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A Social Gradient of Cortical Thickness Relative to Age in Adolescence:
Relations Among Neighborhood Socioeconomic Disadvantage, Advanced Cortical Thinning,
and Depressive Symptoms

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Abstract

Background: Mental and physical health are affected by family and neighborhood socioeconomic status (SES). Accelerated biological aging in the context of lower SES is one mechanism that might contribute to underlying health disparities; few studies, however, have considered neighborhood SES in relation to neural markers of biological aging in adolescents.

Methods: In 120 adolescents 13-18 years of age, we examined family and neighborhood SES in relation to biological aging in the brain, indexed by cortical thickness relative to chronological age. We also examined whether advanced cortical thinning relative to age was related to depressive symptoms and explored regions of interest.

Results: Neighborhood socioeconomic disadvantage was uniquely associated with advanced cortical thinning in the left hemisphere ($\beta = -.20$), which was related to more severe depressive symptoms ($\beta = -.33$). In contrast, family income-to-needs was not significantly associated with cortical thickness age after controlling for relevant covariates. In exploratory, covariate-adjusted analyses of cortical thickness relative to age at the regional level, neighborhood socioeconomic disadvantage was associated with advanced cortical thinning in the left superior frontal gyrus ($\beta = -.27$), fusiform gyrus ($\beta = -.20$), and insula ($\beta = -.21$). Of these regions, only advanced cortical thinning in the left superior frontal gyrus was associated with more severe depressive symptoms ($\beta = -.18$).

Conclusion: Our findings provide evidence for a social gradient of accelerated biological aging at the neural level during adolescence. Adolescents living in less advantaged communities have a thinner left hemisphere cortex than expected given their chronological age. Advanced cortical thinning may increase risk for depression in adolescence.

**A Social Gradient of Cortical Thickness Relative to Age in Adolescence:
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Physical and mental health vary by socioeconomic status (SES) (1,2), often defined as the combined access to social prestige, education, and material wealth (3). Compared to individuals from high-SES backgrounds, individuals with fewer resources are at increased risk for health problems across the lifespan, including cardiovascular disease, cancer, dementia, obesity, and depression (1,4,5). Although the effects of SES on health are strongest at lower levels of SES, it is important to note that they are present across stratum, pointing to a social gradient in health that is relevant to the entire population (6). SES can be assessed at multiple levels, including family and neighborhood (7). Common measures of family SES are household income and education; in contrast, measures of neighborhood SES often consist of census-level data on rates of educational attainment, poverty, and unemployment and data on the shared material infrastructure and social resources within a community (e.g., healthcare access, pollution burden) (8,9). Although family and neighborhood SES are related, their associations with each other are moderate (10). Further, family and neighborhood SES may have dissociable effects on health, be related to distinct health outcomes, and affect health via different pathways (9,11,12). These differences between family and neighborhood SES effects may be particularly relevant during adolescence, given that it is a period of increasing autonomy during which neighborhoods are a primary context outside of the home (13).

One mechanism that might underlie the SES-health gradient is faster versus slower rates of biological aging (14). Accelerated biological aging in childhood and adolescence refers to processes implicated in faster maturation or development (e.g., earlier pubertal onset), or a more

rapid decline in the integrity or functioning of biological systems than occurs simply with increasing chronological age (e.g., faster telomere shortening) (15). Children and adolescents from socioeconomically disadvantaged backgrounds are at greater risk for exposure to negative life events and conditions that are believed to accelerate the pace of biological aging, including early life adversity characterized by threat (16), environmental pollutants and contaminants (17), and reduced access to safe green spaces (18). These SES-related exposures and disparities have been linked to metrics of biological aging such as telomere shortening and advanced pubertal maturation (19–21), which are risk factors for, or possibly consequences of, mental health difficulties (22–25).

