

The role of socioeconomic status in shaping associations between sensory association cortex and prefrontal structure and implications for executive function

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ABSTRACT

Socioeconomic status (SES) is associated with widespread differences in structure of temporal, parietal, occipital, and frontal cortices. Development of sensory processing regions—in particular visual association cortex (VAC) and auditory association cortex (AAC)—may scaffold development of the prefrontal cortex (PFC). Experiences that correlate with SES like cognitive stimulation and language may influence VAC and AAC development, in turn allowing the PFC to resolve conflicts between similar stimuli. SES-related differences in these regions may partly explain differences in executive function (EF) skills. Here, we use structural equation modeling of longitudinal data from the Adolescent Brain and Cognitive Development study to test the hypothesis that SES-related differences in AAC and VAC are associated with differences in structure of the PFC and development of the PFC over time, which in turn are associated with development of EF. We found partial support for this model, demonstrating that SES-related differences in PFC structure are mediated by differences in sensory cortex structure, and that SES-related differences in sensory cortex structure mediate the association between SES and EF. These findings highlight the role sensory processing regions play in SES-related differences in PFC development. Future studies should explore proximal environmental factors driving SES-related differences to inform interventions.

1. Introduction

Socioeconomic status (SES), which refers to access to financial and non-financial home and neighborhood-level resources, has been linked to differences in brain structure during childhood and adolescence (Rakesh, Whittle, et al., 2023; Rakesh and Whittle, 2021). Studies have found widespread associations between SES with both cortical surface area and thickness such that children from lower SES households show thinner cortex and smaller surface area, extending throughout much of the temporal, parietal, occipital, and frontal cortex (Jednorög et al., 2012; Mackey et al., 2015; Noble et al., 2015; Rakesh et al., 2022; Rakesh and Whittle, 2021). These differences in brain structure have been suggested to underlie the well-established socioeconomic disparities in children's cognitive outcomes, including executive function (EF) and language (Machlin et al., 2019; Merz et al., 2019; Rosen et al., 2018; Tomasi and Volkow, 2021). Interestingly, socioeconomic disparities

have been found in structure of the brain that supports executive function and language (e.g. prefrontal and temporal cortices), but also in areas that support more basic sensory processing (e.g., visual association cortex; Mackey et al., 2015; Noble et al., 2015; Piccolo et al., 2016; Rakesh et al., 2022). Our group has previously proposed that these SES-related differences in the development of sensory processing regions (including cortical thickness and surface area as well as functional recruitment) may be related to differences in higher-order regions, which in turn contribute to cognitive development (Rosen et al., 2019). However, to date, studies investigating the link between SES, brain structure, and cognitive outcomes have been limited in their theoretical focus on specific neurobiological systems, and by small cross-sectional samples. This study addresses these gaps by leveraging a large longitudinal dataset to test the possible mediating role of sensory processing regions in SES-related differences in PFC and cognitive development.

SES is associated with widespread differences in brain structure

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across both frontal and sensorimotor regions (Rakesh and Whittle, 2021). While studies have found SES-related differences in frontoparietal network structure and function, robust differences in the structure and function of visual association cortex as a function of SES have also been consistently found. Specifically, lower SES is associated with thinner cortex and reduced surface area in ventral occipital temporal cortex which contains several important visual processing regions and less recruitment of these regions during complex cognitive tasks, including working memory (Finn et al., 2017; Mackey et al., 2015; Noble et al., 2015; Rosen et al., 2018). However, these SES-related differences in visual processing regions have rarely been interpreted or linked to behavioral outcomes (Rakesh and Whittle, 2021), with the exception of work by this group (Lurie et al., 2024; Rosen et al., 2018, 2021). These sensory processing regions develop earlier in life than the PFC (Gogtay et al., 2004; Sowell et al., 2004a), has feed-forward projections to PFC, and receives input through feedback connections from PFC (Gilbert and Li, 2013). Based on these findings, we and others have proposed a theoretical framework that suggests that sensory processing regions—in particular visual association cortex—precedes and scaffolds development of the PFC (Amso and Scerif, 2015; Rosen et al., 2019).

We suggest that experiences that vary with SES, including cognitive stimulation mediated by caregivers, may influence development of the visual system. In the model, we propose that experiences early in development where a caregiver is present and can for example, guide the child's attention to unique features of an object along with an object label (e.g. "That cow has black spots. She's bigger than the sheep. The sheep is fluffy") may impact development of category-preferential visual processing regions. Experiences such as these provide the PFC with initial opportunities to resolve conflicts between similar stimuli (in this example, distinguishing between two farm animals; Rosen et al., 2019). These experiences and other forms of cognitive stimulation are not absent in low-income families. However, they tend to be more prevalent in higher income families largely due to the constraints on time, resources, and energy of low-income families (see Rakesh, McLaughlin, et al., 2024 for a review).

Importantly, the visual association cortex, including areas of the ventral visual stream, have reciprocal connections with the lateral pre-frontal cortex (Gilbert and Li, 2013; Kravitz et al., 2013), thus creating a biologically plausible pathway for lower-order sensory regions to influence PFC development. We have also proposed that SES-related differences in these regions may in part explain SES-related differences in EF skills including working memory, inhibitory control, and cognitive flexibility (Rosen et al., 2019). Indeed, recent studies testing parts of this model have found that functional recruitment of both the frontoparietal network and visual association cortex is positively associated with SES and cognitive performance (Lurie et al., 2021; Rosen et al., 2018, 2021). Together, these findings suggest that the development of the visual association cortex may play a crucial role in explaining socioeconomic disparities in cognitive function. However, these studies have been limited by small sample sizes and cross-sectional designs. Recent work emphasizes the importance of modeling structural changes over time and suggests that longitudinal data is necessary to test mechanisms driving these socioeconomic differences (Rakesh, Whittle, et al., 2023). Further, the conceptual model proposing that sensory processing regions may scaffold development of the PFC has focused on visual processing regions, in part because many functional MRI studies have used visual tasks (e.g., Lurie et al., 2021; Rosen et al., 2021). However, it is also possible that differences in development of *other* sensory association regions are also important for the development of the PFC. Indeed, several studies have found that SES is associated with differences in the structure and function of the auditory association cortex (Jednoróg et al., 2012; Mackey et al., 2015; Merz et al., 2019; Noble et al., 2015; Rakesh and Whittle, 2021). Moreover, these differences may underlie the profound SES-related differences in language development (Romeo, 2019). The auditory association cortex may also be critically involved in SES-related disparities in cognitive outcomes, given that language

development is important for the development of EF (Romeo et al., 2022).

