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DISCUSSION PAPER SERIES

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ABSTRACT

Early-Life Circumstances and Racial Disparities in Cognition for Older Americans: The Importance of Educational Quality and Experiences

Given the critical role of neurocognitive development in early life, this study assesses how racial differences in early-life circumstances are collectively and individually associated with racial disparities in late-life cognition. Leveraging uniquely rich information on life history from the U.S. Health and Retirement Study for non-Hispanic White (White) and non-Hispanic Black (Black) Americans 50 years or older, we employ the Blinder-Oaxaca method to decompose racial gaps in cognitive outcomes into early-life educational experiences, cohort, regional, financial, health, trauma, family relationship, demographic and genetic factors. Overall, differences in early-life circumstances are associated with 61.5% and 82.3% of the racial disparities in cognitive score and impairment, respectively. Early-life educational experience is associated with 35.2% of the disparities in cognitive score and 48.6% in cognitive impairment. Notably, school racial segregation (all segregated schooling before college) is associated with 28.8%-39.7% of the racial disparities in cognition. Policies that improve educational equity have the potential to reduce racial disparities in cognition into older ages. Clinicians may leverage early-life circumstances to promote the screening, prevention, and interventions of cognitive impairment more efficiently, thereby promoting health equity.

JEL Classification: J15, I14, J13, J14, I20, H75

Keywords: early life circumstances, life course, school segregation, quality of education, racial disparity, cognition

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Introduction

There are marked racial disparities in cognitive impairment, with the prevalence of dementia for non-Hispanic Black (Black) adults about twice that of non-Hispanic White (White) adults, after accounting for age, sex, and education.[1–5] The rising proportion of US older adults who are minorities may lead to rising socioeconomic burden associated with cognitive impairment and dementia,[3] with evidence suggesting that low education, smoking, and social isolation are among the strongest risk factors.[6,7] Most research has focused on mid-life to late-life factors, while early-life circumstances through which the racial disparities in cognition may arise and persist have received less attention.

Evidence linking factors associated early-life neurocognitive development with cognitive impairment and dementia in later life is limited. However, brain development is most rapid and plastic early in life.[8,9] Strong early-life brain development supports more complex neuritic and intraneuronal connections and cognition, conferring a young adulthood and middle age advantage[8] that may be associated with a more robust cognitive reserve and a lower risk of dementia in later life.[10,11] Early-life education represents a particularly important modifiable risk factor.[6] In addition to years of education, quality and experience of education may affect brain health. For instance, exposure to a higher concentration of minority students and segregated schools is associated with diminished occupational aspirations, expectations, and achievement among minority students.[12,13] The long-term health consequences of educational factors and their implications for racial inequities are less known, especially when accounting for various other early-life socio-environmental exposures.[14–17]

Using a nationally-representative survey and uniquely comprehensive life history data, this cross-sectional study investigated how racial differences in early-life circumstances are

associated with racial disparities in cognitive status and prevalence of cognitive impairment among older Americans. We tested the following hypotheses: 1) early-life circumstances are significantly and substantively associated with racial disparities in cognitive outcomes; 2) early-life educational experience is the most important early-life factor associated with racial disparities in cognitive outcomes, independent of years of educational attainment and other early-life factors and demographic characteristics.

Methods

Study Design and Participants

We used the Health and Retirement Study (HRS), a large nationally-representative study of older Americans aged 50 years and over. We assembled a large array of factors on early-life circumstances from three HRS components: the core survey (1995-2018); the Life History Mail Survey (LHMS) (2015, 2017); and the Enhanced Face-to-Face (EFTF) Interview (2006-2012). Details of these data sources are provided in eAppendix A and elsewhere.[18] The study followed the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guideline.

Our analysis focused on non-Hispanic White and Black participants who completed at least one cognitive assessment. The sample selection criteria are provided in Figure 1. We excluded participants who self-identified as Hispanic ethnicity and other racial/ethnic groups, and those who did not participate in the EFTF. The resulting analytical sample included 9,015 participants (7,381 White; 1,634 Black) with both LHMS and EFTF data. To optimize sample size, we assessed participants' latest wave of cognitive assessment (i.e., closest to 2018, pre-COVID-19) and corresponding demographic covariates from the same core survey. We did not

exclude participants who died since all participants had matched wave of cognitive assessment. Distribution of survey year when participants' cognition was assessed is provided in eFigure 1.

The HRS study protocol was approved by the Institutional Review Board at the University of Michigan. Data used in this study are de-identified and publicly available.

Cognitive Outcomes

The HRS assessed cognitive function using a range of tests adapted from the Telephone Interview for Cognitive Status, which demonstrated high validity for White and Black adults.[19,20] Specifically, the 27-point cognitive scale includes three cognitive tests: immediate and delayed word recall; serial sevens subtraction; and backward counting. Built on existing criteria, cognitive impairment was determined if participants' 27-point cognitive score fell below 12 (0-11 points), ranging from mild cognitive impairment to dementia.[20,21] Dementia was not separately analyzed due to small numbers.[22] The distribution of cognitive score by race is provided in eFigure 1.

Early-Life Educational Experience

Our primary explanatory variables included comprehensive measures of early-life educational experience, which were uniquely collected in the LHMS. These measures consist of two important components: family education and schooling experience (Figure 1). To assess family education, we included years of parental education, whether the respondent owned at least one shelf of books at age 10, whether the mother spent full time with children, whether she was absent in terms of time/attention, her effort in upbringing, and her teaching before the respondent turned 18. To evaluate the schooling experience, we considered whether the respondent

experienced all segregated schooling before college, experienced all public schooling before college, attended preschool, learned foreign languages in high school, and learned creative arts in high school. The rationale for the selection of variables and domains, as well as their definitions and construction, are provided in eAppendix B.

Traditional Early-Life Factors

As shown in the right panel of Figure 1, our study selected a wide spectrum of early-life circumstances following a thorough literature review, spanning seven key domains that encompassed early-life cohort, regional, financial, health, trauma, family relationship factors, and educational attainment. They are referred to hereafter as traditional early-life factors. A detailed review, justification, and description of these early-life domains and factors are provided in eAppendix B.

Covariates

Demographics (i.e., age, sex), collected at the same wave as the cognitive assessments, and genetic factors were included as covariates. Genetic factors were assessed using polygenic risk scores (PGS) (eAppendix A), enabling adjustment for biological predispositions.[23,24]

Statistical Analysis

To test for differences in sample characteristics between Black and White participants, we used Welch t-tests for continuous variables, Mann-Whitney-Wilcoxon tests for ordinal variables, and Chi-square tests for categorical variables.[25] To evaluate racial differences on a comparable scale,

we also estimated the Cohen's d standardized mean differences for each early-life circumstance between Black and White participants.

We performed a Blinder-Oaxaca Decomposition (BOD) to evaluate the association between racial differences in early-life circumstances and racial disparities in cognitive outcomes.[26] All decompositions were formulated based on Black participants; and racial disparities associated with early-life circumstances were estimated as the predicted change in cognitive outcomes of Black participants if they had the same early-life circumstances as White participants on average.[27] A more detailed description of the BOD and estimation procedures is provided in eAppendix C.

Linear models were used for cognitive score, and logit models were used for cognitive impairment. For each cognitive outcome, we estimated two decomposition models: LifeHistory_1 (Model 1) included traditional early-life factors and other covariates; and LifeHistory_2 (Model 2) additionally included early-life educational experience. In each decomposition, we evaluated the factors at two levels: the overall level, which examines the association of racial differences across all early-life circumstances; and the factor/domain level, which explored the association of individual factors or domains of circumstances, adjusting for the others. Genetic factors were included as covariates only in the sensitivity analysis due to diminished sample size (Figure 1).

To address item-level missingness of early-life factors and covariates, we performed multiple imputation[28] using a sequential regression approach.[29,30] The imputation models included all the early-life factors and demographic covariates in the decomposition analyses, and various other variables that were predictive of missing values, such as cognition, functional limitations and comorbidities. Inclusion of these predictive variables would increase the likelihood that the "missing at random" assumption of multiple imputation holds while also

addressing potential biases.[29,30] Following existing guidelines, 20 imputed datasets were produced and analyzed; and parameter estimates were pooled/combined in the BOD analyses.[31,32] Additional details are provided in eAppendix B.

We conducted a comprehensive set of sensitivity analyses to ensure the robustness of our results. The rationale and details for these analyses are provided in eAppendix D.

The analyses were carried out using STATA (version 17.0) and IVEWare (version 0.3). All tests were two-sided with an alpha level of 0.05 for statistical significance.

Results

Characteristics of the Study Population

Table 1 shows descriptive statistics for cognitive outcomes, covariates, and early-life circumstances of the study population. Black participants were on average younger than White participants, with an average age gap of 4.0 (95% CI, 3.5-4.5; *P*<.001) years. Compared to White participants, Black participants had lower cognitive scores and higher proportion of cognitive impairment. The differences in cognitive scores between White and Black participants were 2.3 (95% CI, 2.1-2.6; *P*<.001) points for cognitive score, and 17.2 (95% CI, 14.8-19.6; *P*<.001) percentage points (pp) for cognitive impairment. The cognitive racial disparities pertained to the entire distributions of cognitive score (eFigure 1).

Black and White participants had many differences in early-life circumstances (Figure 2). Black participants were younger (*P*<.001) and more likely to be born in the South (*P*<.001) and living there at age 10 (*P*<.001) than White participants. They had less favorable early-life socioenvironmental factors than White participants. Notably, they were more likely to relocate due to financial difficulties (*P*=.02) and receive financial help (*P*<.001) during childhood than

White participants. Moreover, they had a larger household size at age 10 (P<.001) and were more likely to experience early-life trauma (P<.001 for LHMS items) than White participants.

Stark racial differences were observed in educational attainment and educational experience. Black participants had significantly lower educational attainment (P<.001) and parental educational attainment (P<.001) than White participants. A lower proportion of Black participants owned books at age 10 (P<.001), and their mothers were less likely to spend full time with them during childhood (P<.001). The schooling experience was also less advantaged among Black participants. Specifically, relative to White participants, a lower proportion of Black participants had learned any foreign languages in high school (P<.001), and a much higher proportion experienced all segregated schooling (P<.001) and all public schooling before college (P<.001). These differences did not change substantively after adjustment for survey weights (eFigure 2).

Association of Racial Differences Across All Early-Life Circumstances with Racial Disparities in Cognition

As shown in Figure 3 and eTables 1-2, racial differences in early-life circumstances were associated with 0.81 (95% CI, 0.41-1.2) points, or 35.0% of the racial disparities in cognitive score between White and Black participants in decomposition analyses that included only traditional early-life factors (LifeHistory_1) and demographic covariates. The association substantially increased when educational experience was also included, with racial differences in early-life circumstances being associated with 1.4 (95% CI, 0.88-2.0) points or 61.5% of the racial disparities in cognitive score (LifeHistory_2).

The magnitude of these associations was greater for cognitive impairment. Across $LifeHistory_1$ and $LifeHistory_2$, racial differences in early-life circumstances were associated with 6.4 (95% CI, 2.0-10.8) pp or 37.4%, and 14.2 (95% CI, 8.8-19.5) pp or 82.3% of the racial disparities in cognitive impairment, respectively.

Association of Individual Factors or Domains of Early-Life Circumstances with Racial Disparities in Cognition

Figure 4 and eTable 2 show the association of individual factors or domains of early-life circumstances with racial disparities in cognition in $LifeHistory_2$.

Among all early-life factors, racial differences in educational experience were most substantively associated with the racial disparities in cognition, collectively accounting for 0.81 (95% CI, 0.37-1.3) points or 35.2% in cognitive score and 8.4 (95% CI, 4.0-12.7) pp or 48.6% in cognitive impairment, independent of traditional early-life factors and covariates. Collectively, the magnitude of these associations was smaller for the traditional early-life factors, which accounted for 0.61 (95% CI, 0.20-1.0) points or 26.3% in cognitive score, and 5.8 (95% CI, 2.0-9.6) pp or 33.7% in cognitive impairment, independent of educational experience and covariates.

Racial differences in all segregated schooling before college was associated with the greatest racial disparities, accounting for 0.66 (95% CI, 0.26-1.1) points or 28.8% of the racial disparities in cognitive score and 6.8 (95% CI, 2.6-11.0) pp or 39.7% of the racial disparities in cognitive impairment, independent of other early-life circumstances and covariates (see eFigure 3 for differences in cognitive outcomes by segregated schooling and race). Racial differences in years of educational attainment were associated with the second greatest racial disparities in cognitive outcomes, independently accounting for 0.55 (95% CI, 0.43-0.67) points or 24.0% of

the racial disparities in cognitive score, and 4.4 (95% CI, 3.2-5.6) pp or 25.6% of the racial disparities in cognitive impairment. Additionally, racial differences in learning foreign languages in high school was independently associated with 2.7% of the racial disparities in cognitive score and 2.9% of the racial disparities in cognitive impairment. In contrast, racial differences in factors from the other domains were not independently associated with the racial disparities in cognition (Figure 4, eTable 2).

Our findings were consistent across a series of sensitivity analyses that: (1) included PGS (eFigures 4-5) to adjust for biological predispositions; (2) incorporated additional regional indicators to account for disparities in public education resources (eFigure 6); (3) used a streamlined set of early-life factors to address potential multicollinearity (eFigure 7) and mitigate overfitting (eFigure 8); (4) adjusted for survey weights using the same wave of cognitive assessment to account for non-response and sampling/survey design (eFigure 9 for 2016, and eFigure 10 for 2018); and (5) leveraged baseline cognitive assessment to address sample dropout and mortality selection (eFigure 11). Notably, the associations were more pronounced when participants with dementia were excluded, suggesting that our primary estimates may have been conservative due to potential recall bias (eFigure 12). Lastly, a final set of analyses that used alternative exposure definitions indicated that the associations involving segregated schooling likely accumulated, with a critical period observed during primary education (eFigure 13).

Discussion

Few prior studies have evaluated associations between a rich set of early-life circumstances and racial disparities in cognition. This study evaluated whether and how two clinically meaningful cognitive outcomes differed between Black and White older Americans, and quantified how

much early-life circumstances, including educational experience, were collectively and individually associated with the racial disparities. Three major findings warrant comment.

First, our study revealed substantial disparities between Black and White adults in both early-life circumstances and late-life cognition. The presence of less favorable early-life circumstances among Black older adults than their White counterparts,[33,34] was associated with a clinically meaningful racial disparities in cognition, with Black older adults doing more poorly. Our findings underscore the substantial role of early-life circumstances in elucidating racial disparities in cognition. From a policy perspective, addressing adverse early-life circumstances has the potential to alleviate racial disparities in late-life cognition.[33]

Second, among these early-life circumstances less advantaged educational experiences among Black children were the greatest contributors to racial disparities in cognitive outcomes, independent of educational attainment and other early-life factors and demographic characteristics. Educational factors may work through a set of channels to influence racial disparities, such as brain development, employment/income, access to health care, health literacy and lifestyle choices.[35] For example, attending segregated schools may expose Black children to discrimination and racism that contributed to stress, traumatized brain development, and behavioral problems.[36,37] Moreover, attending segregated schools may adversely affect late-life cognition by diminishing school resources, such as spending per pupil, student-teacher ratio, term length, combined grades in classroom, and teacher training experience, as shown in prior studies.[15,38–43] Overall, our results suggest that policies implemented to improve educational equity may generate long-lasting impacts on reducing racial disparities in cognition into older ages.[39,43]

Third, relative to the pivotal role played by educational factors, a majority of traditionally measured early-life exposures showed modest role, and almost no independent associations with cognitive outcomes were discovered. This finding aligns with evidence that underscores the mediating role of educational attainment in connecting various early-life circumstances to late-life cognition.[6,44] Our findings suggest that these additional factors do not have direct, independent associations with racial disparities in cognition after accounting for educational attainment. In contrast, the robust association between school segregation and racial disparities in cognition after accounting for educational attainment highlights the inadequacy of relying solely on educational attainment to assess the relationship between education and late-life cognition.

School racial segregation can be attributed to two distinct historical sources. One is the codified de jure segregation that persisted prior to the landmark Brown v. Board of Education case, while the second arises from modern-day de facto racial segregation driven primarily by economic factors.[45] Despite increases in migration and implementation of desegregation policies in recent decades,[45] US schools continue to struggle with segregation, even decades after the historic Brown v. Board ruling.[46] In addition, early-life disadvantages for the offspring of younger minority parents has been exacerbated over the last four decades due to a rise in socioeconomic inequality[33,47], potentially amplifying racial disparities in both educational experiences and cognitive outcomes.[48-49] Given these complex dynamics, continuously monitoring the trend in racial disparities among upcoming age cohorts and generations is warranted.

Our current analysis builds on prior work that has evaluated the association between education and late-life cognitive outcomes. An important strength is the focus on the role of early-life circumstances prior to full educational attainment. Our findings may guide medical

professionals during review of social history to consider early-life signals of elevated risks, such as segregated schooling experiences, thereby allowing them to prioritize screening, prevention, and interventions. A second strength relates to our accounting for a uniquely rich set of early-life circumstances, which enabled us to better understand how they were collectively and individually associated with racial disparities in late-life cognition. Our in-depth investigation into the important but often neglected role played by educational experience offers novel evidence.

Limitations

This study has limitations. First, persons with impaired cognition or other health problems, as well as those with more disadvantaged early-life circumstances, may have been less likely to participate, suggesting that the associations between early-life circumstances and late-life racial disparities in cognition may be conservative. Second, due to the small sample size for other racial/ethnic groups, the current study focused solely on comparisons between non-Hispanic White and Black adults. Third, because of its low prevalence, it was not possible to evaluate dementia as an outcome. Fourth, we were unable to assess the variation in educational quality among segregated schools or investigate the tangible factors that may have contributed to the substandard quality in these schools. Fifth, it is possible that unobservable early-life factors may have biased our estimates. For example, Black participants in the earlier cohorts of HRS might have experienced the transition to desegregated schools, which could have led to harmful experiences either from firsthand mistreatment or broader opposition to desegregation. This trauma from adolescence could have an enduring adverse impact on cognition. Finally, while

early-life circumstances long preceded the cognitive outcomes, the associations identified in the current study are not causal. Additional research is needed to identify potential causal links.

Conclusions

This study identified early-life circumstances, especially educational factors, that are strongly associated with racial disparities in cognitive outcomes among older Americans. To slow cognitive decline and address racial disparities, additional research is needed to elucidate the mechanisms and inform the development of early-life interventions.