A growing body of research has investigated the relations between socioeconomic disadvantage and brain-based measures of accelerated biological aging (10). Cortical thickness decreases with age (26,27), possibly due to increases in synaptic pruning or in myelination across development (28), and has been used as a neural marker of accelerated aging or maturation (19,29,30). A recent systematic review of the literature suggests that low SES is consistently associated with reduced cortical thickness relative to higher SES peers (19); however, the majority of prior studies have focused on family income and parent education measures of SES (31–34). For example, Mackey and colleagues found that students who were eligible for free or reduced lunch based on family income had reduced cortical thickness in all lobes of the brain (32). Using a large sample of individuals 3–20 years of age, Piccolo and colleagues found steeper age-related decreases in global cortical thickness during childhood that level off later in adolescence, at low compared to high levels of family income and parent education (35). Conversely, Lawson and colleagues found that less parent education, but not family income, was associated with reduced cortical thickness in specific frontal regions, including the right anterior

cingulate and left superior frontal gyrus (33). These SES-related alterations in cortical thickness may have implications for adolescent mental health, given that advanced cortical thinning has been associated with the development of depressive symptoms (36,37).

More recently, researchers have considered whether neighborhood socioeconomic disadvantage is uniquely associated with neural measures of biological aging (38–41). Compared to the literature on family SES and cortical thickness, however, findings concerning the nature of the relation between neighborhood SES and cortical thickness are less clear. Researchers have found that, in adults, socioeconomic disadvantage at the neighborhood level is associated with greater cortical thinning (39,42). Similarly, using data from the Adolescent Brain Cognitive Development (ABCD) Study, Vargas and colleagues found in adolescents that neighborhood socioeconomic disadvantage was associated with less cortical thickness in prefrontal regions such as the left lateral orbitofrontal, right medial orbitofrontal, right superior frontal, and right rostral middle frontal gyrus (43). In contrast, however, some studies of children and adolescents have found that neighborhood disadvantage is not directly associated with cortical thickness in prefrontal and parietal regions (40,44), or have found that neighborhood disadvantage is associated with *increased* cortical thickness in temporal lobe regions (45). Taken together, it is unclear whether neighborhood SES is associated with either global or regional measures of age-related cortical thickness in adolescents.

In this study, we examined associations of family and neighborhood SES with advanced cortical thinning in a community sample of adolescents. We defined advanced cortical thinning as less cortical thickness than would be expected based on an individual's chronological age (i.e., residual of cortical thickness regressed on age). Given that prior studies have linked measures of family SES and, to a lesser extent, of neighborhood SES to cortical thickness (19,43,46), and that

family and neighborhood SES may make distinct contributions to health and brain structure (9,47), we hypothesized that family and neighborhood SES would be uniquely associated with global measures of cortical thickness age. Further, we expected that advanced cortical thinning relative to chronological age would be associated with increased depressive symptoms. Finally, we conducted exploratory analyses of relations among measures of SES, regional measures of cortical thickness relative to age, and depressive symptoms.

Methods and Materials

Participants and Procedure

Participants were 13- to 18-year-old adolescents from the San Francisco Bay Area who were part of a larger, longitudinal study of the effects of early life stress on neurodevelopment during puberty. Families were recruited from the community using local flyers, media, and online advertisements. Study exclusion criteria at the baseline assessment included inability to undergo MRI (e.g., had metal implants, braces), a history of neurological disorder or major medical illness, serious cognitive or physical challenges that might interfere with the ability to understand or complete procedures, non-fluent English speakers, and, for females, the onset of menses. Participants were re-assessed at approximately 2-year intervals. The current analysis included adolescents who had usable neuroimaging data at the third wave of assessments, conducted between Jan, 2018, and June 2021 ($N=120$). We used residential addresses to compute neighborhood socioeconomic disadvantage at the Census-block group level. Participants and their parents signed assent and consent forms, respectively, and were compensated for their participation. This study protocol was approved by the Stanford University Institutional Review Board.

Neighborhood Socioeconomic Disadvantage

Neighborhood socioeconomic disadvantage was assessed using the Area Deprivation Index (ADI; <https://www.neighborhoodatlas.medicine.wisc.edu>) (48). As described in (49), the ADI maps neighborhood socioeconomic disadvantage at the Census-block group level using Census and 2018 American Community Survey data. The ADI uses a factor analysis of measures of educational attainment, poverty, housing quality, and unemployment to identify overall neighborhood socioeconomic disadvantage. The ADI converts these factor scores into deciles representing the amount of socioeconomic disadvantage in a given neighborhood relative to other neighborhoods in California.

