NEW RESEARCH RACE & DISPARITIES

Check for updates

Considerations When Accounting for Race and Ethnicity in Studies of Poverty and Neurodevelopment

Clementine Semanaz[a], MSc, Akhgar Ghassabian[b], MD, PhD, Scott Delaney[a], ScD, JD, MPH, Fang Fang[c], MD, PhD, David R. Williams[a], PhD, MPH, Henning Tiemeier[a,*], MD, PhD

Objective: Poverty and systemic racism are intertwined. Children of marginalized racial and ethnic identities experience higher levels of poverty and adverse psychiatric outcomes. Thus, in models of poverty and neurodevelopment, race and ethnicity, as proxies for exposure to systemic disadvantage, are regularly considered confounders. Recently, however, some researchers have claimed that using race and ethnicity as confounders is statistically dubious, and potentially socially damaging. Instead, they argue for the use of variables measuring other social determinants of health (SDoH). We explore this approach herein.

Method: Data are from 7,836 children 10 years of age in the The Adolescent Brain Cognitive Development℠ (ABCD) Study. We fit mixed regression models for the association of household poverty measures with psychiatric symptoms, magnetic resonance imaging (MRI)–derived cortical measures, and cognition with and without (1) race and ethnicity adjustment, (2) poverty-by–race and ethnicity interaction terms, and (3) alternative SDoH variables. Propensity-based weights were used to calibrate the sample to key US demographics.

Results: For psychiatric and cognitive outcomes, poverty–outcome relationships differed across racial and ethnic groups (interaction of poverty by race and ethnicity, $p < .05$). For MRI-derived outcomes, adjusting for race and ethnicity changed the estimate of the impact of poverty. Alternative SDoH adjustment could not fully account for the impact of race and ethnicity on the associations explored.

Conclusion: Poverty and both race and ethnicity combine to influence neurodevelopment. Results suggest that the effects of poverty are generally inconsistent across race and ethnicity, which supports prior research demonstrating the nonequivalence of SDoH indicators by race and ethnicity. Studies exploring these relationships should assess the interaction between poverty and race and ethnicity and/or should stratify when appropriate. Replacing race and ethnicity with alternative SDoH may induce bias.

Plain language summary: This study examined whether race and ethnicity interact with poverty in measuring psychiatric and neurodevelopment outcomes, and whether alternative social determinants of health (SDoH) variables can instead account for the identified impact of race and ethnicity. Using data from 7,836 10-year-old children in the The Adolescent Brain Cognitive Development℠ (ABCD) Study, researchers compared different regression models for the association of poverty with psychiatric, imaging, and cognitive measures, with and without race and ethnicity adjustments, poverty-by-race and ethnicity interaction terms, and alternative SDoH variables. Results showed a statistically significant interaction between poverty and race and ethnicity in multiple outcome measures, which were not accounted for by alternative SDoH variables. Findings suggest that it is important to assess interactions between poverty and race/ethnicity in research study designs, or apply stratification when appropriate.

Key words: poverty; race; ethnicity; cerebral cortex; adolescent behavior

J Am Acad Child Adolesc Psychiatry 2026;65(1):76-87.

Nearly 11 million children in the United States lived in poverty in 2022.[1] Economically disadvantaged children have fewer opportunities for intellectual stimulation and face greater systemic obstacles to healthy neurodevelopment than children from higher-income homes.[2,3] As a result, children experiencing poverty are more prone to develop emotional and behavioral problems compared to children raised in more affluent families.[4-7] Furthermore, adolescent brain development may be affected by chronic stress, which is consistently associated with poverty.[3,8] Therefore, poverty may have a profound impact on child neurodevelopment.

Separately, studies have consistently documented racial and ethnic differences in the prevalence of internalizing and externalizing symptoms[9] and in subthreshold psychotic symptoms (such as delusion-like thoughts and hallucinations) leading to distress and impairment, known as psychotic-like experiences.[10,11] In most countries, race and ethnicity are closely tied to poverty because of their connection with social factors such as education level and housing location that determine income and wealth. Both systemic and interpersonal racism contribute to poverty[12]; and, because of this association, along with the link between race, ethnicity, and psychiatric symptoms, race and ethnicity are often considered confounding variables in the relationship between poverty and psychiatric outcomes.

Recently, however, some researchers have opposed using race and ethnicity variables as confounders, lodging

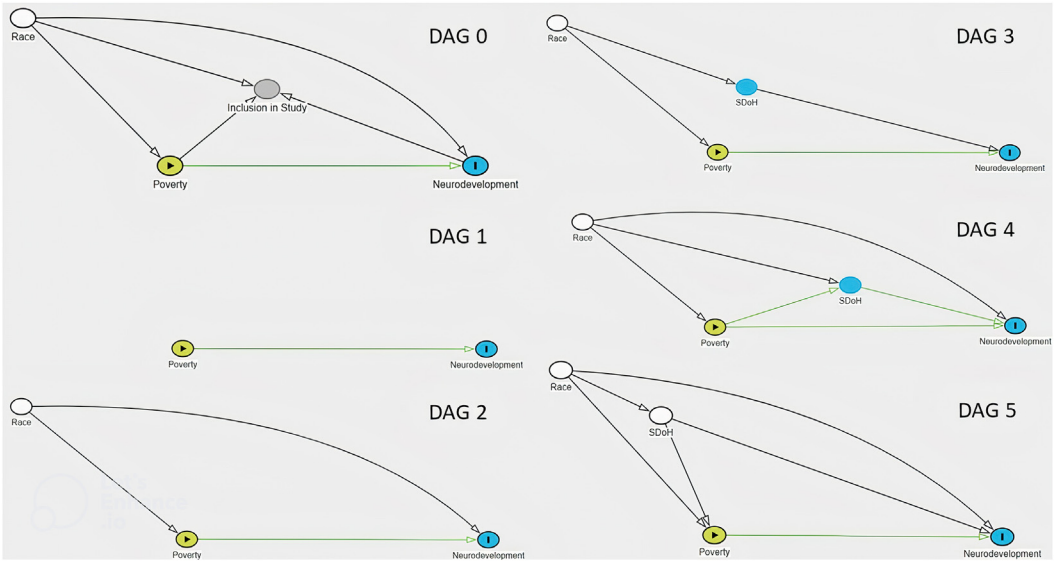
both statistical and philosophical objections. For example, some studies have proffered that poverty status and race and ethnicity are statistically collinear, such that inclusion of the latter may induce statistical challenges, and that using race and ethnicity variables incorrectly presumes a biological basis for racial categories, which can perpetuate racist tropes.[7,13,14] As an alternative, these studies contend that race and ethnicity should be replaced with variables assessing structural inequities that have an impact on health and well-being in children and adults, known as social determinants of health (SDoH), that are presumably mediating factors between race and ethnicity (as a proxy for racism exposure) and poverty or between race and ethnicity and neurodevelopment. SDoH are the conditions in the environments where people are born, live, learn, and play that affect health outcomes and functioning. Domains of SDoH include economic stability, education access, neighborhood environment, and social and community context. Studies espousing this alternative approach include studies that are seeking to develop new measures of exposure to systemic, interpersonal, and internalized racism, as a class of SDoH that have been historically neglected, including a focus on how upstream dimensions of racism shape the distribution of many SDoH. These studies (as well as this one) recognize that this comprehensive approach is critical to advancing sound, antiracist science.[15-17]