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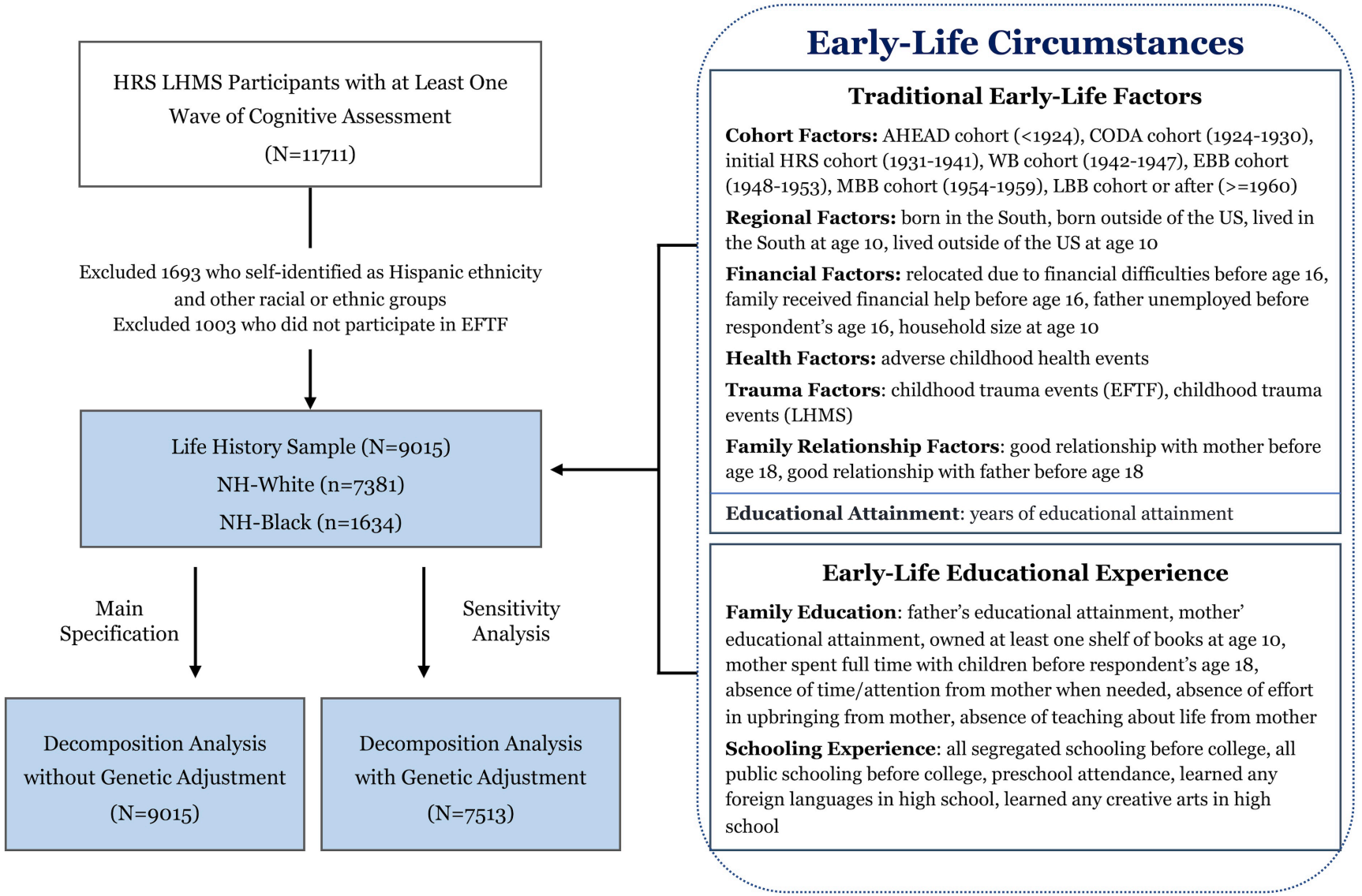
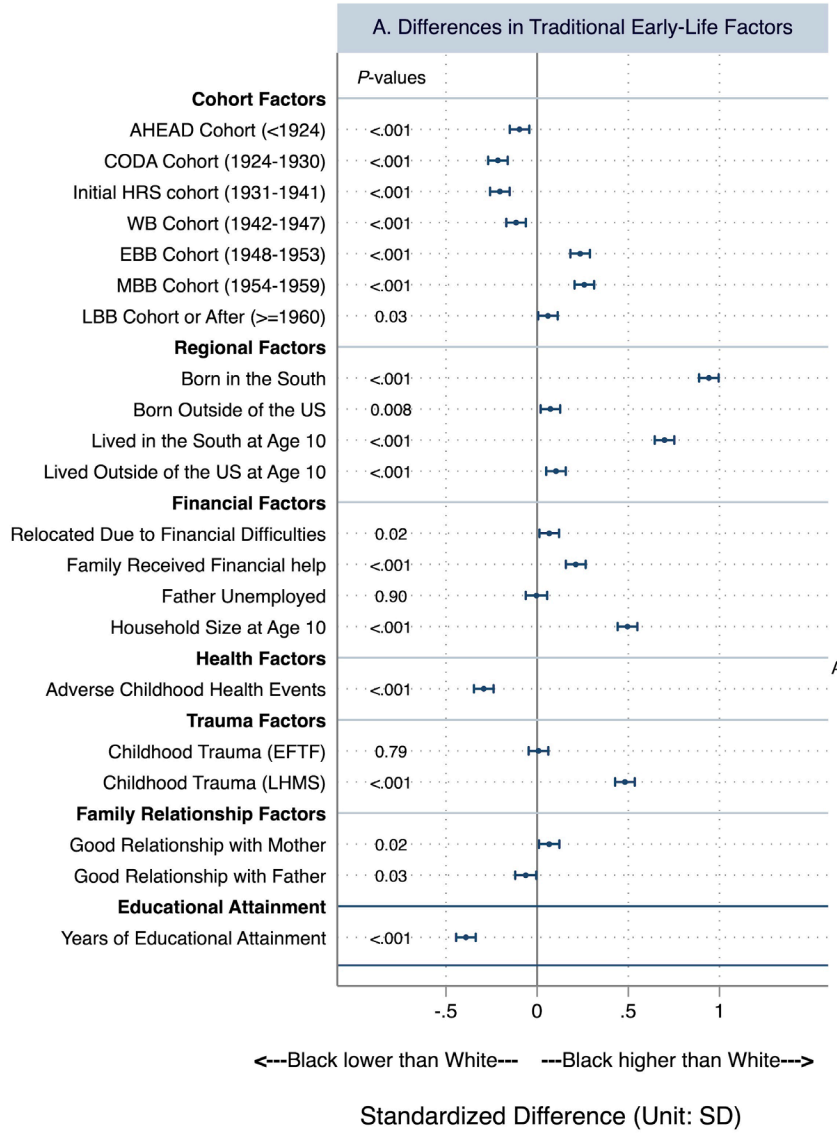
Figure 1. Flow Chart of Sample Selection Process



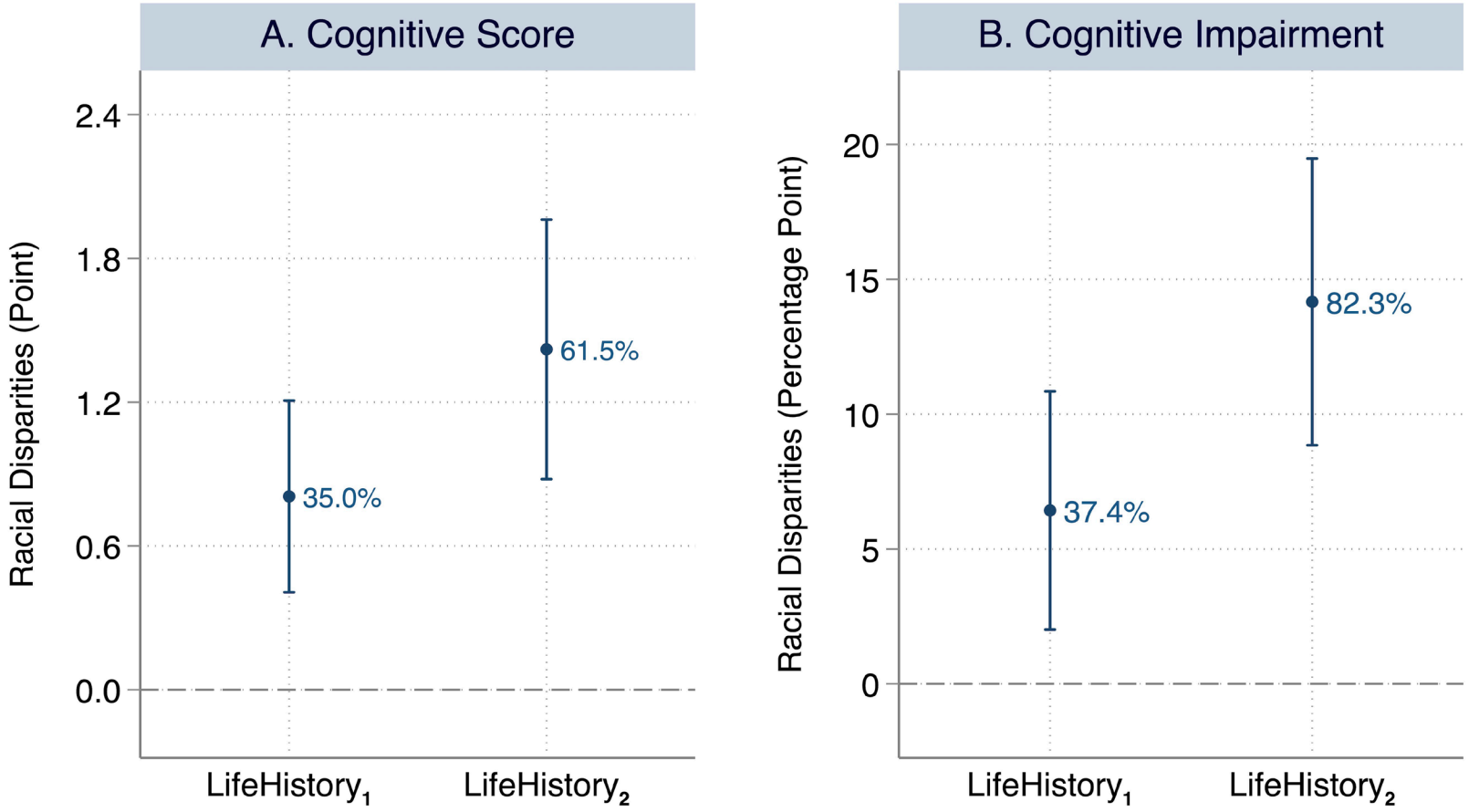
Notes: HRS=Health and Retirement Study; EFTF=Enhanced Face-to-Face; LHMS=Life History Mail Survey; NH=non-Hispanic; AHEAD=Study of Assets and Health Dynamics; CODA=Children of Depression; WB=War Baby; EBB=Early Baby Boomer; MBB=Mid Baby Boomer; LBB=Late Baby Boomer. EFTF and LHMS are separate components conducted within HRS, which are presented in detail in eAppendix A. The early-life circumstances were self-reported. The definition and justification for each of the early-life circumstances are provided in eAppendix B.

Figure 2. Standardized Differences in Early-Life Circumstances between Black and White Participants from the Health and Retirement Study (N=9015)



***Notes*:** The figure presents the Cohen's d standardized mean differences in individual early-life circumstances between Black and White participants. The early-life circumstances were self-reported. Panel A presents the estimates for traditional early-life factors; and Panel B presents the estimates for early-life educational experience. The scales of X axis for the two panels are different. For each early-life factors, the mean difference between Black and White participants was divided by their pooled standard deviation (SD) to obtain the standardized difference (unit: SD); and the estimates were visualized in the figure. The standardized differences were calculated to be comparable across various factors of different scales. The dotted points denote the Cohen's d standardized differences between Black and White participants for each individual early-life factors, and the horizontal line denotes the 95% confidence interval. A positive (negative) value of differences indicates Black participants had higher (lower) mean value of early-life factor than White participants. The P-values listed alongside the Y axis denote the statistical significance of the differences, estimated by Mann-Whitney-Wilcoxon tests for ordinal variables and Chi-square tests for binary and categorical variables. The differences were estimated using sample without data imputation, and sample size for each factor can be slightly less than 9,015 due to item-level missingness (see eAppendix B for more details). The results were similar when using imputed data or adjusting for sample demographics (available upon request); and the weighted estimates using the 2016 survey weight (N=8334) were very consistent except for the cohort factors as survey weights have already adjusted for the cohort differences (eFigure 2). Abbreviations: AHEAD=Study of Assets and Health Dynamics; CODA=Children of Depression; WB=War Baby; EBB=Early Baby Boomer; MBB=Mid Baby Boomer; LBB=Late Baby Boomer.

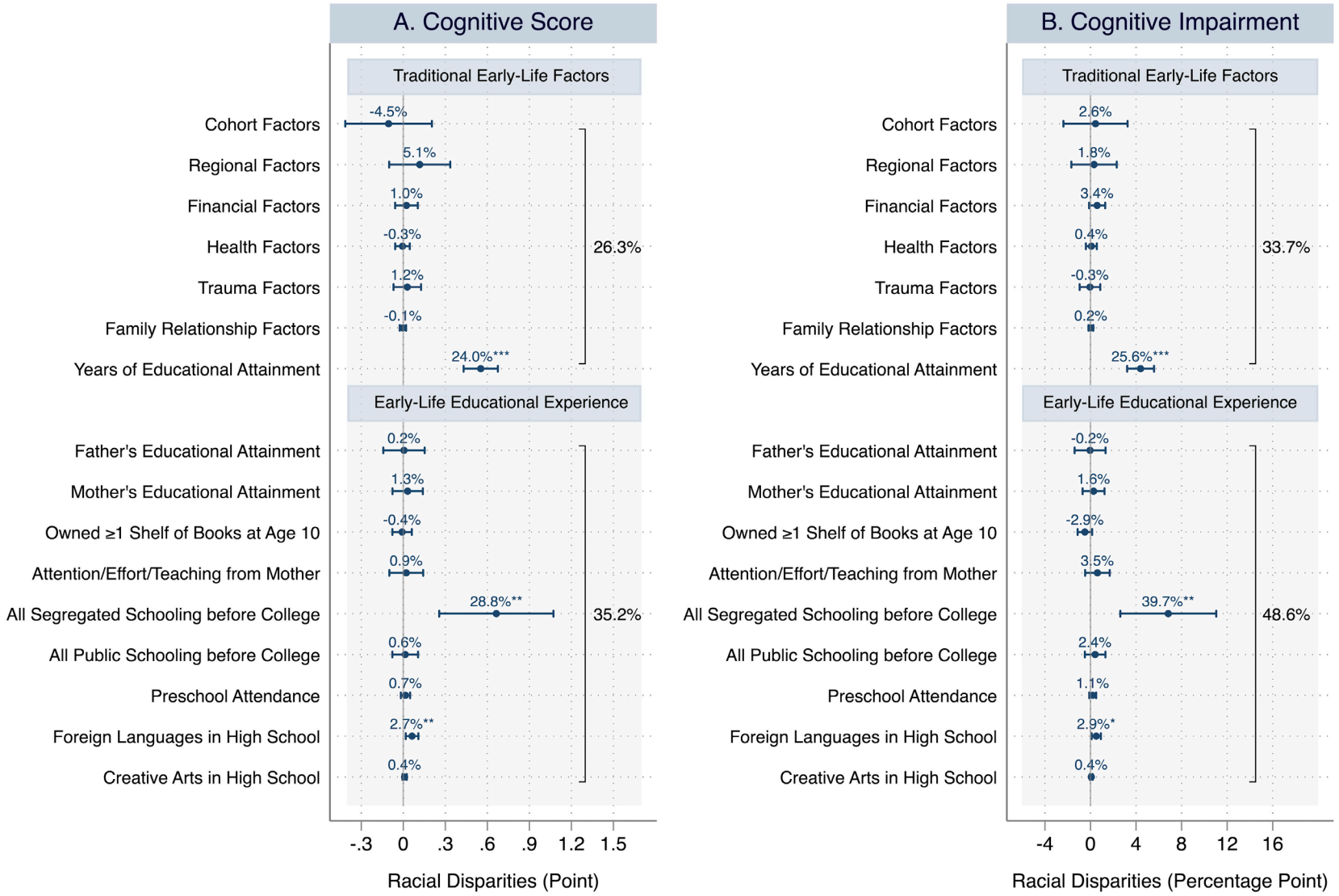
Figure 3. Association of Racial Differences Across All Early-Life Circumstances with Racial Disparities in Cognition between White and Black Participants (N=9015)



***Notes*:** Panel A presents the decomposition results for cognitive score, a continuous variable ranging from 0 to 27; and Panel B presents the decomposition results for cognitive impairment, a dichotomous variable indicating whether individuals were classified as cognitive impaired (0/1). For each cognitive outcome, X axis denotes the models being examined, including $LifeHistory_1$ and $LifeHistory_2$. In $LifeHistory_1$, traditional early-life factors were included to perform the decomposition. In $LifeHistory_2$, early-life educational experience were also included. All decompositions

adjusted for demographic covariates including age and sex. In Panels A and B, Y axis denotes the association of racial differences across all early-life circumstances with the racial disparities in cognition between White and Black participants. In Panel A, the unit of cognitive score is point; and in Panel B, the unit of cognitive impairment is percentage point (pp). The point estimates are plotted as circles and their 95% confidence interval are plotted as vertical lines. In each setting, the relative magnitude of racial disparities, expressed as percentage (%), is displayed alongside the horizontal lines. These percentages indicate the extent to which racial differences in early-life circumstances were associated with the total racial disparities in cognition between White and Black participants. Linear models were used for cognitive score, and logit models were used for cognitive impairment. The pooled estimates were obtained using multiple imputation with 20 imputed datasets. The early-life circumstances were self-reported.

Figure 4. Association of Racial Differences in Individual Factors or Domains of Early-Life Circumstances with Racial Disparities in Cognition between White and Black Participants, LifeHistory$_2$ (N=9015)



***Notes*:** Panel A shows the decomposition results for cognitive score, a continuous variable ranging from 0 to 27; Panel B shows the decomposition results for cognitive impairment, a dichotomous variable indicating whether individuals were classified as cognitive impaired or not (0/1). For each cognitive outcome, Y axis denotes the individual factors or domains being examined; X axis denotes the association of racial differences in individual factors or domains of early-life circumstances with the racial disparities between White and Black participants. A positive value signifies that equalizing racial differences in individual early-life circumstances was positively associated with the reduction of racial disparities in cognitive outcomes. In Panel A, the unit of cognitive score is point; and in Panel B, the unit of cognitive impairment is percentage point (pp). The point estimates are plotted as circles and their 95% confidence interval are plotted as horizontal lines. The relative magnitude of racial disparities, expressed as percentage (%), is displayed alongside the horizontal lines. These percentages indicate the extent to which racial differences in individual early-life circumstances were associated with the total racial disparities in cognition between White and Black participants. For each domain of factors (e.g., financial factors), its association with racial disparities were estimated by summing the racial disparities linked to each individual factor within that domain, thereby capturing the total racial disparities associated with all the factors in that domain. This additive calculation was also applied when assessing the total racial disparities associated with early-life traditional factors, as well as educational experience. Linear models were used for cognitive score, and logit models were used for cognitive impairment. The decompositions were performed using the Life History sample without genetic adjustment (LifeHistory_2), and the numerical results can be found in Supplementary eTable 2. The pooled estimates were obtained using multiple imputation with 20 imputed datasets. The early-life circumstances were self-reported. Asterisks denote the statistical significance of the association: *** $P < 0.001$, ** $P < 0.01$, * $P < 0.05$.