Structural Magnetic Resonance Image Acquisition and Processing

All participants completed a T1-weighted magnetic resonance imaging (MRI) scan on a 3T GE Discovery MR750 (GE Medical Systems, Milwaukee, WI, USA) with a 32-channel head coil (Nova Medical, Wilmington, MA, USA) at the Stanford Center for Cognitive and Neurobiological Imaging. T1-weighted images were collected using the following spoiled gradient echo pulse sequence: 186 sagittal slices; TR (repetition time)/TE (echo time)/TI (inversion time) = 6.24/2.34/450ms; flip angle=12°; voxel size = 0.9 mm x 0.9 mm x 0.9 mm; scan duration = 5.15 minutes.

The T1-weighted structural brain images were processed using the ‘recon-all’ feature of FreeSurfer version 6.0.1 (<http://surfer.nmr.mgh.harvard.edu/fswiki/recon-all>) to perform tissue segmentation and to estimate cortical thickness according to the Desikan-Killiany atlas (50). Each segmentation was visually inspected for quality assurance according to the protocols established by the global consortium, Enhancing Neuroimaging Genetics through Meta-Analysis (ENIGMA) (<http://enigma.ini.usc.edu/protocols/imaging-protocols>). Poorly segmented regions were excluded from further analysis only after careful visual inspection.

Depressive Symptoms

We assessed depressive symptoms using the 10-item version of the self-report Children's Depression Inventory (CDI) (51). Participants reported on their depressive symptoms over the preceding 2 weeks. For each item, participants endorsed one of three statements indicating graded severity of symptoms. We summed responses to yield a score of depressive symptom severity. Symptom scores were available for 116 of the 120 participants.

Covariates

Prior studies have found evidence of sex differences in cortical thinning (52). Further, being a person of color who experiences discrimination, or exposure to early life stress more broadly, can influence biological aging (19,53), including aging as indexed by cortical thickness (54). Finally, neighborhood and family-level SES are often correlated. Therefore, in regression models, we adjusted for sex, race/ethnicity, severity of early life stress, and a ratio of family income-to-needs in order to test unique effects of neighborhood socioeconomic disadvantage.

Biological female or male sex was coded as 0 and 1, respectively. We created dummy coded variables for each race/ethnicity category except for White, which was used as the reference category. We operationalized family-level SES as the ratio of the participant's household income to the low-income limit for Santa Clara county based on the number of people living in the household (i.e., income-to-needs). The caregiver who brought the adolescent to the laboratory self-reported household income in the past year and the number of people living in the household. Rather than report an exact dollar amount, caregivers reported household income in bins on a 10-point scale: $\leq \$5000$; \$5001-\$10,000; \$10,001-\$15,000; \$15,001-\$25,000; \$25,001-\$35,000; \$35,001-\$50,000; \$50,001-\$75,000; \$75,001-\$100,000; \$100,001-\$150,000; $\geq \$150,001$. The low-income limit for Santa Clara county was calculated as 80% of the median

income by number of people living in the household. These values are set by the U.S. Department of Housing and Urban Development for the year 2018, when the median income was \$125,200 and the low-income limit for a family of four was \$94,450. We divided the midpoint value of the endorsed household income bin by the low-income limit for the household size to produce the income-to-needs ratio. Income-to-needs data were available for 110 of the 120 participants. As reported in (55,56), early life stress severity was measured at the baseline assessment when adolescents were 9-13 years of age. Adolescents completed an interview assessing exposure to 30 different types of stressful events using a modified version of the Traumatic Events Screening Inventory for Children. A panel of coders rated interview responses for severity using a modified version of the UCLA Life Stress Interview coding system (57); endorsed events were rated on a scale ranging from 0 (non-event or no impact) to 4 (extremely severe impact) with half-point increments. Ratings were summed across stressful life events to create a cumulative measure of early life stress severity.