Racial and ethnic identity are closely related to poverty, as well as to the gradient of socioeconomic status (SES) more generally, and the above-cited concerns raise both theoretical and empirical questions of their own. First, they overlook evidence demonstrating the effects of structural racism on health.[18,19] Race and ethnicity are social constructs. They are a way that humans have been categorized as a consequence of colonialism, oppression, and discrimination.[20] This interpretation of physical and other personal features does not arise from biological reality but, rather, from complex social processes. Over the centuries, race has become a social reality that shapes how we experience the world.[13,21] In this regard, both race and racism are real, and racism has tangible biological consequences that should be considered.[20]

Second, the arguments above do not accommodate evidence demonstrating the nonequivalence of SDoH across racial and ethnic groups.[22] Most social assets—including income, education and many others—confer fewer benefits to racially minoritized (vs White) adults. For example, there are racial differences in income at every level of education and racial differences in wealth at every level of income.[17] In this way, racism—whether structural, interpersonal, or internalized—is an SDoH that affects the distribution and effects of nearly all other SDoH. For example, geographic segregation in the United States, which is itself a product of structural racism, leads to racial differences in the purchasing power of income. There are also often fewer services in highly segregated Black areas, and the available ones tend to be poorer in quality and higher in price. On average, Black (vs White) families pay higher prices for many goods and services, including food and housing. Separately, higher levels of schooling often do not confer the same health advantage to racially minoritized adults, such that Black–White health inequities are often largest among higher educated Americans. Geographic segregation also alters many facets of social life for children of differing racial and ethnic identities. A White child growing up in a poor household in an affluent White area might have a relative experience of poverty different from that of a Black child growing up in an equally poor household in a less affluent Black neighborhood. This relative difference between one's household income and that of one's peers can lead to feelings of exclusion or social isolation, a phenomenon referred to as relative deprivation.[23]

Third, although a range of variables representing downstream effects of structural racism are available in most neuroscientific and epidemiologic datasets, it is unclear whether these indicators, such as material hardship or area deprivation, can fully explain the impact of race and ethnicity (and thus racism) on neurodevelopment. Dumornay *et al.*[24] examined several SDoH-relevant factors and racial differences in brain structures in the Adolescent Brain Cognitive Development℠ (ABCD) Study. They found that disparities in childhood adversity only partially accounted for race-related differences in gray matter volume. This suggests that the SDoH factors typically assessed insufficiently account for the totality of effects of structural, interpersonal, and internalized racism. In an editorial, Barch and Luby[13] noted that the analyses did not include some key SDoH variables. Whether these additional factors are sufficient to account for the residual confounding has not been tested. Prior research demonstrating the racial nonequivalence of SDoH factors suggests that this approach is unlikely to have the effect that its proponents intend.[22] In addition, many of the proposed SDoH indicators are mediators between poverty and neurodevelopmental outcomes, such that including them in models quantifying the unmediated impacts of poverty would be inappropriate. In this study, we examine the impact of different approaches of accounting for race and ethnicity (as a proxy for racism). We explore the interplay of race, poverty, and neurodevelopment in 3 different scenarios (Figure 1 provides an illustration using directed acyclic graphs [DAG]): (1) race and ethnicity confound the relationship between race and neurodevelopment (DAG 1 and 2); (2) the effect of poverty

FIGURE 1 Directed Acyclic Graphs (DAGs) Depicting Different Causal Models



Note: *Here the term "poverty" refers to the income status of the family, the term "race" to parent-reported race and ethnicity. The term "social determinants of health" (SDoH) denotes determinants of health other than family income; SDoH include material hardship, traumatic event, family conflict, area deprivation, child opportunity, perceived discrimination, and poverty level at school. DAG 1 represents the association between poverty and neurodevelopment assuming no confounding. This corresponds to model 1: no adjustment for race and ethnicity. DAG 2 represents the associations assuming that race is a confounder. This corresponds to model 2: adjustment for race and ethnicity. DAG 3 represents the association assuming that SDoH can replace the variable race in the model. Importantly, race cannot be directly (independently) associated with neurodevelopment, and there is no association of poverty as an antecedent with SDoH. This corresponds to model 4: adjusted for SDoH but not for race. DAG 4 represents the association with the variable SDoH but assumes that SDoH are a consequence of both race and poverty. This corresponds to model 2, as adjusting for SDoH would constitute overadjustment. DAG 5 represents the association assuming that poverty is a consequence of SDoH. This corresponds to model 5/A; adjustment for both race and SDoH are adequate if this assumption is correct. Please note color figures are available online.*

on neurodevelopment can differ by race and ethnicity, thus justifying race and ethnicity being modeled as an effect modifier; (3) and, finally, race and ethnicity can be entirely left out of models with the assumption that the SDoH variables could replace them (DAG 3, race not independently associated with neurodevelopment anymore). For comparison, we also tested a model with both SDoH and race and ethnicity as confounders in the model.

METHOD

Participants

We used data from the ABCD Study® (Curated Annual Release 4.0), an investigation of neurodevelopment across 21 US sites.[25] Consenting parents and assenting children 9 to 10 years of age were recruited through a school-based sampling strategy (Supplement 1, available online). We used baseline data collected from September 2016 to October 2018, and 1-year follow-up mental health data.[26] We excluded participants with missing data on parental education, sex, age, race and ethnicity, family income, or number of people in the household, or if they were identified by their parents as Asian or Mixed Race. One participant was randomly selected per family to account for intra-sibling correlations. A total of 7,836 children were included in one or more analyses (Figure S1, available online).

Measures

Race and Ethnicity. Children's race and ethnicity were parent-reported. We assumed that parents' responses aligned with children's self-perceptions, as child-reported data were not collected. Response options for race were White, Black/African American, American Indian/Native American, Alaska Native, Native Hawaiian, Guamanian, Samoan, Other Pacific Islander, Asian Indian, Chinese, Filipino, Japanese, Korean, Vietnamese, Other Asian, and Other. Parents selected as many as applicable. Hispanic/Latino ethnicity was assessed separately using the following categories: White Non-Hispanic, Black Non-Hispanic, Hispanic/Latino, Asian, and Other Mixed Race and Other Races. Because of the small sample size and heterogeneity, the Asian and Others groups were excluded from analyses.