In the present study, we address the limitations of previous work by leveraging the Adolescent Brain and Cognitive Development (ABCD) Study to investigate how SES is related to differences in development of sensory association cortices (auditory and visual) and how these differences may explain SES related differences in structure of the PFC as well as change in PFC development over time. Furthermore, we investigate whether these neural differences explain the well-documented SES-related differences in EF (Lawson et al., 2018; Rakesh, Lee, et al., 2024). We use structural equation modeling to test the overall hypothesis that SES is associated with differences in structure (i.e. cortical thickness and surface area) of visual and auditory association cortices, which in turn explain development of the PFC, ultimately contributing to SES-related differences in EF development.

2. Methods

Our hypotheses, methods and analytic plan were preregistered on Open Science Framework: <https://osf.io/quy2n/>.

2.1. Participants

We leveraged data from the ongoing Adolescent Brain Cognitive Development (ABCD) study. The ABCD study recruited over 11,500 children aged 9–10 years across 21 sites in the US with the goal to comprehensively characterize psychological and neurobiological development from late childhood to early adulthood. The 21 ABCD study sites consist of universities and research institutes selected via a competitive grant application process. Metropolitan areas within their reach were chosen based on demographic criteria resembling those of the entire US population. These sites encompass around 20 % of 9- to 10-year-olds nationwide. Within these locations, public, public charter, and private schools within a 50-mile radius of the data collection site were identified, and participant recruitment schools were chosen randomly. Participants underwent comprehensive neuroimaging and neurocognitive assessments. All parents or caregivers provided written informed consent, and all children provided assent. Participant rights were safeguarded by local institutional review boards. Following exclusion based on imaging quality control, missing all three SES measures at both time points, missing Flanker task at both time points, and/or missing usable brain structure data at both time points., the final sample for the primary analysis comprised $n = 11,566$ children (47.95 % assigned female at birth, 52.05 % assigned male at birth; see Table 1 for

Table 1
Descriptive Statistics.

	Mean	SD	Max
Parent EDU	16.39	2.69	21
Income-to-Needs Ratio	3.72	2.45	12.32
ADI	39.95	26.89	100
Age T1 (months)	118.98	7.49	133
Age T2 (months)	143.21	7.76	166
Months between T1 and T2	24.28	2.32	41
Flanker T1	95.46	13.64	171
Flanker T2	96.57	14.31	146
VAC Thickness T1	2.70	0.10	3.10
VAC Thickness T2	2.66	0.10	3.05
AAC Thickness T1	2.90	0.12	3.38
AAC Thickness T2	2.87	0.12	3.33
PFC Thickness T1	2.75	0.09	3.10
PFC Thickness T2	2.72	0.09	3.07
VAC Surface Area T1	12669	1489	18408
VAC Surface Area T2	12693	1491	19019
AAC Surface Area T1	18514	2232	30586
AAC Surface Area T2	18516	2233	30095
PFC Surface Area T1	21314	2610	32267
PFC Surface Area T2	21469	2614	32383

additional demographic information).

2.2. Measures

2.2.1. Socioeconomic status

A SES composite was created by standardizing and averaging parent education, income-to-needs ratio, and reversed ADI (described below) measured at T1. Participants needed at least one SES measure to be included in the analyses. Sensitivity analyses were also conducted separating household and neighborhood SES measures (see Supplemental; Materials).

2.2.1.1. Household SES. The mean educational attainment of parents or caregivers (in years) was calculated based on the educational levels of both individuals or from data for one when information for both was unavailable. The income-to-needs ratio was determined by dividing the median value of the income band by the federal poverty line corresponding to the household size. A value of 1 indicates that the household is at the poverty threshold, with values above or below 1 signifying being above or below the threshold, respectively.

2.2.1.2. Neighborhood SES. We used a composite measure of neighborhood disadvantage—the area deprivation index (ADI), assessed at the census tract level using the participant's primary residential address. The ADI is a composite measure based on data from 17 different factors, including neighborhood-level income, education, and employment. Neighborhoods are ranked on a national percentile scale where higher values indicate greater disadvantage. The ADI was reverse-scored before being included in the SES composite.

2.2.2. Executive function

The ABCD cognitive battery at baseline consisted of seven tasks from the NIH Toolbox (Picture Vocabulary Test, Oral Reading Recognition Test, Flanker Task, List Sorting working Memory Test, Dimensional Change Card Sort, Pattern Comparison Processing Speed Test, and the Picture Sequence Memory Test). However, not all tasks were administered at subsequent time points. Notably, the List Sorting Working Memory and Dimensional Card Sort Task were omitted from follow-up assessments. Consequently, we opted to utilize the only EF assessment that was conducted both at baseline and during the 2-year follow-up: the Flanker Task, which measures cognitive control/attention (Luciana et al., 2018). Performance on the Flanker is measured by the difference in accuracy and reaction times for incongruent versus congruent trials. A total computed score indexes accuracy only if $\leq 80\%$ of trials were answered correctly. We use the total “age corrected” score, which accounts for age differences amongst all participants in the sample based on a normative sample (Casaletto et al., 2015). Participants needed at least one valid Flanker score (i.e., at either time 1 or time 2) to be included in the analyses. Scores were standardized across both time points to ensure that growth was represented in the same units.