Statistical Analyses

We first tested associations between age and cortical thickness averaged within each hemisphere. When age was significantly associated with cortical thickness ($p < .05$), we created unstandardized residual scores (cortical thickness residualized on age) for each participant. In these instances, negative residual scores represent thinner cortex than what would be expected given a participant's chronological age. We adopted this approach from prior studies that have used age residuals to measure accelerated epigenetic aging and pubertal maturation (58,59). We first tested correlations between neighborhood socioeconomic disadvantage and cortical thickness residuals, and then conducted regression analyses to examine whether these associations were present after adjusting for the covariates described above. We conducted a

follow-up regression analysis to test whether cortical thickness residuals were associated with depressive symptoms. In exploratory analyses, we repeated these analytic steps for cortical thickness residuals of individual regions as defined by the Desikan-Killiany atlas. We conducted all analyses using the lavaan package in R software (60) and used full-information maximum likelihood estimation to account for missing data (61).

Results

Participant characteristics are presented in Table 1. On average, participants came from relatively advantaged socioeconomic backgrounds in terms of income-to-needs ratio and neighborhood SES. Nevertheless, the sample included a wide range of neighborhood disadvantage scores, and 22% of participants were from families who were considered low income based on having an income-to-needs ratio less than 1.

Neighborhood Disadvantage and Residuals of Mean Cortical Thickness in the Left and Right Hemispheres

Age was negatively associated with mean cortical thickness in the left ($r=-.25, p=.004$) and right hemispheres (both $r=-.25, p=.004$). Thus, we used the unstandardized residuals of cortical thickness in left and right hemispheres on age as metrics of advanced cortical thinning. Neighborhood socioeconomic disadvantage was negatively associated with cortical thickness residuals in the left and right hemispheres ($r=-.23, p=.011$ and $r=-.20, p=.027$, respectively), indicating that more disadvantage in a neighborhood was related to thinner-than-expected cortex; in contrast, income-to-needs ratio was positively associated with cortical thickness residuals in the right hemisphere ($r=.24, p=.013$), indicating that low family SES was also related to advanced cortical thinning. As expected, neighborhood socioeconomic disadvantage and family income-to-needs were moderately negatively intercorrelated ($r=-.39, p<.001$).

We conducted regression analyses to determine whether the observed correlations between neighborhood socioeconomic disadvantage and cortical thickness residuals remained significant after adjusting for income-to-needs, sex, race/ethnicity, and severity of early life stress. In adjusted models, the effect of neighborhood socioeconomic disadvantage on cortical thickness residuals in the left hemisphere remained statistically significant ($\beta = -.20$, $p = .038$); however, the effects of neighborhood and family SES on cortical thickness residuals in the right hemisphere were no longer significant after adjusting for covariates (both $ps > .067$). The fully adjusted models are presented in Table 2.

We conducted follow up regression analyses to test whether cortical thickness residuals were associated with concurrent depressive symptoms. Cortical thickness residuals (indicating advanced cortical thinning) in the left hemisphere were associated with more severe depressive symptoms ($\beta = -.33$, $p = .014$). This relation was independent of the effects of sex ($\beta = .26$, $p = .002$), income-to-needs ratio ($\beta = -.22$, $p = .031$), and severity of early life stress ($\beta = .20$, $p = .033$). Cortical thickness residuals in the right hemisphere, neighborhood socioeconomic disadvantage, and race/ethnicity were not significantly associated with depressive symptoms in this model (all $ps > .064$).

Exploratory Analyses of Neighborhood Disadvantage and Regional Residuals of Cortical Thickness

We conducted exploratory analyses of associations between age and cortical thickness in each Desikan-Killiany atlas region. Age was significantly and negatively associated with thickness in 17 frontal regions, 6 parietal regions, 6 temporal regions, 2 occipital regions, 2 cingulate regions, and the left insula (see Table 3). Thus, we used unstandardized residuals of cortical thickness regressed on age for these regions in analyses exploring associations with SES.

