HHS Public Access

Author manuscript

Biol Psychiatry Glob Open Sci. Author manuscript; available in PMC 2022 August 25.

Published in final edited form as:

Biol Psychiatry Glob Open Sci. 2022 July ; 2(3): 253–262. doi:10.1016/j.bpsgos.2022.03.005.

A Social Gradient of Cortical Thickness in Adolescence: Relationships With Neighborhood Socioeconomic Disadvantage, Family Socioeconomic Status, and Depressive Symptoms

Jonas G. Miller,

Vanessa López,

Jessica L. Buthmann,

Jordan M. Garcia,

Ian H. Gotlib

Department of Psychology, Stanford University, Stanford, California

Abstract

BACKGROUND: Mental and physical health are affected by family and neighborhood socioeconomic status (SES). Accelerated maturation 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 putative markers of brain maturation in adolescents.

METHODS: In 120 adolescents 13 to 18 years of age, we examined family and neighborhood SES in relation to cortical thickness adjusted for age. We also examined whether cortical thickness was related to depressive symptoms and explored regions of interest.

RESULTS: Controlling for age, neighborhood socioeconomic disadvantage was associated with a thinner cortex in the left hemisphere (standardized $\beta = -0.20$), which was related to more severe depressive symptoms (standardized $\beta = -0.33$). Family SES was not significantly associated with age-adjusted mean cortical thickness in either hemisphere after controlling for relevant covariates. In exploratory, covariate-adjusted analyses of cortical thickness at the regional level, neighborhood socioeconomic disadvantage was associated with reduced cortical thickness in the left superior frontal gyrus (standardized $\beta = -0.27$), fusiform gyrus (standardized $\beta = -0.20$), and insula (standardized $\beta = -0.21$), whereas family SES was positively associated with cortical thickness in the right lateral and right medial orbitofrontal cortex (standardized $\beta = 0.21$ and standardized $\beta = 0.19$, respectively) and left transverse temporal gyrus (standardized $\beta = 0.22$).

CONCLUSIONS: Our findings provide evidence for a social gradient of cortical thickness during adolescence. Adolescents living in less advantaged community or family contexts appear to have a thinner cortex according to global and regional measures. Reduced cortical thickness in the left hemisphere may indicate increased risk for depression in adolescence.

Address correspondence to Jonas G. Miller, Ph.D., at jgmiller@stanford.edu.

DISCLOSURES

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

Supplementary material cited in this article is available online at https://doi.org/10.1016/j.bpsgos.2022.03.005.

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 with individuals from high-SES backgrounds, individuals with fewer resources are at increased risk for health problems across the lifespan, including cardiovascular disease, dementia, obesity, and depression (1,4,5). Although the effects of SES on health are strongest at lower levels of SES (6,7), it is important to note that they are present across strata, pointing to a social gradient in health that is relevant to the entire population (8). SES can be assessed at multiple levels, including family and neighborhood (9). 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., health care access, pollution burden) (10,11). Although family and neighborhood SES are related, their associations with each other are moderate (12). Further, family and neighborhood SES may have dissociable effects on health, may be related to distinct health outcomes, and may affect health via different pathways (11,13,14). These differences between family and neighborhood SES effects may be particularly relevant during adolescence, a period of increasing autonomy during which neighborhoods are a primary context outside of the home (15).

One mechanism that might underlie the SES–health gradient is rates of biological aging (16), including brain maturation (17,18). 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) (19). Children and adolescents from socioeconomically disadvantaged backgrounds are at greater risk for exposure to events and conditions that are believed to accelerate the pace of development, including early-life adversity characterized by threat (20), environmental pollutants and contaminants (21), and reduced access to safe green spaces (22). These SES-related exposures and disparities have been linked to biological aging metrics such as telomere shortening and advanced pubertal maturation (23–25), which are risk factors for, or possibly consequences of, mental health difficulties (26–29).

A growing body of research has investigated the relationship between socioeconomic disadvantage and brain-based measures of maturation (12,18). Cortical thickness decreases with age (30,31), possibly due to increases in synaptic pruning or in myelination across development (32), and has been used as a neural marker of maturation (17,23,33,34). A recent systematic review of the literature suggests that low SES is consistently associated with reduced cortical thickness (23). The majority of prior studies have focused on family-level measures of SES (35–38). For example, Mackey *et al.* (36) 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. Using a large sample of individuals 3 to 20 years of age, Piccolo *et al.* (39) found steeper age-related decreases in global cortical thickness during childhood that level off later in adolescence at low, compared with high, levels of family income and parent education. Conversely, Lawson *et al.* (37) found that less parent education was associated with reduced cortical thickness in specific frontal regions, including the right anterior cingulate and left superior frontal gyrus. These SES-related differences in cortical

thickness may have implications for adolescent mental health, given that advanced cortical thinning has been associated with the development of depressive symptoms (40,41) and with altered attention and arousal processes that are implicated in depression (42).

More recently, researchers have considered whether neighborhood socioeconomic disadvantage is associated with putative measures of brain maturation (43–47). Compared with the literature on family SES and cortical thickness, however, findings concerning the nature of the relationship between neighborhood SES and cortical thickness are less clear. Researchers have found that socioeconomic disadvantage at the neighborhood level is associated with reduced cortical thickness in adults (44,48). Similarly, using data from the Adolescent Brain Cognitive Development (ABCD) Study, Vargas *et al.* (49) 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. 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 (45,50) or have found that neighborhood disadvantage is associated with increased cortical thickness in temporal lobe regions (51). Taken together, it is unclear whether neighborhood SES is associated with either global or regional measures of cortical thickness in adolescents.

In this study, we examined associations of family and neighborhood SES with cortical thickness, controlling for age, in a community sample of adolescents. Given that prior studies have linked measures of family SES and, to a lesser extent, neighborhood SES to cortical thickness (23,49,52) and that family and neighborhood SES may make distinct contributions to health and brain structure (11,53), we hypothesized that family and neighborhood SES would be independently associated with global measures of cortical thickness. Further, we expected that advanced cortical thinning would be associated with increased depressive symptoms. Finally, we conducted exploratory analyses of relationships among measures of SES, regional measures of cortical thickness, 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 when adolescents were 9 to 13 years of age included inability to undergo magnetic resonance imaging (e.g., had metal implants, braces), a history of neurologic disorder or major medical illness, serious cognitive or physical challenges that might interfere with the ability to complete procedures, nonfluent speaker of English, and, for females, the onset of menses. Participants were reassessed at approximately 2-year intervals. This analysis included adolescents who had usable neuroimaging data at the third wave of assessments, conducted between January 2018 and June 2021 ($N = 120$). We used residential addresses to compute neighborhood socio-economic disadvantage at the Census-block group level. We used data on parental

education and family income-to-needs ratios to create an index of family SES. 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) (54). As described in (55), ADI maps neighborhood socioeconomic disadvantage at the Census-block group level using Census and 2018 American Community Survey data. ADI uses a factor analysis of measures of educational attainment, poverty, housing quality, and unemployment to identify overall neighborhood socio-economic disadvantage. ADI converts these factor scores into deciles representing the amount of socioeconomic disadvantage in a given neighborhood relative to other neighborhoods in California.