2.3. Imaging acquisition, preprocessing, and quality control

Neuroimaging procedures were conducted with standardized protocols across all sites, utilizing either a 3 T Siemens, Phillips, or General Electric magnetic resonance imaging (MRI) scanner equipped with a 32-channel head coil. A 3-dimensional T1-weighted image with a voxel resolution of 1 mm was acquired for each participant. The preprocessing steps were carried out by the ABCD Data Analysis, Informatics & Resource Center using a uniform pipeline (for comprehensive details and quality control procedures, refer to Hagler et al., 2019). Real-time motion detection and correction software programs were utilized at Siemens and GE sites. Both manual and automatic techniques were employed to assess data quality, with trained professionals examining all images for artifacts and abnormalities. The extent of artifact in the

cortical reconstruction of post-processed images was assessed on a scale from 0 to 3, with 3 indicating the highest level. Based on these ratings, technicians provided recommendations regarding usability (only images with a rating of 0 were included in this study). Automated processes were employed to calculate signal-to-noise ratio and head motion measurements. Participants that met the ABCD recommended inclusion criteria for scans at either the first or second time point were included in our analyses.

FreeSurfer (version 7.1.1) was used for cortical surface reconstruction and parcellation. We extracted the estimated cortical thickness and surface area of the following bilateral regions of interest (ROIs) using the Destrieux atlas: VAC (middle occipito-temporal gyrus, lateral occipito-temporal gyrus, anterior occipital sulcus, middle occipital-temporal sulcus and lingual gyrus), AAC (supramarginal gyrus, lateral aspect of superior temporal gyrus, planum temporale, superior temporal sulcus, transverse temporal sulcus), and PFC (middle anterior cingulate gyrus and sulcus, triangular part of the inferior frontal gyrus, middle frontal gyrus, inferior frontal sulcus, middle frontal sulcus, superior frontal sulcus). Surface area values for parcels were summed to create composites for the AAC, VAC, and PFC, while cortical thickness values were averaged. Because there were no predictions of lateralized effects, values for the right and left hemispheres were combined for analyses. Structural measures for each of the three ROIs were standardized based on data from both time points to ensure that growth was represented in the same units.

2.4. Final sample inclusion and comparison to full ABCD sample

The full, original ABCD sample includes 11,878 participants. Participants were excluded from the present analytical sample if they were missing all three SES variables ($n = 2$), EF flanker measures at both time points ($n = 69$), and/or usable brain structure measures at both time points ($n = 213$, though $n = 1$ had already been excluded for missing SES, and $n = 1$ had already been excluded for missing EF), leaving a final analytical sample of $n = 11,566$. The analytical sample did not significantly differ from the full sample on any measures (all $p > .6$). However, within the included sample, data was not missing completely at random (15 missing patterns observed). Specifically, participants with usable structural MRI data versus those with missing or poor quality structural MRI data tended to be female (T1: $p = \text{n.s.}$, T2: $p < .001$), older (T1: $p < .001$, T2: $p < .001$), and have higher SES (T1: $p = .035$, T2: $p = .005$), while participants with usable EF data versus those without tended to also have higher SES (T1: $p = \text{n.s.}$, T2: $p < .001$) but be younger (T1: $p = \text{n.s.}$, T2: $p < .001$) at time point 2 only. Additionally, within the inclusion sample, participants who completed the first but not the second time point (as indexed by missing all brain and EF data at time 2) tended to be female ($p = .029$), have lower SES ($p < .001$), and have lower time 1 EF scores ($p < .001$). A sensitivity analysis including all participants, no matter missing data, is included in the supplement for completeness.

2.5. Structural equation model

Two separate structural equation models (one for thickness, one for surface area) were estimated using *lavaan* v0.6.17 (R v4.4.0) using full information maximum likelihood (FIML) for missing data (Li et al., 2024) and allowing exogenous covariates to be estimated (fixed. $x = \text{FALSE}$) to avoid listwise exclusion. First, latent growth curve models were created using T1 and T2 VAC, AAC, PFC, and EF measures, yielding a latent intercept and slope for each (in interpreting results, we refer to the latent intercepts as “baseline” for simplicity). The intercept and slope within each growth model were allowed to freely covary, as were the sensory (VAC, AAC) intercepts and slopes, since they conceptually enter the model at the same level. Negative observed variances for all T1 and T2 indicators were constrained to zero. Then, all direct and indirect effects from SES → sensory regions → PFC → EF were modeled,

excluding the effects of upstream slopes to downstream intercepts (i.e. VAC/AAC slopes predicting PFC intercepts; PFC slope predicting EF intercept—these covariances were constrained to zero). Thus, the following direct effects were modeled: (1) the effect of SES on both sensory region intercepts and slopes, PFC intercept and slope, and EF intercept and slope, controlling for sex, age at T1 scan, age difference between T1 and T2 scans (for slopes only) and whether there was a change in scanner from T1 to T2 (for slopes only); (2) the effect of sensory region intercepts on PFC and EF intercepts and slopes, and of sensory region slopes on PFC and EF slopes; and (3) the effect of PFC intercept on EF intercept and slope, and of PFC slope on EF slope. All possible indirect paths (partial and full) were estimated using 95 % bias-corrected bootstrap confidence intervals based on 10,000 replications. Fig. 1 shows the full tested model.

2.6. Deviations from preregistration

The current analysis plan was preregistered on Open Science Framework. We note some deviations from our planned analysis. First, in the original plan we included two cognitive measures under the umbrella of executive function including the Flanker task, which we still use, and the processing speed task, which we have removed from the analysis. EF is typically defined as working memory, inhibitory control, and cognitive flexibility and does not traditionally include processing speed. We have thus removed it from the analysis. This choice impacted EF as a variable making it a manifest variable rather than a latent factor. We include the analysis with processing speed in the [Supplemental Materials](#) and note that the findings are largely the same across the analyses.

Second, we preregistered using the Flanker score that was uncorrected for age. But because we control for age at scan rather than age at the Flanker, we believe the more correct analysis should use the age-corrected Flanker score and thus include that score in the analysis.

Finally, our original preregistration noted that we would represent EF as factors representing T1 and T2 separately. However, to be consistent with the rest of the model, in the analysis presented here, we represent EF as a growth model with intercept and slope. Critically, all of these choices were made for theoretical reasons and not in response to the results of the analysis.

3. Results

3.1. Sample demographics

Demographic information and descriptive statistics for the included sample has been provided in [Table 1](#).