Neighborhood socioeconomic disadvantage was negatively and significantly associated with cortical thickness residuals in 4 frontal regions: the right caudal middle frontal ($r=-.18, p=.041$), left rostral middle frontal ($r=-.19, p=.032$), and left and right superior frontal gyrus ($r=-.23, p=.009$ and $r=-.20, p=.022$, respectively). Neighborhood socioeconomic disadvantage was also negatively and significantly associated with cortical thickness residuals in 2 temporal regions (the left and right fusiform gyrus, $r=-.25, p=.004$ and $r=-.23, p=.008$, respectively), the left lingual gyrus in the occipital lobe ($r=-.19, p=.032$), and the left insula ($r=-.23, p=.010$). After adjusting for sex, income-to-needs, race/ethnicity, and severity of early life stress, only the associations of socioeconomic disadvantage with the left superior frontal cortex, left fusiform cortex, and left insula remained statistically significant ($\beta=-.27, p=.005$, $\beta=-.20, p=.039$, and $\beta=-.21, p=.030$, respectively). Further, of these regions, only cortical thickness residuals in the left superior frontal cortex were associated with severity of depressive symptoms in models adjusting for covariates ($\beta=-.18, p=.038$).

In exploratory analyses, income-to-needs ratio was positively and significantly associated with cortical thickness residuals in 2 frontal regions (right caudal middle frontal and left lateral orbitofrontal gyrus, $r=.20, p=.031$ for both regions), 3 temporal regions (right fusiform and left and right transverse temporal gyrus, $r=.24, p=.010$, $r=.24, p=.010$, and $r=.22, p=.018$, respectively), and 2 occipital regions (left and right lingual gyrus, $r=.26, p=.005$ and $r=.28, p=.002$, respectively). After adjusting for sex, neighborhood socioeconomic disadvantage, race/ethnicity, and severity of early life stress, none of the associations with income-to-needs ratio remained statistically significant (all $ps>.087$).

Discussion

Accelerated biological aging is one mechanism that might underlie SES-health gradients (14). SES, however, is a complex multilevel construct (7). Family- and neighborhood-level SES are only moderately correlated and may have dissociable effects on health; they may be related to distinct health outcomes or map onto distinct mechanistic pathways. Thus, it is important to consider both family and neighborhood SES to elucidate their potentially unique contributions to biological aging. Here, we found that neighborhood socioeconomic disadvantage is uniquely associated with advanced cortical thinning. In analyses of global cortical thickness relative to age, this relation was specific to the left hemisphere after adjusting for covariates. In contrast, we found associations of neighborhood socioeconomic disadvantage and low family income-to-needs ratio with advanced cortical thinning in the right hemisphere; however, these effects were not statistically significant in a model that included relevant covariates. Our exploratory analyses identified effects of neighborhood socioeconomic disadvantage and family income-to-needs ratio on advanced cortical thinning in distinct brain regions, but only neighborhood disadvantage effects remained significant after adjusting for covariates. Taken together, our findings suggest that neighborhood socioeconomic disadvantage is associated with advanced cortical thinning, both globally in the left hemisphere and within specific regions, including the left superior frontal gyrus, left fusiform gyrus, and left insula. Further, advanced cortical thinning in the left hemisphere and left superior frontal gyrus was related to more severe depressive symptoms, suggesting that neural measures of accelerated biological aging can inform our understanding of risk for depression during adolescence, especially among individuals from socioeconomically disadvantaged communities.

Our findings are consistent with research examining the relation between low neighborhood SES and advanced cortical thinning (39,42,43). Gianaros and colleagues found

