like peer victimization and childhood maltreatment, have also been shown to be related to reduced putamen volume (Edmiston et al., 2011; Quinlan et al., 2018). Thus, should future research aim to establish putamen volume as a potential biomarker of maladaptive outcomes following AEs, considering family income will be necessary for valid translation.

Importantly, previous research has reported differences in putamen activation and volume associated with behavioral output. The putamen has been consistently implicated in reward responses and reward learning (Pascucci et al., 2017), an effect that has been shown to be impacted by AEs. In one study, young adults with maltreatment histories exhibited marginally less activity in the left putamen in response to reward cues than controls in a monetary incentive delay task (Dillon et al., 2009). Similar results have been reported in two prospective studies linking AEs with putamen responses to reward. For example, stressful life events reported at age 7 years were associated with lower putamen responses to reward anticipation at age 10 years in a monetary delay task (Vidal-Ribas et al., 2019). The same pattern of effects has been reported in a sample of adults who completed a semistructured life events interview when they were 10 years old and completed the monetary incentive delay task approximately 10 years later, though the altered putamen activity was specific to loss anticipation (Birn et al., 2017). Further, putamen activation during loss anticipation mediated the link between childhood stress and poor decision making, though this result was only marginally significant. In addition to functional associations, smaller putamen volumes have been associated with increased anhedonia symptoms in 12–14-year-old females (Auerbach et al., 2017). These smaller putamen volumes moderated the association between blunted responses to peer acceptance and anhedonia symptoms such that smaller putamen volume amplified the link between response to peer acceptance and anhedonia symptoms.

While our study is characterized by several strengths, including the socioeconomic diversity of the sample and the prospective nature of the associations reported, there are some limitations. First, despite the prospective design of the study,

brain imaging data was not available until after the preschool period. As such, we were unable to investigate the concurrent relationship between AEs early in development and putamen volume. Second, family INR is a proxy for the environmental features associated with poverty, not a direct measure of these exposures. Future research is needed to determine whether specific aspects of poverty, such as material deprivation, heavy metal or other environmental toxin exposure, or limited access to nutritious food, play specific roles in linking family income to brain development. Third, the sample size of this study was moderate and, despite the ROI approach taken, remains an exploratory investigation of the interaction between family INR and AEs as predictors of subcortical brain structure. Replication is needed in larger study samples. Fourth, our analysis is dependent upon the reliability of our MRI segmentations, which, though generated using a carefully designed longitudinal processing procedure, may introduce a small amount of bias in the results. However, recent work has indicated high levels of test-retest reliability in subcortical regions and confirmed the validity of investigating structural brain volumes in pediatric samples aligned to a common template (Ghosh et al., 2010; Haddad et al., 2022; Madan & Kensinger, 2017). Finally, the study sample was enriched for depression risk at the time of recruitment, which may limit generalizability.

Despite these limitations, the current study suggests an important role for family income as a moderator of the effects of AEs on subcortical brain structure. Specifically, we found that adversity during early and middle childhood was related to smaller putamen volumes but only in children from families with relatively low income. Given previous research linking the structure and function of the putamen with difficulty processing rewarding stimuli, increased anhedonia, and depression diagnosis, the current findings emphasize the importance of considering both life events and the socioeconomic context when assessing risk for psychopathology. Future research should replicate and extend this work with a particular emphasis on the early childhood period and further investigate the role of family income as an important moderator of risk for psychopathology.

CONFLICT OF INTEREST
The authors declare no conflicts of interest.

DATA AVAILABILITY STATEMENT
Data supporting these findings are available from the corresponding author by request.

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REFERENCES
Afifi, T. O., MacMillan, H. L., Boyle, M., Cheung, K., Taillieu, T., Turner, S., & Sareen, J. (2016). Child abuse and physical health in adulthood. *Health Reports*, 27(3), 10–18.

Angold, A., & Costello, E. J. (2000). The child and adolescent psychiatric assessment (CAPA). *Journal of the American Academy of Child and Adolescent Psychiatry*, 39(1), 39–48.

Auerbach, R. P., Pisoni, A., Bondy, E., Kumar, P., Stewart, J. G., Yendiki, A., & Pizzagalli, D. A. (2017). Neuroanatomical prediction of anhedonia in adolescents. *Neuropsychopharmacology*, 42(10), 2087–2095.

