HHS Public Access

Author manuscript

JAMA Pediatr. Author manuscript; available in PMC 2015 December 22.

Published in final edited form as:

JAMA Pediatr. 2015 September ; 169(9): 822–829. doi:10.1001/jamapediatrics.2015.1475.

Association of Child Poverty, Brain Development, and Academic Achievement

Nicole L. Hair, PhD, **Jamie L. Hanson, PhD**, **Barbara L. Wolfe, PhD**, and **Seth D. Pollak, PhD**

Department of Health Management and Policy, School of Public Health, University of Michigan, Ann Arbor (Hair); Department of Psychology and Neuroscience, Duke University, Durham, North Carolina (Hanson); Department of Economics, University of Wisconsin–Madison, Madison (Wolfe); Department of Population Health Sciences, University of Wisconsin–Madison, Madison (Wolfe); La Follette School of Public Affairs, University of Wisconsin–Madison, Madison (Wolfe); Department of Psychology, University of Wisconsin–Madison, Madison (Pollak); Waisman Center, University of Wisconsin–Madison, Madison (Pollak)

Abstract

IMPORTANCE—Children living in poverty generally perform poorly in school, with markedly lower standardized test scores and lower educational attainment. The longer children live in poverty, the greater their academic deficits. These patterns persist to adulthood, contributing to lifetime-reduced occupational attainment.

OBJECTIVE—To determine whether atypical patterns of structural brain development mediate the relationship between household poverty and impaired academic performance.

DESIGN, SETTING, AND PARTICIPANTS—Longitudinal cohort study analyzing 823 magnetic resonance imaging scans of 389 typically developing children and adolescents aged 4 to 22 years from the National Institutes of Health Magnetic Resonance Imaging Study of Normal Brain Development with complete sociodemographic and neuroimaging data. Data collection

Corresponding Author: Seth D. Pollak, PhD, Waisman Center, University of Wisconsin–Madison, 1500 Highland Ave, Madison, WI 53706 (spollak@wisc.edu).

Supplemental content at jamapediatrics.com

Conflict of Interest Disclosures: None reported.

Role of the Funder/Sponsor: The funders had no role in the design and conduct of the study; collection, management, analysis, and interpretation of the data; preparation, review, or approval of the manuscript; and decision to submit the manuscript for publication.

Disclaimer: This article reflects the views of the authors and may not reflect the opinions or views of the National Institutes of Health.

Additional Information: Data used in the preparation of this article were obtained from the Pediatric Magnetic Resonance Imaging Data Repository created by the National Institutes of Health Magnetic Resonance Imaging Study of Normal Brain Development. A listing of the participating sites and a complete listing of the study investigators can be found at http://www.bic.mni.mcgill.ca/nihpd/info/participating_centers.html.

Author Contributions: Drs Pollak and Wolfe had full access to all of the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis.
Study concept and design: All authors.
Acquisition, analysis, or interpretation of data: All authors.
Drafting of the manuscript: All authors.
Critical revision of the manuscript for important intellectual content: All authors.
Statistical analysis: Hair, Wolfe.
Obtained funding: Wolfe, Pollak.
Administrative, technical, or material support: Hanson, Wolfe.
Study supervision: Wolfe, Pollak.

began in November 2001 and ended in August 2007. Participants were screened for a variety of factors suspected to adversely affect brain development, recruited at 6 data collection sites across the United States, assessed at baseline, and followed up at 24-month intervals for a total of 3 periods. Each study center used community-based sampling to reflect regional and overall US demographics of income, race, and ethnicity based on the US Department of Housing and Urban Development definitions of area income. One-quarter of sample households reported the total family income below 200% of the federal poverty level. Repeated observations were available for 301 participants.

EXPOSURE—Household poverty measured by family income and adjusted for family size as a percentage of the federal poverty level.

MAIN OUTCOMES AND MEASURES—Children's scores on cognitive and academic achievement assessments and brain tissue, including gray matter of the total brain, frontal lobe, temporal lobe, and hippocampus.