that neighborhood socioeconomic disadvantage was negatively associated with a global measure of mean cortical thickness in adults (39). In our unadjusted correlation analyses, we similarly observed reduced mean cortical thickness relative to age in adolescents who were living in more disadvantaged neighborhoods. This relation was specific to the left hemisphere after adjusting for relevant covariates. One explanation for this unexpected lateralization finding is that we might have needed a larger sample size to reliably assess the effects of SES on average cortical thickness relative to age in the right hemisphere in a covariate-adjusted analysis. Alternatively, this finding may reflect the effects of neighborhood SES on the development of specific neurocognitive processes that rely on regions and connections in the left hemisphere. For example, given the role of the left hemisphere in language processing (62), and given that youth from disadvantaged neighborhoods score lower on language assessments (47), our left-lateralized effects of neighborhood SES may have implications for understanding SES-related differences in the development of language and related cognitive skills. Indeed, structural abnormalities, including in left prefrontal regions, may explain the relation between neighborhood SES and language skills (47). Certainly, this interpretation is speculative, and further research is necessary to replicate our finding that neighborhood SES is uniquely related to advanced cortical thinning in the left hemisphere, and to assess the relation between advanced cortical thinning and cognitive development.

Our exploratory analyses identified relations between neighborhood SES and cortical thickness relative to age in some of the same prefrontal regions that were reported in analyses of adolescents in the ABCD Study (43), including the superior frontal and rostral middle frontal gyrus. Frontal lobe regions are involved in processes that are central to mental health, such as emotion regulation (63), and structural abnormalities in these regions, such as reduced cortical

thickness, have been associated with psychopathology (64,65). Consistent with these findings, we found that advanced cortical thinning in the left superior frontal gyrus was associated with more severe depressive symptoms. Future longitudinal research should test the possibility that advanced cortical thinning in the left hemisphere and left superior frontal gyrus mediates the association between neighborhood SES and depressive symptoms in adolescence.

The specific mechanisms by which neighborhood SES might affect advanced cortical thinning are still unclear. Prior research suggests that structural abnormalities in some of the regions identified in this study are linked to events and experiences that are more prevalent in low-SES contexts, such as life stress (37), childhood maltreatment (66), and reduced cognitive stimulation (44). In low-SES neighborhoods, increased exposure to these kinds of experiences may accelerate synaptic pruning, leading to advanced cortical thinning. Deprivation in socioeconomically disadvantaged neighborhoods characterized by less exposure to cognitive stimulation and fewer positive social experiences may lead to accelerated pruning of underutilized neural regions and connections (67). Conversely, it is also possible that accelerated pruning reflects the strengthening of neural processes that are used more frequently in socioeconomically disadvantaged neighborhoods. Further research is needed to clearly identify the psychosocial, environmental, and biological paths that explain more precisely how living in more disadvantaged neighborhoods leads to advanced cortical thinning.

Our results add to a growing literature suggesting that low SES is associated with accelerated cortical maturation (19). Interestingly, our measure of neighborhood SES was associated with advanced cortical thinning more robustly than was our measure of family SES. This finding was surprising given that most prior studies linking SES to advanced cortical thinning have focused on family SES (32,35). There are three possible explanations for this

inconsistency. First, the association between family SES and cortical thickness may vary over the course of development (19). Indeed, Piccolo and colleagues found that family SES-related differences in cortical thickness were more pronounced in early than in mid- to late-adolescence (35). Our participants were 13-18 years of age, and greater statistical power may be necessary to identify family SES effects on advanced cortical thinning at this later developmental stage.

Second, it is possible that family SES effects on cortical development are a proxy for neighborhood SES effects. Most of the studies on family SES and cortical thickness did not control for neighborhood SES and thus were unable to test this possibility. A third explanation involves the fact that participants in our sample, on average, came from relatively advantaged backgrounds. It is possible that family SES effects are stronger at lower SES strata, whereas neighborhood disadvantage may more effectively capture nuances of social inequality that are relevant to cortical maturation at moderate to high levels of SES.

We should note three limitations of our study. First, we had 120 adolescents in our sample, but it is possible that we needed a larger sample to identify reliably unique effects of neighborhood and family SES. Second, our findings do not provide causal evidence for the relations among neighborhood SES, cortical thickness relative to age, and depressive symptoms, nor do they yield specific mechanistic pathways between these variables. As a related point, although we covaried for race/ethnicity, early life stress, and family SES, we cannot rule out all possible confounds or selection variables that may lead individuals to live in more or less disadvantaged neighborhoods (68). Finally, although our participants came from a wide range of socioeconomic backgrounds, our sample, on average, was relatively advantaged. Relations of neighborhood and family SES with cortical thickness relative to age may be more robust at lower levels of SES, which has been reported in the literature examining SES-related health and

developmental disparities (6). Nevertheless, the SES-health gradient appears to be relevant to the entire population (69,70). Our findings suggest that even across neighborhoods with moderate to high levels of SES, variation in disadvantage has meaningful implications for cortical thickness aging during adolescence.