Table 1. Characteristics of Study Participants from the Health and Retirement Study [a]

	Life History Sample [b] (N=9015)	
Characteristic	**Black**	**White**
No. of participants	1634	7381
Outcomes and Demographic Covariates		
Cognitive score, mean (SD)	13.5 (4.8)	15.8 (4.4)
Cognitive impairment prevalence, No. (%)	549 (33.6)	1210 (16.4)
Age, mean (SD)	69.2 (9.2)	73.2 (10.1)
Female, No. (%)	1094 (67.0)	4410 (59.7)
Traditional Early-Life Factors		
AHEAD cohort (born before 1924), No. (%)	10 (0.6%)	135 (1.8%)
Children of Depression (CODA) cohort (1924-1930), No. (%)	45 (2.8%)	616 (8.3%)
Initial HRS cohort (1931-1941), No. (%)	390 (23.9%)	2461 (33.3%)
War Baby (WB) cohort (1942-1947), No. (%)	215 (13.2%)	1289 (17.5%)
Early Baby Boomer (EBB) cohort (1948-1953), No. (%)	454 (27.8%)	1356 (18.4%)
Mid Baby Boomer (MBB) cohort (1954-1959), No. (%)	435 (26.6%)	1228 (16.6%)
Late Baby Boomer (LBB) cohort or after (born in or after 1960), No. (%)	85 (5.2%)	296 (4.0%)
Born in the South, No. (%)	1087 (66.6)	1850 (25.1)
Born outside of the US, No. (%)	89 (5.5)	294 (4.0)
Lived in the South at age 10, No. (%)	974 (59.6)	2051 (27.8)
Lived outside of the US at age 10, No. (%)	72 (4.4)	196 (2.7)
Relocated due to financial difficulties before age 16, No. (%)	292 (18.0)	1147 (15.6)

Family received financial help before age 16, No. (%)	347 (21.9)	1032 (14.2)
Father unemployed before respondent’s age 16, No. (%)	276 (20.8)	1465 (21.0)
Household size at age 10, median (IQR)	6 (3)	5 (2)
Adverse childhood health events, No. (%)	1167 (72.1)	6087 (83.5)
Childhood trauma events (EFTF), No. (%)	557 (34.5)	2515 (34.1)
Childhood trauma events (LHMS), No. (%)	908 (56.4)	2433 (33.4)
Good relationship with mother before age 18, No. (%)	1203 (80.9)	5538 (78.2)
Good relationship with father before age 18, No. (%)	956 (69.1)	4975 (71.9)
Years of educational attainment, median (IQR)	12 (2)	13 (4)
Early-Life Educational Experience		
Father's educational attainment (years), median (IQR)	8 (6)	12 (4)
Mother's educational attainment (years), median (IQR)	10 (4)	12 (4)
Owned at least one shelf of books at age 10, No. (%)	863 (54.1)	5038 (69.7)
Mother spent full time with children before respondent’s age 18, No. (%)	349 (22.3)	3251 (44.7)
Absence of time/attention from mother when needed, No. (%)	58 (3.7)	219 (3.0)
Absence of effort in upbringing from mother, No. (%)	55 (3.6)	179 (2.5)
Absence of teaching about life from mother, No. (%)	95 (6.1)	427 (6.0)
All segregated schooling before college, No. (%)	1011 (73.5)	44 (0.6)
All public schooling before college, No. (%)	1294 (89.6)	5337 (76.9)
Preschool attendance, No. (%)	290 (18.5)	894 (12.4)
Learned any foreign languages in high school, No. (%)	662 (42.9)	3656 (51.6)
Learned any creative arts in high school, No. (%)	950 (60.7)	4507 (63.5)

Abbreviations: SD, standard deviation; IQR, interquartile range; HRS; Health and Retirement Study; LHMS, Life History Mail Survey (2015, 2017); EFTF, Enhanced Face-to-Face (2006-2012); AHEAD, Study of Assets and Health Dynamics; CODA, Children of Depression; WB, War Baby; EBB, Early Baby Boomer; MBB, Mid Baby Boomer; LBB, Late Baby Boomer.

[a] The descriptive statistics presented in the table were obtained using non-missing data before imputation. The sample size for each variable might be lower than the total sample size, though the difference was minimal (see eAppendix B for details). The early-life circumstances were self-reported. The definition and justification for each of the early-life circumstances are provided in eAppendix B.

[b] Life History sample refers to non-Hispanic White and Black participants who had valid cognitive measure and participated in the LHMS and EFTF.

Supplementary Online Content

Title: Assessing Early-Life Contributions to Racial Disparities in Cognition for Older Americans: The Importance of Educational Experience

eAppendix A. Data Source

Data Source 1. The HRS Core Survey

The Health and Retirement Study (HRS) has biennial interviews for collecting a wide range of information, including economics, health, marital, family status, and public and private support systems since 1992.[1] Although the HRS has grown with the addition of new cohorts, the contents of the core survey have remained mostly consistent. In particular, the HRS core survey generally included multiple sections such as demographics and background, health, cognition, family structure and transfers, functional limitations, housing, physical measures, employment and pensions, disability, health services and insurance, expectations, assets and income, assets change, widowhood and divorce, and insurance. Additionally, the HRS has some experimental modules on specialized topics as part of the core survey. These modules only target a random subsample at the end of the core survey.[1,2]

In this study, variables of early-life demographics and socioeconomic status (SES) were constructed using the measures from the HRS core survey and/or related modules. Many of the variables were assembled from the RAND HRS files since RAND HRS groups have created user-friendly files with cleaned and processed variables with consistent and intuitive naming conventions, as well as model-based imputations.[3] Otherwise, we assembled variables from the original HRS released core data from 1995 (when the variables were available) to 2018.

Data Source 2. The Life History Mail Survey

The HRS Life History Mail Survey (LHMS) contains additional questions about respondents' residential history, educational history, and other important early-life and family events. The 2015, 2017 Spring, and 2017 Fall versions were conducted in subsamples of HRS participants. The target subsample for the 2015 wave included all living HRS participants who were not included in the 2015 Consumption and Activities Mail Survey (CAMS) and who completed their most recent HRS core survey interview in English (rather than Spanish). The 2017 Spring wave included participants who were 2015 CAMS Sample Members who were still alive in 2017 and whose household was considered finalized on their 2016 core interview(s) by early March 2017 (members of finalized households either had completed core interviews or were considered final refusals for the core that wave). Lastly, the 2017 Fall sample included participants who were not included in the 2017 CAMS sample, and who did not return a 2015 LHMS questionnaire. The response rates and total enrolled participants for the 2015, 2017 Spring, and 2017 Fall waves respectively were 58% and 6,481 participants, 74% and 3,844 participants, and 28% and 1,444 participants.[4–6] We assembled a range of variables for early-life circumstances surveyed in all three waves of the LHMS.

Data Source 3. The Enhanced Face-to-Face Interview

In 2006, HRS initiated the Enhanced Face-to-Face (EFTF) interview with a mixed-mode design during the follow-up period in which a random half of HRS participants were assigned a face-to-face interview with physical and biological measures (e.g., salivary DNA samples) and a psychosocial questionnaire. The other half completed only the Core survey but were selected for the next (i.e., 2008) EFTF interview. A similar method was applied to the subsequent HRS survey. Several early-life circumstance variables in the present study related to trauma were assembled from this psychosocial questionnaire from 2006 to 2012.

In addition, polygenic risk scores (PGS) for a variety of phenotypes from respondents who provided salivary DNA between 2006 and 2012 were included in this study. Genotyping was conducted by the Center for Inherited Disease Research (CIDR) in 2011, 2012, and 2015, and principal component analysis was performed to identify population group outliers and to provide sample eigenvectors for association testing to adjust for potential population stratification. The final European American sample included all participants who self-reported as non-Hispanic White that had PC loadings within ± one standard deviations of the mean for eigenvectors 1 and 2 in the PC analysis of all unrelated study subjects. The final African American sample included all self-reported non-Hispanic Black Americans within two standard deviations of the mean of all self-identified Black Americans for eigenvector 1 and ± one standard deviation of the mean for eigenvector 2 in the PC analysis of all unrelated study subjects.

Genetic factors were included as additional covariates in our sensitivity analysis. They were assessed using PGS, which is a score constructed based on variation in multiple genetic loci and their associated weights. It serves as the best prediction for the trait that can be made when accounting for variation in multiple genetic variants. All phenotypes for the four domains for genetic factors are listed below, and more information can be found in the document from HRS.[7] Potential population stratification were accounted for.[8]

PGS Domains	**Traits/phenotypes**
Anthropometric	(1) Height; (2) Body mass index; (3) Waist circumference; (4) Waist-to-Hip ratio
Disease/cognition/longevity	(1) Coronary artery disease; (2) Myocardial infarction; (3) Type II diabetes; (4) Alzheimer's disease IGAP 2013 with APOE; (5) Alzheimer's disease IGAP 2019 with APOE; (6) General cognition 2015; (7) General cognition 2018; (8) Longevity
Mental health/personality	(1) Attention deficit/hyperactivity disorder PGC 2010; (2) Attention deficit/hyperactivity disorder PGC 2017; (3) Autism; (4) Bipolar disorder; (5) Mental health cross disorder; (6) Major depressive disorder PGC 2010; (7) Major depressive disorder PGC 2018; (8) Subjective wellbeing; (9) Neuroticism; (10) Depressive symptoms; (11) Schizophrenia; (12) Extraversion
Smoking	(1) Smoking initiation (ever/never); (2) Number of cigarettes smoked per day TAG 2010; (3) Number of cigarettes smoked per day GSCAN 2019

eAppendix B. Early-Life Circumstances

Early-life circumstances involve all exposures during individuals' early stages of life that may have profound impact on their long-term health outcomes. Based on a comprehensive review of literature, we summarized seven domains of *early-life* factors that have been traditionally identified to be important for individuals' cognition, including early-life cohort, regional, financial, health, trauma, family relationship factors, and educational attainment. These factors and domains were classified as traditional early-life factors in our study. Moreover, emerging evidence suggests that early-life educational experience can play a very critical role in cognitive development and have long-lasting associations with later-life cognitive health even after adjusting for educational attainment. This motivates us to leverage the comprehensive educational history data in the HRS to investigate this domain of factors.

In this section, we provided a comprehensive review of existing evidence linking each domain of early-life factors to the later-life cognitive health and disparities, providing both theoretical and empirical bases for this study. Moreover, we discussed how HRS data were utilized in this study as well as the conceptualizations and definitions of each variable we included.

Traditional Early-life Factors

1. Early-Life Cohort Factors

Literature review and theoretical justification: Birth cohorts represent the specific time period when a group of individuals were born who share the common characteristics, experience, and exposures.[9–12] The early-life environment impacting prenatal and postnatal exposures may differ greatly across birth cohorts and exert differential effects on individuals' cognitive health.[9–11] Existing work has identified associations between birth period and dementia incidence among older American adults, which persist despite the inclusion of more granular early-life educational and environmental factors.[11] The inclusion of birth cohort as a variable may hence capture the influence of broader historical and societal-level changes on later incidence of cognitive impairment. For instance, a prior study employing HRS data identified early-life exposure to the Great Depression as being associated with changes in fluid cognition, consistent with a critical period model, with Black study participants affected more adversely.[10] To assess birth cohort, we included the birth cohort indicators as delineated by the HRS in the analysis following prior literature, to account for underlying cohort differences.[12,13]

Measurement: The seven dichotomous cohort indicators encompassed:

- AHEAD cohort: born before 1924
- Children of Depression (CODA) cohort: born in 1924-1930
- Initial HRS cohort: born in 1931-1941
- War Baby (WB) cohort: born in 1942-1947
- Early Baby Boomer (EBB) cohort: born in 1948-1953
- Mid Baby Boomer (MBB) cohort: born in 1954-1959
- Late Baby Boomer (LBB) cohort or after: born in or after 1960.

2. Early-Life Regional Factors

Literature review and theoretical justification: Place of birth and childhood residence have been identified in existing work as being associated with cognition in late adulthood, suggesting that childhood exposures may establish cognitive reserve in a highly geographically patterned manner.[14–21] As where people are born is not in the realm of individuals' own choices, place of birth and childhood residence are often considered as crucial sources of inequalities later in life.[15] Notably, birth in the U.S. South—more common among Black study participants—has been found in several studies to be associated with elevated rates of dementia and Alzheimer's mortality, regardless of future migration patterns.[14,16–18,20,21] Moreover, compared with their U.S.-born counterparts, adults in the U.S. who were born in a foreign country have been found to have elevated dementia and cognitive impairment rates.[16,19] Given this evidence, we employed two birthplace factors in our analysis: born in the South, and born outside of the US.

Moreover, the geographical effects of place of residence may extend to late childhood. Early-life exposures during childhood are significantly shaped by regional policies, resources, and social environments, such as insurance coverage, nutrition, and healthcare resources, which may profoundly impact individuals' early cognitive development, resulting in cognitive disparities later in life.[21–27] This geographic difference may also apply to the socioeconomic conditions and social contexts that affect individuals' health and health behaviors, as well as labor market outcomes, such as educational attainment. All of these may consequently impact cognitive health in the long term[15,24,28–31]. Therefore, we additionally included regional indicators at age 10 to capture such differences: whether the respondent lived in the South at age 10, and whether the respondent lived outside of the US at age 10.

Measurement: In our study, regional factors measured respondents' early-life aggregate exposure to regional contextual factors. In HRS public data files, respondents' birthplace and residence place at age 10 were reported at the census region level, including New England, Mid Atlantic, East North Central, West North Central, South Atlantic, East South Central, West South Central, Mountain, Pacific, other US/NA division, and not in US/US territory. Following existing literature, we defined participants' birthplace (and childhood residence place) based on whether they were born (lived at age 10) in the South (including South Atlantic, East South Central, West South Central) or not.[16,21]

The four dichotomous regional indicators included:

- Born in the U.S. South
- Born outside of the US
- Lived in the South at age 10
- Lived outside of the U.S. at age 10

The findings were similar when incorporating a more granular and comprehensive list of regional indicators (eFigure 6).

3. Early-life Financial Factors

Literature review and theoretical justification: Family socioeconomic status during childhood has been extensively associated with later life cognitive outcomes.[11,20,21,24,27,28,32–38] It may impact individuals' prenatal, postnatal, and childhood exposures as well as access to resources which are crucial for cognitive development, consequently impacting their cognitive health over the life course.[20,21,24,28,29,39] Given that Black Americans have disproportionately experienced financial instability and poverty during childhood compared to their White counterparts, racial disparities in financial factors may be linked to the observed racial disparities in cognition.[20] To identify childhood financial instability and access to resources, we followed prior literature to include measures of whether the respondent relocated due to financial difficulties before age 16, whether their family had received financial assistance before respondent's age 16, whether the respondent's father experienced a significant unemployment spell ("several months or more"), and household size at age 10. Similar variables have all been employed as measures of childhood economic hardship in existing work on cognition, including work using HRS data.[36–38] For instance, one study shows that relocation due to financial difficulties and parental unemployment are associated with poorer cognitive outcomes,[36] which motivates us to further explore the associations of these financial factors with racial disparities in cognition.

Measurement: The four financial factors we considered included:

- Relocated due to financial difficulties before age 16 (dichotomous)
- Family received financial help before age 16 (dichotomous)
- Father unemployed before respondent's age 16 (dichotomous)
- Household size at age 10 (continuous/ordinal)

4. Early-Life Health Factors

Literature review and theoretical justification: The existing body of literature has widely documented the detrimental effects of childhood health adversity on cognitive health in later life.[21,24,28,31,35–38,40–44] Factors such as childhood illness, disability, and unhealthy behaviors can directly impede brain development, particularly during early developmental stages. When the brain fails to reach its full potential due to such adversities and illnesses, it may result in diminished cognitive reserve, making individuals less resilient to the aging process and more susceptible to cognitive impairment.[21,24,28,31,35–38,41,42,44,45]

Furthermore, in accordance with the cumulative disadvantage theory, the adverse effects of childhood health adversity may accumulate and persist throughout one's life, affecting both health and cognition over the life course.[24,31,46] For instance, childhood illness can increase the risk of chronic diseases such as hypertension, diabetes, cardiovascular diseases and depression, all of which constitute critical risk factors for cognitive impairment and dementia.[20,24,28,29,41,46] Similarly, unhealthy behaviors like smoking can persist over time, exerting a lasting negative impact on cognitive health.[29,39,47,48]

Taken together, these childhood health adversities play a pivotal role in shaping later-life cognition.

Measurement: To assess early-life health, we included 16 important health events occurring prior to age 18 from the Core, such as disabled for six months and severe head injury.[41,45] Each variable for childhood health was coded as a dichotomous variable (0=no; 1=yes). All health outcomes which we included are as follows:

- Disabled for six months or more prior to age 18
- Injury to head which resulted in the loss of consciousness prior to age 18
- High blood pressure prior to age 18
- Heart issues prior to age 18
- Respiratory issues prior to age 18
- Diabetes prior to age 18
- Epilepsy prior to age 18
- Problems with speech prior to age 18
- Problems with hearing prior to age 18
- Problems with vision prior to age 18
- Depressed prior to age 18
- Other emotional or psychological disorder prior to age 18
- Learning disability prior to age 18
- Usage of drugs or alcohol prior to age 18
- Smoking cigarettes prior to age 18
- Parents smoked cigarettes prior to age 18

Respondents who had at least one of the aforementioned health events were coded as 1 and 0 otherwise. We employed this construction because the prevalence of individual indicators was low (mostly <10%, with some <2% for both White and Black); and this more simplified and dichotomous construction had the advantage of more direct interpretation. We also tested the models using the continuous specification (total number of adverse events) of health events as well as treating them as individual factors. The findings were similar and the estimates are available upon request.

5. Early-Life Trauma Factors

Literature review and theoretical justification: Growing evidence highlights a compelling association between adverse traumatic events and reduced cognitive function in later life.[49–57] Such adversities can result in elevated and prolonged exposure to glucocorticoids during early childhood, leading to permanent loss of neurons and cognitive dysfunction.[28,49,50,58–63] These effects cumulatively impact an individuals' long-term cognitive function.[24,27,49,50]

Specifically, recent research indicates that threat-related events, such as childhood abuse, harm and violence, may have detrimental effects on brain regions responsible for emotional processing and regulation.[50,53,54,64–67] Similarly, deprivation-related events, including childhood neglect, separation, and the absence of a parent, can result in reduced cognitive stimulation during early development, leading to underdeveloped brain networks associated with language development and executive function.[50,53,54,64–67] These biological pathways collectively contribute to an increased risk of cognitive impairment and dementia in later life.