FreeSurfer Processing

Scan acquisition parameters are presented in the Supplement. 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 (56). Each segmentation was visually inspected for quality assurance according to the protocols established by the global consortium ENIGMA (Enhancing Neuro Imaging Genetics through Meta Analysis) (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 Children's Depression Inventory (57). Participants reported on their depressive symptoms over the preceding 2 weeks. For each item, participants endorsed 1 of 3 statements indicating graded severity of symptoms. We summed responses to yield a score of depressive symptom severity.

Covariates

Prior studies have found evidence of sex differences in cortical thinning (58). Further, being a person of color in the United States who experiences culture-dependent discrimination may affect the pace of biological aging (59). Finally, neighborhood- and family-level SES are often correlated. Therefore, in regression models, we tested whether neighborhood socioeconomic disadvantage is independently associated with cortical thickness over and above age, sex, race/ethnicity, and family SES effects.

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 using measures of parental education and family income-to-needs ratio. Specifically, we computed average parental education and 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 reported the educational attainment of both parents, household income in the past year, and the number of people living in the household. Parent education levels ranged from no General Education Development/high school diploma (coded as 1) to professional/doctorate degree (coded as 8). Rather than report an exact dollar amount, parents reported household income in bins on a 10-point scale: ≤$5000; $5001 to $10,000; $10,001 to $15,000; $15,001 to $25,000; $25,001 to $35,000; $35,001 to $50,000; $50,001 to $ $75,000; $75,001 to $100,000; $100,001 to $150,000; and ≥$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 United States 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 $94,450. We divided the midpoint value of the endorsed household income bin by the low-income limit for the household size to produce the income-to-needs ratio. Parental education and income-to-needs ratio were significantly correlated ($r = 0.47$, $p < .001$); thus, we standardized and averaged these measures to create a single index of family SES to compare with our single index of neighborhood disadvantage.

Statistical Analyses

We first tested the associations between age and cortical thickness averaged within each hemisphere. When age was significantly associated with cortical thickness, we tested associations between neighborhood socioeconomic disadvantage and cortical thickness controlling for age and then conducted 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 was associated with depressive symptoms. In exploratory analyses, we repeated these analytic steps for cortical thickness of individual regions as defined by the Desikan-Killiany atlas. We conducted all analyses using the lavaan package in R software (60) to use full-information maximum likelihood estimation to account for missing data (61). We present information on missing data in the Supplement.

RESULTS

Participant characteristics are presented in Table 1. On average, participants came from relatively advantaged socio-economic 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 <1.

Neighborhood Disadvantage and Age-Adjusted Cortical Thickness in the Left and Right Hemispheres

Age was negatively associated with mean cortical thickness in the left ($r = −0.25$, SE = 0.084, 95% CI = −0.42 to −0.09, $p = .003$) and right ($r = −0.29$, SE = 0.082, 95% CI = −0.45 to −0.13, $p < .001$) hemispheres (Figure 1). Controlling for age, neighborhood socioeconomic disadvantage was negatively associated with cortical thickness in the left and right hemispheres (β = −0.22, SE = 0.084, 95% CI = −0.38 to −0.05, $p = .010$ and

β = −0.17, SE = 0.085, 95% CI = −0.33 to 0.00, p = .051, respectively). Family SES was positively associated with cortical thickness in the left and right hemispheres after controlling for age (β = 0.18, SE = 0.085, 95% CI = 0.02 to 0.35, p = .033 and β = 0.22, SE = 0.083, 95% CI = 0.05 to 0.37, p = .012, respectively). As expected, neighborhood socioeconomic disadvantage and family SES were negatively intercorrelated (r = −0.49, SE = 0.066, 95% CI = −0.62 to −0.36, p < .001). Age was not associated with neighborhood socioeconomic disadvantage, family SES, or depressive symptoms (all p values >.103). Correlations among all study variables are available at https://osf.io/gxd39/.

We conducted regression analyses to determine whether the observed associations between neighborhood socioeconomic disadvantage and age-adjusted cortical thickness remained significant after controlling for family SES, sex, and race/ethnicity. The results of the fully adjusted models are presented in Table 2. The effect of neighborhood socio-economic disadvantage on cortical thickness in the left hemisphere remained statistically significant; however, the effects of neighborhood on cortical thickness in the right hemisphere and the effects of family SES on cortical thickness in both hemispheres were no longer statistically significant after adjusting for covariates. Figure 2 shows the association between neighborhood socioeconomic disadvantage and left hemisphere cortical thickness.

We conducted follow-up regression analyses to test whether cortical thickness values were associated with concurrent depressive symptoms. Adjusting for covariates, lower cortical thickness in the left hemisphere was associated with more severe depressive symptoms (β = −0.32, SE = 0.147, 95% CI = −0.61 to −0.04, p = .027). This relationship was independent of the effects of sex (β = 0.23, SE = 0.087, 95% CI = 0.06 to 0.40, p = .008) and family SES (β = −0.31, SE = 0.101, 95% CI = −0.51 to −0.11, p = .002). Age, cortical thickness in the right hemisphere, neighborhood socioeconomic disadvantage, and race/ethnicity were not significantly associated with depressive symptoms in this model (all p values >.072).

Exploratory Analyses of Neighborhood Disadvantage and Family SES With Age-Adjusted Regional Cortical Thickness

We conducted exploratory analyses of associations between age and cortical thickness in each Desikan-Killiany atlas region (68 total). Age was significantly and negatively associated with thickness in 18 frontal regions, 6 parietal regions, 6 temporal regions, 2 occipital regions, 3 cingulate regions, and the left insula (Table 3). We used these regions in analyses exploring associations with SES (Table 3). Controlling for age, neighborhood socioeconomic disadvantage was negatively and significantly associated with cortical thickness in four frontal regions: the right caudal middle frontal, left rostral middle frontal, and left and right superior frontal gyrus. Neighborhood socioeconomic disadvantage was also negatively and significantly associated with cortical thickness in 2 temporal regions (the left and right fusiform gyrus), the left and right lingual gyrus in the occipital lobe, and the left insula. It should be noted, however, that these effects were not significant using false discovery rate (FDR)–adjusted p values. After adding sex, family SES, and race/ethnicity as additional covariates, only the associations of socioeconomic disadvantage with the left superior frontal gyrus (β = −0.25, SE = 0.097, 95% CI = −0.44 to −0.06, p = .011, FDR p = .081), left fusiform gyrus (β = −0.23, SE = 0.097, 95% CI = −0.42 to −0.04, p = .018, FDR

p = .081), and left insula (β = −0.20, SE = 0.096, 95% CI = −0.39 to −0.01, p = .042, FDR p = .126) remained statistically significant. Cortical thickness in these 3 regions were weakly, and not significantly, negatively associated with severity of depressive symptoms in models adjusting for covariates (all uncorrected p values <.051).