3.2. Model Fit

Fit indices were excellent for both the cortical thickness ($CFI = .998$, $TLI = .991$, $RMSEA = .024$, $SRMR = .013$) and surface area models ($CFI = 1.000$, $TLI = .998$, $RMSEA = .016$, $SRMR = .006$). For direct effects, we report the unstandardized parameter estimates (B), the fully standardized (latent and observed) estimates (β) and p -values. For indirect effects, we additionally report 95 % bias-corrected bootstrap confidence intervals. [Fig. 2](#) highlights the results of the models.

3.3. Associations of the structure of sensory association regions with PFC development

Before describing associations between SES, EF, and brain development, we first interpret the “center” of the model, or the direct effects of sensory association development (VAC, AAC) on PFC development. Results were consistent for both cortical thickness and surface area. The intercept of each sensory region was significantly positively associated with the PFC intercept (VAC thickness: $B = .296$, $\beta = .297$, $p < .001$; VAC surface area: $B = .278$, $\beta = .278$, $p < .001$; AAC thickness: $B = .398$, $\beta = .409$, $p < .001$; AAC surface area: $B = .501$, $\beta = .502$, $p < .001$). Additionally, the slope of each sensory region was significantly positively associated with the PFC slope (VAC thickness: $B = .267$, $\beta = .228$, $p < .001$; VAC surface area: $B = .224$, $\beta = .153$, $p < .001$; AAC thickness: $B = .556$, $\beta = .449$, $p < .001$; AAC surface area: $B = .275$, $\beta = .211$, $p < .001$). For neither thickness nor surface area was any sensory intercept directly associated with the PFC slope. Together, this indicates that baseline structural measures of sensory association and prefrontal cortices are positively related, and changes over time in these regions are also positively correlated. However, baseline sensory cortex structure does not predict change in PFC structure over time.

3.4. SES and cortical development

After controlling for participant age and sex, SES was significantly associated with the intercept for VAC thickness ($B = .203$, $\beta = .172$, $p < .001$) and surface area ($B = .208$, $\beta = .174$, $p < .001$), AAC thickness ($B = .096$, $\beta = .080$, $p < .001$) and surface area ($B = .213$, $\beta = .178$, $p < .001$), and PFC surface area ($B = .048$, $\beta = .040$, $p < .001$). This indicates that higher SES is associated with greater baseline thickness and surface area in both sensory association regions, as well as higher baseline surface area in the PFC. Additionally accounting for the age difference between T1 and T2 and whether there was a change in scanner, SES was also associated with the slopes of VAC thickness ($B = .021$, $\beta = .032$, $p = .040$), AAC thickness ($B = .023$, $\beta = .037$, $p = .003$) and surface area ($B = .010$, $\beta = .034$, $p = .008$), and PFC thickness ($B = .018$, $\beta = .023$, $p = .019$). This indicates that higher SES is positively correlated with change in cortical thickness and cortical surface area over time in both sensory regions, and change in PFC cortical thickness

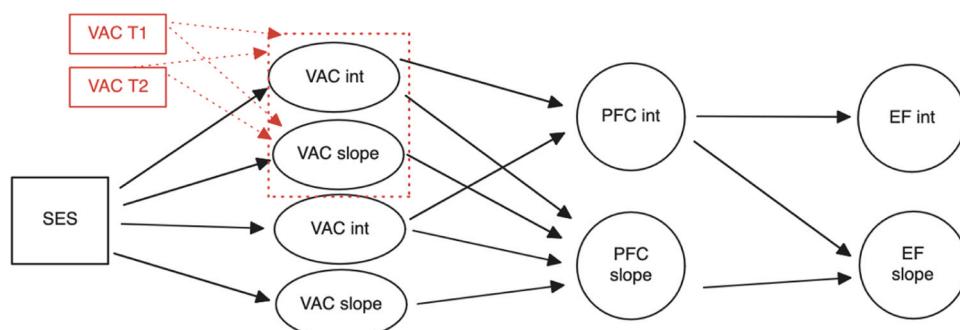


Fig. 1. A visual representation of the tested model. SES represents the composite scores described above. Values from T1 and T2 contribute to the intercept and slope values in all cases, demonstrated for the VAC as an example in red with dashed lines. Covariances, variances and residuals were modeled but are not drawn. AAC = auditory association cortex, int = intercept, SES = socioeconomic status, VAC = visual association cortex.