Moreover, research suggests potential psychosocial pathways linking childhood adverse traumatic events to later-life cognitive health. These adversities may indirectly affect individuals' educational attainment, occupational opportunities, subsequent socioeconomic outcomes, overall health, health behaviors, and meatal well-being, thereby increasing the risk of cognitive aging (i.e., cumulative risk model).[27,49,50,55–57,68] For instance, evidence has indicated associations between adverse traumatic events and later-life health and health behaviors such as smoking, obesity, and depression, all of which are significant risk factors for dementia.[29,49,50,52]

Measurement: We assembled several indicators of childhood traumatic events and experience from the EFTF and the LHMS, which have been used before.[44,49,50,57,69–71] Each variable for childhood trauma was coded as a dichotomous variable based on the answer (0=no; 1=yes). Since these trauma variables were from two different data sources (i.e., the EFTF interview as well as the 2015 Fall, 2017 Spring, and 2017 Fall LHMS), there was a slight difference in age frame in the questions (18 years old vs. age 16); hence we constructed two dichotomous trauma variables based on measures from each respective source. Specifically, respondents who had at least one of the trauma events from the EFTF were coded as 1 and 0 otherwise. Similarly, respondents with at least one of the trauma events from the LHMS were coded as 1 and 0 otherwise. We adopted this variable construction for the same reasons as adverse health events; the results were similar when using the continuous specification of trauma events and when treating them as individual factors (available upon request). The trauma events included in our study from each data source are provided below:

Traumas included from the EFTF

- Physical abused prior to age 18
- Parents used drugs or alcohol which caused problems prior to age 18
- Trouble with police prior to age 18
- Repeated school prior to age 18

Traumas included from the LHMS

- Spent any amount of time in an orphanage prior to age 16
- Spent any amount of time in a foster home prior to age 16
- One or more parents died prior to age 16
- Parents divorced prior to age 16
- Separated from mother for more than 6 months prior to age 16
- Separated from father for more than 6 months prior to age 16

6. Early-Life Family Relationship

Literature review and theoretical justification: The quality of childhood family relationships with parents holds significant importance, as it mirrors the degree of emotional attachments and support that individuals perceived during their formative years.[72–81] The existing body of literature underscores the crucial role of parent-child interactions and attachments in shaping children's

social-emotional competence, physical health, mental well-being, and cognitive development.[72–81] A nurturing, secure, and enduring parent-child relationship can prove highly advantageous for a child's future socialization and emotional well-being, directly contributing to positive mental and cognitive stimulation and enhancing an individual's resilience against cognitive decline and impairment.[72–81] Moreover, these early relationships can exert a profound and lasting impact on an individual's cognitive health, potentially influencing a chain of risk factors throughout their life course.[24,28,77]

Recent evidence has further highlighted the connection between childhood relationship with parents and later-life cognitive functioning and cognitive decline.[24,28] These findings have spurred our motivation to delve deeper into the family relationship factors during childhood.

Measurement: Specifically, the relationship measures were coded as dichotomous variables (0=no; 1=yes) to indicate whether the respondent had good relationship with parents or not before age 18. The relationships were assessed respectively for father and mother.

- Good relationship with mother before age 18
- Good relationship with father before age 18

7. Educational Attainment

Literature review and theoretical justification: Educational attainment plays a pivotal role in individuals' cognitive aging process and has been recognized as the most influential early-life factor associated with cognitive impairment and dementia.[29] Extensive literature has consistently demonstrated robust positive associations between education and cognitive functioning.[11,20,35,36,82–100] Specifically, educational attainment exerts a substantial impact on individuals' problem-solving strategies, cognitive capacity, and cognitive reserve.[90,101] Studies have consistently revealed enduring relationship between educational attainment, cognitive function[101–103], and dementia in later life.[104] Evidence has further established a causal link between educational attainment and cognitive abilities among older adults.[82,86,98] Significantly, the Lancet Commission Report on dementia prevention has identified education as the single most important modifiable risk factor for dementia in early life, based on a comprehensive literature review.[29]

Education can influence cognitive aging through multiple pathways. First, it exerts a direct and profound impact on cognitive development.[28,29,85,90] Formal education exposes individuals to active and extensive cognitive stimulation, facilitating neurological developments and the acquisition of cognitive abilities and skills. This process contributes to the buildup of cognitive reserve, leading to enhanced cognitive function in later life, and increased resilience against the cognitive aging process.[28,29,85,90]

Additionally, educational attainment enhances individuals' potential to engage in intellectually and cognitively stimulating occupations and activities throughout their lifespan. This creates more opportunities for cognitive maintenance and reduces the risks of cognitive impairment.[28,29,86,98]

Moreover, educational attainment has been linked to various health outcomes and modifiable risk factors of dementia over the life course, including hypertension, diabetes, cardiovascular diseases,

physical health, and mental well-being. Higher educational attainment is associated with lower risks of these modifiable factors, resulting in improved cognitive health in later life.[11,20,35,36,82–100]

Measurement: In this study, we adopted the definition of educational attainment commonly used in prior literature,[11,20,35,36,82–100] which refers to the number of years of formal schooling completed.

- Years of educational attainment

Early-Life Educational Experience

In addition to educational attainment, family education and schooling experience have been increasingly considered as important educational factors that contribute to cognitive health in older age. As educational attainment only pertains to the years of formal education, it does not differentiate between differential schooling experience and fails to account for the education received at home. Therefore, we leveraged a rich set of measures of educational history that were uniquely collected in the HRS LHMS to assess the early-life educational experience.

To minimize the potential impact of recall bias, we defined dichotomous variables carefully to differentiate those with or without a particular experience/exposure. We did not classify variables based on the degree of experience/exposure because such a classification might suffer from recall bias and could be less accurate due to ambiguity of the questions and answers.[105] The classifications were defined based on the answer choices, such as "not at all" and "none", which can be clearly differentiated among others.

Below we provided the theoretical review and justification for each domain respectively, as well as the detailed definitions and explanations for each factor included.

1. Family Education

Literature review and theoretical justification: Family education, particularly when imparted from parents, plays a pivotal role in shaping cognitive development in childhood, thereby exerting a consequential influence on cognitive aging in later life.[28,106–109] A substantial body of research has established a link between parental educational attainment and cognition, even after accounting for various social, economic and demographic factors, including the educational attainment of the individuals themselves.[28,35,91,96,106–116]

One major explanation of these findings is that educated parents are more likely to provide cognitive stimulation that proves highly beneficial during the critical period of synaptic development in childhood.[107–109,111–113,115,116] Notably, parental education has consistently emerged as a predictor of childhood and later-life cognitive outcomes, independent of other factors. For example, children of educated parents tend to exhibit a richer vocabulary, and the quality of mother-infant interactions has been shown to have enduring associations with cognition in later life.[107–109,111–113,116] Given that mothers often play a central role as caregivers at home, they tend to invest more time in educating and interacting with children within the family context.[107–109,117] For example, mothers' time spent with children in educationally oriented activities (e.g., studying,

reading and doing homework) has been found to be positively correlated with child development.[107,109] Additionally, mothers often take on the role of the primary decision-maker regarding children's education.[118] The importance of maternal education and teaching has been underscored by evidence, which has highlighted the profound and significant impact of a mother on the educational and cognitive development of their children.[106,107,109]

Given the mounting evidence regarding the crucial educational role of mothers, we have included additional factors in our study which reflect the level of positive stimulation and teaching received by the respondents at home during childhood.[107–109,117] Quasi-experimental evidence, in particular, has demonstrated that time mothers invest in their children is a quantitatively important determinant of children's skill development, especially in the realm of cognitive skills.[107,109] This maternal time investment represents a vital facet of family education and is pivotal for the development of cognitive reserve, particularly during early childhood. The cognitive skills and reserve acquired through a mother's time investment in teaching, nurturing, attention, and care, subsequently serve as a buffer against rapid cognitive aging, promoting elevated levels of cognitive reserve and sustained cognitive stimulations throughout the life course.[28,107–109]

Therefore, in our study, we have measured family education through not only the years of maternal and paternal educational attainment, but also several indicators that capture the time and teaching received from mothers.[28,107–109] These indicators encompass whether the mother spent full time with the children, whether the respondent experienced an absence of time or attention from their mother, whether the respondent perceived an absence of effort in upbringing from their mother, and whether the respondent reported an absence of "teaching about life" from their mother.

Measurement: In our study, the family education was defined by following factors:

- Father's educational attainment (years): respondents' reported father's educational attainment (in years)
- Mother's educational attainment (years): respondents' reported mother's educational attainment (in years)
- Owned at least one shelf of books at age 10: respondents were asked "When you were 10 years old, approximately how many books were in the place you lived (not counting magazines, newspapers, or your school books): none, enough to fill one shelf (11-25 books), enough to fill one bookcase (26-100 books), enough to fill two bookcase (101-200 books), or enough to fill more than two bookcase (more than 200 books)." Those who reported to have at least one shelf of books were coded as 1, and 0 otherwise.
- Mother spent full time with children before respondent's age 18: respondents were asked "What portion of the time did your mother work outside the home when you were growing up: all of the time, some of the time, or not at all?". Those who answered "not at all" were coded as 1, and 0 otherwise.
- Absence of time/attention from mother when needed: respondents were asked "How much time and attention did your mother give you when you needed it (before you were 18 years old): a lot, some, a little, or not at all?" Those who answered "not at all" were considered to be absence of time/attention from mother and were coded as 1, and 0 otherwise.

- Absence of effort in upbringing from mother: respondents were asked "How much effort did your mother put into watching over you and making sure you had a good upbringing (before you were 18 years old): a lot, some, a little, or not at all?" Those who answered "not at all" were considered to be absence of effort from mother and were coded as 1, and 0 otherwise.
- Absence of teaching about life from mother: respondents were asked "How much did you mother teach you about life (before you were 18 years old): a lot, some, a little, or not at all?" Those who answered "not at all" were considered to be absence of teaching about life from mother and were coded as 1, and 0 otherwise.

In the primary decomposition results, we reported the joint contribution (i.e., sum) of the latter four factors: 1) Mother spent full time with children before respondent's age 18; 2) Absence of time/attention from mother when needed; 3) Absence of effort in upbringing from mother; 4) Absence of teaching about life from mother, and defined the domain as "Attention/Effort/Teaching from Mother". We reported their contribution jointly because they all reflected similar attributes. The estimation of joint contribution is further described in eAppendix C. The individual estimates are also available upon request.

2. Schooling Experience

Literature review and theoretical justification: While the number of years of education has long been associated with cognitive outcomes in adulthood, a growing body of research has shifted its focus toward examining the impact of the schooling and educational experience on cognition.[83,84,119–127] For instance, research that incorporated a range of measures related to schooling quality, such as student-teacher ratio and spending per student, has revealed significant associations between school advantage and later-life cognitive performance.[83,84,120,125,126] Furthermore, multiple studies have underscored the pivotal role of school quality in shaping cognitive abilities and addressing disparities in cognition.[83,84,120,125,126] For instance, one study revealed that accounting for educational experience (measured by the percentage of white students, urban or rural school settings, and classroom grade combinations), accounted for a large portion of the disparities between Black and White older adults in general cognitive functioning.[83] Another study identified several measures of high school quality, most notably the prevalence of teachers holding graduate degrees, as being influential in later-life cognitive outcomes.[120]

Considering that Black study participants were often exposed to lower-quality schools, research suggests that disparities in school quality may contribute to the observed differences in the prevalence of cognitive impairment between Black and White older adults.[83,84,120,125,126] In a separate study, attending a highly segregated school was associated with racial disparities in cognition, even after accounting for school type (e.g., public, private, vocational), the variety of available courses, and socioeconomic neighborhood indicators.[119]

Based on this evidence, our analysis incorporated measures related to whether respondents exclusively attended segregated schools and attended public schools prior to college. Additionally, we included measures related to course offerings, which may serve as indicators of higher school quality, such as whether respondents learned any foreign languages in high school and whether the respondent learned any creative arts in high school.[123] Lastly, while the majority of research

on school quality and experience has focused on high school and beyond, it is worth noting that the quality of preschool has also been found to be associated with cognitive outcomes. Hence, we additionally included a measure of preschool attendance.[128]

Measurement: In our study, schooling experience was assessed primarily based on respondents' education history data, which included the following:

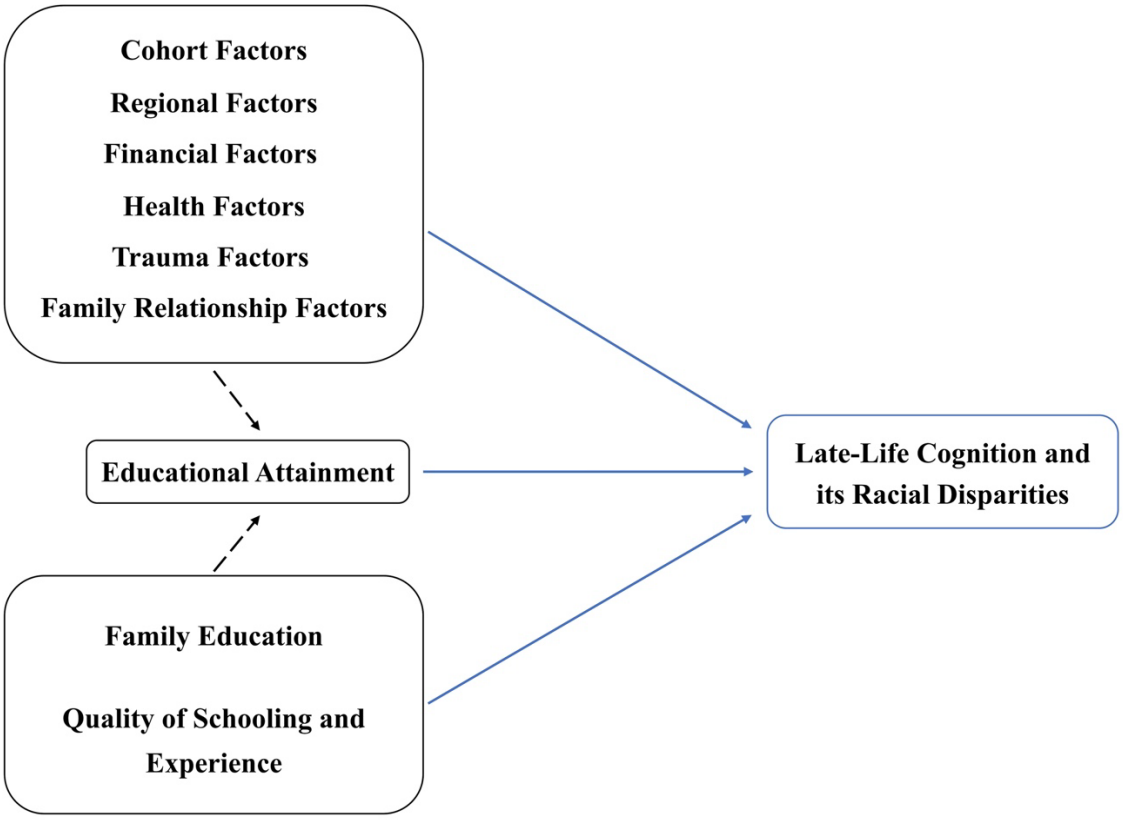
- All segregated schooling before college: respondents were asked to list all of the primary, elementary, middle, junior high and high schools they attended; and for each school, they were asked whether "most children in the school were White, Black, Hispanic or others". The school was considered to be a segregated school if most children in the school were Black, Hispanic or others. If all the schools the respondents attended before college were segregated schools, they were coded as 1 (and 0 otherwise).
- All public schooling before college: respondents were asked to list all of the primary, elementary, middle, junior high and high schools they attended; and for each school, they were asked "Was this a public or private/religious school?". If all the schools the respondents attended before college were public schools, they were coded as 1 (and 0 otherwise).
- Preschool attendance: respondents were asked "Did you attend a pre-school, nursery school, or other program before primary/elementary school?". Those who answered "yes" were coded as 1, and 0 otherwise.
- Learned any foreign languages in high school: respondents were asked "Did you study a foreign language in high school?". Those who answered "yes" were coded as 1, and 0 otherwise. Respondents who did not attend high school were coded as 0.
- Learned any creative arts in high school: respondents were asked "In high school, did you take classes or spend time to do the following: 1) learn to play a musical instrument; 2) take singing lessons or sing in a chorus or choir; 3) learn woodwork or carpentry; 4) learn a craft (e.g., knitting, quilting, embroidery); 5) learn ballet or dance; and 6) learn to paint or draw or other art". Those who learned any of the aforementioned creative arts in high school were coded as 1, and 0 otherwise. Respondents who did not attend high school were coded as 0.

Relationship between Early-Life Circumstances and Cognition and Its Racial Disparities

Among the various early-life circumstances examined, educational attainment stands out as a distinct factor. This distinction arises because many other early-life circumstances may exert indirect effects on later-life cognition through their influence on educational attainment, in addition to their direct effect on cognition. For instance, early-life financial and health adversity can limit individuals' educational opportunities and attainment, resulting in diminished cognitive reserve and an elevated risk of cognitive impairment in later life.[27,28,46,96] Similarly, lower-quality schooling experience and segregated education can negatively affect educational attainment, particularly among Black older adults, thereby contributing to more significant racial disparities in cognition.[83,84,119–127] This relationship is further illustrated in the diagram below.

In our study, we primarily focused on the independent association of early-life circumstances (represented by the blue solid lines in the diagram). If an early-life factor demonstrates a direct, significant, and clinically meaningful association with later-life cognition and its racial disparities, independent of educational attainment and other early-life factors, then, from a clinical perspective, gathering additional data on such a factor can yield crucial insights for disease prevention, diagnosis, and intervention. Therefore, we have included all early-life factors in a model to estimate their independent association with the racial disparities, even considering the potential mediating role of educational attainment.

eAppendix B Figure: Diagram presenting the relationship between educational attainment and other early-life circumstances as well as their associations with racial disparities in cognition.



Notes: This diagram shows the conceptual relationship among educational attainment and other early-life circumstances, as well as their associations with racial disparities in cognition. Blue solid lines represent the identified associations, which represents the independent associations for each domain of factors. Black dashed lines represent the indirect association of other early-life circumstances on cognitive outcomes potentially mediated through educational attainment.

Variable Missingness and Multiple Imputation

The table below summarizes the N (%) of missingness for outcomes, covariates, and early-life circumstances. Since we required participants to have at least one wave of valid cognitive assessment between 1995 to 2018, participants had no missing data regarding cognitive outcomes. In the Life History sample (N=9015), covariates had either no or limited missingness (<0.2%); and

for 33 early-life circumstances included, 27 factors had 0-5% missingness, 5 factors had 5-10% missingness, and only 1 factor (years of father's educational attainment) had slightly above 10% missingness. Similar patterns of missingness were observed in the Life History sample for respondents who also had PGS data (N=7513).

Considering the large number of early-life circumstances included in the analysis, we performed multiple imputation to address item-level missingness of early-life factors.[129] We followed a multivariate imputation procedure adopted in the HRS to impute the early-life factors, which is a sequential regression approach (i.e., chained equations) that creates imputations through a sequence of multiple regressions.[130,131]

The imputation models included all the early-life factors (except genetic factors) and covariates included in the decomposition analyses, as well as a variety of variables that were predictive of missing values.[130,131] As the missingness of self-reported early-life factors and covariates could be associated with cognitive status, we followed the existing guideline to include our outcome variables of cognition as important predictors in the imputation models.[129] Moreover, we included a wide range of variables associated with the missingness in our imputation model to increase the likelihood that the "missing at random" assumption of multiple imputation holds while also addressing potential biases. These variables included birth year and month, highest degree obtained, college completion, working status, wealth, Medicare enrollment, Medicaid enrollment, military health plan enrollment, private health insurance coverage, employer-based health insurance coverage, hypertension, diabetes, cancer, lung diseases, heart diseases, stroke, psychiatric disorders, arthritis, ADL functional limitations, IADL functional limitations, and depressive symptoms.