In exploratory analyses controlling for age, family SES was positively and significantly associated with cortical thickness in ten frontal regions (right caudal middle frontal, bilateral lateral orbitofrontal, right medial orbitofrontal, bilateral pars opercularis, right pars orbitalis, left rostral middle frontal, and right superior frontal gyrus), 3 temporal regions (right fusiform, right superior temporal, and right transverse temporal gyrus), and 2 occipital regions (left and right lingual gyrus) (Table 4). After adding sex, neighborhood socioeconomic disadvantage, and race/ethnicity as additional covariates, only the associations of family SES with the right lateral and right medial orbitofrontal cortex (β = 0.21, SE = 0.100, 95% CI = 0.02 to 0.41, p = .034, FDR p = .212 and β = 0.19, SE = 0.097, 95% CI = 0.00 to 0.38, p = .049, FDR p = .212, respectively) and left transverse temporal gyrus (β = 0.22, SE = 0.102, 95% CI = 0.02 to 0.42, p = .033, FDR p = .212) remained statistically significant. Cortical thickness in these 3 regions was weakly, and not significantly, negatively associated with severity of depressive symptoms in models adjusting for covariates (all uncorrected p values >.089).

We present tests of nonlinear associations between age and cortical thickness, tests of interaction effects between age and SES variables, and exploratory analyses of cortical thickness in regions that were not significantly associated with age in the Supplement.

DISCUSSION

Accelerated maturation is one mechanism that might underlie SES–health gradients (16,17). SES, however, is a complex multilevel construct (9). Family- and neighborhood-level SES are moderately correlated but may have dissociable effects on health, 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 independent relationships with development. Here, we found that neighborhood socioeconomic disadvantage is associated with reduced cortical thickness over and above the effects of family SES. In analyses of age-adjusted global cortical thickness, this relationship was only significant for the left hemisphere after including covariates. In contrast, the associations of lower family SES with lower age-adjusted cortical thickness in the left and right hemispheres were not significant in models that included covariates. Our exploratory analyses identified effects of neighborhood socioeconomic disadvantage and family SES on age-adjusted cortical thickness in distinct brain regions. Although the majority of tested associations between SES measures and regional cortical thickness did not reach statistical significance, they were consistently inverse for neighborhood disadvantage and positive for family SES. Taken together, our findings suggest that neighborhood socioeconomic disadvantage is associated with advanced cortical thinning globally in the left hemisphere and that neighborhood and family SES may be associated with cortical thickness within different regions. Further, lower cortical thickness in the left hemisphere was related to more severe depressive symptoms, suggesting that putative measures of brain maturation can inform our understanding of risk

for depression during adolescence, especially among individuals from socioeconomically disadvantaged communities.

Our findings are consistent with research suggesting a relationship between low neighborhood SES and advanced cortical thinning (44,48,49). Gianaros *et al.* (44) found that neighborhood socioeconomic disadvantage was negatively associated with global cortical thickness in adults. Here, we similarly observed reduced age-adjusted mean cortical thickness in adolescents who were living in more disadvantaged neighborhoods. After including covariates, this relationship was only significant for the left hemisphere. One explanation for this lateralized finding is that we might have needed a larger sample size to reliably detect the effects of SES on average cortical thickness 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 (53), 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 in left prefrontal regions may explain the relationship between neighborhood SES and language skills (53). Certainly, this interpretation is speculative, and further research is necessary to replicate our findings and to assess the relationship between advanced cortical thinning and cognitive development.

Our exploratory analyses identified relationships between neighborhood SES and cortical thickness in some of the same prefrontal regions that were reported in analyses of adolescents in the ABCD Study (49), 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). In our study, associations between depressive symptoms and regional cortical thickness were weak and not statistically significant. Future longitudinal research with larger samples should test the possibility that global and regional measures of advanced cortical thinning mediate the associations between SES and depressive symptoms in adolescence.

The specific mechanisms by which neighborhood and family 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, experiences, and environmental exposures that are more prevalent in low-SES contexts, such as life stress (41), childhood maltreatment (66), reduced cognitive stimulation (50), and pollution (67). In low-SES contexts, 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 (68). Conversely, it is also possible that accelerated pruning reflects the strengthening of neural processes that are used more frequently in socioeconomically disadvantaged neighborhood and family contexts. Further research is needed to clearly

identify the psychosocial, environmental, and biological paths that explain more precisely how living in more disadvantaged neighborhoods or less affluent family contexts leads to advanced cortical thinning.

Our results add to a growing literature suggesting that low SES is associated with accelerated cortical maturation (18,23). Interestingly, neighborhood SES was more consistently associated with age-adjusted cortical thickness at significant levels across analyses than was family SES. This finding was surprising given that most prior studies linking SES to advanced cortical thinning have focused on family SES (36,39). There are 3 possible explanations for this inconsistency. First, the association between family SES and cortical thickness may vary over the course of development (23). Indeed, Piccolo *et al.* (39) found that family SES-related differences in cortical thickness were more pronounced in early than in mid- to late adolescence. Our participants were 13 to 18 years of age, and we may have been underpowered to identify statistically significant family SES effects on global metrics of 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 five study limitations. First, it is possible that we needed a larger sample to identify statistically significant effects of both neighborhood and family SES. Second, our findings do not provide causal evidence for the relationships among SES, cortical thickness, and depressive symptoms, nor do they yield specific mechanistic pathways between these variables. As a related point, although we covaried for race/ethnicity 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 (69). Third, our analyses are cross-sectional. We collected neuroimaging data at earlier assessments, but our T1-weighted scan parameters, combined with scanning younger participants, led to poor cortical segmentation in FreeSurfer. Thus, we do not have the longitudinal data to examine changes in cortical thickness over time, which is important for fully testing differences in pace of development. Fourth, our analysis focused on cortical thickness as a putative measure of brain maturation (17,23). Recently, investigators have combined multiple brain morphology measures with machine learning to consider deviations between brain-predicted-age and chronological age (brainAGE) (70). Further, Rakesh *et al.* (47) found that youth living in more disadvantaged neighborhoods exhibit increased brainAGE earlier in adolescence followed by decreases such that, by late adolescence, their brainAGE is similar to levels observed in peers from more advantaged neighborhoods. Thus, considering trajectories versus cross-sectional assessments may uncover different patterns of relationships with SES. Longitudinal studies of the associations between SES and brainAGE metrics will continue to be an important direction for the field to explore. Finally, although our participants came from a wide range of socioeconomic backgrounds, our sample, on average, was relatively advantaged and not representative of the U.S. population. Relations of neighborhood and family SES with

cortical thickness may be more robust at lower levels of SES, which has been reported in the literature examining SES-related health and developmental disparities (8). Nevertheless, the SES–health gradient appears to be relevant to the entire population (71,72). Our findings suggest that even across neighborhoods with moderate to high levels of SES, variation in disadvantage has meaningful implications for cortical thickness during adolescence.