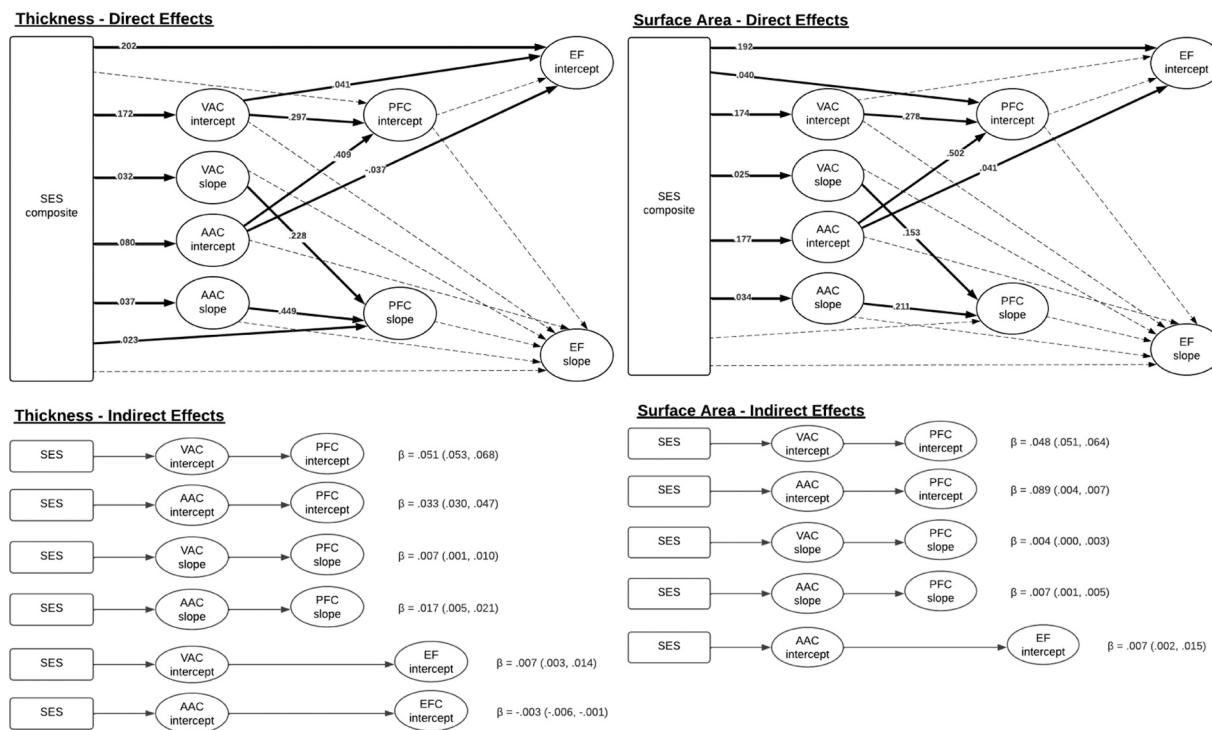


Fig. 2. SEM model results. In the depiction of direct effects (top), thick lines represent significant paths (values represent standardized coefficients), and dotted lines represent non-significant paths. Only significant indirect effects are shown (bottom), and values represent standardized coefficients and their bootstrapped 95 % confidence intervals.

over time.

In terms of indirect paths (Fig. 2, bottom) from SES to PFC through sensory association cortices, all intercept-only paths were significant. For thickness: SES → VAC intercept → PFC intercept ($B = .060$, $\beta = .051$, $p < .001$, 95 %CI = [.053,.067]) and SES → AA intercept → PFC intercept ($B = .038$, $\beta = .003$, $p < .001$, 95 % CI = [.029,.047]); for surface area: SES → VAC intercept → PFC intercept ($B = .058$, $\beta = .049$, $p < .001$, 95 %CI = [.052,.064]) and SES → AAC intercept → PFC intercept ($B = .107$, $\beta = .089$, $p < .001$, 95 %CI [.096,.117]).

Additionally, all slope-only paths were significant: For thickness: SES → VAC slope → PFC slope ($B = .006$, $\beta = .007$, $p = .013$, 95 %CI = [.001,.010]) and SES → AAC slope → PFC slope ($B = .013$, $\beta = .017$, $p = .003$, 95 %CI = [.005,.021]); for surface area: SES → VAC slope → PFC slope ($B = .001$, $\beta = .004$, $p = .045$, 95 %CI = [.000,.003]) and SES → AAC slope → PFC slope ($B = .003$, $\beta = .007$, $p = .009$, 95 %CI = [.001,.005]). No models in which sensory intercepts mediated PFC slopes were significant. Together, these results indicate that the baseline structure of sensory association cortex mediates the relationship between SES and the baseline structure of the PFC, and the change in both sensory association cortices mediates the association of SES with change in PFC cortical structure.

3.5. Cortical development and executive functioning

Of the direct paths between cortical structure and EF, only sensory cortex intercepts were significantly or marginally associated with the EF intercept. Specifically, the intercept of each sensory region was significantly associated with the EF intercept (VAC thickness: $B = .042$, $\beta = .041$, $p = .002$; VAC surface area: $B = .023$, $\beta = .023$, $p = .090$; AAC thickness: $B = -.037$, $\beta = -.037$, $p = .007$; AAC surface area: $B = .041$, $\beta = .041$, $p = .006$). No direct associations with EF slope were significant. This indicates that greater baseline VAC thickness and AAC surface area is associated with greater baseline EF, but interestingly, lower baseline AAC thickness was associated with greater baseline EF.

Contrary to predictions, there were no significant direct associations

between any PFC variable (intercept/slope, thickness/surface area) and either the EF intercept or slope. Additionally, there were no significant indirect pathways from any sensory cortex variable through PFC variables to EF variables.

3.6. SES, cortical development, and executive functioning

Across both models, SES was significantly directly associated with the EF intercept (thickness: $B = .243$, $\beta = .203$, $p < .001$; surface area: $B = .231$, $\beta = .193$, $p < .001$), but not the EF slope. This indicates that SES is associated with baseline EF measures, but not EF change during this time period.

For indirect relationships between SES, sensory cortex, and EF, all intercept-only paths were either significant or marginally significant. For thickness: SES → VAC intercept → EF intercept ($B = .009$, $\beta = .007$, $p = .002$, 95 %CI = [.003,.014]) and SES → AAC intercept → EF intercept ($B = -.004$, $\beta = -.003$, $p = .010$, 95 %CI = [-.006, -.001]); for surface area: SES → VAC intercept → EF intercept ($B = .005$, $\beta = .004$, $p = .091$, 95 %CI = [-.001,.010]) and SES → AAC intercept → EF intercept ($B = .009$, $\beta = .007$, $p = .007$, 95 %CI = [.002,.015]). No models with sensory cortex slopes and/or EF slopes were significant. Additionally, no PFC variable (intercept/slope, thickness/surface area) significantly mediated relationships between SES and either the EF intercept or slope. Together, this indicates that baseline VAC thickness and AAC surface area mediate the relationship between SES and baseline EF. Also, interestingly, although SES was positively associated with intercepts for both AAC thickness and EF, the negative relationship between AAC thickness and EF intercepts contributed to this one significant mediation in which lower baseline AAC thickness mediated the positive relationship between SES and baseline EF.

Finally, serial mediation models investigated the indirect paths from SES, through sensory cortex variables (VAC/AAC, intercept/slope, thickness/surface area) and PFC cortex (intercept/slope, thickness/surface area), to EF (intercept/slope). No serial mediation models were significant.