Following existing guidelines, 20 imputed datasets were produced and analyzed; and the parameter estimates for each imputed dataset were pooled/combined in the BOD analyses based on Rubin's Rules.[132,133] Genetic factors were not imputed.

The primary findings were not sensitive to complete case analysis (with no variable imputation) and the estimates are available upon request.

	Missingness in the Life History Sample (N=9015), No. (%)
Outcomes and Covariates	
Cognitive score	0 (0.00%)
Cognitive impairment prevalence	0 (0.00%)
Age	0 (0.00%)
Sex	0 (0.00%)
Early-Life Circumstances	
AHEAD cohort (born before 1924)	0 (0.00%)
Children of Depression (CODA) cohort (1924-1930)	0 (0.00%)
Initial HRS cohort (1931-1941)	0 (0.00%)
War Baby (WB) cohort (1942-1947)	0 (0.00%)

Early Baby Boomer (EBB) cohort (1948-1953)	0 (0.00%)
Mid Baby Boomer (MBB) cohort (1954-1959)	0 (0.00%)
Late Baby Boomer (LBB) cohort or after (born in or after 1960)	0 (0.00%)
Born in the South	2 (0.02%)
Born outside of the US	2 (0.02%)
Lived in the South at age 10	1 (0.01%)
Lived outside of the US at age 10	1 (0.01%)
Relocated due to financial difficulties before age 16	39 (0.43%)
Family received financial help before age 16	159 (1.8%)
Father unemployed before respondent's age 16	704 (7.8%)
Household size at age 10, median (IQR)	95 (1.1%)
Adverse childhood health events	103 (1.1%)
Childhood trauma events (EFTF)	29 (0.32%)
Childhood trauma events (LHMS)	127 (1.4%)
Good relationship with mother before age 18	446 (4.9%)
Good relationship with father before age 18	712 (7.9%)
Years of educational attainment	38 (0.42%)
Years of father's educational attainment	1174 (13.0%)
Years of mother's educational attainment	666 (7.4%)
Owned at least one shelf of books at age 10	193 (2.1%)
Mother spent full time with children before age 18	171 (1.9%)
Absence of time/attention from mother when needed	282 (3.1%)
Absence of effort in upbringing from mother	288 (3.2%)
Absence of teaching about life from mother	289 (3.2%)
All segregated schooling before college	863 (9.6%)
All public school before college	632 (7.0%)
Preschool attendance	238 (2.6%)
Learned any foreign languages in high school	383 (4.2%)
Learned any creative arts in high school	353 (3.9%)

eAppendix C. Decomposition Analysis

Blinder-Oaxaca Decomposition (BOD) was used to measure the extent to which early-life circumstances may collectively and individually explain the racial disparities in cognitive outcomes, including cognitive score and cognitive impairment, between *White* and *Black* participants. The BOD decomposes mean differences in regression outcomes in a counterfactual manner and is widely used in economics to understand racial disparities.[134–144] It allows us to separate cognitive disparities between White and Black participants into a part that is associated with racial differences in early-life circumstances, versus a part that cannot be accounted for by such differences.[136,145] We tested the hypothesis that racial differences in early-life circumstances are significantly and sizably associated with racial disparities in cognitive outcomes.

We focused on comparing racial disparities in cognitive outcomes between White and Black participants and examined the extent to which racial differences in early-life circumstances were associated with racial disparities in cognitive outcomes.[146,147] Formally, the racial disparities in cognitive outcomes between White (W) and Black (B) participants can be formulated as,

$$R = E(Y_W) - E(Y_B) = E(X_W'\beta_W + \epsilon_W) - E(X_B'\beta_B + \epsilon_B) = E(X_W)'\beta_W - E(X_B)'\beta_B$$

where $E(Y_l)$ denotes the expected value of cognitive outcomes for race l, $l \in (W, B)$; and X_l is a matrix that contains early life circumstances and constants. The second and the third equalities hold under the assumptions of linear model, $Y_l = X_l'\beta_l + \epsilon_l,\ E(\epsilon_l) = 0$. The equation can be decomposed as follows.

$$R = \{E(X_W) - E(X_B)\}'\beta_B + E(X_B)'(\beta_W - \beta_B) + \{E(X_W) - E(X_B)\}'(\beta_W - \beta_B)$$

The first component $\{E(X_W) - E(X_B)\}'\beta_B$ represents the part of the racial disparities in outcome that is associated with the racial differences across all early-life circumstances. The second component $E(X_B)'(\beta_W - \beta_B)$ measures the associated racial disparities with racial differences in coefficients. The third component $\{E(X_W) - E(X_B)\}'(\beta_W - \beta_B)$ is an interaction term accounting for both the differences in early-life circumstances and coefficients. Our parameter of interest is the first component (i.e., endowment effects), which measures the expected changes in Black participants' mean cognition if they had the same levels of early life circumstances as White participants on average (i.e., the overall contribution of early-life circumstances). To understand how racial differences in individual characteristics are associated with racial disparities in cognitive outcomes, this component can be further decomposed as,

$$\{E(X_W) - E(X_B)\}'\beta_B = [E(X_{1W}) - E(X_{1B})]\beta_{1B} + [E(X_{2W}) - E(X_{2B})]\beta_{2B} + \cdots$$

where $E(X_1), E(X_2), \ldots$ are the expectation of individual characteristics; and $\beta_1, \beta_2, \ldots$ are the associated coefficients. $[E(X_{1W}) - E(X_{1B})]\beta_{1B}$, thus captures the part of racial disparities that is associated with the racial difference in individual characteristic X_1.

In this study, the decompositions were conducted at both the overall and the variable levels to evaluate the collective and individual contribution of early-life circumstances to racial disparities in cognition. For continuous outcome (i.e., cognitive score), BOD was conducted using a linear decomposition method (i.e., linear model), while for dichotomous outcome (i.e., cognitive

impairment), a nonlinear decomposition method (i.e., logit model) was employed.[134–144,148] Robust standard errors were estimated.

Grouping of individual factors: In our main findings, we present the racial disparities in cognition that were associated with specific domains of traditional early-life factors (e.g., financial factors). This estimation was achieved by summing the racial disparities linked to each individual factor within that domain, thereby capturing the total racial disparities associated with all the factors in that domain. Similarly, in our decomposition analysis, we computed the racial disparities associated with traditional early-life factors by summing the racial disparities related to each individual traditional early-life factor (i.e., Traditional = Cohort + Regional + Financial + Health + Trauma + Family Relationship + Educational Attainment). This additive calculation method was also applied when assessing the racial disparities associated with early-life educational experience, as well as the overall racial disparities associated with all included early-life factors.

Comparing Blinder-Oaxaca Decomposition and Mediation Analysis: The Blinder-Oaxaca decomposition (BOD) has been increasingly used in the public health literature for examining the sources of racial disparities[149–151] and has been featured in leading medical journals, such as the JAMA Network series.[152–156] An alternative approach commonly used in epidemiology is mediation analysis. While these two methods share similarities and are even considered equivalent in certain contexts,[145] they serve somewhat distinct purposes and come with their own sets of advantages and disadvantages.

BOD is primarily employed to decompose and quantify the extent to which various factors explain observed racial disparities. It offers a clear, direct, and quantitative breakdown of disparities between two racial groups, all without imposing strong assumptions regarding causal relationships or the functional form of variable relationships[145,150]. In contrast, mediation analysis primarily investigates the causal pathways through which one or more variables may mediate the relationship between race and the outcome[145]. It places a stronger emphasis on understanding the causal relationships and may include additional assumptions regarding functional forms.

Given that our study is focused on a comprehensive set of early-life circumstances associated with cognition without delving into causal relationships, the BOD is the more appropriate choice compared to mediation analysis. Several advantages support the use of BOD in our context:

- **Simplicity**: BOD is relatively simpler and more intuitive to use compared to mediation analysis. It provides an overview of numerous potential explanatory early-life factors and is more straightforward to interpret and communicate regarding the contributions of these factors.
- **Quantitative Attribution of Individual Factors**: BOD is better suited to quantify and comprehend the role of each individual factor or domain, offering a clear quantitative assessment and facilitating comparisons among various early-life factors.
- **No Assumption of Causality:** BOD does not require strong assumptions about causal relationships between variables during the decomposition process. Given the complexity of causal pathways in our setting, BOD is better suited for this comprehensive analysis with exploratory objectives.

Limitations of BOD: A key contribution of our work lies in providing novel evidence on the most comprehensive set of early-life circumstances and their associations with racial disparities in cognitive outcomes. However, two limitations of BOD should be acknowledged.

- **Differences in Mean Predicted Outcomes**: BOD provides information about the difference in mean predicted outcome between the two groups, which may differ from the crude difference due to variations in the distribution of other covariates between the two groups.[150]
- **Lack of Formal Causal Interpretation**: The BOD lacks a formal causal interpretation because the explanatory variables we are interested in may be confounded by unobserved variables [145]. When the focus is on interventions to equalize risk factors associated with certain early-life circumstances, the causal interpretations after the BOD can be questionable, as the early-life circumstances can be confounded.[145] Unlike BOD, mediation analysis does explicitly adjust for their confounders, but it often does so in a manner that effectively equalizes confounders across racial groups, potentially deviating from the intended intervention.[145]

Future research could consider focusing on particular early-life factors and implementing the causal mediation analysis to enable a more formal causal interpretation of the decompositions and to advance our understanding of potential mechanisms.

eAppendix D. Sensitivity Analysis

A comprehensive set of sensitivity analyses were performed in the study to demonstrate the robustness of the findings. Specifically, our findings proved robust to the adjustment of generic risks (eFigures 4-5), the inclusion of additional regional indicators to account for potential disparities in public education resources (eFigure 6); the use of a more streamlined set of early-life factors to address multicollinearity (eFigure 7) and mitigate overfitting (eFigure 8). We also adjusted for survey weights using the same wave of cognitive assessment to account for non-response and sampling and survey design (eFigure 9 for 2016, and eFigure 10 for 2018); and leveraged baseline cognitive assessment to account for potential sample dropout and mortality selection (eFigure 11).

Notably, when we excluded dementia patients from the analysis to reduce recall bias, the observed associations were more pronounced and robust, suggesting that our findings may have been somewhat conservative due to the presence of recall bias (eFigure 12). This is consistent with existing work that shows measurement errors originate from retrospective early-life measures in HRS modestly attenuate estimates of their associations with later-life outcomes.[157] Finally, our analysis, using alternative definitions of segregated schooling, indicated that the associations of segregated schooling likely accumulated, with a critical period observed during primary education (eFigure 13).

The detailed explanation and description for each sensitivity analyses are provided below:

1. Adjusting for Genetic Risks

As the racial disparities in cognition could be attributable to their differential biological predispositions, we adjusted for polygenic risk scores as genetic factors in the sensitivity analysis to account for such differences (see eAppendix A for the data and variables used for genetic factors). While the sample size slightly declined when using genetic data, the results were consistent (eFigures 4-5, N=7513).

2. Accounting for Potential Disparities in Public Education Recourses

The early-life factors included in our study, particularly educational experience, may reflect some of the regional disparities in public education resources. Data on the public funding of the school areas of the participants or other proxy measures such as zip code average household income may be linked to account for these regional disparities. However, we are unable to perform this analysis because publicly available HRS data do not include where participants resided during their schooling at the county or zip code level. Moreover, historical data at such granular geographic levels were difficult to obtain, especially for older cohorts. As an alternative approach, we performed a sensitivity analysis using additional regional indicators provided by the HRS public data file to account for regional differences more explicitly. Specifically, we additionally included (1) rural/urban status during individuals' school age (i.e., age 10); (2) rural/urban by census region indicators during school age; (3) census region indicators during school age; and (4) census region indicators. These elements constituted the early-life domain of regional factors. The findings remained unchanged (eFigure 6, N=9023).

3. Addressing Multicollinearity

Despite the number of early-life factors included, we found little concern of multicollinearity for most of these variables except for cohort factors. Specifically, we have checked multicollinearity by estimating the variance inflation index (VIF). A rule of thumb is that the VIF should be less than 10, and the lower the VIF, the less likely multicollinearity exists. We found that the VIFs were generally very small for most variables included (VIF<5, mean VIF=3.76). The only exception was cohort indicators that were included to account for cohort effects, which were correlated with age and had relatively large VIF. To address this issue, in the sensitivity analysis, we removed two cohort indicators, i.e., AHEAD cohort (0/1) and initial HRS cohort (0/1), that were strongly correlated with age. The variance inflation index (VIF) was small after excluding the two cohort indicators (all variable VIFs<5, mean VIF=1.75), indicating limited multicollinearity for this setting. The findings remained unchanged (eFigure 7, N=9015).

4. Mitigating Overfitting

A total of 32 early-life factors were included in our main setting. While the number of predictors were far less than the sample size ($p<<n$), overfitting may still be a possible concern for readers. Therefore, in the sensitivity analysis, we reduced the granularity of early-life factors to reduce overfitting. Specifically, for cohort factors, we only included two cohort indicators representing the cohorts that experienced the most critical historical events that might most substantially impact their early-life exposures, i.e., Children of Depression (CODA) cohort and War Baby (WB) cohort. For regional factors, we included whether the participants were born in the South and whether they were born outside of the US. For financial factors, we construct a dichotomous variable to denote whether participants had any of the three adverse financial events we considered, including relocated due to financial difficulties, family received financial help, father unemployed. For health factors, we did not change the definition. For trauma factors, we constructed a dichotomous variable to denote whether participants experienced any of the ten trauma events collected in the HRS survey. For parental educational attainment, we used the average years of mother's and father's educational attainment. All other factors remained unchanged. The number of early-life factors were reduced by half from 32 factors to 17 factors, which directly attenuates the potential concern of overfitting. The findings remained unchanged (eFigure 8, N=9015)

5. Weighted Analysis Using the Same Wave of Cognitive Assessment

In this study, as we aimed to retain all HRS life history participants, we matched participants with their latest cognitive assessments. This reduced sample attrition and maximized the sample size but made it challenging to apply HRS survey weights as they were not extracted from the same survey wave. Hence, in our main analysis, the results were unweighted.

Nonetheless, we tested the robustness of the results when accounting for sampling and survey design, non-response, and attrition by using survey weight. Specifically, we used the same HRS core survey for cognitive assessment and covariates and applied HRS survey weights in our decomposition analyses. We provided two weighted estimates based on the two latest waves of the survey prior to the COVID pandemic, including the 2016 core survey (eFigure 9, N=8334), and the 2018 core survey (eFigure 10, N=7338). While the sample size and statistical power were reduced due to the use of a single survey wave, the findings remained consistent using the weighted design.

6. Further Addressing Sample Selection Bias

Our study matched participants with their latest wave of cognitive assessment and covariates to maximize sample size. However, sample selection bias, such as differential timing of sample dropout and death, might be a potential concern. In the sensitivity analysis, we matched participants to their baseline (earliest) wave of cognitive assessment and covariates. The baseline wave was the time when the participants first entered the HRS survey and completed the cognitive assessment, which had very limited selection biases, i.e., no selection related to loss to follow-up, drop-out, and death. Although this pertains to the racial comparisons of much younger cohorts, our primary findings still hold (eFigure 11, N=9015).

7. Excluding Persons Living with Dementia to Investigate Recall Bias

Recall bias could be a potential concern for our estimates as it might directly relate to cognitive impairment. To examine how recall bias might affect our results, we performed a sensitivity analysis that excluded persons living with dementia (cognitive score range: 0-6). Persons living with dementia were most likely to suffer from recall biases and excluding them may provide insights on the potential influence of recall bias on our main estimates. As shown in eFigure 12 (N=8641), when we excluded dementia patients from the analysis to reduce recall bias, our observed associations were more pronounced and robust, suggesting that our main estimates (Figures 3 & 4, N=9015) may have been somewhat conservative due to the presence of recall bias. This is consistent with existing work that shows measurement errors originate from retrospective early-life measures in HRS modestly attenuate estimates of their associations with later-life outcomes, thereby making the results more conservative.[157]

8. Examining the Alternative Definitions of Segregated Schooling

In our study, segregated schooling was defined as whether the respondent experienced all segregated schooling before college (0/1). The reference group was those who did not experience all their education in segregated schools before college. We used this specification because Black adults who had this "extreme" school segregation experience can be more clearly defined and identified. They constituted a large proportion of Black older adults (>70% among Black adults) and have policy relevance due to their excessive vulnerability. Moreover, they would have been more likely to benefit from policy measures and interventions to reduce racial inequities. To further examine this variable, we present the decomposition estimates for two alternative specifications of segregated schooling with less "extreme" exposures. Specifically, in the first setting, we defined the exposure as all segregated schooling during primary education (0/1) or not, which encompasses the most critical period of cognitive development. In the second setting, we defined the exposure as attending any segregated school, regardless of degree and timing of exposure, or not (i.e., any segregated schooling vs. not). We expected the associations would diminish with the loose definition of the exposure. As shown in eFigure 13 (N=9015), all segregated schooling during primary education was still associated with large racial disparities in cognitive impairment but not cognitive score. This finding may imply the potential significance of the critical period (i.e., during primary school education) and the cumulative nature of the association. By contrast, for the least restrictive setting (any vs. no), we observed no association. This finding indicates that the degree of segregated schooling exposure matters, which further enriches our understanding of the policy question.