Avants, B. B., Hackman, D. A., Betancourt, L. M., Lawson, G. M., Hurt, H., & Farah, M. J. (2015). Relation of childhood home environment to cortical thickness in late adolescence: Specificity of experience and timing. *PLoS ONE*, 10(10), 1–10.

Barch, D. M., Donohue, M. R., Elsayed, N. M., Gilbert, K., Harms, M. P., Hennefield, L., Herzberg, M., Kandala, S., Karcher, N. R., Jackson, J. J., Luking, K. R., Rappaport, B. I., Sanders, A., Taylor, R., Tillman, R., Vogel, A. C., Whalen, D., & Luby, J. L. (2021). Early childhood socioeconomic status and cognitive and adaptive outcomes at the transition to adulthood: The mediating role of gray matter development across 5 scan waves. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*, 7(1), 34–44.

Bates, D., Mächler, M., Bolker, B., & Walker, S. (2015). Fitting linear mixed-effects models using lme4. *Journal of Statistical Software*, 67(1), 1–48.

Birn, R. M., Roeber, B. J., & Pollak, S. D. (2017). Early childhood stress exposure, reward pathways, and adult decision making. *Proceedings of the National Academy of Sciences of the United States of America*, 114(51), 13549–13554.

Blair, C., & Raver, C. C. (2016). Poverty, stress, and brain development: New directions for prevention and intervention. *Academic Pediatrics*, 16(3), S30–S36.

Brody, G. H., Gray, J. C., Yu, T., Barton, A. W., Beach, S. R. H., Galvan, A., MacKillop, J., Windle, M., Chen, E., Miller, G. E., & Sweet, L. H. (2017). Protective prevention effects on the association of poverty with brain development. *JAMA Pediatrics*, 171(1), 46–52.

Carrion, V. G., Weems, C. F., Eliez, S., Patwardhan, A., Brown, W., Ray, R. D., & Reiss, A. L. (2001). Attenuation of frontal asymmetry in pediatric posttraumatic stress disorder. *Biological Psychiatry*, 50(12), 943–951.

Dahmen, B., Puetz, V. B., Scharke, W., Von Polier, G. G., Herpertz-Dahlmann, B., & Konrad, K. (2018). Effects of early-life adversity on hippocampal structures and associated HPA axis functions. *Developmental Neuroscience*, 40(1), 13–22.

Danese, A., & McEwen, B. S. (2012). Adverse childhood experiences, allostasis, allostatic load, and age-related disease. *Physiology and Behavior*, 106(1), 29–39.

De Brito, S. A., Viding, E., Sebastian, C. L., Kelly, P. A., Mechelli, A., Maris, H., & McCrory, E. J. (2013). Reduced orbitofrontal and temporal grey matter in a community sample of maltreated children. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, 54(1), 105–112.

Dillon, D. G., Holmes, A. J., Birk, J. L., Brooks, N., Lyons-Ruth, K., & Pizzagalli, D. A. (2009). Childhood adversity is associated with

left basal ganglia dysfunction during reward anticipation in adulthood. *Biological Psychiatry*, *66*(3), 206–213.

Doom, J. R., & Cicchetti, D. (2020). The developmental psychopathology of stress exposure in childhood. In K. L. Harkness & E. P. Hayden (Eds.), *The Oxford* handbook of stress and mental health (pp. 264–286). Oxford University Press.

Dufford, A. J., Kim, P., & Evans, G. W. (2020). The impact of childhood poverty on brain health: Emerging evidence from neuroimaging across the lifespan. *International Review of Neurobiology*, *150*, 77–105.

Edmiston, E. E., Wang, F., Mazure, C. M., Guiney, J., Sinha, R., Mayes, L. C., & Blumberg, H. P. (2011). Corticostriatal-limbic gray matter morphology in adolescents with self-reported exposure to childhood maltreatment. *Archives of Pediatrics and Adolescent Medicine*, *165*(12), 1069–1077.

Egger, H. L. (2009). Psychiatric assessment of young children. *Child and Adolescent Psychiatric Clinics of North America*, *18*(3), 559–580.

Egger, H. L., Erkanli, A., Keeler, G., Potts, E., Walter, B. K., & Angold, A. (2006). Test-retest reliability of the Preschool Age Psychiatric Assessment (PAPA). *Journal of the American Academy of Child and Adolescent Psychiatry*, *45*(5), 538–549.