3.7. Sensitivity analyses to test specificity of the model

To test the specificity of our model, we have conducted a sensitivity analysis comparing the model fit between our predicted models (where the intercepts and slopes of the AAC/VAC predict the intercept and slope of the PFC) and a model not predicted by the theory in which we reverse the location of the PFC and sensory association cortex in the model (where the intercept and slope of the PFC predict the intercepts and slopes of the AAC/VAC). The results provide some direct support for our theoretical model, and some inconclusive findings. Specifically, the surface area model provides support for our model in that there is a poorer fit for the reverse model ($\text{PFC} \rightarrow \text{AAC/VAC}$) compared to the predicted model ($\text{AAC/VAC} \rightarrow \text{PFC}$; AIC = 182819 for the predicted model vs. AIC = 182918 for the reverse model). This finding provides support for our predicted model over the reverse model. For the thickness data, the model comparison between the predicted and the reverse models is inconclusive because AIC values are nearly identical (Predicted model AIC = 218545 vs. reverse model AIC = 218540). This finding limits our ability to make a strong argument on the directionality of the associations based on the data.

Additionally, we conducted sensitivity analyses removing ADI from our measure of SES and including only individual-level SES variables as well as analyses where we include only ADI as a measure of SES. We note that results are largely unchanged in these analyses compared to the main analyses, with minor differences (see [Supplemental Materials](#) for details).

4. Discussion

The goal of the present study was to investigate the neural mechanisms underlying SES-related differences in EF development in a large longitudinal sample. We find that development of SES-related differences in sensory association cortices, including the visual and auditory cortex, would be associated with differences in structure of the PFC, which in turn would be associated with development of EF over time. While we did not find support for the entire model, results supported key parts of the hypothesized model. Most notably, socioeconomic differences in PFC structure were mediated by sensory structure. Additionally, we found some support for the hypothesis that SES-related differences in EF are mediated by sensory cortex structure.

4.1. SES and cortical structure of sensory association cortex and PFC

We found that children from higher SES homes show greater thickness and surface area in sensory association regions and higher PFC surface area in early adolescence. This finding is consistent with many previous studies which find SES-related differences in brain structure in these regions (Jednorög et al., 2012; Machlin et al., 2020; Mackey et al., 2015; Merz et al., 2020; Noble et al., 2015; Piccolo et al., 2016; Rakesh et al., 2022; Rakesh and Whittle, 2021). Moreover, SES was positively associated with the slope of cortical thickness and cortical surface area in the AAC and cortical surface area in the VAC over the two-year period. There was a negative association for each structural measure between the slope and the intercept, such that individuals with higher cortical thickness or surface area tended to show less change in these measures over time. This negative association between slope and intercept makes the positive association between SES and both the slope and intercept of these structural measures particularly notable, highlighting the strength of the association between SES and brain structure.

4.2. Structure of sensory association cortices mediate SES-related differences in PFC structure

Here, we demonstrate that SES-related differences in PFC structure are mediated by the structure of sensory processing regions, a key aspect of our model. We found support for this hypothesis both cross-

sectionally and when investigating growth over time such that baseline structure of the visual and auditory association cortex mediated SES-related differences in baseline PFC structure and change in sensory association cortex structure mediated SES-related differences in change in the PFC over time. These results provide support for our theoretical model that predicts that development of sensory processing regions scaffolds development of the prefrontal cortex in late childhood and early adolescence (Amso and Scerif, 2015; Rosen et al., 2019) and explains SES-related differences in PFC development. These findings align with the observed spatiotemporal variation in cortical development, whereby sensory regions develop earlier than regions that support higher order cognitive function such as the PFC (Norbom et al., 2021; Rakesh, Dehestani, et al., 2024).

Socioeconomic status is a broad factor that is correlated with many different environmental experiences more proximal to the child including experiences in the home and school. For instance, financial constraints can cause parental stress which can in turn contribute to differences in parent-child interactions (Martins et al., 2023) and ultimately children's brain and behavior development (Farber et al., 2020; Whittle et al., 2022). Income has a role to play in where a family lives, and in the US, public schools are funded in large part by property taxes. This link leads to lower income neighborhoods tending to have lower quality schools (Wodtke et al., 2023), which are in turn related to children's brain structure and function (Piccolo et al., 2019; Rakesh, Zalesky, et al., 2023). This developmental systems framework is critical to determining how macro-level socioeconomic factors influence children's more proximal environments and how those experiences may in turn contribute to individual differences in brain development. The theoretical model on which this work is based posits that cognitive stimulation may be an important factor in explaining SES-related differences in sensory cortex development (Rosen et al., 2019). However, at the time points included in this analysis, no measure of cognitive stimulation was collected in ABCD and therefore we are unable to directly test this model. Indeed, we have recently shown across two studies that SES-related differences in the functional recruitment of visual processing regions during cognitively demanding tasks (e.g. visual attention and visual working memory) is explained by lower levels of cognitive stimulation (Lurie et al., 2021; Rosen et al., 2021). Future work should investigate whether cognitive stimulation or other more proximal factors explain SES-related differences in structural development of the visual association cortex. The ABCD study does include a wide array of other environmental experiences (e.g. violence exposure, environmental toxins) that correlate with SES and could be playing a role in the associations explored in the current analyses. However, the current study takes a hypothesis-driven rather than a data-driven approach and therefore we do not explore these other environmental factors. Future exploratory work should focus on whether other environmental experiences correlated with SES play a role in SES-related differences in cortical structure and EF development.

Importantly, our theoretical model only focused on the role of visual association cortex in scaffolding development of the PFC. Here, we extend our previous theoretical work by demonstrating that *auditory* association cortex regions also play a significant role in these associations and scaffold the development of the PFC. It will be important for future studies to investigate the possible proximal factors that play a role in SES-related differences in structure of the auditory association cortex and in turn development of the PFC. One likely candidate is language exposure. Language exposure varies as a function of SES and has been found to be associated with brain structure (Cheng et al., 2023; Gilkerson et al., 2017; Merz et al., 2020; Romeo, 2019). Exposure to a complex linguistic environment and conversational turns with adults—both of which are positively associated with socioeconomic status—may contribute to the development of auditory processing regions. This increasingly complex information may then be passed to areas of the PFC and allow children the opportunity to practice increasingly complex cognitive skills including EF (Merz et al., 2020).

Future work will need to directly investigate the potential role that language exposure and experience play in this pathway explaining SES-related differences in development of auditory processing regions and in turn development of the PFC. Here, we provide the first evidence to our knowledge that auditory association cortex is playing a key role in explaining SES-related differences in PFC development.

