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Timing-specific associations between income-to-needs ratio and hippocampal and amygdala volumes in middle childhood: A preliminary study

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Abstract

It is well known that financial disadvantage is associated with alterations in brain development in regions critical to socioemotional well-being such as the hippocampus and the amygdala. Yet little is known about whether family income at different points in development is differentially associated with these structures. Furthermore, little is known about which environmental factors statistically mediate associations between income and subcortical structure. Using a longitudinal birth cohort and linear mixed-effects models, we identified associations between income-to-needs ratio (INR) at 6 timepoints throughout childhood and hippocampal and amygdala volumes at age 7–9 years (n = 41; 236 INR measurements; 41 brain measurements). Mediation analysis identified environmental sequelae of income that statistically accounted for INR–brain associations. Lower INR prior to age 4 was associated with smaller hippocampal volumes, whereas lower INR prior to age 2 was associated with smaller right amygdala volume. These associations were mediated by unmet basic needs (e.g., food, housing). These findings delineate the temporal specificity of associations between income and hippocampal and amygdala structures.

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AUTHOR CONTRIBUTIONS

B.R. was associated with conception, analysis, and interpretation of data, drafting, and revising the manuscript. D.P. was associated with analysis and interpretation of data, revising the manuscript. J.H. was associated with conception and design, analysis and interpretation of data, and drafting and revising the manuscript. J.D.D. was associated with analysis and interpretation of data and revising the manuscript. K.G.N. was associated with analysis and interpretation of data, and revising the manuscript. A.E.M. was associated with conception and design, analysis and interpretation of data, and revising the manuscript.

CONFLICTS OF INTEREST

The authors report no biomedical financial interests or potential conflicts of interest.

DATA SHARING STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

SUPPORTING INFORMATION

Additional supporting information may be found in the online version of the article at the publisher’s website.

Keywords

amygdala; hippocampus; income; material hardship; sensitive period

1 | INTRODUCTION

Approximately 13 million children in the United States (18%) live in poverty (United States Census Bureau, 2018c). An additional 16 million are low-income (income<200% of the federal poverty line; United States Census Bureau, 2018a). Financial disadvantage is associated with a wide range of poor mental health outcomes including depression, anxiety, and attention-deficit hyperactivity disorder (McLaughlin et al., 2011; Peverill et al., 2020; Sareen et al., 2011; Yoshikawa et al., 2012). One biological pathway by which income may be related to mental illness is through a reduction in hippocampal and amygdala volumes (Barch et al., 2020; Brito& Noble, 2014; Dufford et al., 2020), two subcortical brain regions critical to a wide range of cognitive, social, and emotional abilities (Adolphs, 2010; Eichenbaum, 2004; Hamilton et al., 2008; Videbech & Ravnkilde, 2004). Many studies that have found associations between income and these structures examined income at a single timepoint, whether prospectively measured (Ellwood-Lowe et al., 2018; Hair et al., 2015; McDermott et al., 2019), retrospectively reported (Staff et al., 2012), or measured concurrently with neuroimaging (Dufford et al., 2019; Hanson et al., 2011; Noble et al., 2012; Uban et al., 2020). Furthermore, the sign of income–amygdala associations has been inconsistent (Noble et al., 2012), perhaps due to the unique effects of income timing and chronicity (Hanson&Nacewicz, 2020; Merz et al., 2018). Prospective longitudinal approaches are needed to understand whether there exists a sensitive period (a developmental period of heightened sensitivity) for the association between income and the volumes of these subcortical structures in childhood. Longitudinal studies have shown that early childhood often constitutes a sensitive period for the neural correlates of early life adversity in general (Humphreys et al., 2019; Luby et al., 2016; Lupien et al., 2009). However, little is known about the effects of income at various developmental stages on subcortical volumes. Given the rapid rate of structural brain development during the first years of life (Knickmeyer et al., 2008), this may be a period of heightened vulnerability to the influence of financial disadvantage on the brain.

Also understudied are the environmental factors that mediate income–subcortex associations. In this context, mediators are environmental characteristics that are associated with income and are more temporally proximal to brain volumes. Importantly, statistical mediation does not prove causation, but identifying statistical mediators may inform future, experimental studies on the mechanisms linking income and poor outcomes. Possible mediators between income and subcortical volumes include environmental adversities that might vary with income such as material hardship (unmet basic needs; Mayer & Jencks, 1989), and parental distress (Goosby, 2007).