Despite these limitations, our findings provide evidence for a social gradient of cortical thickness during adolescence. Based on global and regional measures of cortical thickness, adolescents living in less advantaged communities have a thinner cortex. Left hemisphere cortical thickness may contribute to or be a consequence of heightened risk for depression in adolescence.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

ACKNOWLEDGMENTS

This research was supported by the National Institutes of Health (Grant No. R37MH101495 [to IHG]) and the Stanford University Precision Mental Health and Integrated Diagnostics Center (to IHG).

We thank Sache Coury, Rachel Weisenburger, and Johanna Walker for their assistance with data collection and organization, as well as the families who participated in this study.

REFERENCES

1. Reiss F (2013): Socioeconomic inequalities and mental health problems in children and adolescents: A systematic review. Soc Sci Med 90:24–31. [PubMed: 23746605]
2. Adler NE, Boyce T, Chesney MA, Cohen S, Folkman S, Kahn RL, Syme SL (1994): Socioeconomic status and health. The challenge of the gradient. Am Psychol 49:15–24. [PubMed: 8122813]
3. Hackman DA, Farah MJ (2009): Socioeconomic status and the developing brain. Trends Cogn Sci 13:65–73. [PubMed: 19135405]
4. Goodman E (1999): The role of socioeconomic status gradients in explaining differences in US adolescents' health. Am J Public Health 89:1522–1528. [PubMed: 10511834]
5. Kivimäki M, Batty GD, Pentti J, Shipley MJ, Sipilä PN, Nyberg ST, et al. (2020): Association between socioeconomic status and the development of mental and physical health conditions in adulthood: A multicohort study. Lancet Public Health 5:e140–e149. [PubMed: 32007134]
6. Backlund E, Sorlie PD, Johnson NJ (1996): The shape of the relationship between income and mortality in the United States. Evidence from the National Longitudinal Mortality Study. Ann Epidemiol 6:12–20; discussion 21–22. [PubMed: 8680619]
7. Ecob R, Smith GD (1999): Income and health: What is the nature of the relationship? Soc Sci Med 48:693–705. [PubMed: 10080369]
8. Hackman DA, Farah MJ, Meaney MJ (2010): Socioeconomic status and the brain: Mechanistic insights from human and animal research. Nat Rev Neurosci 11:651–659. [PubMed: 20725096]
9. Krieger N, Williams DR, Moss NE (1997): Measuring social class in US public health research: Concepts, methodologies, and guidelines. Annu Rev Public Health 18:341–378. [PubMed: 9143723]
10. Macintyre S, Ellaway A, Cummins S (2002): Place effects on health: How can we conceptualise, operationalise and measure them? Soc Sci Med 55:125–139. [PubMed: 12137182]
11. Chen E, Paterson LQ (2006): Neighborhood, family, and subjective socioeconomic status: How do they relate to adolescent health? Health Psychol 25:704–714. [PubMed: 17100499]

12. Farah MJ (2017): The neuroscience of socioeconomic status: Correlates, causes, and consequences. Neuron 96:56–71. [PubMed: 28957676]
13. Leventhal T, Dupéré V (2011): Moving to Opportunity: Does long-term exposure to "low-poverty" neighborhoods make a difference for adolescents? Soc Sci Med 73:737–743. [PubMed: 21821323]
14. Pickett KE, Pearl M (2001): Multilevel analyses of neighbourhood socioeconomic context and health outcomes: A critical review. J Epidemiol Community Health 55:111–122. [PubMed: 11154250]
15. Boardman JD, Saint Onge JM (2005): Neighborhoods and adolescent development. Child Youth Environ 15:138–164. [PubMed: 21984874]
16. Adams JM, White M (2004): Biological ageing: A fundamental, biological link between socio-economic status and health? Eur J Public Health 14:331–334. [PubMed: 15369043]
17. Gur RE, Moore TM, Rosen AFG, Barzilay R, Roalf DR, Calkins ME, et al. (2019): Burden of environmental adversity associated with psychopathology, maturation, and brain behavior parameters in youths. JAMA Psychiatry 76:966–975. [PubMed: 31141099]
18. Tooley UA, Bassett DS, Mackey AP (2021): Environmental influences on the pace of brain development. Nat Rev Neurosci 22:372–384. [PubMed: 33911229]
19. Belsky J (2019): Early-life adversity accelerates child and adolescent development. Curr Dir Psychol Sci 28:241–246.
20. Foster H, Brooks-Gunn J, Martin A (2007): Poverty/socioeconomic status and exposure to violence in the lives of children and adolescents. In: Flannery DJ, Vazsonyi AT, Waldman ID, editors. The Cambridge Handbook of Violent Behavior and Aggression New York: Cambridge University Press, 664–687.
21. Adler NE, Stewart J (2010): Health disparities across the lifespan: Meaning, methods, and mechanisms. Ann N Y Acad Sci 1186:5–23. [PubMed: 20201865]
22. Wen M, Zhang X, Harris CD, Holt JB, Croft JB (2013): Spatial disparities in the distribution of parks and green spaces in the USA. Ann Behav Med 45(suppl 1):S18–S27. [PubMed: 23334758]
23. Colich NL, Rosen ML, Williams ES, McLaughlin KA (2020): Biological aging in childhood and adolescence following experiences of threat and deprivation: A systematic review and meta-analysis. Psychol Bull 146:721–764. [PubMed: 32744840]
24. Tyrka AR, Price LH, Kao HT, Porton B, Marsella SA, Carpenter LL (2010): Childhood maltreatment and telomere shortening: Preliminary support for an effect of early stress on cellular aging. Biol Psychiatry 67:531–534. [PubMed: 19828140]
25. Miri M, de Prado-Bert P, Alahabadi A, Najafi ML, Rad A, Moslem A, et al. (2020): Association of greenspace exposure with telomere length in preschool children. Environ Pollut 266:115228. [PubMed: 32763773]
26. Hamilton JL, Hamlat EJ, Stange JP, Abramson LY, Alloy LB (2014): Pubertal timing and vulnerabilities to depression in early adolescence: Differential pathways to depressive symptoms by sex. J Adolesc 37:165–174. [PubMed: 24439622]
27. Lee Y, Styne D (2013): Influences on the onset and tempo of puberty in human beings and implications for adolescent psychological development. Horm Behav 64:250–261. [PubMed: 23998669]
28. Henje Blom E, Han LKM, Connolly CG, Ho TC, Lin J, LeWinn KZ, et al. (2015): Peripheral telomere length and hippocampal volume in adolescents with major depressive disorder. Transl Psychiatry 5:e676. [PubMed: 26556285]
29. Humphreys KL, Sisk LM, Manczak EM, Lin J, Gotlib IH (2020): Depressive symptoms predict change in telomere length and mitochondrial DNA copy number across adolescence. J Am Acad Child Adolesc Psychiatry 59:1364–1370.e2. [PubMed: 31628984]
30. Wierenga LM, Langen M, Oranje B, Durston S (2014): Unique developmental trajectories of cortical thickness and surface area. Neuroimage 87:120–126. [PubMed: 24246495]
31. Parker N, Patel Y, Jackowski AP, Pan PM, Salum GA, Pausova Z, et al. . (2020): Assessment of neurobiological mechanisms of cortical thinning during childhood and adolescence and their implications for psychiatric disorders [published correction appears in JAMA Psychiatry 2020; 77:1195]. JAMA Psychiatry 77:1127–1136. [PubMed: 32584945]