Despite these limitations, our findings provide evidence for a social gradient of accelerated biological aging at the neural level during adolescence. Adolescents living in less advantaged communities have thinner cortex than would be expected given their chronological age, and this advanced cortical thinning may contribute to heightened risk for depression in adolescence.

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Table 1. Participant characteristics

	Mean or % (SD)	Range
Sex (male)	53.3%	Female/male
Age	15.58 (1.10)	13-18
Ethnicity/race		
White	46.7%	
Black	8.3%	
Hispanic or Latinx	9.2%	
Asian	11.7%	
Biracial	17.5%	
Other	6.7%	
Income-to-needs ratio	1.24 (0.45)	0.07-1.98
Neighborhood disadvantage (state decile)	2.77 (2.07)	1-10

Table 2. Regression models for associations of neighborhood socioeconomic disadvantage and income-to-needs ratio with cortical thickness residuals in the left and right hemispheres

	Left hemisphere cortical thickness residual				Right hemisphere cortical thickness residual			
	B	SE	β	<i>p</i>	B	SE	β	<i>p</i>
Intercept	0.03	0.04		0.549	-0.01	0.04		.785
Sex	-0.02	0.03	-.08	.368	-0.01	0.02	-.04	.633
Race/ethnicity (compared to White)								
Black	-0.03	.004	-.08	.373	-0.04	0.03	-.11	.261
Hispanic	-0.09	0.03	-.25	.006**	-0.04	0.03	-.12	.212
Asian	-0.09	0.03	-.26	.003**	-0.06	0.03	-.19	.033
Biracial	-0.06	0.03	-.22	.016**	-0.05	0.03	-.20	.030
Other	-0.02	0.04	-.07	.441	-0.03	0.04	-.06	.487
Early life stress severity	0.00	0.00	.15	.110	0.00	0.00	.02	.837
Income-to-needs ratio	0.02	0.02	.09	.371	0.04	0.02	.19	.068
Neighborhood disadvantage	-0.01	0.00	-.20	.038*	-0.00	0.00	-.10	.336

Note. * $p < .05$, ** $p < .01$.

Table 3. Regions significantly associated with age that were used in exploratory analyses.

Region	Association of cortical thickness with age	p-value
Frontal		
R caudal middle frontal	-.19	.032
L lateral orbitofrontal	-.34	<.001
R lateral orbitofrontal	-.26	.004
L medial orbitofrontal	-.34	<.001
R medial orbitofrontal	-.41	<.001
L paracentral	-.20	.028
R paracentral	-.28	.001
R pars opercularis	-.35	<.001
L pars orbitalis	-.21	.019
R pars orbitalis	-.26	.003
L pars triangularis	-.23	.008
R pars triangularis	-.35	<.001
L rostral middle frontal	-.22	.014
R rostral middle frontal	-.27	.003
L superior frontal	-.30	<.001
R superior frontal	-.23	.009
Parietal		
L inferior parietal	-.23	.008
R inferior parietal	-.22	.014
L precuneus	-.27	.002
R precuneus	-.31	<.001
L superior parietal	-.24	.008
R superior parietal	-.32	<.001
Temporal		
R bankssts	-.27	.003
L fusiform	-.23	.011
R fusiform	-.35	<.001
R superior temporal	-.20	.028
L transverse temporal	-.26	.004
R transverse temporal	-.28	.002
Occipital		
L lingual	-.23	.009
R lingual	-.22	.014
Cingulate and insula		
L posterior cingulate	-.23	.011
R posterior cingulate	-.23	.009
L insula	-.23	.009

Note. L=left; R=right; bankssts=Banks of the Superior Temporal Sulcus.