Using a prospective longitudinal birth cohort, we sought to delineate timing-specific effects of income across the first seven years of life on hippocampal and amygdala volumes at age 7–9 years. We hypothesized: (a) that INR in the first years of life would be more

strongly associated with hippocampal and amygdala volumes in middle childhood than later poverty and (b) that INR-associated environmental adversities would mediate INR-brain associations. Identifying sensitive periods and mediators of INR-subcortex associations would have implications for the timing and types of interventions required to address neural and psychiatric disparities.

2 | MATERIALS AND METHODS

2.1 | Participants

Prenatal recruitment for the Sibling–Hermanos birth cohort (SHBC) began in 2008, and participants were a community sample of pregnant women from the South Bronx, Harlem, and Northern Manhattan who were already part of the Mothers and Newborns birth cohort (started in 1998; Cowell et al., 2017; Perera et al., 2006). Participants were excluded on the basis of prenatal maternal active smoking or if they gave birth before their scheduled third-trimester visit (third-trimester data are critical to the main aims of the SHBC).

Participants in the current sub-study included 53 children aged 7–9 years from the SHBC. Five participants opted against participating in the neuroimaging portion of our study. A trained research assistant visually inspected each T1-weighted image for head motion-related artifacts (e.g., ringing, blurring) and identified seven participants who did not provide artifact-free images. In total, 41 participants provided useable hippocampal and amygdala volumetric data. This study was approved by the institutional review boards at Columbia University and New York State Psychiatric Institute. All children and guardians provided written informed assent and consent, respectively. All research conforms to the recognized standards outlined in the Declaration of Helsinki and the U.S. Federal Policy for the Protection of Human Subjects.

2.2 | Income-to-needs ratio

The income-to-needs ratio (INR) was calculated as mother-reported household income divided by the federal poverty threshold for each participant's household size at child age 6 months and 1, 2, 3, 5, and 7 years (Supporting Information for additional details). INR was also measured at one timepoint during the third trimester. Our primary analyses focused on associations between postnatal INR and subcortical volumes because the putative mechanisms linking postnatal income (direct experience of adversity) to altered brain development are distinct from those of prenatal income (i.e., transplacental conferral of stress hormones, inflammation, and nutrition; Hantsoo et al., 2019; Lefmann & Combs-Orme, 2014; O'Donnell et al., 2009; Ramphal et al., 2020; Spann et al., 2020). Nevertheless, supplementary analyses included the prenatal INR timepoint.

2.3 | Potential mediators linking INR and brain volumes

At each timepoint, mothers participated in a structured interview that included questions about environmental adversities. Potential mediators included material hardship (Mayer & Jencks, 1989) and two measures of maternal psychological distress: demoralization (Dohrenwend et al., 1980) and perceived stress (Cohen et al., 1994). A timeline of data collection (Table S1) is presented in the Supporting Information.

2.4 | MRI acquisition and processing

Neuroimaging data were collected using a 3T GE 750 scanner with a 32-channel head coil at one timepoint between child age 7 and 9 years. Two structural T1 images were collected per participant using a 3D FSPGR sequence (11° flip angle, TE = 2.588 ms, TR = 6.412 ms, 180 slices, 1 mm isotropic resolution). Structural images were visually inspected, and subjects with apparent motion artifacts were excluded. T1 images were anatomically segmented using the recon-all command in FreeSurfer v6.0, segmentations were visually inspected by a trained research assistant for quality assurance, and amygdala and hippocampal volumes were extracted (Fischl et al., 2002, 2004).

2.5 | Statistical analyses

Total (sum of left and right) hippocampal and total amygdala volumes were calculated and residualized for age, sex assigned at birth, and estimated total intracranial volume, given the effects of these variables on regional brain volumes (Koolschijn & Crone, 2013; Pintzka et al., 2015). These residualized volumes were used in all analyses to preserve degrees of freedom and because we did not have hemisphere-specific hypotheses. To test our first hypothesis, we used a linear mixed-effects model with a random effect for participant and tested the interaction between timepoint of INR measurement (age, ranging from 0.5 to 7 years) and hippocampus/amygdala volumes at age 7–9 years on INR. This method of analysis, which permits missing data, tests whether the slope relating brain volumes to INR varies linearly with the timepoint of INR measurement. INR was the dependent variable because it was the repeated measure, while timepoint of INR measurement, brain volumes, and their interaction were the independent variables. Bonferroni correction was applied for two brain regions (α = .025). Post hoc Johnson–Neyman analyses were performed to determine the developmental window during which INR-brain associations were significant. Although this approach of switching the independent and dependent variables is counterintuitive, it allows a parsimonious examination of timing-specific associations between INR and subcortical volumes and has been previously used (Chen et al., 2015).