Poverty

Poverty was estimated using baseline income-to-needs ratio (INR), which captures the annual income that a family

earns relative to the federal poverty line for a family of that size. INR was calculated by dividing total family income by the 2017 poverty threshold provided by the US Department of Health and Human Services.[27] Total family income and number of persons in the household were parent-reported. To operationalize poverty, a binary poverty variable was created (below/above 100% of the poverty line). We also performed supplemental analyses using an alternative poverty cut-off (200% of the poverty line) and a continuous INR measure.

Neurodevelopmental Outcomes

The Child Behavior Checklist (CBCL) was used to measure parent-reported externalizing and internalizing symptoms. The questionnaire comprised 112 statements, such as "Destroys their own things," with 3 possible responses: not true, sometimes true, and very true. Internalizing raw scores were derived from the following syndrome scores: anxious/depressed, withdrawn/depressed, somatic complains, social problems, and thought problems; externalizing raw scores were derived from the rule-breaking behavior and aggressive behavior subscales.[28] The internal consistency of the CBCL as measured by Cronbach alpha was 0.95.

Child-reported externalizing and internalizing symptoms were measured with the Brief Problem Monitor (BPM) form completed by the participants at 1-year follow-up. The 19-item form included statements, such as "I argue a lot," which could be rated as not true, sometimes true, and very true.[29] Although the BPM was filled out 1 year after the poverty assessment, we assume that potential changes in the families' economic situations in this time interval did not significantly affect symptoms. The internal consistency of the BPM as measured by Cronbach alpha was 0.80.

Teachers reported externalizing and internalizing symptoms of the child at baseline through the BPM–Teacher form (available in less than half of the participants) (Figure S1, available online). The 18-item form included statements such as "Disobedient at school," which could then be rated as not true, sometimes true, or very true.[29] We used raw externalizing and internalizing symptom scores. The internal consistency of the BPM–Teacher form as measured by Cronbach alpha was 0.91. Outcome information from multiple reporters were included to assess whether reporter bias may have determined the result pattern.

Children completed the Prodromal Questionnaire–Brief Child Version, a 21-item self-report questionnaire (eg, Do familiar surroundings sometimes seem strange, confusing, threatening, or unreal to you?). Consistent with previous research, standardized distress scores were calculated as the sum of endorsed questions weighted by level of distress.[30] The internal consistency as measured by Cronbach alpha was 0.89.

We examined cortical thickness and surface area from baseline magnetic resonance imaging (MRI) scans. MRI data were pre-processed using the ABCD Study pipeline as detailed by Hagler *et al.*[31] Cortical thickness and surface area can be used to predict inter-individual variability in basic and higher cognitive functions, such as perception, motor control, memory, and the ability to introspect.[32]

Cognition was evaluated using the NIH Toolbox Cognitive Battery, a comprehensive set of neurobehavioral tests that assess motor, emotional, sensory, and cognitive function. We used standardized age-corrected total cognition (Supplement 1, available online).

Additional Social Determinants of Health

The following SDoH variables were selected as race-related measures of deprivation as recommended by Barch and Luby[13]: neighborhood disadvantage, child opportunity, perceived discrimination, family conflict, family material hardship, trauma history of the child, and school's poverty (Supplement 1, available online).

Covariates

We adjusted for child age and sex and parental partnership status because the variables are likely associated with psychiatric symptoms. We also adjusted for parental education as a confounder because it is typically an antecedent of income. Including extraneous determinants of the outcome may result in more efficient estimates of the associations of interest.[33]

Statistical Analysis

Demographic characteristics of the study population are described (Table 1). Linear regression was used to estimate associations of poverty with outcomes, adjusting for age, sex, parental educational levels, and partnership status. We started our analyses with minimally adjusted models (model 1: Figure 1, DAG 1) and then introduced the race and ethnicity covariate (model 2: Figure 1, DAG 2) to test the confounding effect of the race and ethnicity variable. Next, we examined the interaction between poverty and race and ethnicity (model 3, no DAG) to test whether the effect of poverty differed by race and ethnicity.[34] Finally, for exposure–outcome associations confounded by race and ethnicity, we included the additional SDoH variables (model 4: Figure 1, DAG 3) to test whether they could accurately replace race and ethnicity. In further analyses, we ran sequences of models under the assumption that all SDoHs are determinants of poverty

TABLE 1 Study Population Characteristics by Poverty Level (N = 7,791)

	Below poverty line (n = 1,149)	Total (N = 7,791)
Age, mean (SD)	9.9 (0.6)	9.9 (0.6)
Sex		
Female, n (%)	553 (48.1)	3,693 (47.4)
Race and ethnicity		
White non-Hispanic, n (%)	232 (20.2)	4,760 (61.1)
Black non-Hispanic, n (%)	474 (41.3)	1,236 (15.8)
Hispanic/Latino, n (%)	443 (38.6)	1,795 (23.1)
Highest parental educational level		
Less than high school diploma, n (%)	90 (7.8)	145 (1.9)
High school diploma, n (%)	786 (68.4)	2,920 (37.5)
Some college, n (%)	180 (15.7)	1,137 (14.6)
Bachelor's degree or higher, n (%)	93 (8.1)	3,589 (46.1)
Parental partnership status		
Living with a partner, n (%)	452 (39.3)	5,771 (74.1)
Externalizing score, mean (SD)		
Parent-reported, standardized	0.3 (1.3)	0.0 (1.0)
Teacher-reported, standardized	0.4 (1.3)	0.0 (1.0)
Child-reported, standardized	0.2 (1.2)	0.0 (1.0)
Internalizing score, mean (SD)		
Parent-reported, standardized	0.1 (1.1)	0.0 (1.0)
Teacher-reported, standardized	0.3 (1.2)	0.0 (1.0)
Child-reported, standardized	0.2 (1.0)	0.0 (1.0)
PLEs score, mean (SD)		
Child-reported, standardized	0.2 (1.2)	0.0 (1.0)
Cortical measures, mean (SD)		
Cortical surface area, in cm^2	1,827.2 (179.5)	1,899.0 (180.8)
Cortical thickness, in μm	2,702.6 (81.6)	2,727.6 (79.7)
Cognitive score, standardized mean (SD)	−0.7 (0.9)	0.0 (1.0)
additional SDoH, mean (SD)		
Material hardship	0.8 (1.5)	0.0 (1.0)
Traumatic event	0.3 (1.3)	0.0 (1.0)
Family conflict	0.2 (1.1)	0.0 (1.0)
Area deprivation	0.6 (0.7)	0.0 (1.0)
Child opportunity	−1.0 (1.0)	0.0 (1.0)
Perceived discrimination	0.4 (1.4)	0.0 (1.0)
Poverty level at school	1.0 (0.8)	0.0 (1.0)

Note: *Percentages are presented per column. PLEs = psychotic-like experiences; SDoH = social determinants of health.*

(model 5/A: Figure 1, DAG 5 with both race and SDoH in the model). Without this assumption, adjustment for SDoH as confounder in the association between poverty and neurodevelopment would not be appropriate. All SDoH measures were standardized and included as continuous variables. Missing SDoH variables, assumed to be missing at random, were imputed using multiple imputation by chained equations.[35] The White non-Hispanic group was used as the reference, as it was the largest group. All statistical analyses were performed using Stata 17.0.