32. Sowell ER, Peterson BS, Thompson PM, Welcome SE, Henkenius AL, Toga AW (2003): Mapping cortical change across the human life span. Nat Neurosci 6:309–315. [PubMed: 12548289]
33. Proskovec AL, Rezich MT, O'Neill J, Morsey B, Wang T, Ideker T, et al. (2020): Association of epigenetic metrics of biological age with cortical thickness. JAMA Netw Open 3:e2015428. [PubMed: 32926115]
34. Aycheh HM, Seong JK, Shin JH, Na DL, Kang B, Seo SW, Sohn KA (2018): Biological brain age prediction using cortical thickness data: A large scale cohort study. Front Aging Neurosci 10:252. [PubMed: 30186151]
35. Noble KG, Houston SM, Brito NH, Bartsch H, Kan E, Kuperman JM, et al. (2015): Family income, parental education and brain structure in children and adolescents. Nat Neurosci 18:773–778. [PubMed: 25821911]
36. Mackey AP, Finn AS, Leonard JA, Jacoby-Senghor DS, West MR, Gabrieli CFO, Gabrieli JDE (2015): Neuroanatomical correlates of the income-achievement gap. Psychol Sci 26:925–933. [PubMed: 25896418]
37. Lawson GM, Duda JT, Avants BB, Wu J, Farah MJ (2013): Associations between children's socioeconomic status and prefrontal cortical thickness. Dev Sci 16:641–652. [PubMed: 24033570]
38. McDermott CL, Seidlitz J, Nadig A, Liu S, Clasen LS, Blumenthal JD, et al. (2019): Longitudinally mapping childhood socioeconomic status associations with cortical and subcortical morphology. J Neurosci 39:1365–1373. [PubMed: 30587541]
39. Piccolo LR, Merz EC, He X, Sowell ER, Noble KG, Pediatric Imaging, Neurocognition, Genetics Study (2016): Age-related differences in cortical thickness vary by socioeconomic status. PLoS One 11:e0162511. [PubMed: 27644039]
40. Bos MGN, Peters S, van de Kamp FC, Crone EA, Tamnes CK (2018): Emerging depression in adolescence coincides with accelerated frontal cortical thinning. J Child Psychol Psychiatry 59:994–1002. [PubMed: 29577280]
41. Bartlett EA, Klein DN, Li K, DeLorenzo C, Kotov R, Perlman G (2019): Depression severity over 27 months in adolescent girls is predicted by stress-linked cortical morphology. Biol Psychiatry 86:769–778. [PubMed: 31230728]
42. Peterson BS, Warner V, Bansal R, Zhu H, Hao X, Liu J, et al. (2009): Cortical thinning in persons at increased familial risk for major depression. Proc Natl Acad Sci U S A 106:6273–6278. [PubMed: 19329490]
43. Ramphal B, DeSerisy M, Pagliaccio D, Raffanello E, Rauh V, Tau G, et al. (2020): Associations between amygdala-prefrontal functional connectivity and age depend on neighborhood socioeconomic status. Cereb Cortex Commun 1:tgaa033. [PubMed: 32984815]
44. Gianaros PJ, Kuan DCH, Marsland AL, Sheu LK, Hackman DA, Miller KG, Manuck SB (2017): Community socioeconomic disadvantage in midlife relates to cortical morphology via neuroendocrine and cardiometabolic pathways. Cereb Cortex 27:460–473. [PubMed: 26498832]
45. Wrigglesworth J, Ryan J, Vijayakumar N, Whittle S (2019): Brain-derived neurotrophic factor DNA methylation mediates the association between neighborhood disadvantage and adolescent brain structure. Psychiatry Res Neuroimaging 285:51–57. [PubMed: 30771753]
46. Tooley UA, Mackey AP, Ciric R, Ruparel K, Moore TM, Gur RC, et al. (2020): Associations between neighborhood SES and functional brain network development [published correction appears in Cereb Cortex 2021; 31:2307]. Cereb Cortex 30:1–19.
47. Rakesh D, Cropley V, Zalesky A, Vijayakumar N, Allen NB, Whittle S (2021): Neighborhood disadvantage and longitudinal brain-predicted-age trajectory during adolescence. Dev Cogn Neurosci 51:101002. [PubMed: 34411954]
48. Krishnadas R, Kim J, McLean J, Batty GD, McLean JS, Millar K, et al. (2013): The envirome and the connectome: Exploring the structural noise in the human brain associated with socioeconomic deprivation. Front Hum Neurosci 7:722. [PubMed: 24273501]
49. Vargas T, Damme KSF, Mittal VA (2020): Neighborhood deprivation, prefrontal morphology and neurocognition in late childhood to early adolescence. Neuroimage 220:117086. [PubMed: 32593800]