RESULTS—Poverty is tied to structural differences in several areas of the brain associated with school readiness skills, with the largest influence observed among children from the poorest households. Regional gray matter volumes of children below 1.5 times the federal poverty level were 3 to 4 percentage points below the developmental norm ($P < .05$). A larger gap of 8 to 10 percentage points was observed for children below the federal poverty level ($P < .05$). These developmental differences had consequences for children's academic achievement. On average, children from low-income households scored 4 to 7 points lower on standardized tests ($P < .05$). As much as 20% of the gap in test scores could be explained by maturational lags in the frontal and temporal lobes.

CONCLUSIONS AND RELEVANCE—The influence of poverty on children's learning and achievement is mediated by structural brain development. To avoid long-term costs of impaired academic functioning, households below 150% of the federal poverty level should be targeted for additional resources aimed at remediating early childhood environments.

Low-income students are now a majority of schoolchildren attending public schools in the United States. Data collected by the National Center for Education Statistics show that 51% of students across US public schools were from low-income families in 2013.[1] Socioeconomic disparities in school readiness and academic performance are well documented. Children living in poverty have lower scores on standardized tests of academic achievement, poorer grades in school, and lower educational attainment.[2,3] These patterns persist into adulthood, ultimately contributing to low wages and income.[4,5] Moreover, increased exposure to poverty in childhood is tied to greater deficits in these domains.[6,7] Despite numerous studies demonstrating the relationship between family resources and children's educational outcomes, little is known about mechanisms underlying the influence of poverty on children's learning and achievement. In the current study, we tested whether atypical structural development in several areas of the brain tied to school readiness skills may have mediated the relationship between childhood poverty and impaired academic performance. Our hypotheses were motivated by the widespread environmental inequities (both physical and psychological) faced by children living in poverty along with increasing evidence that environmental stimulation, parental nurturance, and early life stress affect brain growth and functioning.

Socioeconomic Status Disparities in Academic Achievement

Children living in poverty tend to fare poorly across a variety of academic measures beginning in early childhood,[8] with consequences found to persist to adulthood.[4,5] A study of adopted children by Duyme et al[9] provides some of the most compelling evidence that parental financial resources have a causal effect on children's cognitive performance. In that study,[9] the IQs of more than 5000 children were assessed prior to adoption and again in adolescence. Compared with children adopted into lower socioeconomic status (SES) families, the IQs of children adopted into higher SES families were 13 points higher in adolescence. Additional studies that exploit variation in the types of public programs that target low-income families, such as the Earned Income Tax Credit[10] and Welfare to Work experiments,[11] also point to the influence of increased parental income on children's outcomes.

Brain Plasticity and Environments of Poverty

Research involving nonhuman animals (where the environment can be experimentally manipulated, controlled, and precisely measured) demonstrates that environmental stimulation, parental nurturance, and early life stress affect brain structure and functioning.[12–14] These kinds of early experiences map adversities characteristic of poverty environments. When compared with their more-advantaged peers, children living in poverty experience less parental nurturance while confronting elevated levels of life stress, increased family instability, and greater exposure to violence. Their homes are more crowded and often provide less-cognitive stimulation.[15]

Initial efforts to understand the effects of poverty on the human brain structure and development used neurocognitive tests to assay functions associated with specific areas of the brain.[16] There is strong evidence that poverty influences language (tied to the temporal lobe) and executive functioning (related to the frontal lobe).[17–19] Deficits in the executive functioning of individuals in poverty have been found during the life course in studies conducted during infancy[20] as well as in childhood, adolescence,[21] and adulthood.[22] Motivated by these findings, a growing number of studies have used neuroimaging and found smaller volumes in the frontal and temporal lobes for children and adolescents living in poverty.[23,24] Different facets of poverty, including elevated life stress and less caregiving support,[25,26] may uniquely or interactively contribute to such differences in neurobiology.