Our target population for this study was all children in the United States 9 to 10 years of age. The ABCD Study, however, is not representative of our target population, and factors driving selection bias may differ by race and ethnicity (Figure 1, DAG 0).[36] To strengthen the generalizability of ABCD Study–based results, researchers have calculated propensity-based weights for the baseline sample so that it approximates key demographic characteristics of our target population. We used these weights in all analyses as recommended by the ABCD Research Consortium. Thus, all presented results are from models using design-based weights described by Gard *et al.*[36]

In a post hoc analysis, we fitted models with interactions between race and ethnicity and our additional

SDoH to assess the extent to which SDoH variables may have different associations with outcomes in different racial groups. In theory, such interaction could confound the association between poverty and neurodevelopmental outcomes. We performed these post hoc analyses for a single outcome, namely, parent-reported externalizing symptoms, which offered both a large sample size and significant interactions between race and ethnicity and poverty.

RESULTS

Sociodemographic Characteristics

Our study population included 4,760 White non-Hispanic children (61.1%) 1,236 Black non-Hispanic children (15.8%), and 1,795 Hispanic/Latino children (23.1%). Of these 7,791 children, 1,149 (14.7%) lived below the poverty threshold between 2016 and 2018; 3,693 were girls (47.4 %); and the mean age was 9.9 years (SD = 0.6) (Table 1; Table S1, available online).

Psychiatric Symptoms

Poverty was positively associated with self-, parent-, and teacher-reported psychiatric symptoms, after adjusting for age, sex, and highest parental educational level (Table 2, model 1). When race and ethnicity were added as covariates, the associations showed only modest changes (Table 2, model 2). For brevity, we focus here on parent-reported outcomes. The coefficient estimating the association between poverty and parent-reported externalizing symptoms increased by less than 25% between the model not adjusted for race and ethnicity ($\beta 1 = 0.3$, 95% CI = 0.2-0.5, $p < .05$) and the model adjusted for race and ethnicity ($\beta 2 = 0.4$, 95% CI = 0.2-0.5, $p < .05$). Figure 2 graphs the score difference associated with poverty in relation to internalizing and externalizing symptoms by different reporters without (pink) and with (blue) adjustment for race and ethnicity. However, we found significant interactions between poverty and race and ethnicity in the associations between poverty and psychiatric symptoms (Table S2, available online), showing that model 2 (without the interaction term) was misspecified. The association between poverty and parent-reported externalizing symptoms was less marked among both the non-Hispanic Black (β for interaction = −0.3, 95% CI = −0.5 to 0.0, $p < .05$) and the Hispanic/Latino (β for interaction = −0.4, 95% CI = −0.7 to −0.2), $p < .05$) children than in non-Hispanic White children. As shown in Figure 2, regardless of reporters, the association of poverty with psychiatric symptoms was stronger among non-Hispanic White children than among non-Hispanic Black children or Hispanic/Latino children. Results were consistent when using a continuous INR measure (Figure S2, available online) and a poverty cutoff of 200% of the poverty line (Table S3, available online).

Brain Morphology and Cognition

Poverty was negatively associated with brain volume (cortical surface area: Table 3, model 1 and cortical thickness: Table S4, model 1) and cognitive functioning (Table S5, model 1). When adding the race and ethnicity variable to the models (model 2 in the respective tables), associations of poverty with brain morphology and cognition diminished. For example, the coefficient estimating the association between poverty and cortical surface area was reduced by 50% between the model not adjusted for race and ethnicity ($\beta 1 = -43.6$, 95% CI = − 57.1 to −30.0, $p < .05$) and the model adjusted for race and ethnicity ($\beta 2 = -22.2$, 95% CI = − 35.9 to −8.5, $p < .05$).

When examining interactions in these associations, the association between poverty and cognition differed by race and ethnicity. We also found some evidence that the association between poverty and cortical thickness differed by race and ethnicity (Table S4, model 3), although the interaction terms were not significant. In contrast, we found no evidence of effect modification by race and ethnicity in the association between poverty and cortical surface area (Table 3, model 3).

SDoH as Proxies for Race and Ethnicity

We replaced race and ethnicity with SDoH in models that showed confounding by race in the association of poverty and neurodevelopment (ie, brain morphology and cognition). The associations between poverty and brain morphology (Table 3, model 4; Table S4, model 4) or cognition (Table S5, model 4) diminished by more than 20% when including SDoH variables (see Methods) as covariates compared to model 1 (no race and ethnicity). Next, we included both the SDoH variables and the race and ethnicity variable in the model. We observed a 67% decrease in the association between poverty and cortical surface area (Table 3, model 5) and a 36% decrease in the association between poverty and cortical thickness (Table S4, model 5) compared to model 1. SDoH variables were therefore not able to fully capture the race and ethnicity effect, as residual confounding remained when these variables were used instead of race and ethnicity. Regardless, adjusting only for race and ethnicity or the SDoH variables did not explain as much variance in the outcomes as the model adjusted for both SDoH and race and ethnicity variables (models 2 and 4 vs model 5 in Table 3 and in Table S4 and S5, available online). This nonadditive pattern suggests that SDoH variables also account for non–race-related factors. Not all the effect

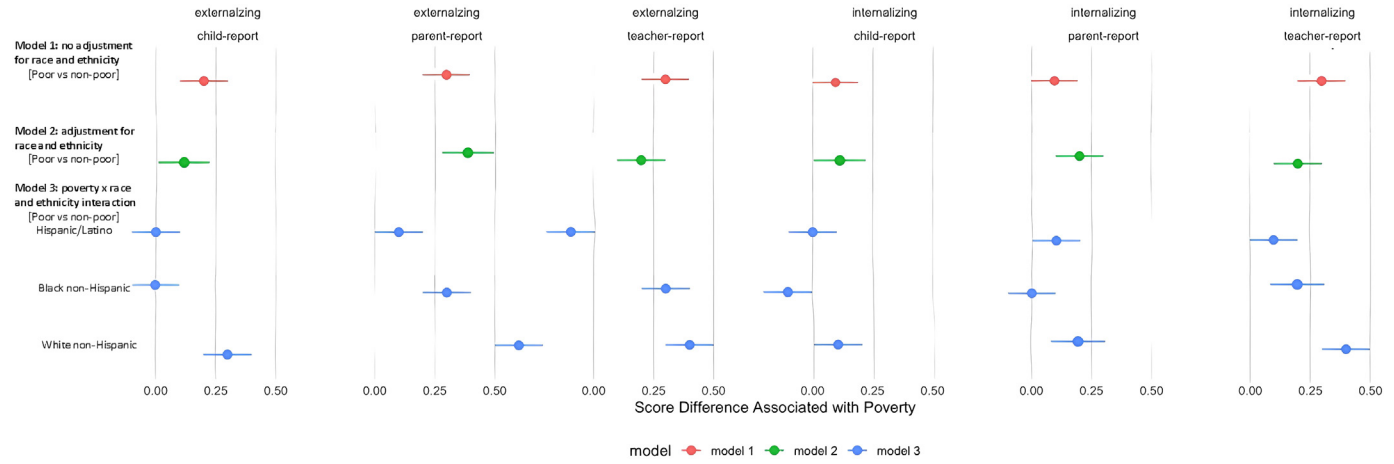
TABLE 2 Association of Poverty With Parent-Reported, Child-Reported, and Teacher-Reported Psychiatric Symptoms