We also used a complementary approach that preserves intuitive independent and dependent variable assignments and employs generalized estimating equations to simultaneously regress subcortical volumes on INR at multiple timepoints (Supporting Information). To ensure that our findings were due to financial dynamics rather than family compositional changes, we tested our model using raw income bins instead of derived INR values. To examine whether maternal education drove any observed effects, we implemented our main model again using subcortical volumes that were further residualized for years of maternal education.

Supplementary analyses examined how results varied when each nuisance covariate was residualized individually, the effects of prenatal INR on the estimation of INR's sensitive periods, whether results varied by brain hemisphere, and whether the inclusion of timepoint^2 and brain volume × timepoint^2 term improved our models estimating INR's sensitive periods.

To test our second hypothesis, we first identified which environmental adversities (measured at the timepoint after which INR first had an effect) were correlated with INR. Bonferroni correction was applied for four possible mediators (α = .0125). Next, we tested whether these mediated any associations between INR and subcortical volumes. The indirect effect of INR on brain volumes through a mediator was defined as the average causal mediation effect (ACME). The ACME equals the product, *ab*, of the regression coefficient, *a*, relating the independent variable (INR) to the mediator and the regression coefficient, *b*, relating the mediator to the dependent variable (brain volume) with the independent variable as a covariate. Whether the ACME was significantly different from zero was tested using a bootstrapping method implemented in the R *mediation* package with 10,000 resamples (Tingley et al., 2014). Bonferroni correction was applied for two brain regions (α = .025). All analyses were performed using R v4.0.0 (Core Team, 2015), employed pairwise deletion, statistical tests were two-sided, and model diagnostics are reported in the Supporting Information.

3 | RESULTS

3.1 | Participants

Sample characteristics are presented in Table 1, and correlations among all variables of interest in this study are presented in Table S2. Participants were predominantly low-income (INR < 2, i.e., having income less than twice the poverty line for their household size) with some participants being middle-income (2 < INR < 4; Figure S1). While those who were low- or middle- income at age 6 months predominantly remained in the same category at age 7 years (Figure S2), INR did fluctuate between timepoints for all participants.

3.2 | Income-to-needs ratio over the first seven years of life and hippocampal and amygdala volumes

The hippocampus × timepoint (β = −0.079, p = .020), and amygdala × timepoint (β = −0.108, p = .002) interactions were associated with INR (Figure 1, see Figure S3 for simple slopes), indicating that INR–brain associations varied based on the child age at which INR was measured. These results were corroborated with a complementary approach using generalized estimating equations (Table S3). Additionally, when a variable representing income bins (1–10) was used instead of INR, our findings persisted (Table S4). Finally, when subcortical volumes were additionally residualized for years of maternal education, our income findings remained significant (Table S5).

Johnson–Neyman analyses indicated that hippocampal volumes were positively associated with INR when it was measured before age 4.5 years, and amygdala volumes were positively associated with INR when it was measured before age 1.2 years (Figure S4).

In sensitivity analyses, effect sizes were similar to our main analyses when subcortical volumes were residualized for intracranial volume and reduced when not (Table S6). Including prenatal INR in our main model produced a similar pattern of results as the main analysis, though with attenuated effect sizes (Table S7). The bivariate association between prenatal INR and hippocampal volume was significant (r = .33, p = .04), whereas prenatal

INR was not associated with amygdala volume ($r = .19$, $p = .25$). Multiple regression models with subcortical volumes as the dependent variable and both prenatal and 6-month INR as predictors reveal larger effect sizes for 6 month INR than prenatal INR (hippocampus: $\beta_{\text{prenatal}} = 0.16$, $\beta_{\text{6-month}} = 0.37$; amygdala: $\beta_{\text{prenatal}} = 0.01$, $\beta_{\text{6-month}} = 0.35$).

In supplementary analyses, there was no evidence for hemisphere-specific effects (volume × timepoint × hemisphere effect p-values > .5). Including additional terms for timepoint^2 and timepoint^2 × volume terms did not improve model fit (hippocampus: $\chi^2(2) = 2.0$, $p = .73$; amygdala: $\chi^2(2) = 2.6$, $p = .62$).