4.3. SES, cortical structure and executive function

A main goal of this study was to investigate whether SES-related differences in EF are explained by differences in sensory cortical and PFC structure. However, this investigation only provided limited support for our hypotheses. The only significant associations between cortical structure and EF performance were found for baseline thickness of the AAC. Furthermore, we found that AAC and VAC thickness and AAC surface area, but not VAC surface area or either measure of PFC structure, mediates the association between SES and EF at baseline. Our results indicate that structure of the auditory association cortex may be a mechanism explaining socioeconomic differences in EF. Other studies exploring language development have found SES-related disparities in language development are mediated by structure of language processing regions that abut the auditory association cortex we investigated, indicating that these regions play a role in disparities in cognitive outcomes (Merz et al., 2020). Critically the associations here were only found cross-sectionally, and results were mixed between measures of cortical structure (thickness and surface area), and thus should be replicated.

Additionally, contrary to our hypotheses, we did not find significant associations between cortical thickness or surface area in the PFC and EF performance and thus did not find significant support for our entire model. Indeed, another study using small subsample of ABCD found positive associations between cortical thickness in part of the PFC and performance on the flanker at one time point (Sarabin et al., 2023). Additionally, the relation between EF performance and cortical thickness in the PFC is mixed in this age range, with some studies finding negative (Kharitonova et al., 2013; Tamnes et al., 2010) and others finding positive associations (e.g. Lawson et al., 2013; Sarabin et al., 2023). Furthermore, some studies demonstrate that SES *moderates* the association between cortical thickness and cognitive performance. For example, one study found that among high-SES children, there was a negative association between cortical thickness in the lateral PFC and reasoning skills, whereas among low-SES children, this association was reversed such that children with thicker lateral PFC had better reasoning skills (Leonard et al., 2019). It will be important for future studies to continue to track this association over time and with additional measures of EF in the ABCD sample.

Importantly, we found that SES was positively associated with EF performance at baseline, indicating that children from higher SES households performed better on the EF tasks. This finding aligns with many studies and meta-analyses highlighting socioeconomic disparities in EF performance (Hackman et al., 2015; Last et al., 2018; Lawson et al., 2018; Lengua et al., 2015; Noble et al., 2005; Rosen et al., 2020). Interestingly, we did not find that SES was associated with growth in EF over time, which is consistent with work in younger children showing that SES-related differences in EF emerge early and remain relatively stable over time (Lengua et al., 2015). We did, however, find significant increases in EF over time. This finding is consistent with a recent study involving over 10,000 children, adolescents, and adults demonstrating the age range covered in this study (9–13 years) represents a period of significant growth in EF development (Tervo-Clemmens et al., 2023). It will be interesting and important to continue to follow the children in this study to determine whether these SES-related differences remain stable over longer periods or if significant associations in *change* in EF emerge as they develop.

5. Limitations and open questions

The current study has several strengths including a large sample size with both EF and brain structure measured over time, being preregistered, and testing a strong theoretical framework. However, there are several limitations that should be considered when interpreting the results. First, our longitudinal approach constrained us to the EF measures administered at both timepoints. While the baseline assessment encompassed a diverse battery of EF tasks, including the dimensional change card sort task and the list sorting working memory task, they were omitted at the follow-up assessment. Consequently, only the Flanker task was retained for both time points. Unfortunately, this measures do not fully capture the breadth of EF skills (Miyake et al., 2000). If a wider battery of EF tasks is administered at future time points of ABCD, it will be important to investigate whether cortical structure mediates the association between SES and these core EF constructs over time.

Relatedly, SES-related differences in EF emerge early in development and remain relatively stable over time (Lengua et al., 2015). The children in this study were 9–10 years old at the first time point, with the second time point occurring only two years later. This short interval, combined with the relatively older age of the sample, makes it challenging to detect significant changes over time. To address this limitation, future studies should leverage the Healthy Brain and Cognitive Developmental (HBCD) which starts prenatally and tracks children over the first years of life to better capture the emergence of differences in brain structure and EF (Jordan et al., 2020).

Importantly, these differences in brain structure do not imply deficits in children from low-income families. Contemporary models in developmental cognitive neuroscience highlight that a “deficit-based” approach can be harmful and exclusionary (DeJoseph et al., 2024; Nketia et al., 2021). Instead, it is important to note that SES-related differences in brain structure can reflect adaptations to the environment that facilitate skills for that particular context or simply reflect the diversity of neurodevelopmental trajectories. In the current study, we focus on SES-related differences in brain structure that support EF development and do replicate the well-documented negative associations between SES and EF. Importantly however, this does not preclude the idea that SES-related differences in brain structure could be correlated with higher performance on some other cognitive tasks not measured in the current study.

We are limited in our ability to make claims about the directionality of the associations. Our model is predicated on the idea that AAC and VAC structure predict the PFC structure. However, it is also possible that individual differences in overall baseline brain structure and development over time may be driving these associations. In sensitivity analyses (see *Supplemental Materials* for more details) we tested the reverse association such that PFC structure predicted AAC/VAC structure in the models. Model fit comparisons between our predicted and reversed models provide partial support for our theoretical framework. Specifically, the model fit is better for the theory-based model than the reverse model for surface area. However, the findings are inconclusive for cortical thickness where the predicted and reverse models have nearly identical model fit. Given these findings and the availability of only two timepoints of data, we are limited in our ability to make claims about the directionality of these associations. We do note that our model is based on theory and evidence from developmental studies that demonstrate that the sensory association cortices develop before the PFC (Norbom et al., 2021; Rakesh, Dehestani, et al., 2024).

Additionally, it is important to note that while we found significant results consistent with several hypothesized associations, the effect sizes were quite small. The effect sizes we report here are consistent with those found in other studies using structural MRI and cognitive data from ABCD and are expected in large samples (Marek et al., 2022). Critically, these small effect sizes at the individual level can have large effects over time at the population level (Funder and Ozer, 2019).