		Parent-reported (n = 7,789)		Child-reported (n = 6,695)		Teacher-reported (n = 3,300)	
		Model 1	Model 2	Model 1	Model 2	Model 1	Model 2
Symptoms	**Variables**	**β [95% CI]**	**β [95% CI]**	**β [95% CI]**	**β [95% CI]**	**β [95% CI]**	**β [95% CI]**
Externalizing symptoms	Poverty	0.3 [0.2; 0.5]*	0.4 [0.2; 0.5]*	0.2 [0.1; 0.3]*	0.1 [0.1; 0.3]*	0.3 [0.1; 0.4]*	0.2 [0.0; 0.4]*
	Race and ethnicity						
	White non-Hispanic		0 (Ref)		0 (Ref)		0 (Ref)
	Black non-Hispanic		−0.1 [−0.2; 0.1]		0.1 [0.0; 0.2]*		0.5 [0.3; 0.7]*
	Hispanic/Latino		−0.1 [−0.2; 0.0]*		−0.1 [−0.1; 0.0]		0.0 [−0.1; 0.2]
Internalizing symptoms	Poverty	0.1 [0.0; 0.2]*	0.2 [0.1; 0.3]*	0.1 [0.0; 0.1]	0.1 [0.0; 0.1]	0.3 [0.1; 0.4]*	0.2 [0.1; 0.4]*
	Race and ethnicity						
	White non-Hispanic		0 (Ref)		0 (Ref)		0 (Ref)
	Black non-Hispanic		−0.3 [−0.4; −0.2]*		0.0 [0.0; 0.1]		0.0 [−0.1; 0.2]
	Hispanic/Latino		0.0 [−0.1; 0.1]		0.1 [0.0; 0.1]*		0.0 [−0.1; 0.2]
Psychotic- like experiences	Poverty			0.1 [0.0; 0.3]*	0.1 [0.0; 0.2]		
	Race and ethnicity						
	White non-Hispanic				0 (Ref)		
	Black non-Hispanic				0.2 [0.1; 0.4]*		
	Hispanic/Latino				0.1 [0.0; 0.3]		

Note: *Results are from 13 separate mixed-regression models. Age, sex, and highest parental education level and parental partnership status were included as covariates, and site location was incorporated as a random effect. Poverty was defined as 100% of the poverty line. Model 1: no adjustment for race/ethnicity. Model 2: adjustment for race/ethnicity. Table S3, available online, shows results of using a different poverty cut-off. These results reflect cross-sectional analyses at an average age of 10 years and do not account for environmental influences known to have an impact on psychiatric symptoms that social structural forces, including structural racism. may pattern.*

*$p < .05$.

FIGURE 2 Score Difference Associated With Poverty in Mixed-Effect Analysis Predicting Parent-Reported (n = 7,789), Child-Reported (n = 6,695), and Teacher-Reported (n = 3,300) Externalizing and Internalizing Symptom Scores



Note: *Please note color figures are available online.*

estimate change observed when adjusting for SDoH can thus be attributed to race and ethnicity. This also suggests that controlling for SDoH may introduce meaningful overadjustment if poverty is considered an antecedent to certain SDoH.

In a post hoc analysis, we fitted different models adjusting for SDoH and allowing SDoH to interact with race. We present the analyses of parent-reported externalizing symptoms with a number of statistically significant interactions between race and ethnicity and SDoH (Table S6, available online), suggesting that the association of SDoH with neurodevelopmental outcomes can vary by race and ethnicity. However, these interactions did not confound the association of poverty with externalizing symptoms, as we observed a constant poverty coefficient across models with and without SDoH interactions.

DISCUSSION

In this study, we observed that race and ethnicity confounded the association of poverty with neurodevelopmental outcomes. For all behavioral and cognitive outcomes, regardless of reporter, as well as for cortical thickness, we also found evidence suggesting that the effects of poverty differ for children of varying race and ethnicity. For the remaining outcome (cortical surface area), adjusting models for race and ethnicity substantially changed the estimate of the neurodevelopmental impact of poverty, but there was no evidence of effect modification. The additional SDoH variables used could not fully account for the confounding by race and ethnicity in the association of poverty with any of the outcomes tested. These results are fully consistent with social theory and prior empirical research, and they demonstrate that failing to account for race and ethnicity in studies of poverty and neurodevelopment can lead to biased estimates.

Race and Ethnicity Interactions and Confounding

Although race and ethnicity as a proxy of a broader social construct such as racism can confound the relationship between poverty and neurodevelopmental outcomes, the effect of poverty on neurodevelopment could also vary by race and ethnicity, making them effect modifiers as well. We found interactions between poverty and race and ethnicity for all psychiatric symptoms and cognition. The effect of poverty was stronger in the non-Hispanic White population. This pattern is consistent with the "diminishing returns hypothesis," in which non-Hispanic Black (vs White) individuals benefit less from increasing SES.[17,37,38] Several mechanisms may explain this pattern. One potential explanation is the effect of relative deprivation, which the American Psychological Association defines as the perception by an individual that the amount of a desired resource (eg, money) that they possess is less than some comparison standard.[39] For many Black children living in segregated areas, being poor is common. These children are surrounded by families in similar financial situations, which normalizes the experience of poverty and attenuates its consequences. Other possible mechanisms include the nonequivalence of SES across racial groups and, perhaps most significantly, the burden of racism that even high-SES Black children face. In the latter instance, structural racism restricts opportunities for healthy neurodevelopment even for high-SES Black (vs White) children; thus, poverty has fewer opportunities to steal from them.