3.3 | Material hardship mediates INR-subcortex associations

Income-to-needs ratio at age 6 months, the timepoint at which INR was most strongly associated with hippocampal and amygdala volumes at age 7–9 years, was used for subsequent analyses examining the mediating role of later stressors. INR at age 6 months was not correlated with maternal demoralization at age 1 ($r = -.02$, $p = .90$), or maternal perceived stress at age 1 ($r = .02$, $p = .89$); however, 6-month INR was negatively correlated with material hardship at child age 1 year ($r = -.41$, $p = .009$, Table S2). This time-staggered approach best described the INR-material hardship association (Table S8).

Material hardship at age 1 year was negatively correlated with both hippocampal ($r = -.59$, $p < .001$) and amygdala ($r = -.5$, $p = .001$) volumes. In multiple regression specificity analyses, material hardship at age 1-year was negatively associated with subcortical volumes, while material hardship in the third trimester and at age 5 years were not (Table S9).

The indirect effect of INR at 6 months on brain volumes through material hardship at age 1 year was significant for the hippocampus (average causal mediation effect (ACME) = 0.20, $p = .009$; proportion mediated = 0.49, $p = .02$), as well as the amygdala (ACME = 0.18, $p = .008$; proportion mediated = 0.60, $p = 0.15$).

4 | DISCUSSION

4.1 | Summary of results

In the current study, which is preliminary due to its small sample size, we demonstrate that positive associations between INR and hippocampal and amygdala volumes depend on when INR is measured. Specifically, we find that INR prior to age 4.5 years may be most critical for hippocampal volumes in middle childhood and prior to age 1.2 years may be most critical for amygdala volumes. We further show that material hardship mediates INR–brain associations.

4.2 | Sensitive periods

The hippocampus and the amygdala are two brain regions commonly implicated as vulnerable to the effects of financial disadvantage, yet most previous studies on this topic measure income at a single timepoint (Dufford et al., 2019; Ellwood-Lowe et al., 2018; Hair et al., 2015; Hanson et al., 2011; McDermott et al., 2019; Noble et al., 2012; Staff et al., 2012; Uban et al., 2020). By leveraging a predominantly low-income longitudinal

birth cohort with frequent income measurements, we demonstrate that earlier income is more associated with subcortical volumes than later income. Although very few participants moved from low- to middle-income or vice versa, our study was nevertheless able to detect differential effects between earlier and later INR. This suggests that the development of the hippocampus and the amygdala may be highly sensitive to small variation in income in this low-income sample. Indeed, small fluctuations in income can have substantive effects on a family's ability to meet their needs in low-income contexts (Brownell et al., 2018; Rojas et al., 2020). Consistent with the idea that income variation can be impactful for low-income households, Noble and colleagues have previously shown that the association between income and cortical surface area is most pronounced in low-income children (Noble et al., 2015).

Our finding that early income is most related to brain volumes in middle childhood is likely due to the rapid rate of brain growth during the first few years of life (Knickmeyer et al., 2008). One study showed that prior to age five represents a sensitive period for associations between early life stressful events and hippocampal volumes (Humphreys et al., 2019). We extend this finding to income, to the amygdala, and provide further temporal specificity. Convergent with our findings, longitudinal studies have shown that INR prior to age five is more detrimental to physical (Kalil et al., 2016) and mental health (Mazza et al., 2017; McFarland, 2017) than later INR. Furthermore, it has been shown that adversity prior to age 3 years may constitute a sensitive period for DNA methylation (Dunn et al., 2019), a mechanism by which early experience may confer its effects on hippocampal volumes (Davis et al., 2017; Jia et al., 2019).

The sensitive period identified for the amygdala was more circumscribed than that of the hippocampus, potentially due to the earlier development of the amygdala's stress response (Tottenham & Sheridan, 2010). Animal evidence suggests that by early postnatal life, the amygdala exhibits increased corticotropin-releasing hormone mRNA production following a stressor (Avishai-Eliner et al., 1996), while this phenomenon does not emerge in the hippocampus until substantially later (Fenoglio et al., 2004). In humans, the amygdala volume increases by approximately 105% in the first year of life, while the hippocampus grows by 84% (Gilmore et al., 2012). Thus, the relatively protracted functional and structural development of the hippocampus may explain its wider sensitive period.