50. Rosen ML, Sheridan MA, Sambrook KA, Meltzoff AN, McLaughlin KA (2018): Socioeconomic disparities in academic achievement: A multimodal investigation of neural mechanisms in children and adolescents. Neuroimage 173:298–310. [PubMed: 29486324]
51. Whittle S, Vijayakumar N, Simmons JG, Dennison M, Schwartz O, Pantelis C, et al. (2017): Role of positive parenting in the association between neighborhood social disadvantage and brain development across adolescence. JAMA Psychiatry 74:824–832. [PubMed: 28636697]
52. Brito NH, Noble KG (2014): Socioeconomic status and structural brain development. Front Neurosci 8:276. [PubMed: 25249931]
53. Taylor RL, Cooper SR, Jackson JJ, Barch DM (2020): Assessment of neighborhood poverty, cognitive function, and prefrontal and hippocampal volumes in children. JAMA Netw Open 3:e2023774. [PubMed: 33141160]
54. Kind AJH, Buckingham WR (2018): Making neighborhood-disadvantage metrics accessible - The neighborhood atlas. N Engl J Med 378:2456–2458. [PubMed: 29949490]
55. Miller JG, Dennis EL, Heft-Neal S, Jo B, Gotlib IH (2022): Fine particulate air pollution, early life stress, and their interactive effects on adolescent structural brain development: A longitudinal tensor-based morphometry study. Cereb Cortex 32:2156–2169. [PubMed: 34607342]
56. Desikan RS, Ségonne F, Fischl B, Quinn BT, Dickerson BC, Blacker D, et al. (2006): An automated labeling system for subdividing the human cerebral cortex on MRI scans into gyral based regions of interest. Neuroimage 31:968–980. [PubMed: 16530430]
57. Kovács M (2003): Children's Depression Inventory (CDI): Technical Manual Update. North Tonawanda, NY: Multi-Health Systems.
58. Mutlu AK, Schneider M, Debbané M, Badoud D, Eliez S, Schaer M (2013): Sex differences in thickness, and folding developments throughout the cortex. Neuroimage 82:200–207. [PubMed: 23721724]
59. Rewak M, Buka S, Prescott J, De Vivo I, Loucks EB, Kawachi I, et al. (2014): Race-related health disparities and biological aging: Does rate of telomere shortening differ across blacks and whites? Biol Psychol 99:92–99. [PubMed: 24686071]
60. Rosseel Y (2012): lavaan: An R package for structural equation modeling. J Stat Softw 48:1–36.
61. Kline RB (2011): Principles and Practice of Structural Equation Modeling, 3rd ed. New York: Guilford Press.
62. Vigneau M, Beaucousin V, Hervé PY, Duffau H, Crivello F, Houdé O, et al. (2006): Meta-analyzing left hemisphere language areas: Phonology, semantics, and sentence processing. Neuroimage 30:1414–1432. [PubMed: 16413796]
63. Ohira H, Nomura M, Ichikawa N, Isowa T, Iidaka T, Sato A, et al. (2006): Association of neural and physiological responses during voluntary emotion suppression. Neuroimage 29:721–733. [PubMed: 16249100]
64. Asami T, Takaishi M, Nakamura R, Yoshida H, Yoshimi A, Whitford TJ, et al. (2018): Cortical thickness reductions in the middle frontal cortex in patients with panic disorder. J Affect Disord 240:199–202. [PubMed: 30077161]
65. Suh JS, Schneider MA, Minuzzi L, MacQueen GM, Strother SC, Kennedy SH, Frey BN (2019): Cortical thickness in major depressive disorder: A systematic review and meta-analysis. Prog Neuro-psychopharmacol Biol Psychiatry 88:287–302.
66. Kelly PA, Viding E, Wallace GL, Schaer M, De Brito SA, Robustelli B, McCrory EJ (2013): Cortical thickness, surface area, and gyrification abnormalities in children exposed to maltreatment: Neural markers of vulnerability? Biol Psychiatry 74:845–852. [PubMed: 23954109]
67. Beckwith T, Cecil K, Altaye M, Severs R, Wolfe C, Percy Z, et al. (2020): Reduced gray matter volume and cortical thickness associated with traffic-related air pollution in a longitudinally studied pediatric cohort. PLoS One 15:e0228092. [PubMed: 31978108]
68. McLaughlin KA, Sheridan MA, Nelson CA (2017): Neglect as a violation of species-expectant experience: Neurodevelopmental consequences. Biol Psychiatry 82:462–471. [PubMed: 28392082]
69. Oakes JM (2004): The (mis)estimation of neighborhood effects: Causal inference for a practicable social epidemiology. Soc Sci Med 58:1929–1952. [PubMed: 15020009]

70. Cole JH, Marioni RE, Harris SE, Deary IJ (2019): Brain age and other bodily 'ages': Implications for neuropsychiatry. Mol Psychiatry 24:266–281. [PubMed: 29892055]

71. Adler NE, Rehkopf DH (2008): U.S. disparities in health: Descriptions, causes, and mechanisms. Annu Rev Public Health 29:235–252. [PubMed: 18031225]

72. Noble KG, McCandliss BD, Farah MJ (2007): Socioeconomic gradients predict individual differences in neurocognitive abilities. Dev Sci 10:464–480. [PubMed: 17552936]

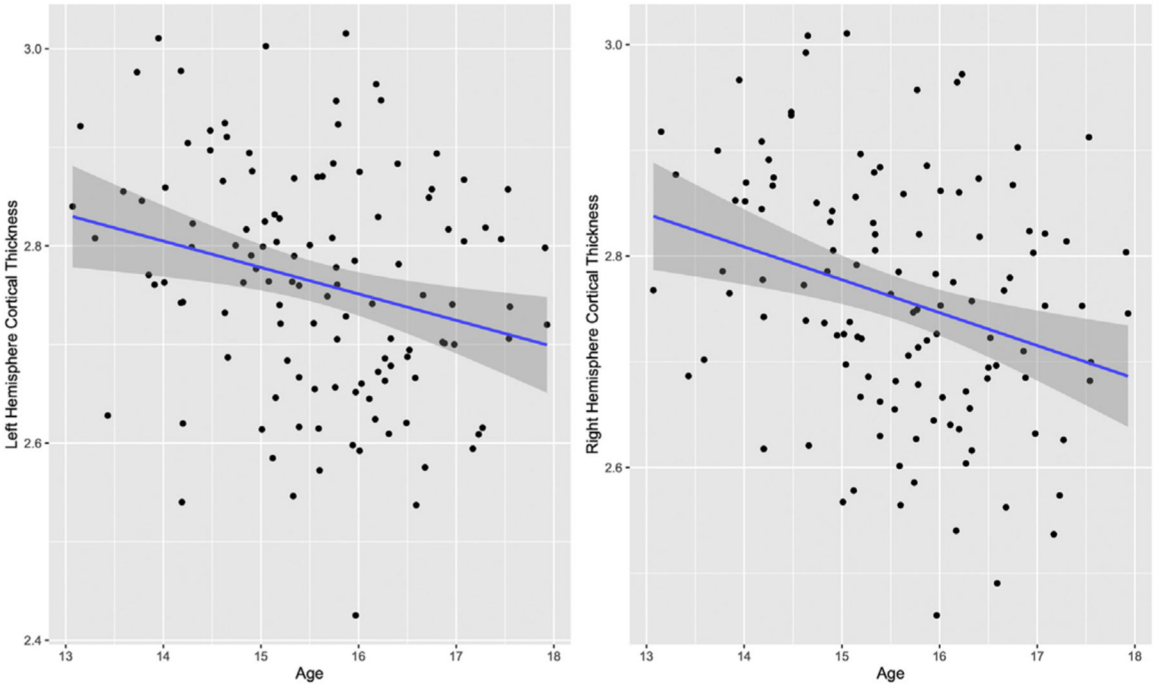

Figure 1.
Associations of age and cortical thickness in the left and right hemispheres.