TABLE 3 Association of Poverty With Cortical Surface Area (n = 7,496)

	Model 1 β [95% CI]	Model 2 β [95% CI]	Model 3 β [95% CI]	Model 4 β [95% CI]	Model 5 β [95% CI]
Poverty	−43.6 [−57.1; −30.0]*	−22.2 [−35.9; −8.5]*	−27.4 [−62.5; 7.8]	−24.8 [−36.4; −13.1]*	−14.5 [−26.0; −3.1]
Race and ethnicity					
White non-Hispanic		0 (Ref)	0 (Ref)		0 (Ref)
Black non-Hispanic		###*	###*		###*
Hispanic/Latino		###*	###*		###*
Poverty and race/ethnicity interaction					
Black non-Hispanic below the poverty line			11.0 [−24.3; 46.2]		
Hispanic/Latino below the poverty line			5.6 [−39.5; 50.7]		
Omnibus Wald test of interaction			*p* = .67		
Additional SDoH					
Material hardship				−7.0 [−10.9; −3.1]*	−4.8 [−8.7; −1.0]*
Traumatic event				1.4 [−2.2; 5.1]	1.5 [−2.1; 5.0]
Family conflict				−7.6 [−11.1; −4.0]*	−7.0 [−10.5; −3.5]*
Area deprivation				−1.3 [−5.9; 3.2]	−3.7 [−8.2; 0.8]
Child opportunity				20.9 [14.5; 27.2]*	5.1 [−1.4; 11.7]
Perceived discrimination				−6.9 [−10.7; −3.1]*	−3.6 [−7.4; 0.2]
Poverty level at school				−10.6 [−16.5; −4.7]*	−5.3 [−11.3; 0.8]*

Note: Results are from 16 separate mixed-regression models. Age, sex, highest parental education level, and parental partnership status were included as covariates, and site location was incorporated as a random effect. Poverty was defined as 100% of the poverty line. Model 1: no adjustment for race/ethnicity. Model 2: adjustment for race/ethnicity. Model 3: poverty by race/ethnicity interaction included. Model 4: additional SDoH adjusted. Model 5: adjustment for race/ethnicity and additional SDoH. These results reflect cross-sectional analyses at an average age of 10 years and do not account for environmental influences known to affect cortical surface area that social structural forces, including structural racism, may pattern. To avoid perpetuating discriminatory ideas/belief, estimates by racial and ethnic groups are not presented. Ref = reference; SDoH = social determinants of health; ### = hidden values.
**p < .05.*

Future studies of poverty and neurodevelopment should test for interactions with race and ethnicity. If interactions are found, analyses should be stratified by race and ethnicity. For analyses of brain morphology, stratification by race and ethnicity may still be necessary, even though we did not find significant interactions between poverty and race and ethnicity in this study. At a minimum, race and ethnicity adjustment as a potential confounder in brain morphology analyses should be considered.

SDoH as Proxies for Race and Ethnicity

Although the SDoH variables used to replace race and ethnicity appeared to be important mediators of the relationship between poverty and neurodevelopment, they explained only part of the race and ethnicity effect. Our indicators of material hardship, traumatic events, family conflict, area deprivation, child opportunity, perceived discrimination, and school-level poverty appear not to fully capture all of the information needed to explain confounding by race and ethnicity. Remaining differences between racial and ethnic groups do not imply biological differences, but simply that the included SDoH variables do not fully reflect the effects of current and historic structural, interpersonal, and internalized racism. Perceived discrimination (ie, interpersonal racism) is a measure of one's personal experiences of discrimination, and fails to capture the systemic level at which structural racism operates. The introduction of material hardship, traumatic events, family conflict, area deprivation, and child opportunity attempted to encompass more of the effects of upstream aspects of racism. However, the present results suggest that these variables explained only part of the structural racism experienced by racial and ethnic minorities. Using only these variables could leave race and ethnicity–related residual confounding. Also, these measures likely adjust for some of the downstream mediating effects of poverty, such as material hardship, and SDoH adjustment would constitute overadjustment.

Finally, the significant interactions between race and ethnicity and SDoH further suggest that associations of SDoH with neurodevelopment differ by race and ethnicity, either because these SDoH variables (like poverty) do not measure the same phenomena across different groups or that the impact of SDoH varies by race and ethnicity. The nonequivalence of different SDoH indicators across racial and ethnic groups has been noted in previous literature.[22] Even if a variable is assessed uniformly in different groups, the information obtained may not reflect the same reality. Material hardship, for example, could reflect different purchasing power of income, as some goods may be more expensive in some segregated areas. Alternatively, having a different SES may not have the same impact on the relationship between poverty and neurodevelopmental outcomes between races and ethnicities. This can also apply to some SDoH variables such as family conflict or corporal punishment. It is well known that the impact of these factors on, for example, externalizing problems can differ by race and ethnicity and social context.[40] However, the interaction between SDoH covariates and race and ethnicity had negligible effects on the association between poverty and the outcome (eg, externalizing) (Table S6, available online). This does not rule out the possibility that if SDoH other than poverty were defined as the exposure, there would be an complex interplay with race and ethnicity equal to that observed for poverty.[34] Although methods to measure structural racism directly are rapidly advancing, as of now, the measures available to researchers in nearly all psychological, neuroscientific, and epidemiological datasets are insufficient to fully explain the effects of racism.[15,41,42] Therefore, we recommend using a race and ethnicity variable when studying poverty and neurodevelopment until validated, comprehensive measures of multiple levels of racism are widely collected and made available. Researchers interested in the pathway between poverty and neurodevelopment might also want to study mediation by other SDoH.

This study has some limitations. First, the ABCD Study did not use probability sampling and is not representative of the US population. Poverty is not only related to nonparticipation, but selection effects may differ by racial and ethnic group. We mitigated this with propensity score weighting. However, other unmeasured selection characteristics of the ABCD Study population may differ by race/ethnicity and poverty. Under these circumstances, the stratified analyses would be preferred to deal with confounding by race. Second, the categorization of race and ethnicity used in this study fails to capture the full complexity of US backgrounds linked to the historical, life course, and migration factors. A more nuanced classification could allow for better control for the confounding effect of race, ethnicity, and racism. Future studies should investigate mixed racial and ethnic identities when possible, and incorporate migration history.[17] Third, poverty is an important but only one marker of SES, and does not capture all aspects of social and economic stratification and the risk factors and resources linked to them. Fourth, we did not account for regional differences in the cost of living and purchasing power, therefore failing to capture, for example, the significant financial strain of many families in some metropolitan areas. Consequently, we might have overlooked that poverty and race and ethnicity are collinear in certain regions. Moreover, the lack of strong variability and equal representation at both tails of the income spectrum across all race and ethnicity categories, along with the

significant differences in sample sizes across some subgroup analyses, needs to be acknowledged. Fifth, despite our comprehensive assessment of SDoH, we may not have fully captured all aspects of current and life course risk factors and resources linked to living and working conditions.[17] Of note, some prior research has sought to investigate factors that are downstream of both poverty and racism (eg, healthy eating).[43] This approach can be helpful when exploring pathways through which upstream determinants may have an impact on child health. However, when studying poverty, including these mediating variables may bias estimates of the impact of poverty. Finally, given our cross-sectional design, we must assume that family financial hardships are relatively stable and are not a result of child behavior.