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eTable 1. Numerical Estimates of the Association between Racial Differences in Early-Life Circumstances and Racial Disparities in Cognition between White and Black Participants, LifeHistory1 (N=9015)

	Cognitive Score (Point)		Cognitive Impairment (Percentage Point, pp)	
	Mean (95% CI)		**Mean (95% CI)**	
White participants	15.8 (15.7, 15.9)		16.4 (15.6, 17.2)	
Black participants	13.5 (13.3, 13.7)		33.6 (31.3, 35.9)	
Total disparities between White and Black participants	2.3 (2.1, 2.6)		-17.2 (-19.6, -14.8)	
Decomposition Estimates: Association between Racial Differences in Early-Life Circumstances and Racial Disparities in Cognition				
	Associated Racial Disparities (95% CI)	**% Disparities Explained**	**Associated Racial Disparities (95% CI)**	**% Disparities Explained**
All early-life circumstances (combined)	0.81 (0.41, 1.2)	35.0%***	-6.4 (-10.8, -2.0)	37.4%**
Cohort factors	-0.09 (-0.40, 0.22)	-3.8%	-0.57 (-3.2, 2.0)	3.3%
Regional factors	0.21 (0.004, 0.42)	9.2%*	-0.81 (-2.7, 1.1)	4.7%
Financial factors	0.03 (-0.05, 0.11)	1.2%	-0.50 (-1.2, 0.16)	2.9%
Health factors	-0.006 (-0.06, 0.05)	-0.2%	-0.09 (-0.53, 0.34)	0.5%
Trauma factors	0.04 (-0.06, 0.14)	1.7%	-0.01 (-0.82, 0.79)	0.1%
Family relationship factors	-0.004 (-0.03, 0.02)	-0.2%	-0.02 (-0.21, 0.16)	0.1%
Years of educational attainment	0.63 (0.50, 0.75)	27.1%***	-4.4 (-6.1, -2.8)	25.7%***
Decomposition Estimates: Association between Racial Differences in Demographic Covariates and Racial Disparities in Cognition				
Age	-0.49 (-0.80, -0.18)		3.9 (1.2, 6.5)	
Sex	-0.05 (-0.08, -0.01)		0.09 (-0.18, 0.35)	

Notes: The decomposition analyses were performed using the Life History sample with traditional early life factors and covariates. Linear model was used for cognitive score, and logit model was used for cognitive impairment. For each domain of factors (e.g., financial factors), its association with racial disparities were estimated by summing the racial disparities linked to each individual factor within that domain, thereby capturing the total racial disparities associated with all the factors in that domain. This additive calculation was also applied when assessing the total racial disparities associated with all included early-life circumstances. Specifically, cohort factors

included AHEAD cohort (<1924), Children of Depression (CODA) cohort (1924-1930), initial HRS cohort (1931-1941), War Baby (WB) cohort (1942-1947), Early Baby Boomer (EBB) cohort (1948-1953), Mid Baby Boomer (MBB) cohort (1954-1959), Late Baby Boomer (LBB) cohort or after (>=1960). Regional factors included born in the South, born outside of the US, lived in the South at age 10, and lived outside of the US at age 10. Financial factors included relocated due to financial difficulties before age 16, family received financial help from relatives before age 16, father unemployed before age 16, and household size at age 10. Health factors included adverse health events before age 18. Trauma factors included childhood trauma events from EFTF, and childhood trauma events from LHMS. Family relationship factors included good relationship with mother before age 18, and good relationship with father before age 18. The early-life circumstances were self-reported. The unit of cognitive score is point; and the unit of cognitive impairment is percentage point (pp). The association of racial differences across all early-life circumstances and racial disparities in cognition is visualized in Figure 2, where we reverse the signs of absolute disparities in cognitive impairment for better visualization (i.e., same directions for both outcomes); and the interpretations are similar. Statistical significance of the contribution was denoted as asterisks in the table as: *** $P < 0.001$, ** $P < 0.01$, * $P < 0.05$.

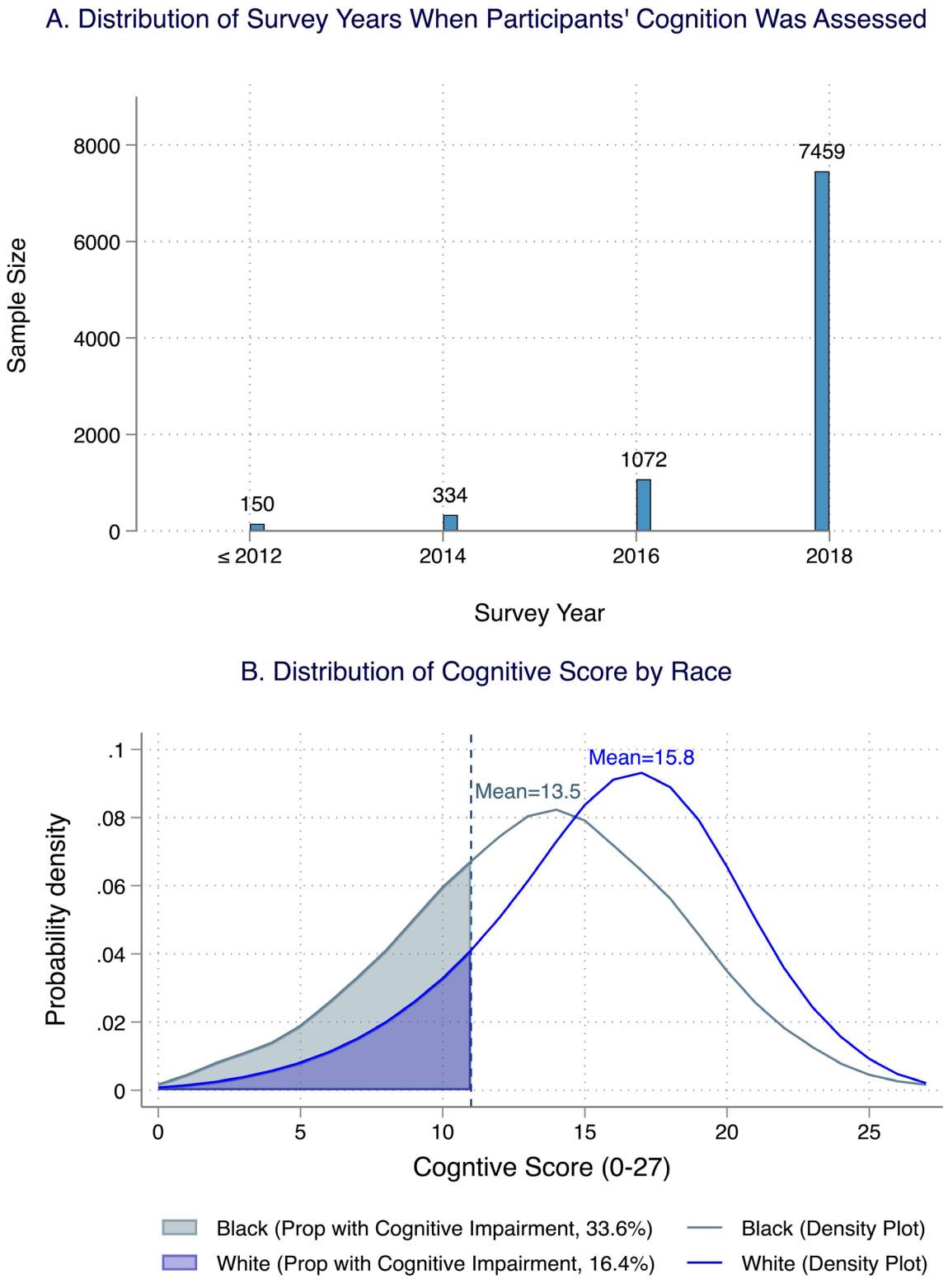
eTable 2. Numerical Estimates of the Association between Racial Differences in Early-Life Circumstances and Racial Disparities in Cognition between White and Black Participants, LifeHistory$_2$ (N=9015)

	Cognitive Score (Point)		**Cognitive Impairment (Percentage Point, pp)**	
Variable	**Mean (95% CI)**		**Mean (95% CI)**	
White participants	15.8 (15.7, 15.9)		16.4 (15.6, 17.2)	
Black participants	13.5 (13.3, 13.7)		33.6 (31.3, 35.9)	
Total disparities between White and Black participants	2.3 (2.1, 2.6)		-17.2 (-19.6, -14.8)	
Decomposition Estimates: Association between Racial Differences in Early-Life Circumstances and Racial Disparities in Cognition				
	Associated Racial Disparities (95% CI)	**% Disparities Explained**	**Associated Racial Disparities (95% CI)**	**% Disparities Explained**
All early-life circumstances (combined)	1.4 (0.88, 2.0)	61.5%***	-14.2 (-19.5, -8.8)	82.3%***
Traditional early-life factors (combined)	0.61 (0.20, 1.0)	26.3%**	-5.8 (-9.6, -2.0)	33.7%**
Cohort factors	-0.10 (-0.41, 0.20)	-4.5%	-0.44 (-3.3, 2.4)	2.6%
Regional factors	0.12 (-0.10, 0.34)	5.1%	-0.31 (-2.3, 1.7)	1.8%
Financial factors	0.02 (-0.06, 0.10)	1.0%	-0.58 (-1.3, 0.12)	3.4%
Health factors	-0.006 (-0.06, 0.05)	-0.3%	-0.08 (-0.56, 0.40)	0.4%
Trauma factors	0.03 (-0.07, 0.13)	1.2%	0.05 (-0.86, 0.95)	-0.3%
Family relationship factors	-0.003 (-0.03, 0.02)	-0.1%	-0.04 (-0.25, 0.18)	0.2%
Years of educational attainment	0.55 (0.43, 0.67)	24.0%***	-4.4 (-5.6, -3.2)	25.6%***
Early-life educational experience (combined)	0.81 (0.37, 1.3)	35.2%***	-8.4 (-12.7, -4.0)	48.6%***
Father's educational attainment	0.005 (-0.14, 0.15)	0.2%	0.04 (-1.3, 1.4)	-0.2%
Mother's educational attainment	0.03 (-0.08, 0.14)	1.3%	-0.27 (-1.2, 0.69)	1.6%
Owned at least one shelf of books at age 10	-0.009 (-0.08, 0.06)	-0.4%	0.49 (-0.14, 1.1)	-2.9%
Attention/effort/teaching from mother	0.02 (-0.10, 0.14)	0.9%	-0.61 (-1.7, 0.47)	3.5%

All segregated schooling before college	0.66 (0.26, 1.1)	28.8%**	-6.8 (-11.0, -2.6)	39.7%**
All public schooling before college	0.01 (-0.08, 0.11)	0.6%	-0.41 (-1.3, 0.50)	2.4%
Preschool attendance	0.02 (-0.02, 0.05)	0.7%	-0.19 (-0.50, 0.11)	1.1%
Learned any foreign languages in high school	0.06 (0.02, 0.11)	2.7%**	-0.51 (-0.91, -0.10)	2.9%*
Learned any creative arts in high school	0.009 (-0.006, 0.02)	0.4%	-0.07 (-0.20, 0.06)	0.4%
Decomposition Estimates: Association between Racial Differences in Demographic Covariates and Racial Disparities in Cognition				
Age	-0.44 (-0.75, -0.14)		4.0 (1.1, 6.8)	
Sex	-0.05 (-0.08, -0.01)		0.11 (-0.18, 0.40)	

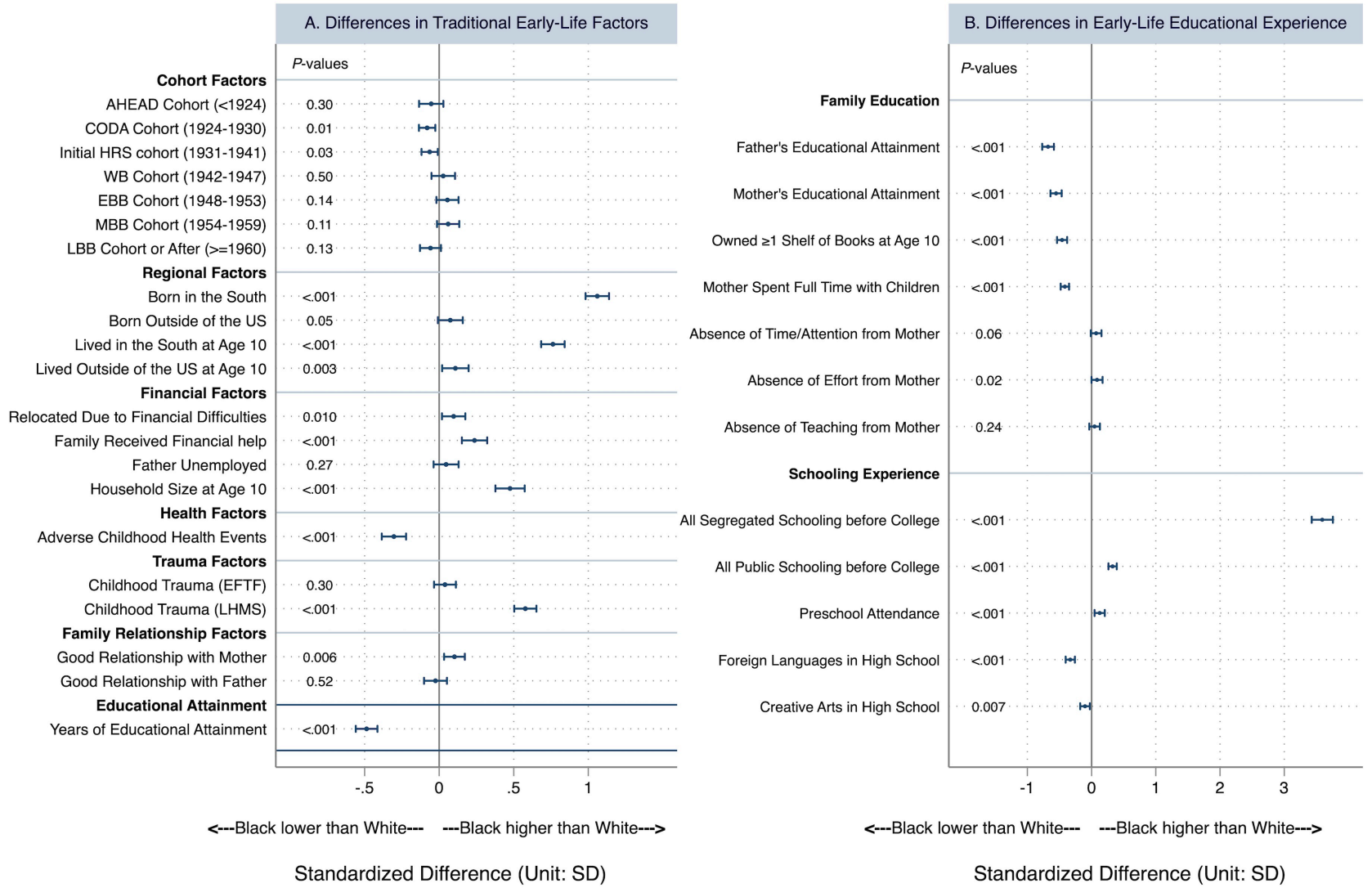
Notes: The decomposition analyses were performed using the Life History sample with traditional early life factors, additional early-life educational factors, and covariates. Linear model was used for cognitive score, and logit model was used for cognitive impairment. For each domain of factors (e.g., financial factors), its association with racial disparities were estimated by summing the racial disparities linked to each individual factor within that domain, thereby capturing the total racial disparities associated with all the factors in that domain. This additive calculation was also applied when assessing the total racial disparities associated with early-life traditional factors, educational experience, as well as the total racial disparities associated with all included early-life circumstances. Specifically, cohort factors included AHEAD cohort (<1924), Children of Depression (CODA) cohort (1924-1930), initial HRS cohort (1931-1941), War Baby (WB) cohort (1942-1947), Early Baby Boomer (EBB) cohort (1948-1953), Mid Baby Boomer (MBB) cohort (1954-1959), Late Baby Boomer (LBB) cohort or after (>=1960). Regional factors included born in the South, born outside of the US, lived in the South at age 10, and lived outside of the US at age 10. Financial factors included relocated due to financial difficulties before age 16, family received financial help from relatives before age 16, father unemployed before age 16, and household size at age 10. Health factors included adverse health events before age 18. Trauma factors included childhood trauma events from EFTF, and childhood trauma events from LHMS. Family relationship factors included good relationship with mother before age 18, and good relationship with father before age 18. The early-life circumstances were self-reported. The unit of cognitive score is point; and the unit of cognitive impairment is percentage point (pp). The estimates are visualized in Figure 2 and 3, where we reverse the signs of absolute disparities in cognitive impairment for better visualization (i.e., same directions for both outcomes); and the interpretations are similar. Statistical significance of the contribution was denoted as asterisks in the table as: *** $P < 0.001$, ** $P < 0.01$, * $P < 0.05$.

eFigure 1. The Distribution of Survey Year When Participants' Cognition Was Assessed and the Distribution of Cognitive Score by Race



***Notes*:** Panel A presents the distribution of survey years when participants' cognitive assessment were assessed. For each participant, their latest wave, i.e., most proximate to 2018, of cognitive assessment and demographic covariates from the same core survey were included to optimize sample size. Panel B presents the distribution of cognitive score (range: 0-27) for our study sample by race. Participants with cognitive scores ranging from 0 to 11 points were classified as having cognitive impairment. As shown in the Panel B, the racial differences in cognition pertain to the entire distributions of cognitive score, rather than a single cutoff point. In particular, the entire cognitive distribution of Black adults shifted to the left of White adults. This distributional difference led to a larger proportion of Black adults shifting to the lower tail of the cognitive distributions and being identified as having cognitive impairment. Such proportional differences pertain to all points and areas at the lower tail of the cognitive distributions (range: 0-11).

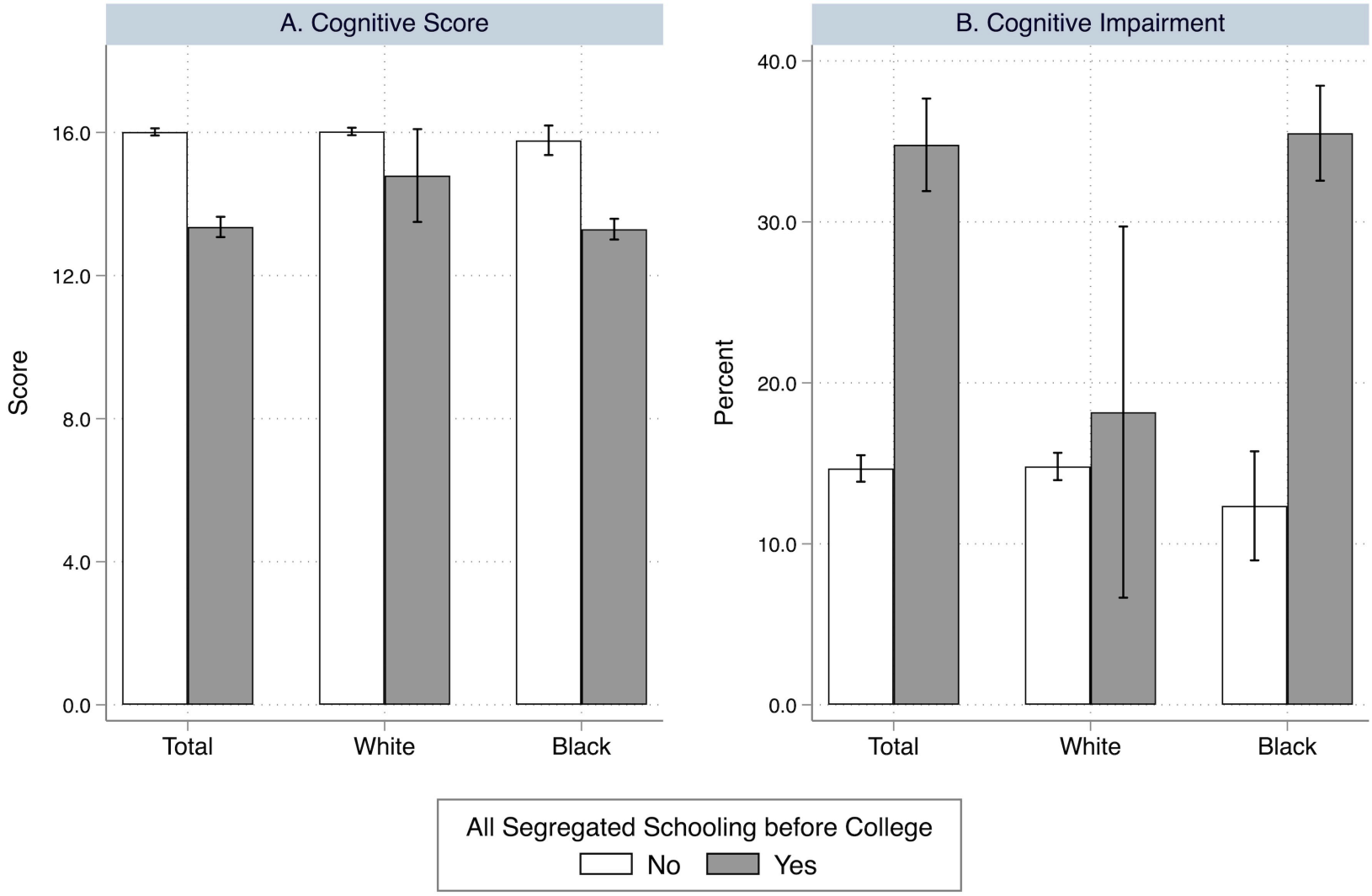
eFigure 2. Sensitivity Analysis: Weighted Standardized Differences in Early-Life Circumstances between Black and White Participants (N=8334)