Ellis, B. J., Bianchi, J. M., Griskevicius, V., Frankenhuis, W. E., Griskevicius, V., & Frankenhuis, W. E. (2017). Beyond risk and protective factors: An adaptation-based approach to resilience. *Perspectives on Psychological Science*, *12*(4), 561–587.

Fennema-Notestine, C., Stein, M. B., Kennedy, C. M., Archibald, S. L., & Jernigan, T. L. (2002). Brain morphometry in female victims of intimate partner violence with and without posttraumatic stress disorder. *Biological Psychiatry*, *52*(11), 1089–1101.

Fischer, A. S., Ellwood-Lowe, M. E., Colich, N. L., Cichocki, A., Ho, T. C., & Gotlib, I. H. (2019). Reward-circuit biomarkers of risk and resilience in adolescent depression. *Journal of Affective Disorders*, *246*, (August 2018), 902–909.

Gehred, M. Z., Knodt, A. R., Ambler, A., Bourassa, K. J., Danese, A., Elliott, M. L., Hogan, S., Ireland, D., Poulton, R., Ramrakha, S., Reuben, A., Sison, M. L., Moffitt, T. E., Hariri, A. R., & Caspi, A. (2021). Long-term neural embedding of childhood adversity in a population-representative birth cohort followed for five decades. *Biological Psychiatry*, *90*(3). https://doi.org/10.1016/j.biopsych.2021.02.971

Ghosh, S. S., Kakunoori, S., Augustinack, J., Nieto-Castanon, A., Kovelman, I., Gaab, N., Christodoulou, J. A., Triantafyllou, C., Gabrieli, J. D. E., & Fischl, B. (2010). Evaluating the validity of volume-based and surface-based brain image registration for developmental cognitive neuroscience studies in children 4 to 11years of age. *NeuroImage*, *53*(1), 85–93.

Gold, A. L., Sheridan, M. A., Peverill, M., Busso, D. S., Lambert, H. K., Alves, S., Pine, D. S., & McLaughlin, K. A. (2016). Childhood abuse and reduced cortical thickness in brain regions involved in emotional processing. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, *57*(10), 1154–1164.

Green, C., Stolicyn, A., Harris, M. A., Shen, X., Romaniuk, L., Barbu, M. C., Hawkins, E. L., Wardlaw, J. M., Steele, J. D., Waiter, G. D., Sandu, A. L., Campbell, A., Porteous, D. J., Seckl, J. R., Lawrie, S. M., Reynolds, R. M., Cavanagh, J., McIntosh, A. M., & Whalley, H. C. (2021). Hair glucocorticoids are associated with childhood adversity, depressive symptoms and reduced global and lobar grey matter in generation Scotland. *Translational Psychiatry*, *11*(1), 1–9.

Green, J. G., McLaughlin, K. A., Berglund, P. A., Gruber, M. J., Sampson, N. A., Zaslavsky, A. M., & Kessler, R. C. (2010). Childhood adversities and adult psychiatric disorders in the national comorbidity survey replication I: Associations with first onset of DSM-IV disorders. *Archives of General Psychiatry*, *67*(2), 113–123.

Gur, R. E., Moore, T. M., Rosen, A. F. G., Barzilay, R., Roalf, D. R., Calkins, M. E., Ruparel, K., Scott, J. C., Almasy, L., Satterthwaite, T. D., Shinohara, R. T., & Gur, R. C. (2019). Burden of environmental adversity associated with psychopathology, maturation, and brain behavior parameters in youths. *JAMA Psychiatry*, *19104*(9), 966–975.

Haddad, E., Pizzagalli, F., Zhu, A. H., Bhatt, R. R., Islam, T., Ba, I., Dixon, D., Thomopoulos, S. I., Thompson, P. M., & Jahanshad, N. (2022). Multisite test-retest reliability and compatibility of brain metrics derived from FreeSurfer Versions 7.1, 6.0, and 5.3. *BioRxiv*, https://doi.org/10.1101/2022.04.13.488251

Hair, N. L., Hanson, J. L., Wolfe, B. L., & Pollak, S. D. (2015). Association of child poverty, brain development, and academic achievement. *JAMA Pediatrics*, *53706*(9), 1–8.

Halfon, N., Larson, K., Son, J., Lu, M., & Bethell, C. (2017). Income inequality and the differential effect of Adverse Childhood Experiences in US children. *Academic Pediatrics*, *17*(7), S70–S78.

Hanson, J. L., Chandra, A., Wolfe, B. L., & Pollak, S. D. (2011). Association between income and the hippocampus. *PLoS ONE*, *6*(5), 1–8.