This study suggests that race and ethnicity substantially influence the relationship between poverty and neurodevelopment. When selecting candidate variables such as race, ethnicity, and racism for adjustment in their analyses, researchers should make their assumptions and choices transparent, for example, with the use of directed acyclic graphs (DAGs), and within the context of specific racial and ethnic identities, which may differ from those considered in this analysis. Specifically, we recommend that studies examining SDoH and neurodevelopment may benefit from testing for interactions between the SDoH indicators of interest and race and ethnicity. If such interactions are found, stratified analyses (if the sample size allows) would be appropriate, even if not prespecified. In cases where no interactions are detected, it may be valuable to include race and ethnicity as covariates. To actively counter individuals who seek to co-opt well-intentioned research on racialized health inequities for racist and white supremacist ends, researchers should consider omitting confounder coefficients from their results tables. Regarding the use of alternative SDoH variables to replace race and ethnicity, the indicators of social disadvantage, discrimination, or inequity currently available to most researchers do not capture all relevant aspects of racism and thus are not sufficient to fully model race and ethnicity. Moreover, SDoH variables may be nonequivalent across different groups, further underscoring the need to consider stratified analyses as one analytic approach and to continue to identify and to empirically verify all aspects of the social environments that can have pathogenic consequences.

CRediT authorship contribution statement

Clementine Semanaz: Writing – review & editing, Writing – original draft, Visualization, Software, Methodology, Formal analysis, Data curation, Conceptualization. **Akhgar Ghassabian:** Writing – review & editing, Methodology, Conceptualization. **Scott Delaney:** Writing – review & editing, Writing – original draft, Data curation, Conceptualization. **Fang Fang:** Writing – review & editing. **David R. Williams:** Writing – review & editing. **Henning Tiemeier:** Writing – review & editing, Supervision, Methodology, Conceptualization.

This article is part of a special series devoted to addressing bias, bigotry, racism, and mental health disparities through research, practice, and policy. The 2024 Race & Disparities Team includes Deputy Editor Lisa R. Fortuna, MD, MPH, MDiv, Consulting Editor Andres J. Pumariega, MD, PhD, Diversity, Equity, and Inclusion Emerging Leaders Fellows Tara Thompson-Felix, MD, and Nina Bihani, MD, Assistant Editor Eraka Bath, MD, Deputy Editor Wanjikū F.M. Njoroge, Associate Editor Robert R. Althoff, MD, PhD, and Editor-in-Chief Douglas K. Novins, MD.

Accepted March 13, 2025.

[a]Harvard T.H. Chan School of Public Health, Boston, Massachusetts; [b]New York University Grossman School of Medicine, New York, New York; [c]Institute of Environmental Medicine, Karolinska Institutet, Stockholm, Sweden.

This project was conceived to guide student community projects and supported by the Health Resources and Services Administration (HRSA) of the U.S. Department of Health and Human Services (HHS) under grant number and title for grant amount (T76 MC000010, Maternal and Child Health Training Grant). This information or content and conclusions are those of the author and should not be construed as the official position or policy of, nor should any endorsements be inferred by HRSA, HHS or the U.S. Government.

Data Sharing: The data belong to the ABCD Consortium and access can be requested.

Disclosure: Clementine Semanaz, Akhgar Ghassabian, Scott Delaney, Fang Fang, David R. Williams, and Henning Tiemeier have reported no biomedical financial interests or potential conflicts of interest.

*Correspondence to Henning Tiemeier, MD, PhD, Department of Social and Behavioral Sciences, Harvard T.H. Chan School of Public Health, 677 Huntington Avenue, Boston, MA 20115; e-mail: tiemeier@hsph.harvard.edu

https://doi.org/10.1016/j.jaac.2025.03.007

REFERENCES

1. United States Census Bureau. Income & poverty data tables. Census.gov. Accessed March 10, 2024. https://www.census.gov/topics/income-poverty/data/tables.html
2. Brito NH, Noble KG. Socioeconomic status and structural brain development. Front Neurosci. 2014;8. Accessed April 10, 2023. https://www.frontiersin.org/articles/10.3389/fnins.2014.00276
3. Blair C, Raver CC. Poverty, Stress, and brain development: new directions for prevention and intervention. Acad Pediatr. 2016;16(3):S30-S36. https://doi.org/10.1016/j.acap.2016.01.010
4. Veldman K, Reijneveld SA, Ortiz JA, Verhulst FC, Bültmann U. Mental health trajectories from childhood to young adulthood affect the educational and employment status of young adults: results from the TRAILS study. J Epidemiol Community Health. 2015;69(6):588-593. https://doi.org/10.1136/jech-2014-204421
5. Slopen N, Fitzmaurice G, Williams DR, Gilman SE. Poverty, food insecurity, and the behavior for childhood internalizing and externalizing disorders. J Am Acad Child Adolesc Psychiatry. 2010;49(5):444-452. https://doi.org/10.1097/00004583-201005000-00005