At a Glance

- This study tests whether structural brain development may mediate the relationship between childhood poverty and impaired academic performance.
- Magnetic resonance imaging brain scans of 389 economically diverse and typically developing children aged 4 to 22 years were analyzed.
- Children from families with limited financial resources displayed systematic structural differences in the frontal lobe, temporal lobe, and hippocampus.

- Developmental differences in the frontal and temporal lobes may explain as much as 20% of low-income children's achievement deficits.

Hypotheses

The focus of this study was to determine whether systematic differences in structural brain development mediate the relationship between poverty and impaired academic performance. We focused on the gray matter tissue of several areas of the brain that are likely vulnerable to early environments (eg, areas that display a protracted period of postnatal development or less heritability) and are believed to have an important role in cognitive abilities that are critical for children's school readiness.[27] Focal brain areas include the frontal lobe because previous research has found that this brain region is particularly important for the top-down control of attention, inhibition, emotion regulation, and complex learning[28]; the temporal lobe because of its importance for memory and language comprehension, such as identifying words, relating heard sounds with letters of the alphabet, and attaching meaning to words[29]; and the hippocampus, a brain structure that plays a critical role in processing spatial and contextual information and has been tied to long-term memory functioning.[30] Taken together, circuits in these areas of the brain influence critical processes and skills, including reading comprehension,[31] language usage,[32] and associative learning.[33] Dysfunction in these processes may significantly affect scholastic and later occupational success.

The current study included a diverse sample of children and adolescents. The broad range of participants aged 4 to 22 years was a novel aspect of this large multisite longitudinal study. Participants were followed up and rescanned across a number of years. Because human gray matter follows a nonlinear developmental trajectory, we established a reference for typical development in focal brain areas and constructed an index that measured whether regional gray matter volume was larger or smaller than expected, comparing children with others of the same sex and age. Thus, structural brain development was assessed in terms of deviations from an expected norm.

Methods

Participants

We used data from the National Institutes of Health Magnetic Resonance Imaging Study of Normal Brain Development (http://pediatricmri.nih.gov/nihpd/info/Documents/Protocol_release_Nov06.pdf). Institutional review board approval was obtained from the University of Wisconsin–Madison. Written informed consent was obtained from parents before screening as well as during on-site visits and magnetic resonance scans. Children aged 6 to 17 years also provided written informed assent.

Following a community-based sampling plan, 433 children aged 4 to 18 years were recruited at 6 study centers across the United States to reflect both regional and US demographic compositions of income and race/ethnicity. Income by race/ethnicity categories were distributed across age, with equal sex representation for each age category. Participants were followed up at 24-month intervals across 3 periods. We analyzed 823 observations of 389 children with complete neuroimaging and sociodemographic information (Table 1).

Participating families were screened for a number of factors suspected to adversely affect brain development. Exclusionary criteria included demographic characteristics (eg, whether the child was adopted); risky pregnancy, birth, and neonatal histories; physical/medical histories (eg, lead treatment or maternal medications during breastfeeding); family psychiatric history; and behavioral/psychiatric measures, including low IQ. Details regarding sampling and recruitment can be found in the Waber et al study.[34]

Family Income

The database included race/ethnicity, family size, parents' education, and household income. Total family income was recorded in 9 categories, ranging from less than $5000 to between $100 000 and $150 000. We adjusted household income measured at the categorical midpoint for family size using federal poverty thresholds. The sample was economically diverse. We observed households well below the federal poverty level (FPL) to families with incomes more than 8 times the FPL. One-quarter of households were classified as poor or near poor (below 200% of the FPL). Reported income was overwhelmingly stable during the sample period, with very few families transitioning in or out of poverty. Mothers' educational attainment in our sample was high; 84.9% of mothers reported at least some college-level education and 22.4% reported at least some graduate-level education. Comparable patterns were observed for sample fathers. Rates of successful recruitment were similar across 3 income groups. However, consistent with elevated morbidity within low-income populations, children from the lowest income category were more likely to meet 1 or more exclusionary criteria during preliminary screening (eTables 1 and 2 in the Supplement).