Sensitivity Analysis: Standardized Differences in Early-Life Factors Using 2016 Survey Weight (N=8334)

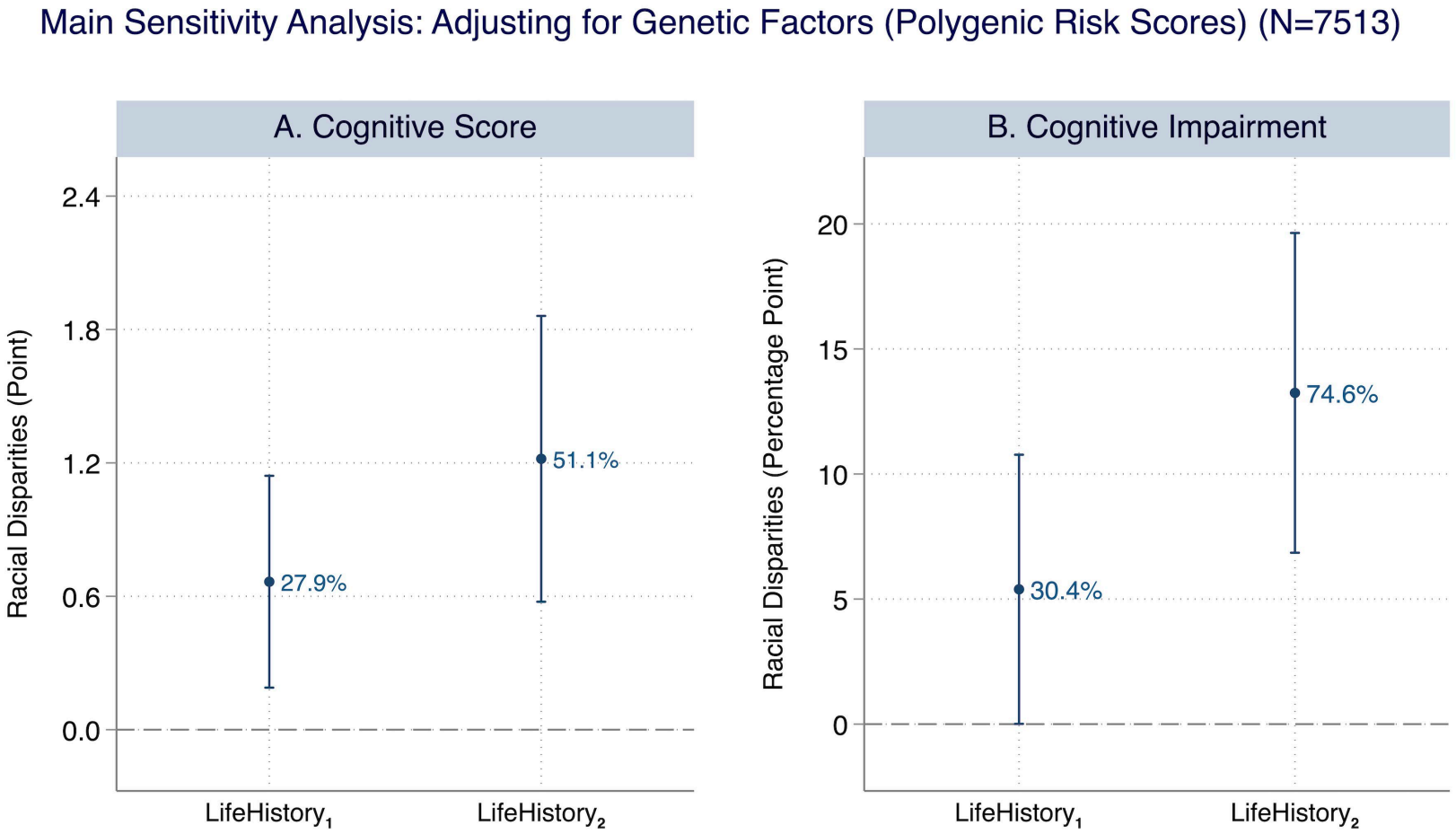
***Notes*:** The figure presents the Cohen's d standardized mean differences in individual early-life circumstances between Black and White participants, weighted by 2016 survey weight. The early-life circumstances were self-reported. The analysis was restricted to participants who participated in the 2016 core survey with valid survey weight (N=8334). In this figure, Panel A presents the estimates for traditional early-life factors; and Panel B presents the estimates for early-life educational experience. The scales of X axis for the two panels are different. For each early-life factors, the mean difference between Black and White participants was divided by their pooled standard deviation (SD) to obtain the standardized difference (unit: SD); and the estimates were visualized in the figure. The standardized differences were calculated to be comparable across various factors of different scales. The dotted points denote the Cohen's d standardized differences between Black and White participants for each early-life factors, and the horizontal line denotes the 95% confidence interval. A positive (negative) value of differences indicates Black participants had higher (lower) mean value of early-life factor than White participants. The P-values listed alongside the Y axis denote the statistical significance of the differences, estimated by Mann-Whitney-Wilcoxon tests for ordinal variables and Chi-square tests for binary and categorical variables. The differences were estimated using sample without data imputation, and sample size for each factor can be slightly less than 8334 due to item-level missingness. The results were similar when using 2018 wave of participants and survey weight (available upon request).

eFigure 3. Differences in Cognitive Outcomes between Participants with and without All Segregated Schooling before College in the Life History Sample



***Notes*:** This figure presents the average cognitive outcomes for participants with (in gray color) and without (in white color) all segregated schooling before college. The estimates were obtained for all participants (Total), White participants, and Black participants in the Life History sample. Panel A shows the average cognitive score; and Panel B shows the average proportion of cognitive impairment (%). The vertical bars represent the mean estimates, and the vertical lines present the 95% confidence interval. Sample sizes were respectively 7,097 (No) and 1055 (Yes) for Total participants, 6733 (No) and 44 (Yes) for White participants; and 364 (No), and 1011 (Yes) for Black participants. The early-life circumstances were self-reported.

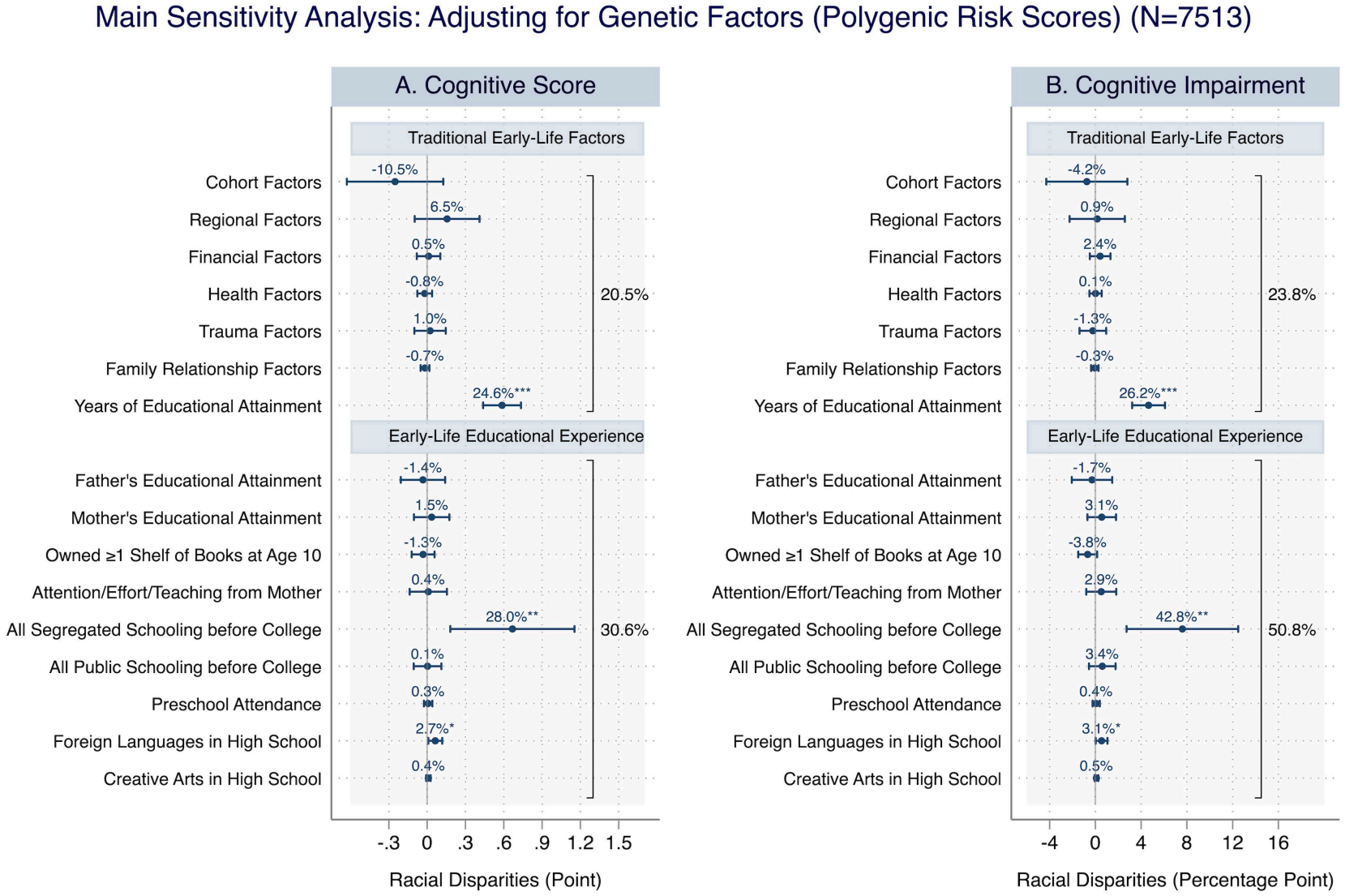
eFigure 4. Sensitivity Analysis: Association of Racial Differences Across All Early-Life Circumstances with Racial Disparities in Cognition between White and Black Participants with Genetic Adjustment (N=7513)



***Notes*:** This figure presents the estimates of main sensitivity analysis that adjusted for genetic factors (i.e., polygenic risk scores), which documents the association of racial differences across all early-life circumstances with racial disparities in cognition between White and Black participants with genetic adjustment (N=7513). Panel A presents the decomposition results for cognitive score, a continuous variable ranging from 0 to 27; and Panel B presents the decomposition results for cognitive impairment, a dichotomous variable

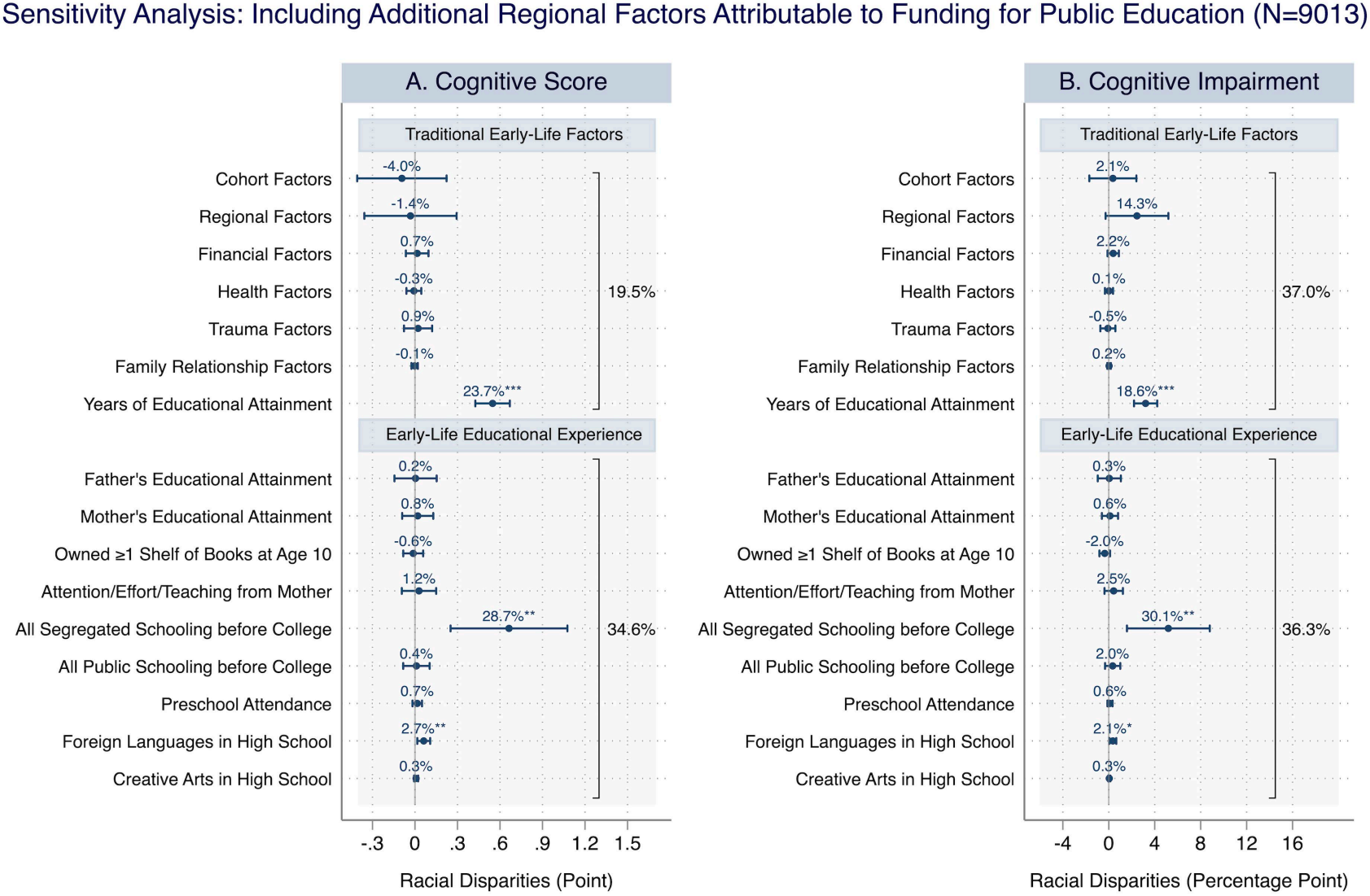
indicating whether individuals were classified as cognitive impaired or not (0/1). For each cognitive outcome, X axis denotes the models being examined, including LifeHistory$_1$ (with genetic adjustment), LifeHistory$_2$ (with genetic adjustment). In LifeHistory$_1$, traditional early-life factors were included to perform the decomposition. In LifeHistory$_2$, early-life educational experience were additionally added. All decompositions adjust for demographic and biological covariates including age, sex, and genetic factors. In Panels A and B, Y axis denotes the association of racial differences across all early-life circumstances with the racial disparities in cognition between White and Black participants. In Panel A, the unit of cognitive score is point; and in Panel B, the unit of cognitive impairment is percentage point (pp). The point estimates are plotted as circles and their 95% confidence interval are plotted as vertical lines. In each setting, the relative magnitude of racial disparities, expressed as percentage (%), is displayed alongside the horizontal lines. These percentages indicate the extent to which racial differences in early-life circumstances were associated with the total racial disparities in cognition between White and Black participants. Linear models were used for cognitive score, and logit models were used for cognitive impairment. The pooled estimates were obtained using multiple imputation with 20 imputed datasets. The early-life circumstances were self-reported.

eFigure 5. Sensitivity Analysis: Association of Racial Differences in Individual Factors or Domains of Early-Life Circumstances with Racial Disparities in Cognition between White and Black Participants with Genetic Adjustment (N=7513)



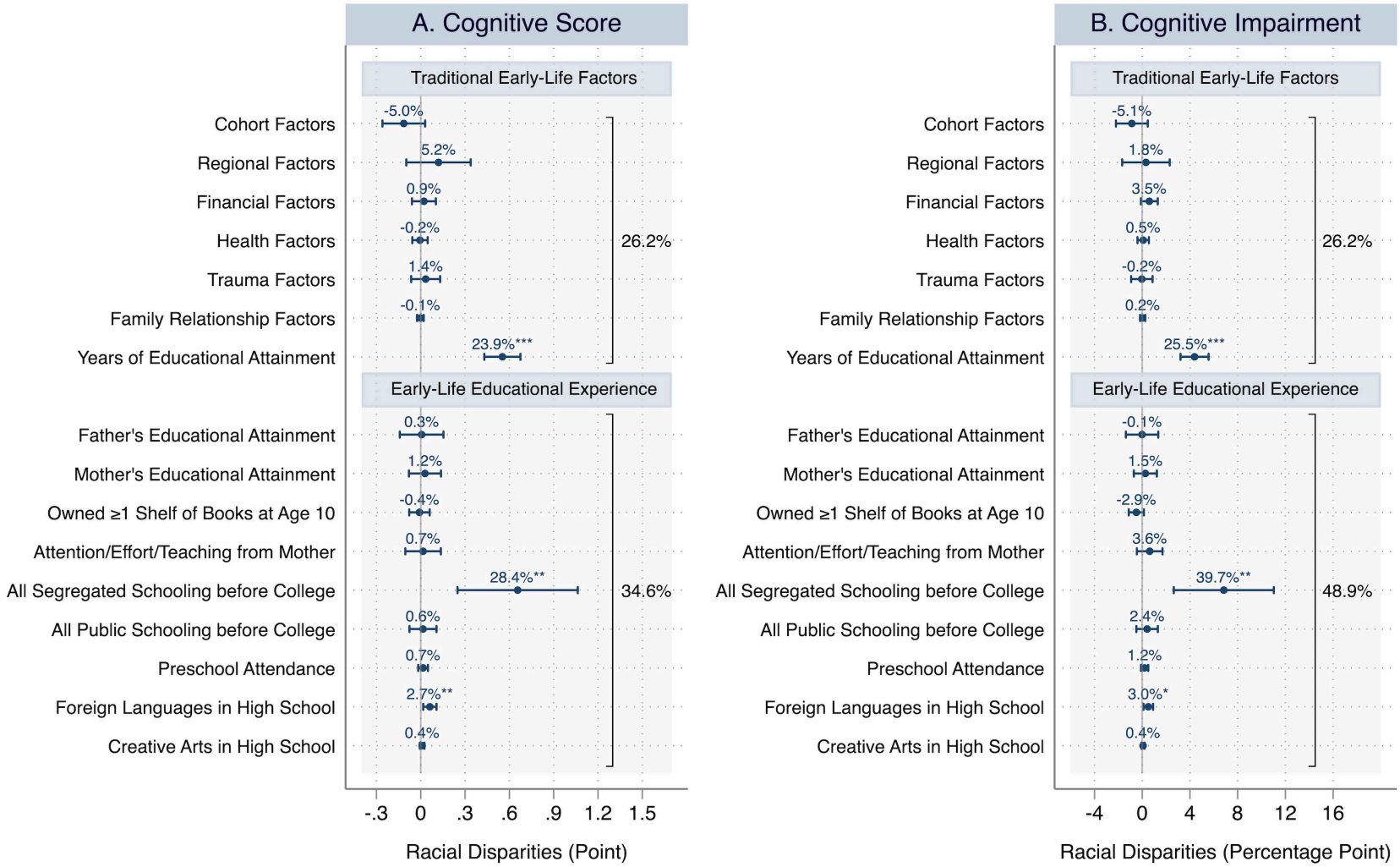
***Notes*:** This figure presents the estimates of main sensitivity analysis that adjusted for genetic factors (i.e., polygenic risk scores), which documents the association of racial differences in individual factors or domains of early-life circumstances with racial disparities in cognition between White and Black participants with genetic adjustment (N=7513). In this figure, Panel A shows the decomposition results for cognitive score, a continuous variable ranging from 0 to 27; Panel B shows the decomposition results for cognitive impairment, a dichotomous variable indicating whether individuals were classified as cognitive impaired (0/1), or not. For each cognitive outcome, Y axis denotes the individual factors or domains being examined; X axis denotes the association of racial differences in individual factors or domains of early-life circumstances with the racial disparities between White and Black participants. A positive value signifies that equalizing racial differences in individual early-life circumstances was positively associated with the reduction of racial disparities in cognitive outcomes. In Panel A, the unit of cognitive score is point; and in Panel B, the unit of cognitive impairment is percentage point (pp). The point estimates are plotted as circles and their 95% confidence interval are plotted as horizontal lines. The relative magnitude of racial disparities, expressed as percentage (%), is displayed alongside the horizontal lines. These percentages indicate the extent to which racial differences in individual early-life circumstances were associated with the total racial disparities in cognition between White and Black participants. For each domain of factors (e.g., financial factors), its association with racial disparities were estimated by summing the racial disparities linked to each individual factor within that domain, thereby capturing the total racial disparities associated with all the factors in that domain. This additive calculation was also applied when assessing the total racial disparities associated with early-life traditional factors, as well as educational experience. Linear models were used for cognitive score, and logit models were used for cognitive impairment. The pooled estimates were obtained using multiple imputation with 20 imputed datasets. The early-life circumstances were self-reported. Asterisks denote the statistical significance of the association: *** $P < 0.001$, ** $P < 0.01$, * $P < 0.05$.