6. Mamah D, Mutiso VN, Ndetei DM. Psychotic-like experiences among 9,564 Kenyan adolescents and young adults. Psychiatry Res. 2021;302:113994. https://doi.org/10.1016/j.psychres.2021.113994
7. Karcher NR, Schiffman J, Barch DM. Environmental risk factors and psychotic-like experiences in children aged 9-10. J Am Acad Child Adolesc Psychiatry. 2021;60(4):490-500. https://doi.org/10.1016/j.jaac.2020.07.003
8. Romeo RD. The impact of stress on the structure of the adolescent brain: implications for adolescent mental health. Brain Res. 2017;1654(Pt B):185-191. https://doi.org/10.1016/j.brainres.2016.03.021
9. McLaughlin KA, Hilt LM, Nolen-Hoeksema S. Racial/ethnic differences in internalizing and externalizing symptoms in adolescents. J Abnorm Child Psychol. 2007;35(5):801-816. https://doi.org/10.1007/s10802-007-9128-1
10. Leaune E, Dealberto MJ, Luck D, *et al*. Ethnic minority position and migrant status as risk factors for psychotic symptoms in the general population: a meta-analysis. Psychol Med. 2019;49(4):545-558. https://doi.org/10.1017/S0033291718002271
11. Earl TR, Fortuna LR, Gao S, *et al*. An exploration of how psychotic-like symptoms are experienced, endorsed, and understood from the National Latino and Asian American Study and National Survey of American Life. Ethn Health. 2015;20(3):273-292. https://doi.org/10.1080/13557858.2014.921888
12. Iceland J. Racial and ethnic inequality in poverty and affluence, 1959-2015. Popul Res Policy Rev. 2019;38(5):615-654. https://doi.org/10.1007/s11113-019-09512-7
13. Barch DM, Luby JL. Understanding social determinants of brain health during development. Am J Psychiatry. 2023;180(2):108-110. https://doi.org/10.1176/appi.ajp.20220991
14. Weissman DG, Hatzenbuehler ML, Cikara M, Barch DM, McLaughlin KA. State-level macro-economic factors moderate the association of low income with brain structure and mental health in U.S. children. Nat Commun. 2023;14(1):2085. https://doi.org/10.1038/s41467-023-37778-1
15. Dean LT, Thorpe RJ. What structural racism is (or is not) and how to measure it: clarity for public health and medical researchers. Am J Epidemiol. 2022;191(9):1521-1526. https://doi.org/10.1093/aje/kwac112
16. Hardeman RR, Homan PA, Chantarat T, Davis BA, Brown TH. Improving the measurement of structural racism to achieve antiracist health policy. Health Aff (Millwood). 2022;41(2):179-186. https://doi.org/10.1377/hlthaff.2021.01489
17. Williams DR, Mohammed SA, Leavell J, Collins C. Race, socioeconomic status and health: complexities, ongoing challenges and research opportunities. Ann N Y Acad Sci. 2010;1186:69-101. https://doi.org/10.1111/j.1749-6632.2009.05339.x
18. Nuru-Jeter AM, Michaels EK, Thomas MD, Reeves AN, Thorpe RJ, LaVeist TA. Relative roles of race versus socioeconomic position in studies of health inequalities: a matter of interpretation. Annu Rev Public Health. 2018;39:169-188. https://doi.org/10.1146/annurev-publhealth-040617-014230
19. Delaney SW. Editorial: are neural biomarkers valid for all children? J Am Acad Child Adolesc Psychiatry. 2022;61(11):1327-1328. https://doi.org/10.1016/j.jaac.2022.08.006
20. Fuentes A, Ackermann RR, Athreya S, *et al*. AAPA statement on race and racism. Am J Phys Anthropol. 2019;169(3):400-402. https://doi.org/10.1002/ajpa.23882
21. Gee GC, Ford CL. Structural racism and health inequities. Bois Rev Soc Sci Res Race. 2011;8(1):115-132. https://doi.org/10.1017/S1742058X11000130
22. Whitfield KE. Closing the Gap: Improving the Health of Minority Elders in the New Millennium. Gerontological Society of America; 2004.
23. Vanneman RD, Pettigrew TF. Race and relative deprivation in the urban United States. 1972. Accessed August 4, 2023. https://journals.sagepub.com/doi/10.1177/030639687201300404
24. Dumornay NM, Lebois LAM, Ressler KJ, Harnett NG. Racial disparities in adversity during childhood and the false appearance of race-related differences in brain structure. Am J Psychiatry. 2023;180(2):127-138. https://doi.org/10.1176/appi.ajp.21090961
25. Compton WM, Dowling GJ, Garavan H. Ensuring the best use of data: the Adolescent Brain Cognitive Development Study. JAMA Pediatr. 2019;173(9):809-810. https://doi.org/10.1001/jamapediatrics.2019.2081
26. Kim HH, McLaughlin KA, Chibnik LB, Koenen KC, Tiemeier H. Poverty, cortical structure, and psychopathologic characteristics in adolescence. JAMA Netw Open. 2022;5(11):e2244049. https://doi.org/10.1001/jamanetworkopen.2022.44049
27. Annual Update of the HHS Poverty Guidelines. Federal Register. January 31, 2017. Accessed April 7, 2023. https://www.federalregister.gov/documents/2017/01/31/2017-02076/annual-update-of-the-hhs-poverty-guidelines
28. Thompson WK, Barch DM, Bjork JM, *et al*. The structure of cognition in 9 and 10 year-old children and associations with problem behaviors: findings from the ABCD Study's baseline neurocognitive battery. Dev Cogn Neurosci. 2018;36:100606. https://doi.org/10.1016/j.dcn.2018.12.004
29. Achenbach TM, McConaughy SH, Ivanova MY, Rescorla LA. Manual for the ASEBA Brief Problem Monitor™ (BPM). ASEBA; 2011.
30. Loewy RL, Pearson R, Vinogradov S, Bearden CE, Cannon TD. Psychosis risk screening with the Prodromal Questionnaire—Brief Version (PQ-B). Schizophr Res. 2011;129(1):42-46. https://doi.org/10.1016/j.schres.2011.03.029
31. Hagler DJ, Hatton SN, Cornejo MD, *et al*. Image processing and analysis methods for the Adolescent Brain Cognitive Development Study. NeuroImage. 2019;202:116091. https://doi.org/10.1016/j.neuroimage.2019.116091
32. Kanai R, Rees G. The structural basis of inter-individual differences in human behaviour and cognition. Nat Rev Neurosci. 2011;12(4):231-242. https://doi.org/10.1038/nrn3000
33. Schisterman EF, Cole SR, Platt RW. Overadjustment bias and unnecessary adjustment in epidemiologic studies. Epidemiology. 2009;20(4):488. https://doi.org/10.1097/EDE.0b013e3181a819a1
34. Clayton D, Hills M. Chapter 26: More About Interaction. In: Clayton D, Hills M, eds. Statistical Models in Epidemiology. Oxford: Oxford University Press; 2013:261-270.
35. Roffman JL, Sipahi ED, Dowling KF, *et al*. Association of adverse prenatal exposure burden with child psychopathology in the Adolescent Brain Cognitive Development (ABCD) Study. PLoS One. 2021;16(4):e0250235. https://doi.org/10.1371/journal.pone.0250235
36. Gard AM, Hyde LW, Heeringa SG, West BT, Mitchell C. Why weight? Analytic approaches for large-scale population neuroscience data. Dev Cogn Neurosci. 2023;59:101196. https://doi.org/10.1016/j.dcn.2023.101196
37. Williams DR, Priest N, Anderson N. Understanding associations between race, socioeconomic status and health: patterns and prospects. Health Psychol. 2016;35(4):407-411. https://doi.org/10.1037/hea0000242
38. Boen C. The role of socioeconomic factors in Black-White health inequities across the life course: point-in-time measures, long-term exposures, and differential health returns. Soc Sci Med. 1982. 2016;170:63-76. https://doi.org/10.1016/j.socscimed.2016.10.008
39. American Psychological Association. Dictionary of Psychology. Accessed April 30, 2023. https://dictionary.apa.org/
40. Lapré GE, Marsee MA. The role of race in the association between corporal punishment and externalizing problems: does punishment severity matter? J Child Fam Stud. 2016;25(2):432-441. https://doi.org/10.1007/s10826-015-0250-3
41. Adkins-Jackson PB, Chantarat T, Bailey ZD, Ponce NA. Measuring structural racism: a guide for epidemiologists and other health researchers. Am J Epidemiol. 2022;191(4):539-547. https://doi.org/10.1093/aje/kwab239
42. Kuban KCK, Boynton-Jarrett R, Heeren T, O'Shea TM. A consideration of racism in pediatric epidemiologic studies. J Pediatr. 2021;239:225-227. https://doi.org/10.1016/j.jpeds.2021.08.004
43. LeWinn KZ, Bush NR, Batra A, Tylavsky F, Rehkopf D. Identification of modifiable social and behavioral factors associated with childhood cognitive performance. JAMA Pediatr. 2020;174(11):1063-1072. https://doi.org/10.1001/jamapediatrics.2020.2904