Procedures

Neuroimaging and neurobehavioral testing batteries were attempted for all participants and intervals. While magnetic resonance imaging scan success rates were high, some neuroimaging data were incomplete owing to artifacts associated with child movement or contraindication for magnetic resonance imaging scanning (eg, missed visit owing to dental braces). Incomplete neuroimaging information was found to be unrelated to socioeconomic characteristics (eTable 3 in the Supplement). Repeated scans were available for 301 children. Neuroimaging data for each participant were processed according to voxel-based morphometry analytic framework with region of interest drawings. The processing of neuroimaging data is described in eAppendix 1 in the Supplement. The Wechsler Abbreviated Scale of Intelligence (WASI) and Woodcock-Johnson III Tests of Achievement (WJ-III) were administered to assess general intelligence and measure language and math achievement. The WASI composite scores included a verbal IQ that measured word knowledge, verbal reasoning, and concept formation and a performance IQ that assessed visual information processing, abstract reasoning, and visual motor coordination. The full-scale IQ combined the verbal IQ and performance IQ.[35] The WJ-III subscales included math computation, letter-word identification, and passage comprehension. The letter-word identification and passage comprehension tests measure a child's word identification skills and ability to understand written text.[36] Both the WASI and WJ-III assessments were standardized with a mean (SD) of 100 (15).

Data Analyses: Modeling Normal Brain Development

Dynamic changes in the brain continue through young adulthood. An initial period of growth is followed by a period of pruning as the brain cuts off unused pathways.[37] To account for the nonmonotonic inverted U-shaped trajectories of gray matter volumes, we first established a reference of typical development for each brain area of interest. We modeled regional gray matter volume trajectories, estimating sex-specific mixed effect linear models, a statistical analysis technique that combined cross-sectional and longitudinal data and accounted for both intraparticipant correlation and unbalanced panel design.[38]

Using the estimated developmental trajectories (eTable 4 and eFigures 1, 2, 3, and 4 in the Supplement), we constructed an index of structural brain development based on an adjusted or normed measure of regional gray matter volume. The participant regional volume was expressed as a percentage of an expected volume given sex and age. This index reflected deviations from normative development. Primary analyses considered whether a region was smaller or larger than expected by comparing a child with others of the same sex and age. Basic summary statistics related to developmental indices are available in eTable 5 in the Supplement.

Modeling Brain Development and Poverty

Using the constructed indices, we examined the influence of socioeconomic status, specifically growing up in or near poverty, on development within focal areas of the brain. Family financial resources were used as an indicator of SES. Low SES was defined using both binary and categorical income measures and we additionally considered the sensitivity of estimates to the selection of particular income thresholds. Specifications with an extended set of covariates controlled for birth weight, race/ethnicity, family size, and maternal education. The results provided evidence of a tie between low income and the gray matter in critical areas of the brain. These results were used in the following analysis of brain development and academic achievement.

Modeling Brain Development in Relation to Poverty and Academic Achievement

As hypothesized, low income was associated with lower WASI and WJ-III scores. To improve our understanding of this relationship between poverty and impaired academic performance, we used mediation analysis.[39] Focusing on areas of the brain where we reported deviations from normative development among low-income children, we tested whether structural brain development (ie, relative regional gray matter) was 1 process or a channel underlying the income achievement gap. The amygdala, a brain structure that was not expected to influence cognition as measured by educational assessments, was presented as a control region. eAppendix 2 in the Supplement includes a detailed discussion of statistical methods.

Results

SES and Anatomical Brain Development

Low SES was associated with atypical gray matter development. Children from families with limited financial resources displayed systematic structural differences in the frontal

lobe, temporal lobe, and hippocampus. The regional gray matter volumes of children below 1.5 times the FPL were, on average, 3 to 4 percentage points below developmental norms for their sex and age (Table 2). The estimated gap increased to 7 to 10 percentage points in children living below the FPL (Table 2).