That individuals raised in low SES households tend to show thinner cortex or different trajectories in cortical thinning has been noted across many studies (Brito, 2017; Mackey et al., 2015; Piccolo et al., 2016; Rakesh, Whittle, et al., 2023; Sanders et al., 2022). One possibility is that children from lower SES-households demonstrate altered pace of brain development, either through accelerated or delayed synaptic pruning. A recent systematic review investigating this question has found that the experiences of low SES is most consistently associated with delayed neural development or simply different trajectories (Rakesh, Whittle, et al., 2023). While cortical thinning is a normal part of neurodevelopment, debate has occurred over the past several decades as to the precise mechanisms through which this occurs. Some researchers have suggested that this occurs through synaptic pruning of underutilized connections (McLaughlin et al., 2014, 2017). Others have suggested that cortical thinning reflects increased cortical myelination, thus reducing the contrast between grey and white matter (Natu et al., 2019; Sowell et al., 2004b). Indeed, recent work on the role of myelination in this process has been a bit mixed. One study found slower myelin growth among individuals who experienced socioeconomic disadvantage as children (Ziegler et al., 2020). Another found that children who experienced low SES had greater overall cortical myelin content, but no difference in the pace of myelin change compared to higher SES peers (Norbom et al., 2022). Yet another study found no significant SES-related differences in myelin content (Weissman et al., 2023). Future studies should further explore the role of changes in myelination across development in explaining SES-related differences in EF.

In the present study we integrate measures of SES on both the individual (parental education and income-to-needs ratio) and the neighborhood level (area deprivation index). Sensitivity analyses (see *Supplemental Materials*) demonstrate that our results are remarkably similar when we use just individual-level measures of SES as when we combine them with ADI. We retain the original preregistered SES factor in the full analysis. While it is true that neighborhood SES, especially when measured at the tract level using metrics like the Area Deprivation Index (ADI), may not strictly conform to traditional definitions of SES, we argue that it remains a crucial component of a child's environment that significantly influences their development. It is critical to recognize the interconnectedness of these factors. Neighborhood characteristics not only directly impact children through other pathways, but also influence household environments and parenting behaviors, which in turn shape child development outcomes. For instance, a disadvantaged neighborhood with limited access to quality education or healthcare facilities may indirectly affect household SES by constraining opportunities for economic advancement or exacerbating stressors within the family unit. Indeed, key to developmental science theories about child development is that distal factors (like neighborhood disadvantage) likely exert their influence on children through more proximal factors (e.g., parents, neighbors, and peers). For example, studies have shown that the utilization of education-focused practices is lower in families that reside in structurally disadvantaged neighborhoods (Greenman et al., 2011). Neighborhood characteristics have also been associated with several parenting behaviors (such as parental warmth and monitoring; Burton and Robin, 2000; Klebanov et al., 1994; Shumow and Lomax, 2009). This could, in part, be due to the existence of role models and socializing influences for parents in the neighborhood (Kohen et al., 2008). Importantly, neighborhood SES has consistently been shown to be associated with brain structure and function over and above household measures of SES and adversity (Gard et al., 2021; Hackman et al., 2021; Rakesh, Cropley, et al., 2021; Rakesh, Seguin, et al., 2021; Rakesh, Zalesky, et al., 2021; Taylor et al., 2020). Therefore, the complex interplay between these factors highlights the need to consider both levels of SES to comprehensively understand child development outcomes.

Another limitation of the study was the inability to account for individuals clustered in the same neighborhoods. For privacy reasons, census-tract data is not linked publicly to individual subject IDs in the ABCD study. Therefore, clustering by neighborhood within the model

was not possible. We do note however that our results remained largely unchanged when we use only person-level indicators of SES (income-to-needs and parental education) and therefore believe that adding this clustering would be unlikely to significantly impact the results.

The current model is also limited in the ability to determine non-linear associations between the variables of interest. Future studies including additional data releases from ABCD—especially once there are four or more time points for both structural brain and behavioral data—should investigate whether there are non-linear associations between SES and brain structure and behavior. Additionally, we test a mediation model here with two time points. We note that the limited number of time points coupled with the potential presence of time-varying confounds make causal inferences difficult to make. We look forward to future analyses with additional data releases from ABCD to test these models further.

Finally, SES is a complex factor that encompasses many aspects of a child's environment and experience. It will be imperative for future studies to explore the more proximal factors in a child's environment that explain these SES-related differences in brain structure and cognitive development including cognitive stimulation, school environment, childhood maltreatment, parenting practices, violence exposure, and toxin exposure (Hackman and Farah, 2009; Rakesh, Elzeiny, et al., 2023; Rakesh, Lee, et al., 2024; Rosen et al., 2019, 2020; Weissman et al., 2022; Whittle et al., 2022).

6. Conclusions

The current study explored the structural cortical mechanisms underlying SES-related differences in EF. While our entire model was not significant, we did find support for the idea that SES-related differences in cortical structure of the PFC are mediated by structure of auditory and visual association cortex and that differences in sensory association cortex structure mediates SES-related differences in EF. This extends the theoretical framework that sensory processing regions in the brain scaffold development of the PFC and may explain socioeconomic disparities in PFC structure.

While the effect sizes in the current study are modest, our models, which seek to understand the neural mechanisms underlying SES-related differences in EF, can have large effects at the population level. The current model extends prior work highlighting the role of PFC development in EF to highlight the additional importance of development of sensory association cortex. Given that the sensory association cortex develops early in life, our findings have implications for early interventions and indicate that including cognitive stimulation that focuses on sensory development may be impactful in supporting structural brain development. Future studies should explore these questions in younger populations as these brain regions are undergoing more rapid development as well as proximal environmental factors that may be driving SES-related differences in brain structure.

CRediT authorship contribution statement

Rosen Maya L.: Writing – review & editing, Writing – original draft, Formal analysis, Data curation, Conceptualization. **Rakesh Divyanganan:** Writing – review & editing, Writing – original draft, Formal analysis, Data curation, Conceptualization. **Romeo Rachel R.:** Writing – review & editing, Writing – original draft, Formal analysis, Data curation, Conceptualization.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at [doi:10.1016/j.dcn.2025.101550](https://doi.org/10.1016/j.dcn.2025.101550).

Data availability

The authors do not have permission to share data.

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