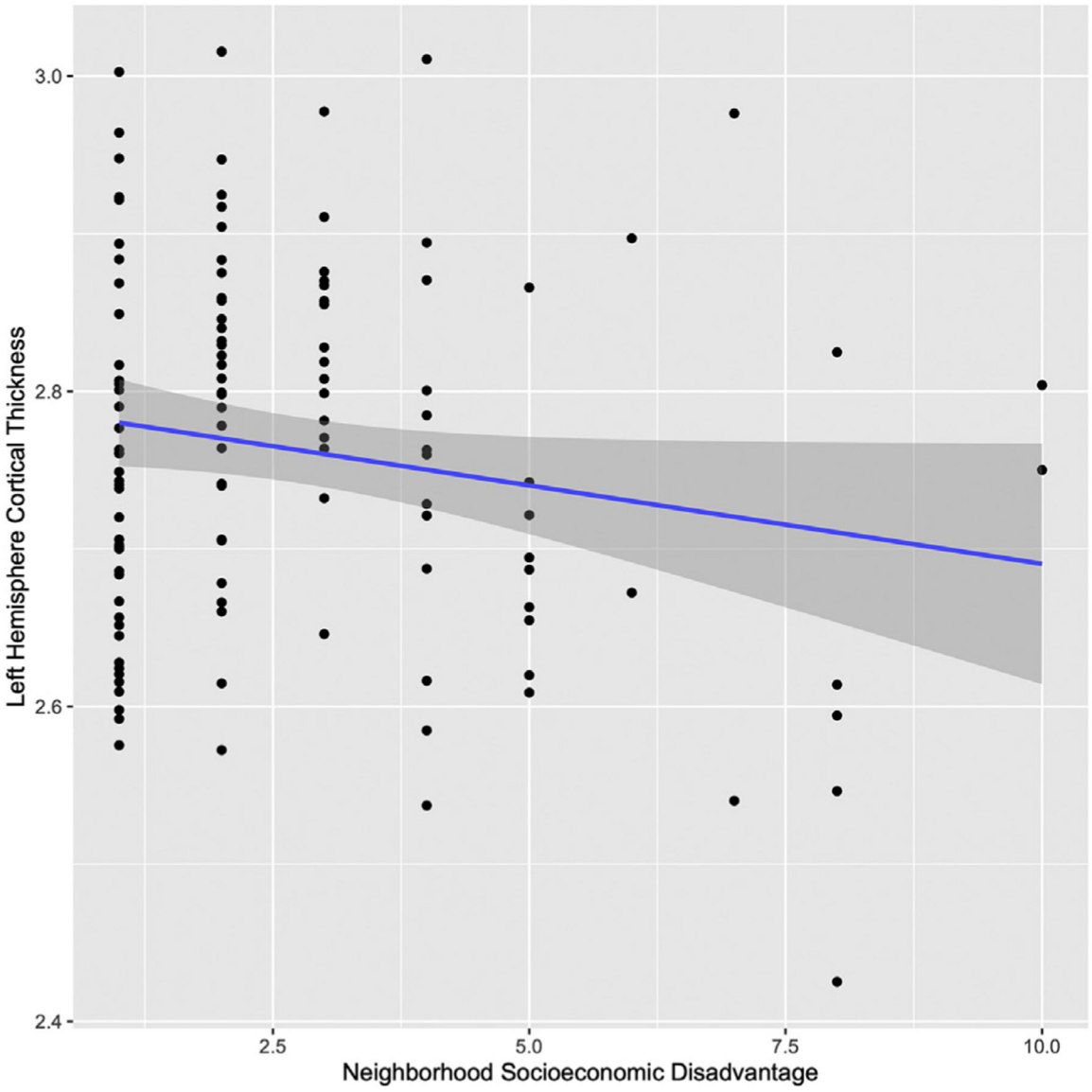

Figure 2.
Association of neighborhood socioeconomic disadvantage with cortical thickness in the left hemisphere.

Table 1.

Participant Characteristics

Characteristics	Mean (SD) [Range] or %
Sex, Male	53.3%
Age at Scan, Years	15.58 (1.10) [13–18]
Ethnicity/Race	
Asian	11.7%
Biracial	17.5%
Black	8.3%
Hispanic	9.2%
Other	6.7%
White	46.7%
Parental Education	
GED/high school diploma	3%
Some college	19%
2-Year college degree	12%
4-Year college degree	38%
Master's degree	19%
Professional degree	8%
Doctorate degree	1%
Income-to-Needs Ratio	1.24 (0.45) [0.07–1.98]
Neighborhood Disadvantage (State Decile)	2.77 (2.07) [1–10]

GED, General Education Development.

Table 2.

Regression Models for Associations of Neighborhood Socioeconomic Disadvantage and Family SES With Cortical Thickness in the Left and Right Hemispheres

	Left Hemisphere Cortical Thickness				Right Hemisphere Cortical Thickness			
Variable	**β**	**SE**	**95% CI**	***p***	**β**	**SE**	**95% CI**	***p***
Age	−0.33	0.078	−0.48 to −0.18	<.001[a]	−0.34	0.079	−0.49 to −0.18	<.001[a]
Sex	−0.12	0.083	−0.28 to 0.04	.154	−0.06	0.085	−0.22 to 0.11	.494
Race/Ethnicity (Compared With White)								
Asian	−0.29	0.080	−0.45 to −0.13	<.001[a]	−0.21	0.084	−0.37 to −0.05	.011[b]
Biracial	−0.17	0.087	−0.34 to −0.003	.046[b]	−0.19	0.088	−0.37 to −0.02	.030[b]
Black	−0.11	0.088	−0.28 to 0.06	.214	−0.13	0.090	−0.31 to 0.04	.138
Hispanic	−0.20	0.086	−0.37 to −0.03	.018[b]	−0.09	0.090	−0.27 to 0.09	.317
Other	−0.09	0.083	−0.25 to 0.07	.276	−0.08	0.085	−0.25 to 0.08	.331
Family SES	0.03	0.100	−0.17 to 0.22	.773	0.13	0.101	−0.07 to 0.32	.214
Neighborhood Disadvantage	−0.19	0.096	−0.37 to −0.002	.047[b]	−0.08	0.099	−0.27 to 0.12	.448

SES, socioeconomic status.

[a] $p < .01$.

[b] $p < .05$.

Table 3.

Regions Significantly Associated With Age That Were Used in Exploratory Analyses of Neighborhood Socioeconomic Disadvantage