Interestingly, although the fetal brain triples in size during the third trimester (Bouyssi-Kobar et al., 2016), the effects of income and material hardship in this time period were minimal. Because any influence of prenatal financial disadvantage on fetal development must be transmitted in the form of glucocorticoids, cytokines, toxicants, nutrition, or other transplacental molecules, any associations between prenatal income and brain outcomes are necessarily limited by the extent to which income is associated with these systemic effects (Gilman et al., 2017; Keenan-Devlin et al., 2017; Miller et al., 2017). Because our sample was predominantly low-income, it is possible that we did not have a sufficient range in prenatal income to detect effects mediated by these transplacental factors. Another possibility is that the contributions of prenatal INR to subcortical volumes in childhood are more complex, whether by exerting effects that are conditional on later disadvantage (O'Donnell & Meaney, 2017) or by exerting opposite effects (Qiu et al., 2013; Rifkin-Graboi

et al., 2018). Future studies with larger samples and greater income ranges may be able to query the influences of prenatal income on brain outcomes via these mechanisms.

4.3 | Material hardship

Material hardship mediated the effects of INR on hippocampal and amygdala volumes. The specific resources queried by our material hardship questionnaire included food, shelter, utilities, clothing, and healthcare. Indeed, when these needs are unmet, children experience more anxiety, depression, inattention, and hyperactivity, and parents face higher rates of mental illness (Knowles et al., 2016; Loukas & Prelow, 2004; Melchior et al., 2009, 2012; Slopen et al., 2010; Whitaker et al., 2006; Zilanawala & Pilkauskas, 2012). Few studies have examined the mediators through which income exerts its effects on hippocampal and amygdala volumes. One study found that harsh parenting mediated the association between income and reduced hippocampal but not amygdala volumes in a sample enriched for preschool depression (Luby et al., 2013). Though we did not examine parenting style, we considered two measures of maternal psychological distress: demoralization and perceived stress. Neither of these was significantly associated with concurrent or antecedent INR or material hardship. One possibility is that material hardship adversely affects parenting in a manner that is not captured by our distress variables. Another possibility is that unmet needs directly affect brain development, given the moderate correlations between material hardship and hippocampal and amygdala volumes. Indeed, animal models have shown that these brain regions are particularly vulnerable to poor nutrition (Coupé et al., 2009; Escobar & Salas, 1993; Hoeijmakers et al., 2015; Pravosudov et al., 2005) and unpredictable stress (Alfarez et al., 2003; Kosten et al., 2008).

4.4 | Limitations

The current study should be interpreted as preliminary given our limited sample size. For example, prior studies suggest that associations between subcortical volumes and mental health (Videbech & Ravnkilde, 2004) and between subcortical volumes and stress (McLaughlin et al., 2019; Wang et al., 2019) are hemisphere- or subregion-specific. Although we tested volume × timepoint × hemisphere interactions, these analyses had limited power to detect potentially extant hemisphere-specific associations between INR and amygdala and hippocampal volumes. Future studies with larger sample sizes are needed to corroborate these findings.

Furthermore, additional factors across development likely contribute to and moderate the association between income-to-needs ratio and subcortical volumes, such as environmental toxicant burden or preterm birth which are known to vary based on socioeconomic factors (Cureton, 2011) or paternal distress, which was not collected in our study. Additionally, it is possible that INR-brain associations may be mediated by different environmental factors at different points in development. Future studies with longitudinal MRI designs are needed to investigate whether the associations demonstrated here persist into adolescence and adulthood.

4.5 | Opportunities for intervention: timing and type

The current study, though limited due to its small sample size, highlights several potential timepoints and modalities for intervention against the neural consequences of financial disadvantage. By identifying sensitive periods for the INR–subcortical volume associations, we suggest that very early life may be a particularly critical time for beginning anti-poverty measures. That these effects are mediated by material hardship provides evidence that guaranteed access to food, housing, utilities, clothing, and healthcare may be beneficial to child brain development. This notion may perhaps seem unsurprising. However, 45% of households are rent-burdened (United States Census Bureau, 2019) and over one million children in the United States are homeless (National Center for Homeless Education, 2020). Four million children are medically uninsured with lower-income families facing lower rates of insurance (United States Census Bureau, 2018b, 2018d); Yet, quasi-experimental human studies have shown that income supplementation can be beneficial for child mental (Akee et al., 2015; Costello et al., 2003) and physical health (Hoynes et al., 2016).