A review of Table 2 suggests that the detrimental influence of growing up in or near poverty was concentrated among those children from the poorest households. When compared with near-poor peers, children below the poverty threshold displayed a significant maturational lag in each brain area of interest. In contrast, a comparison of near-poor children with higher SES peers revealed no significant differences in brain structure (Table 2). This nonlinear income pattern was constant across alternative definitions of SES, including measures based on current income, permanent income, minimum reported income, and family size–adjusted income (eTable 6 in the Supplement).

We considered several alternative hypotheses, such as that the observed structural differences in the brains of children developing in poverty might have been explained by differences in early health or parental education. Study participants were subject to strict eligibility criteria, including family medical, prenatal, birth, and perinatal histories. Additionally, we controlled for birth weight, an indicator of both early health status, and initial head size. Likewise, it is unlikely that the atypical development was driven by SES-associated differences in parental education. Poor families in our sample were highly educated. Estimates of the influence of poverty were consistent in models that were adjusted for the level of maternal education.

SES, Anatomical Brain Development, and Academic Achievement

Children below 1.5 times the FPL scored 4 to 8 points (¼ to ½ of a SD) lower on tests of achievement ($P < .05$). In addition, the structural development of gray matter in brain areas where atypical development has been reported in low-income children was associated with improved test performance. We used mediation analyses to formally test whether differences in neurobiology may help explain the deleterious effects of childhood poverty on academic achievement.

For each focal brain area, we presented estimates of the direct effect of low income on academic achievement alongside estimates of the indirect effect (ie, the portion that may have been explained by poverty's influence on [adjusted] regional gray matter volume). We then calculated the indirect (mediated) effect as a fraction of the total low-income effect. Finally, we presented parallel estimates for 1 additional brain structure. The amygdala provided a point of comparison for the outlined mediation analyses because while the region plays a key role in the processing of emotions, we did not expect it to influence cognition (as measured by the WASI or WJ-III).

We found that developmental differences in the frontal and temporal lobes may have explained as much as 15% (Table 3) to 20% (Table 4) of low-income children's achievement deficits. Analysis of the amygdala provides evidence that we were capturing regionally specific effects (ie, differences in specific brain regions of interest vs the alternative hypothesis that children in poverty have smaller brains overall). In contrast to our

main results, estimates tied to the amygdala (Table 2) were small and statistically indistinguishable from zero. Additional analyses (eTable 7 and eTable 8 in the Supplement) controlled for multiple but nonoverlapping portions of the brain and similarly suggested the importance of the frontal and temporal lobes.

Discussion

Although the income achievement gap is well documented, the question of how childhood poverty is translated into deficits in learning and academic achievement is largely unanswered. With the current data, we demonstrated that children from low-income households exhibit atypical structural development in several critical areas of the brain, including total gray matter and the frontal lobe, temporal lobe, and hippocampus. This maturational lag has implications for children's scholastic success. A typical low-income child scores lower on standardized tests of achievement and 15% to 20% of that developmental difference might be attributed to the deleterious effects of limited family resources on relative brain development. We found that the influence of parental SES on children's anatomical brain development was concentrated among children from the poorest households. No statistically significant differences were found when comparing near-poor children (eg, 150% to 200% of the FPL or $25 000–$35 000) with children from higher SES groups.