Hanson, J. L., Hair, N., Shen, D. G., Shi, F., Gilmore, J. H., Wolfe, B. L., & Pollak, S. D. (2013). Family poverty affects the rate of human infant brain growth. *PLoS ONE*, *8*(12).

Hanson, J. L., Nacewicz, B. M., Sutterer, M. J., Cayo, A. A., Schaefer, S. M., Rudolph, K. D., Shirtcliff, E. A., Pollak, S. D., & Davidson, R. J. (2015). Behavioral problems after early life stress: Contributions of the hippocampus and amygdala. *Biological Psychiatry*, *77*(4), 314–323.

Jednoróg, K., Altarelli, I., Monzalvo, K., Fluss, J., Dubois, J., Billard, C., Dehaene-Lambertz, G., & Ramus, F. (2012). The influence of socioeconomic status on children's brain structure. *PLoS ONE*, *7*(8).

Jenkins, L. M., Chiang, J. J., Vause, K., Hoffer, L., Alpert, K., Parrish, T. B., Wang, L., & Miller, G. E. (2020). Subcortical structural variations associated with low socioeconomic status in adolescents. *Human Brain Mapping*, *41*(1), 162–171.

Jollans, L., & Whelan, R. (2016). The clinical added value of imaging: A perspective from outcome prediction. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*, *1*(5), 423–432.

Kelly-Irving, M., Lepage, B., Dedieu, D., Bartley, M., Blane, D., Grosclaude, P., Lang, T., & Delpierre, C. (2013). Adverse childhood experiences and premature all-cause mortality. *European Journal of Epidemiology*, *28*(9), 721–734.

Kelly, P. A., Viding, E., Puetz, V. B., Palmer, A. L., Mechelli, A., Pingault, J. B., Samuel, S., & McCrory, E. J. (2015). Sex differences in socioemotional functioning, attentional bias, and gray matter volume in maltreated children: A multilevel investigation. *Development and Psychopathology*, *27*, 1591–1609.

Kelly, P. A., Viding, E., Wallace, G. L., Schaer, M., De Brito, S. A., Robustelli, B., & Mccrory, E. J. (2013). Cortical thickness, surface area, and gyrification abnormalities in children exposed to maltreatment: Neural markers of vulnerability? *Biological Psychiatry*, *74*(11), 845–852.

Kessler, R. C., McLaughlin, K. A., Green, J. G., Gruber, M. J., Sampson, N. A., Zaslavsky, A. M., Aguilar-Gaxiola, S., Alhamzawi, A. O., Alonso, J., Angermeyer, M., Benjet, C., Bromet, E., Chatterji, S.,

De Girolamo, G., Demyttenaere, K., Fayyad, J., Florescu, S., Gal, G., Gureje, O., … Williams, D. R. (2010). Childhood adversities and adult psychopathology in the WHO world mental health surveys. *British Journal of Psychiatry*, *197*(5), 378–385.

Kraemer, H. C., Kiernan, M., Essex, M., & Kupfer, D. J. (2008). How and why criteria defining moderators and mediators differ between the Baron & Kenny and MacArthur approaches. *Health Psychology*, *27*(2 SUPPL. 2), 101–108.

Lawson, G. M., Duda, J. T., Avants, B. B., Wu, J., & Farah, M. J. (2013). Associations between children's socioeconomic status and prefrontal cortical thickness. *Developmental Science*, *16*(5), 641–652.

Lim, L., Radua, J., & Rubia, K. (2014). Gray matter abnormalities in childhood maltreatment: A voxelwise metaanalysis. *American Journal of Psychiatry*, *171*(8), 854–863.

Luby, J. L., Belden, A., Botteron, K., Marrus, N., Harms, M. P., Babb, C., Nishino, T., & Barch, D. (2013). The effects of poverty on childhood brain development: The mediating effect of caregiving and stressful life events. *JAMA Pediatrics*, *167*(12), 1135–1142.

Luby, J. L., Heffelfinger, A., Koenig-McNaught, A. L., Brown, K., & Spitznagel, E. (2004). The preschool feelings checklist: A brief and sensitive screening measure for depression in young children. *Journal of the American Academy of Child and Adolescent Psychiatry*, *43*(6), 708–717.