Interventional studies are needed to more directly establish causality among the longitudinal associations we have demonstrated between income, material hardship, and brain structure (Luby et al., 2020). Nevertheless, in the absence of such studies, we have ample evidence that financial disadvantage is associated with child cognition, emotional wellbeing, and brain development (Farah, 2017; McLaughlin et al., 2011; Sareen et al., 2011; Yoshikawa et al., 2012). Furthermore, while individual-level intervention may be useful, it is equally important not to construe these disparities as individual-level phenomena. Thus, individual-level approaches must be coupled with policy interventions against the distal, clandestine, structural factors that permit and recapitulate neural and psychiatric disparities (Lende, 2012; Link & Phelan, 1995; Macvarish et al., 2014; Phelan et al., 2010). Resource inequality is rampant and expanding in the context of the COVID-19 pandemic (Coughlin et al., 2020; Dunn et al., 2020; Kinsey et al., 2020; Patrick et al., 2020). Yet, small experiments involving resource redistribution have produced powerful results for children (Akee et al., 2015; Costello et al., 2003). The adoption of alternative, equitable economic norms would necessarily be more far-reaching than ad-hoc individual-based intervention and perhaps more effective at alleviating health disparities more broadly (Link & Phelan, 1995; Phelan et al., 2010).

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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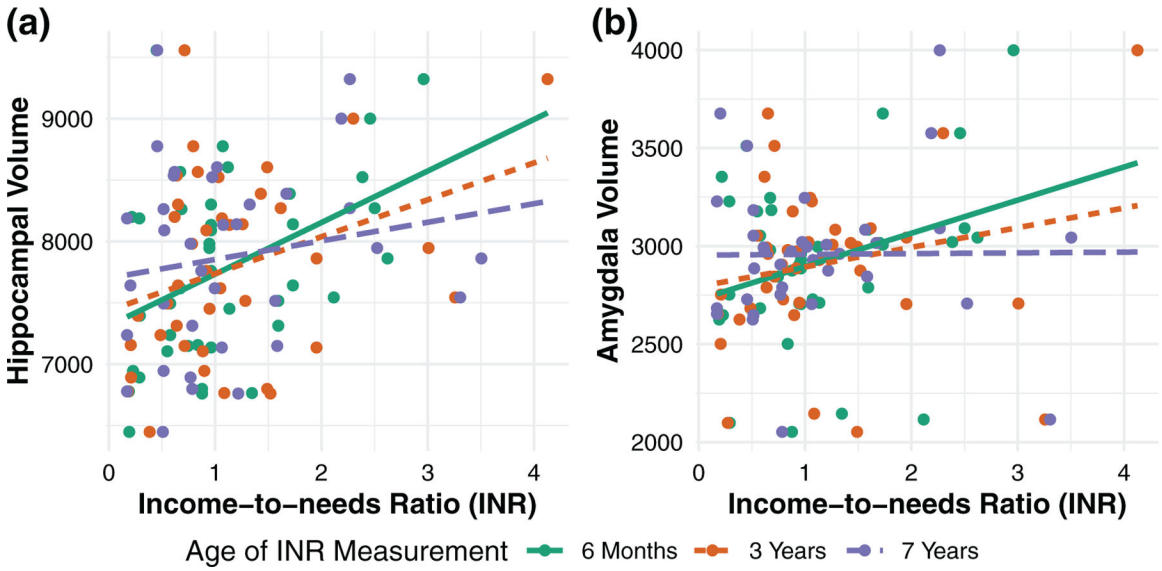

FIGURE 1.

The association between income-to-needs ratio (INR) and residualized subcortical volumes (mm^2) depends on when INR is measured. Earlier INR is more strongly associated with hippocampal and amygdala volumes than later INR

TABLE 1

Sample characteristics

N = 41	Mean (SD)/N (%)	Range
Sex assigned at birth (Female)	24 (59%)	Female/Male
Age (Years)	8.6 (0.7)	7.0–9.8
Income-to-needs ratio		
Prenatal	1.4 (1.0)	0.2–4.3
6 months	1.1 (0.7)	0.2–3.0
1 year	1.2 (0.8)	0.2–3.4
2 years	1.2 (0.8)	0.2–1.6
3 years	1.1 (0.8)	0.2–4.1
5 years	1.1 (0.7)	0.2–4.0
7 years	1.1 (0.7)	0.2–3.5
Across all timepoints	1.2 (0.8)	0.2–4.3
Self-reported ethnicity		
African-American	19 (46%)	African-American/Hispanic
Maternal education (in third trimester)		
No high school or equivalency	12 (29%)	
High school or equivalency	8 (20%)	
Some college	11(27%)	
Associate’s degree	6 (15%)	
Bachelor’s degree	3 (7%)	
Graduate degree	1 (2%)	