Our study had 2 limitations worth noting. First, it is possible that reported differences across socioeconomic groups could have been caused by a third factor tied both to family poverty and smaller regional gray matter volumes, such as a genetic predisposition that might have led an individual to become poor. Our analyses mitigated concerns related to this competing explanation. We focused on regions of the brain known to undergo a protracted period of postnatal development (most likely to be influenced by environmental conditions), specifically, the brain's gray matter tissue, which previous work suggests is likely affected by early environment and less heritable than other brain tissues. Second, the National Institutes of Health study was designed specifically to study typical development; therefore, children were screened based on factors thought to adversely affect brain development. However, such adversities are disproportionately represented among impoverished children, meaning that this study examined a sample of children who were likely doing better than most children living in poverty. Our analyses likely understated the full effects of poverty on children's development. The strict exclusionary criteria were beneficial in that they allowed us to rule out a number of potentially confounding factors, particularly a child's early or initial health status, as influencing reported associations with family income or socioeconomic status and mitigated the potential for adverse selection of sample families based on unobserved factors (eg, families who may volunteer out of concern for a child's health or developmental progress). However, a true representative sample of children in poverty is likely to reveal even greater deficiencies than those reported in this relatively healthy sample of impoverished children, who, despite meeting the study's inclusionary criteria, still evinced striking neurocognitive delays.

Conclusions

While brain structure and development may not be the only mechanism underlying the income achievement gap, the novel evidence presented in this study seems to suggest that 1 component linking parental SES to children's achievement and human capital more broadly operates through a neurobiological mechanism. Our work suggests that specific brain structures tied to processes critical for learning and educational functioning (eg, sustained attention, planning, and cognitive flexibility) are vulnerable to the environmental circumstances of poverty, such as stress, limited stimulation, and nutrition. If so, it would appear that children's potential for academic success is being reduced at young ages by these circumstances. Such understanding should lead to public policy initiatives aimed at improving and decreasing disparities in human capital. Development in these brain regions appears sensitive to the child's environment and nurturance. These observations suggest that interventions aimed at improving children's environments may also alter the link between childhood poverty and deficits in cognition and academic achievement.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

Funding/Support: This multisite longitudinal study of typically developing children, including newborns through children in young adulthood, was conducted by the Brain Development Cooperative Group and supported by the National Institute of Child Health and Human Development, the National Institute on Drug Abuse, the National Institute of Mental Health, and the National Institute of Neurological Disorders and Stroke (grants N01-HD02-3343; N01-MH9-0002; and N01-NS-9-2314, -2315, -2316, -2317, -2319, and -2320).

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Table 1

Summary of Sample Characteristics in the National Institutes of Health Magnetic Resonance Imaging Study of Normal Brain Development[a]

Variable	Mean (Range)
Male	0.475 (0–1)
Nonwhite	0.147 (0–1)
Hispanic	0.122 (0–1)
Birth weight, oz	126 (86–182)
Age, y	12 (4–22)
Scans, No.	2.12 (1–3)
Age at first scan, y	11.1 (4–20)
Family size	5.39 (2–14)
Education level	
Less than high school	0.008 (0–1)
High school	0.144 (0–1)
Some college	0.302 (0–1)
College	0.323 (0–1)
Some graduate school	0.056 (0–1)
Graduate school	0.168 (0–1)
Income	
Relative to the FPL, %	360.7 (10.7–838.9)
Below 100% of the FPL	0.056 (0–1)
Between 100% and 150% of the FPL	0.100 (0–1)
Between 150% and 200% of the FPL	0.104 (0–1)
Above 200% of the FPL	0.740 (0–1)
WASI	
Full-scale IQ	112 (75–160)
Performance IQ	111 (72–157)
Verbal IQ	110.4 (73–156)
WJ-III	
Math computation	110.3 (74–156)
Letter-word identification	108.6 (71–151)
Passage comprehension	107.7 (71–140)

Abbreviations: FPL, federal poverty level; WASI, Wechsler Abbreviated Scale of Intelligence; WJ-III, Woodcock-Johnson III Tests of Achievement.

[a] Analysis sample comprised 823 observations of 389 children with neuroimaging and sociodemographic information. Family income assigned the value of the categorical midpoint. Household income levels were overwhelmingly stable across the sample period, with very few families observed to transition into or out of poverty. Mean (SD) scores on both the WASI and WJ-III were standardized (100 [15]). The WASI and WJ-III batteries were administered to children who were aged at least 5 and 6 years, respectively.