Luby, J. L., Tillman, R., & Barch, D. M. (2019). Association of timing of adverse childhood experiences and caregiver support with regionally specific brain development in adolescents. *JAMA Network Open*, *2*(9). https://doi.org/10.1001/jamanetworkopen.2019.11426

Lupien, S. J., McEwen, B. S., Gunnar, M. R., & Heim, C. (2009). Effects of stress throughout the lifespan on the brain, behaviour and cognition. *Nature Reviews Neuroscience*, *10*(6), 434–445.

Mackes, N. K., Golm, D., Sarkar, S., Kumsta, R., Rutter, M., Fairchild, G., Mehta, M. A., & Sonuga-Barke, E. J. S. (2020). Early childhood deprivation is associated with alterations in adult brain structure despite subsequent environmental enrichment. *Proceedings of the National Academy of Sciences of the United States of America*, *117*(1), 641–649.

Madan, C. R., & Kensinger, E. A. (2017). Test–retest reliability of brain morphology estimates. *Brain Informatics*, *4*(2), 107–121.

Marshall, N. A., Marusak, H. A., Sala-Hamrick, K. J., Crespo, L. M., Rabinak, C. A., & Thomason, M. E. (2018). Socioeconomic disadvantage and altered corticostriatal circuitry in urban youth. *Human Brain Mapping*, *39*(5), 1982–1994.

Masten, A. S., & Cicchetti, D. (2010). Developmental cascades. *Development and Psychopathology*, *22*(03), 491–495.

Maxwell, E. (1992). *Manual for the family interview for genetic Studies (FIGS)*. Clinical Neurogenetics Branch, Intramural Research Program, National Institute of Mental Health.

McDermott, C. L., Seidlitz, J., Nadig, A., Liu, S., Clasen, L. S., Blumenthal, J. D., Reardon, P. K., Lalonde, F., Greenstein, D., Patel, R., Chakravarty, M. M., Lerch, J. P., & Raznahan, A. (2019). Longitudinally mapping childhood socioeconomic status associations with cortical and subcortical morphology. *The Journal of Neuroscience*, *39*(8), 1365–1373.

McLaughlin, K. A., Green, J. G., Gruber, M. J., Sampson, N. A., Zaslavsky, A. M., Kessler, R. C., Jennifer, G., Green, G., Gruber, M. J., Sampson, N. A., Zaslavsky, A. M., & Kessler, R. C. (2012). Childhood adversities and first onset of psychiatric disorders in a national sample of US adolescents. *Archives of General Psychiatry*, *69*(11), 1151–1160.

McLaughlin, K. A., Sheridan, M. A., Gold, A. L., Duys, A., Lambert, H. K., Peverill, M., Heleniak, C., Shechner, T., Wojcieszak, Z., & Pine, D. S. (2016). Maltreatment Exposure, Brain Structure, and Fear Conditioning in Children and Adolescents. *Neuropsychopharmacology*, *41*(8), 1956–1964.

McLaughlin, K. A., Sheridan, M. A., & Lambert, H. K. (2014). Childhood adversity and neural development: Deprivation and threat as distinct dimensions of early experience. *Neuroscience and Biobehavioral Reviews*, *47*, 578–591.

McLoyd, V. C. (1998). Socioeconomic disadvantage and child development. *American Psychologist*, *53*(2), 185–204.

Mehta, M. A., Golembo, N. I., Nosarti, C., Colvert, E., Mota, A., Williams, S. C. R. R., Rutter, M., & Sonuga-Barke, E. J. S. S. (2009). Amygdala, hippocampal and corpus callosum size following severe early institutional deprivation: The English and Romanian adoptees study pilot. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *50*(8), 943–951.

Merz, E. C., Tottenham, N., & Noble, K. G. (2018). Socioeconomic status, amygdala volume, and internalizing symptoms in children and adolescents. *Journal of Clinical Child and Adolescent Psychology*, *47*(2), 312–323.

Noble, K. G., Houston, S. M., Brito, N. H., Bartsch, H., Kan, E., Kuperman, J. M., Akshoomoff, N., Amaral, D. G., Bloss, C. S., Libiger, O., Schork, N. J., Murray, S. S., Casey, B. J., Chang, L., Ernst, T. M., Frazier, J. A., Gruen, J. R., Kennedy, D. N., Van Zijl, P., … Sowell, E. R. (2015). Family income, parental education and brain structure in children and adolescents. *Nature Neuroscience*, *18*(5), 773–778.

Pascucci, D., Hickey, C., Jovicich, J., & Turatto, M. (2017). Independent circuits in basal ganglia and cortex for the processing of reward and precision feedback. *NeuroImage*, *162*(August), 56–64.