Region	Association of Cortical Thickness With Age	*p*	Association of Neighborhood Disadvantage With Cortical Thickness (Controlling for Age)	SE	95% CI	*p*	FDR *p*
Frontal							
R caudal middle frontal	−0.19	.028	−0.19	0.087	−0.35 to −0.01	.034[a]	.149
L lateral orbitofrontal	−0.34	<.001	−0.14	0.085	−0.31 to 0.02	.091	.268
R lateral orbitofrontal	−0.26	.002	0.01	0.089	−0.17 to 0.18	.926	.953
L medial orbitofrontal	−0.34	<.001	−0.02	0.087	−0.19 to 0.15	.821	.889
R medial orbitofrontal	−0.41	<.001	−0.05	0.064	−0.22 to 0.11	.516	.753
L paracentral	−0.20	.023	−0.13	0.089	−0.30 to 0.05	.159	.327
R paracentral	−0.28	.001	−0.12	0.087	−0.29 to 0.05	.157	.327
L pars opercularis	−0.18	.047	−0.15	0.088	−0.32 to 0.03	.100	.269
R pars opercularis	−0.36	<.001	−0.08	0.086	−0.25 to 0.09	.336	.521
L pars orbitalis	−0.23	.006	−0.02	0.090	−0.20 to 0.16	.828	.889
R pars orbitalis	−0.26	.003	−0.12	0.088	−0.29 to 0.06	.187	.364
L pars triangularis	−0.23	.008	−0.04	0.090	−0.21 to 0.14	.669	.807
R pars triangularis	−0.35	<.001	−0.04	0.086	−0.21 to 0.13	.646	.807
L rostral middle frontal	−0.22	.011	−0.19	0.086	−0.36 to −0.02	.026[a]	.130
R rostral middle frontal	−0.27	.001	−0.11	0.088	−0.28 to 0.06	.199	.367
L superior frontal	−0.30	<.001	−0.23	0.082	−0.39 to −0.06	.006[b]	.061
R superior frontal	−0.23	.007	−0.20	0.085	−0.37 to −0.04	.017[a]	.119
Parietal							
L inferior parietal	−0.23	.006	−0.10	0.089	−0.28 to 0.07	.244	.413
R inferior parietal	−0.22	.014	−0.00	0.090	−0.18 to 0.17	.980	.980
L precuneus	−0.27	.001	−0.10	0.088	−0.27 to 0.07	.248	.413
R precuneus	−0.32	<.001	−0.04	0.087	−0.21 to 0.13	.653	.807
L superior parietal	−0.24	.005	−0.08	0.089	−0.26 to 0.09	.343	.522

Region	Association of Cortical Thickness With Age	*p*	Association of Neighborhood Disadvantage With Cortical Thickness (Controlling for Age)	SE	95% CI	*p*	FDR *p*
R superior parietal	−0.32	<.001	−0.02	0.087	−0.19 to 0.15	.838	.889
Temporal							
R bankssts	−0.27	.001	−0.05	0.089	−0.22 to 0.13	.593	.798
L fusiform	−0.23	.008	−0.25	0.083	−0.41 to −0.09	.002[b]	.061
R fusiform	−0.35	<.001	−0.22	0.081	−0.38 to −0.07	.006[b]	.061
R superior temporal	−0.20	.023	−0.15	0.088	−0.32 to 0.02	.092	.268
L transverse temporal	−0.26	.002	−0.13	0.087	−0.30 to 0.04	.131	.317
R transverse temporal	−0.28	.001	−0.16	0.086	−0.33 to 0.01	.057	.199
Occipital							
L lingual	−0.23	.007	−0.19	0.086	−0.36 to −0.02	.026[a]	.130
R lingual	−0.22	.011	−0.17	0.087	−0.34 to −0.00	.046[a]	.179
Cingulate and Insula							
L posterior cingulate	−0.23	.008	−0.02	0.090	−0.20 to 0.15	.809	.889
R posterior cingulate	−0.23	.006	−0.05	0.089	−0.22 to 0.13	.586	.798
R isthmus cingulate	−0.18	.046	0.13	0.089	−0.04 to 0.31	.136	.317
L insula	−0.23	.007	−0.23	0.084	−0.39 to −0.06	.007[b]	.061

bankssts, banks of the superior temporal sulcus; FDR, false discovery rate; L, left; R, right.

[a] $p < .05$.

[b] $p < .01$.

Biol Psychiatry Glob Open Sci. Author manuscript; available in PMC 2022 August 25.

Table 4.

Regions Significantly Associated With Age That Were Used in Exploratory Analyses

Region	Association of Family SES With Cortical Thickness (Controlling for Age)	SE	95% CI	*p*	FDR *p*
Frontal					
R caudal middle frontal	0.25	0.083	0.08 to 0.41	.003[a]	.035[b]
L lateral orbitofrontal	0.22	0.081	0.06 to 0.38	.007[a]	.041[b]
R lateral orbitofrontal	0.19	0.085	0.03 to 0.36	.022[b]	.070
L medial orbitofrontal	−0.03	0.086	−0.20 to 0.14	.702	.723
R medial orbitofrontal	0.18	0.080	0.02 to 0.34	.027[b]	.075
L paracentral	0.10	0.089	−0.08 to 0.27	.281	.378
R paracentral	0.11	0.087	−0.06 to 0.28	.190	.293
L pars opercularis	0.20	0.086	0.03 to 0.37	.019[b]	.067
R pars opercularis	0.17	0.083	0.01 to 0.33	.040[b]	.100
L pars orbitalis	0.05	0.090	−0.12 to 0.23	.551	.643
R pars orbitalis	0.17	0.085	0.01 to 0.34	.043[b]	.100
L pars triangularis	0.15	0.087	−0.02 to 0.32	.090	.166
R pars triangularis	0.09	0.085	−0.08 to 0.26	.302	.390
L rostral middle frontal	0.21	0.085	0.04 to 0.38	.013[b]	.051
R rostral middle frontal	0.21	0.085	0.05 to 0.38	.012[b]	.051
L superior frontal	0.11	0.086	−0.06 to 0.28	.193	.294
R superior frontal	0.23	0.083	0.07 to 0.40	.005[a]	.035[b]
Parietal					
L inferior parietal	0.08	0.089	−0.10 to 0.25	.395	.477
R inferior parietal	0.04	0.090	−0.14 to 0.21	.682	.723
L precuneus	0.12	0.087	−0.05 to 0.29	.174	.294
R precuneus	0.12	0.086	−0.05 to 0.28	.178	.294

Region	Association of Family SES With Cortical Thickness (Controlling for Age)	SE	95% CI	*p*	FDR *p*
L superior parietal	0.11	0.088	−0.06 to 0.28	.212	.309
R superior parietal	0.09	0.087	−0.08 to 0.26	.312	.390
Temporal					
R bankssts	0.01	0.090	−0.17 to 0.19	.908	.908
L fusiform	0.10	0.088	−0.07 to 0.27	.250	.350
R fusiform	0.24	0.080	0.09 to 0.40	.002[a]	.035[b]
R superior temporal	0.19	0.087	0.02 to 0.36	.028[b]	.075
L transverse temporal	0.24	0.084	0.08 to 0.40	.004[a]	.035[b]
R transverse temporal	−0.16	0.086	−0.01 to 0.33	.062	.128
Occipital					
L lingual	0.21	0.084	0.05 to 0.38	.012[b]	.051
R lingual	0.25	0.083	0.09 to 0.41	.003[a]	.035[b]
Cingulate and Insula					
L posterior cingulate	0.05	0.089	−0.13 to 0.22	.588	.664
R posterior cingulate	−0.04	0.089	−0.21 to 0.14	.691	.723
R isthmus cingulate	−0.15	0.088	−0.32 to 0.02	.088	.166
L insula	0.17	0.088	−0.00 to 0.33	.056	.123

bankssts, banks of the superior temporal sulcus; L, left; R, right; SES, socioeconomic status.

[a] $p < .01$.

[b] $p < .05$.