JAMA Pediatr. Author manuscript; available in PMC 2015 December 22.

Table 2

Socioeconomic Status and Brain Development in the National Institutes of Health Magnetic Resonance Imaging Study of Normal Brain Development[a]

Variable	Total Gray Matter, β (SE)		Frontal Gray Matter, β (SE)		Temporal Gray Matter, β (SE)		Hippocampus Gray Matter, β (SE)	
Model 1								
Below 200% of the FPL	−2.437[b] (1.098)	−1.581 (1.159)	−2.157[b] (1.24)	−1.19 (1.331)	−2.336[c] (1.285)	−1.165 (1.343)	−1.716 (1.244)	−0.573 (1.302)
Model 2								
Below 150% of the FPL	−3.839[b] (1.432)	−2.816[b] (1.368)	−3.532[b] (1.546)	−2.375 (1.519)	−4.250[b] (1.639)	−3.002[c] (1.528)	−3.710[b] (1.355)	−2.642[c] (1.416)
Model 3								
Below 100% of the FPL	−8.808[c] (2.328)	−7.505[c] (2.298)	−8.383[b] (2.597)	−7.037[b] (2.742)	−9.497[b] (2.600)	−7.844[c] (2.439)	−8.035[b] (1.807)	−6.564[b] (1.946)
Model 4								
Below 150% of the FPL	−3.903[b] (1.436)	−2.859[b] (1.389)	−3.577[b] (1.553)	−2.369 (1.545)	−4.259[b] (1.648)	−2.893[c] (1.562)	−3.661[b] (1.379)	−2.411[c] (1.46)
Between 150% to 200% of the FPL	−0.727 (1.455)	−0.25 (1.566)	−0.502 (1.721)	0.0364 (1.854)	−0.0932 (1.688)	0.634 (1.802)	0.553 (1.739)	1.341 (1.741)
Model 5								
Below 100% of the FPL	−8.953[b] (2.342)	−7.591[b] (2.335)	−8.493[b] (2.615)	−7.024[b] (2.783)	−9.603[b] (2.618)	−7.803[b] (2.483)	−8.079[b] (1.820)	−6.426[b] (1.969)
Between 100% to 150% of the FPL	−1.328 (1.319)	−0.431 (1.244)	−1.07 (1.445)	0.0202 (1.364)	−1.534 (1.603)	−0.373 (1.504)	−1.409 (1.598)	−0.350 (1.635)
Between 150% to 200% of the FPL	−0.727 (1.455)	−0.207 (1.562)	−0.502 (1.722)	0.0782 (1.851)	−0.0932 (1.689)	0.678 (1.799)	0.553 (1.740)	1.377 (1.738)
Extended controls	No	Yes	No	Yes	No	Yes	No	Yes
No. of Observations	823	817	823	817	823	817	823	817

Abbreviation: FPL, federal poverty level.

[a]Measures of brain development are normed. The focal brain area volume is expressed as a percentage of the sex- and age-specific norm. Results related to the estimation of normative developmental curves are available in eTable 4 in the Supplement. Extended controls included birth weight, race/ethnicity, family size, and maternal education.

[b]$P < .05$.

[c]$P < .10$.

Table 3

Socioeconomic Status, Brain Development, and WJ-III Scores in the National Institutes of Health Magnetic Resonance Imaging Study of Normal Brain Development[a]