Quinlan, E. B., Barker, E. D., Luo, Q., Banaschewski, T., Bokde, A. L. W., Bromberg, U., Büchel, C., Desrivières, S., Flor, H., Frouin, V., Garavan, H., Chaarani, B., Gowland, P., Heinz, A., Brühl, R., Martinot, J. L., Martinot, M. L. P., Nees, F., Orfanos, D. P., … Schumann, G. (2018). Peer victimization and its impact on adolescent brain development and psychopathology. *Molecular Psychiatry*, 3066–3076.

R Core Team. (2020). *R: A language and environment for statistical computing*. R Foundation for Statistical Computing. https://www.r-project.org/

Reuter, M., Schmansky, N. J., Rosas, H. D., & Fischl, B. (2012). Within-subject template estimation for unbiased longitudinal image analysis. *NeuroImage*, *61*(4), 1402–1418.

Sacchet, M. D., Camacho, M. C., Livermore, E. E., Thomas, E. A. C., & Gotlib, I. H. (2017). Accelerated aging of the putamen in patients with major depressive disorder. *Journal of Psychiatry and Neuroscience*, *42*(3), 164–171.

Sheridan, M. A., Fox, N. A., Zeanah, C. H., McLaughlin, K. A., & Nelson, C. A. (2012). Variation in neural development as a result of exposure to institutionalization early in childhood. *Proceedings of the National Academy of Sciences of the United States of America*, *109*(32), 12927–12932.

Staff, R. T., Murray, A. D., Ahearn, T. S., Mustafa, N., Fox, H. C., & Whalley, L. J. (2012). Childhood socioeconomic status and adult brain size: Childhood socioeconomic status influences adult hippocampal size. *Annals of Neurology*, *71*(5), 653–660.

Taillieu, T. L., Brownridge, D. A., Sareen, J., & Afifi, T. O. (2016). Childhood emotional maltreatment and mental disorders: Results

from a nationally representative adult sample from the United States. *Child Abuse and Neglect*, *59*, 1–12.

Teicher, M. H., Samson, J. A., Anderson, C. M., & Ohashi, K. (2016). The effects of childhood maltreatment on brain structure, function and connectivity. *Nature Reviews Neuroscience*, *17*(10), 652–666.

Tomalski, P., & Johnson, M. H. (2010). The effects of early adversity on the adult and developing brain. *Current Opinion in Psychiatry*, *23*(3), 233–238.

Tottenham, N. (2014). The importance of early experiences for neuro-affective development. *Current Topics in Behavioral Neurosciences*, *16*, 109–129.

Tottenham, N., Hare, T. A., Quinn, B. T., McCarry, T. W., Nurse, M., Gilhooly, T., Millner, A., Galvan, A., Davidson, M. C., Eigsti, I. M., Thomas, K. M., Freed, P. J., Booma, E. S., Gunnar, M. R., Altemus, M., Aronson, J., & Casey, B. J. (2010). Prolonged institutional rearing is associated with atypically large amygdala volume and difficulties in emotion regulation. *Developmental Science*, *13*(1), 46–61.

Vidal-Ribas, P., Benson, B., Vitale, A. D., Keren, H., Harrewijn, A., Fox, N. A., Pine, D. S., & Stringaris, A. (2019). Bidirectional associations between stress and reward processing in children and adolescents: A longitudinal neuroimaging study. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*, *4*(10), 893–901.

Vlasova, R., Gajawelli, N., Wang, Y., Dirks, H., Dean, D., O'Muircheartaigh, J., Lao, Y., Yoon, J., Nelson, M. D., Deoni, S., & Lepore, N. (2017). Putamen development in children 12 to 21 months old. *12th International Symposium on Medical Information Processing and Analysis*, *10160*(January 2017), 101600E.

Walsh, D., McCartney, G., Smith, M., & Armour, G. (2019). Relationship between childhood socioeconomic position and adverse childhood experiences (ACEs): A systematic review. *Journal of Epidemiology and Community Health*, *73*(12), 1087–1093.

Wierenga, L., Langen, M., Ambrosino, S., van Dijk, S., Oranje, B., & Durston, S. (2014). Typical development of basal ganglia, hippocampus, amygdala and cerebellum from age 7 to 24. *NeuroImage*, *96*, 67–72.

SUPPORTING INFORMATION

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

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