Variable	Frontal Lobe, β	Temporal Lobe, β	Hippocampus, β	Amygdala, β
WJ-III math computation (n = 87)				
Direct effect	−6.18	−6.15	−6.27	−6.64
Indirect effect	−0.95	−1.01	−0.87	−0.54
Percentile 95% CI	−1.72 to −0.32	−1.78 to −0.38	−1.59 to −.28	−1.26 to 0.02
Bias-corrected 95% CI	−1.83 to −0.39	−1.83 to −0.41	−1.66 to −.33	−1.35 to −0.03
Indirect/total effect	0.13	0.14	0.12	0.02
WJ-III letter-word identification (n = 798)				
Direct effect	−3.83	−3.65	−3.97	−4.05
Indirect effect	−0.47	−0.66	−0.33	−0.26
Percentile 95% CI	−0.92 to −0.12	−1.22 to −0.24	−0.77 to −0.02	−0.65 to 0.01
Bias-corrected 95% CI	−0.98 to −0.14	−1.32 to −0.28	−0.83 to −0.05	−0.72 to −0.01
Indirect/total effect	0.11	0.15	0.08	0.06
WJ-III passage comprehension (n = 797)				
Direct effect	−5.15	−4.94	−5.07	−5.43
Indirect effect	−0.4	−0.61	−0.49	−0.13
Percentile 95% CI	−0.83 to −0.08	−1.11 to −0.20	−0.99 to −0.11	−0.43 to 0.05
Bias-corrected 95% CI	−0.91 to −0.11	−1.20 to −0.25	−1.05 to −0.14	−0.52 to 0.01
Indirect/total effect	0.07	0.11	0.09	0.02

Abbreviation: WJ-III, Woodcock-Johnson III Tests of Achievement.

[a]Mediation analyses correspond to specifications in eTable 9 in the Supplement. Estimates of the direct and indirect (mediated through influence on structural brain development) effects of low income on a standardized test of achievement are shown. Mean (SD) tests scores are standardized (100 [15]). Standard errors have been bootstrapped. The 95% CIs were constructed using bootstrap resampling with 5000 iterations.

Table 4

Socioeconomic Status, Brain Development, and WASI Scores in the National Institutes of Health Magnetic Resonance Imaging Study of Normal Brain Development[a]

Variable	Frontal Lobe, β	Temporal Lobe, β	Hippocampus, β	Amygdala, β
WASI full-scale IQ (n = 802)				
Direct effect	−6.92	−6.61	−6.88	−7.62
Indirect effect	−1.08	−1.4	−1.12	−0.41
Percentile 95% CI	−1.96 to −0.34	−2.37 to −0.52	−1.97 to −0.39	−1.00 to 0.03
Bias-corrected 95% CI	−1.98 to −0.36	−2.43 to −0.57	−1.98 to −0.41	−1.09 to −0.001
Indirect/total effect	0.14	0.17	0.14	0.05
WASI performance IQ (n = 802)				
Direct effect	−5.65	−5.32	−5.66	−6.37
Indirect effect	−1.09	−1.43	−1.08	−0.4
Percentile 95% CI	−2.00 to −0.34	−2.47 to −0.57	−1.86 to −0.39	−0.97 to 0.017
Bias-corrected 95% CI	−2.08 to −0.40	−2.56 to −0.63	−1.92 to −0.44	−1.05 to −0.02
Indirect/total effect	0.16	0.21	0.16	0.06
WASI verbal IQ (n = 802)				
Direct effect	−6.67	−6.46	−6.57	−7.2
Indirect effect	−0.83	−1.05	−0.93	−0.33
Percentile 95% CI	−1.53 to −0.26	−1.90 to −0.39	−1.67 to −0.31	−0.86 to 0.015
Bias-corrected 95% CI	−1.58 to −0.29	−1.97 to −0.45	−1.72 to −0.35	−0.95 to −0.02
Indirect/total effect	0.11	0.14	0.12	0.04

Abbreviation: WASI, Wechsler Abbreviated Scale of Intelligence.

[a]Mediation analyses correspond to specifications in eTable 9 in the Supplement. Estimates of the direct and indirect (mediated through influence on structural brain development) effects of low income on a standardized test of achievement are shown. Mean (SD) tests scores are standardized (100 [15]). Standard errors have been bootstrapped. The 95% CIs were constructed using bootstrap resampling with 5000 iterations.