eFigure 6. Sensitivity Analysis: Including Additional Regional Factors Attributable to Funding for Public Education (N=9013)



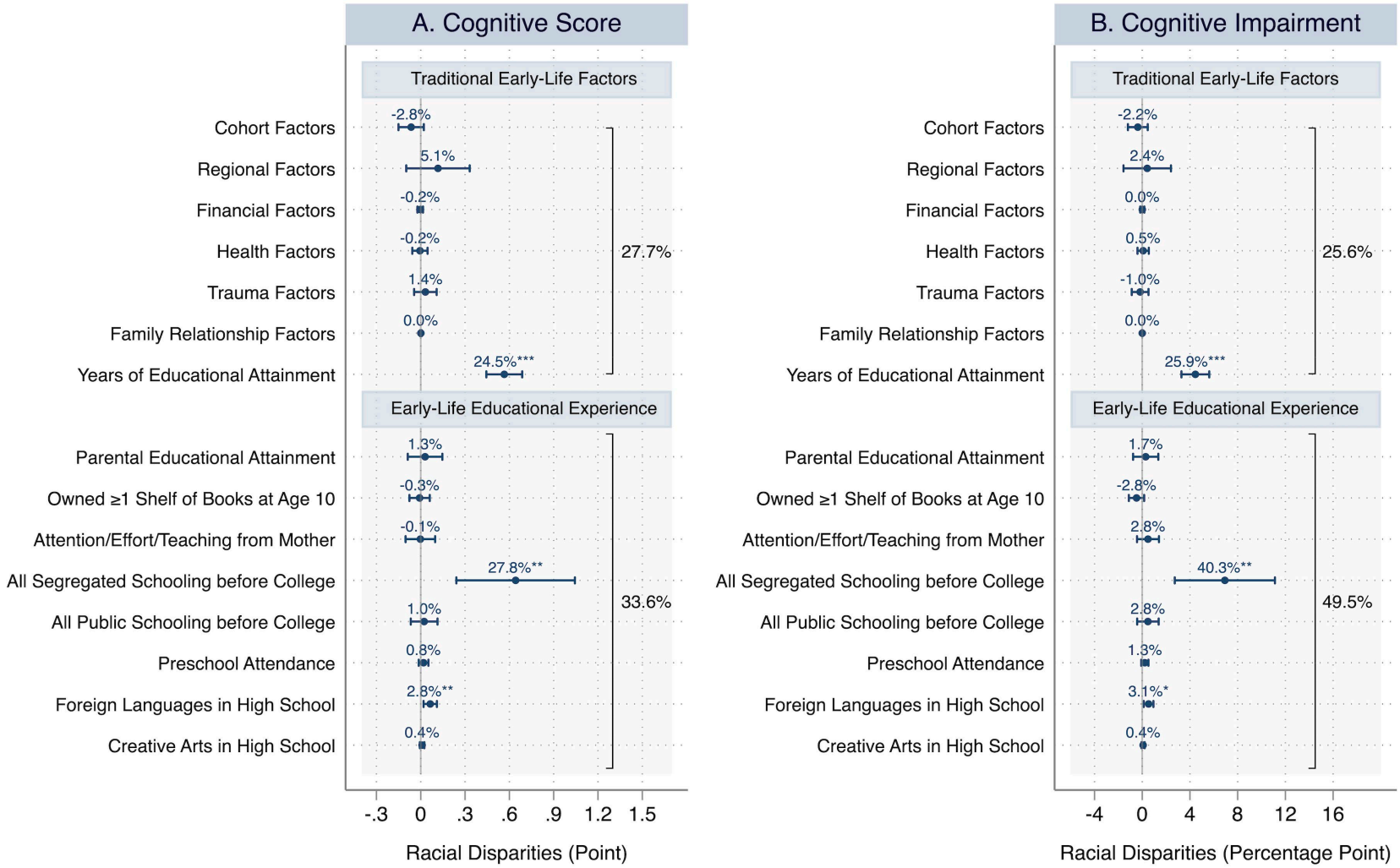
***Notes*:** This figure presents the estimates of sensitivity analysis that included additional regional factors closely related to funding for public education (N=9013). Specifically, we additionally included (1) rural/urban status during individuals' school age (i.e., age 10); (2) rural/urban by census region indicators during school age; (3) census region indicators during school age; and (4) census region indicators at birth. These elements collectively constituted the early-life domain of regional factors. In this figure, Panel A shows the decomposition results for cognitive score, a continuous variable ranging from 0 to 27; Panel B shows the decomposition results for cognitive impairment, a dichotomous variable indicating whether individuals were classified as cognitive impaired or not (0/1). For each cognitive outcome, Y axis denotes the individual factors or domains being examined; X axis denotes the association of racial differences in individual factors or domains of early-life circumstances with the racial disparities between White and Black participants. A positive value signifies that equalizing racial differences in individual early-life circumstances was positively associated with the reduction of racial disparities in cognitive outcomes. In Panel A, the unit of cognitive score is point; and in Panel B, the unit of cognitive impairment is percentage point (pp). The point estimates are plotted as circles and their 95% confidence interval are plotted as horizontal lines. The relative magnitude of racial disparities, expressed as percentage (%), is displayed alongside the horizontal lines. These percentages indicate the extent to which racial differences in individual early-life circumstances were associated with the total racial disparities in cognition between White and Black participants. For each domain of factors (e.g., financial factors), its association with racial disparities were estimated by summing the racial disparities linked to each individual factor within that domain, thereby capturing the total racial disparities associated with all the factors in that domain. This additive calculation was also applied when assessing the total racial disparities associated with early-life traditional factors, as well as educational experience. Linear models were used for cognitive score, and logit models were used for cognitive impairment. The pooled estimates were obtained using multiple imputation with 20 imputed datasets. The early-life circumstances were self-reported. Asterisks denote the statistical significance of the association: *** $P < 0.001$, ** $P < 0.01$, * $P < 0.05$.

eFigure 7. Sensitivity Analysis: Removing Cohort Indicators That Are Susceptible to Multicollinearity (N=9015)



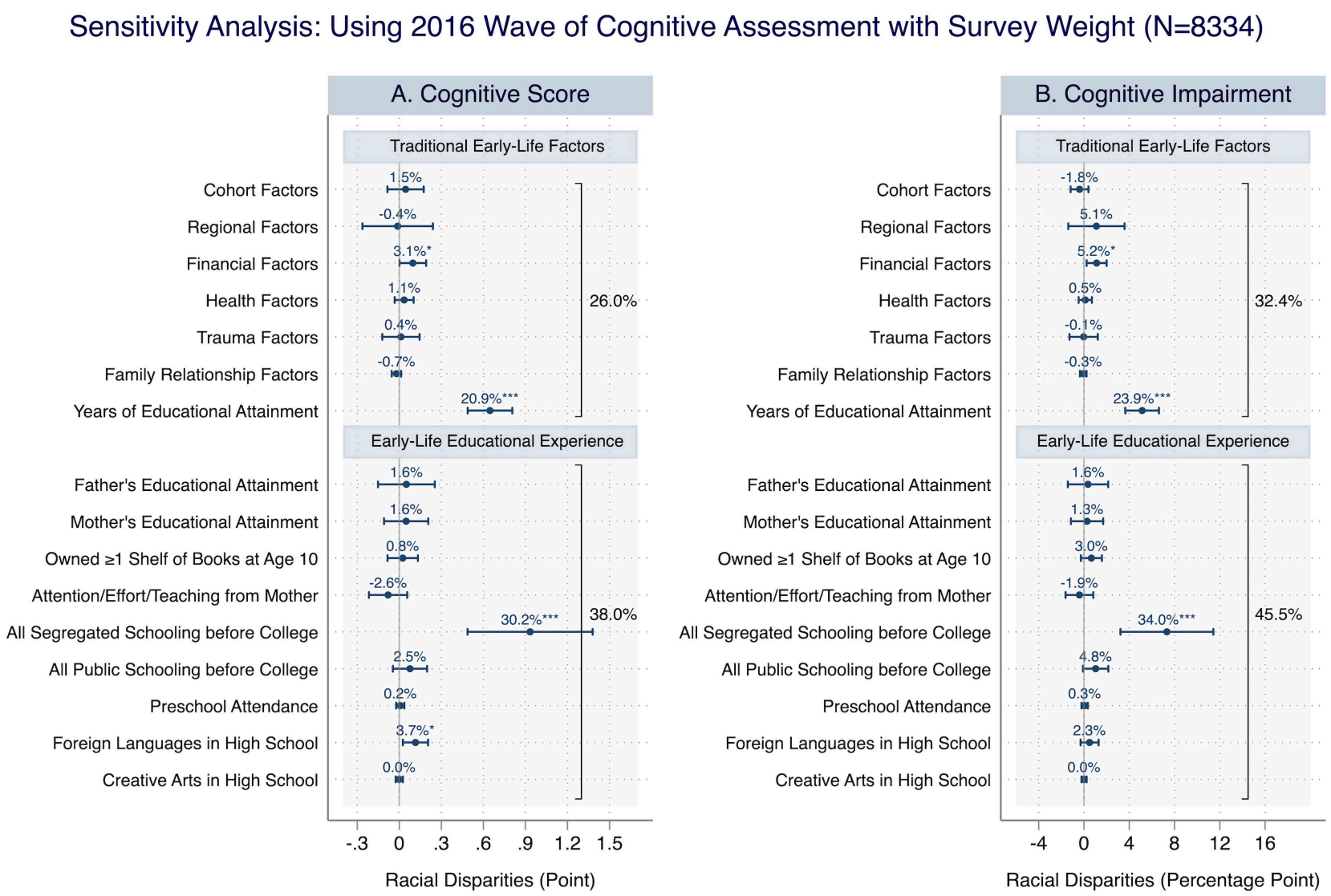
***Notes*:** This figure presents the estimates of sensitivity analysis which removed cohort indicators that are susceptible to multicollinearity (N=9015). Specifically, we excluded two cohort indicators, i.e., AHEAD cohort (0/1) and initial HRS cohort (0/1), that were strongly correlated with age. The variance inflation index (VIF) was small after excluding the two cohort indicators (all variable VIFs<5, mean VIF=1.75), indicating limited multicollinearity for this setting. In this figure, Panel A shows the decomposition results for cognitive score, a continuous variable ranging from 0 to 27; Panel B shows the decomposition results for cognitive impairment, a dichotomous variable indicating whether individuals were classified as cognitive impaired or not (0/1). For each cognitive outcome, Y axis denotes the individual factors or domains being examined; X axis denotes the association of racial differences in individual factors or domains of early-life circumstances with the racial disparities between White and Black participants. A positive value signifies that equalizing racial differences in individual early-life circumstances was positively associated with the reduction of racial disparities in cognitive outcomes. In Panel A, the unit of cognitive score is point; and in Panel B, the unit of cognitive impairment is percentage point (pp). The point estimates are plotted as circles and their 95% confidence interval are plotted as horizontal lines. The relative magnitude of racial disparities, expressed as percentage (%), is displayed alongside the horizontal lines. These percentages indicate the extent to which racial differences in individual early-life circumstances were associated with the total racial disparities in cognition between White and Black participants. For each domain of factors (e.g., financial factors), its association with racial disparities were estimated by summing the racial disparities linked to each individual factor within that domain, thereby capturing the total racial disparities associated with all the factors in that domain. This additive calculation was also applied when assessing the total racial disparities associated with early-life traditional factors, as well as educational experience. Linear models were used for cognitive score, and logit models were used for cognitive impairment. The pooled estimates were obtained using multiple imputation with 20 imputed datasets. The early-life circumstances were self-reported. Asterisks denote the statistical significance of the association: *** $P < 0.001$, ** $P < 0.01$, * $P < 0.05$.

eFigure 8. Sensitivity Analysis: Reducing the Granularity of Early-Life Factors to Reduce Overfitting (N=9015)



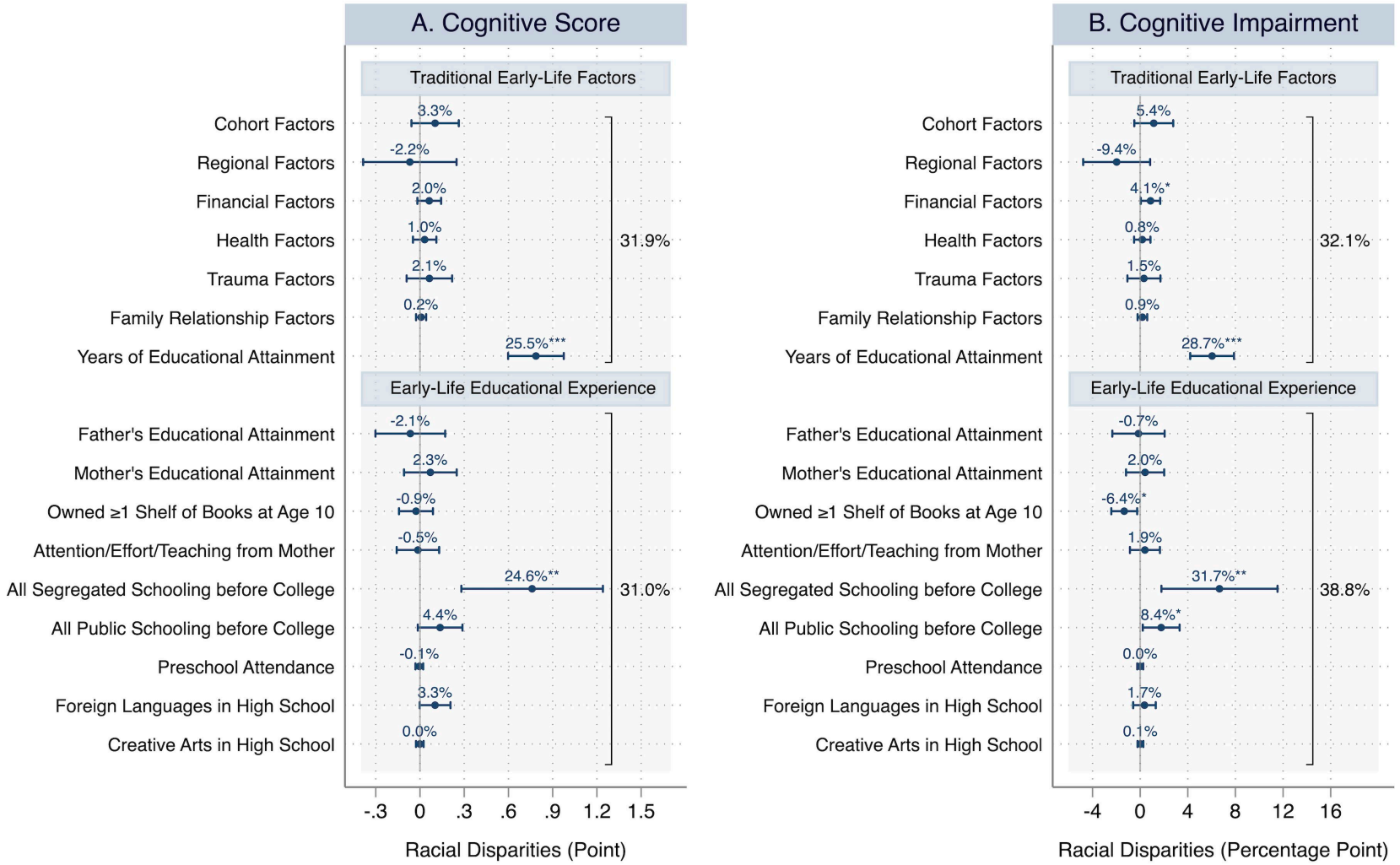
***Notes*:** This figure presents the estimates of sensitivity analysis that reduced the granularity of early-life factors to reduce overfitting (N=9015). Specifically, for cohort factors, we only included two cohort indicators representing the cohorts that experienced the most critical historical events that might most substantially impact their early-life exposures, i.e., Children of Depression (CODA) cohort and War Baby (WB) cohort. For regional factors, we included whether the participants were born in the South and whether they were born outside of the US. For financial factors, we construct a dichotomous variable to denote whether participants had any of the three adverse financial events we considered, including relocated due to financial difficulties, family received financial help, father unemployed. For health factors, we did not change the definition. For trauma factors, we construct a dichotomous variable to denote whether participants experienced any of the ten trauma events collected in the HRS survey. For parental educational attainment, we used the average years of mother's and father's educational attainment. All other factors remained unchanged. The number of early-life factors were reduced by half from 32 factors to 17 factors, which directly attenuate the potential concern of overfitting. In this figure, Panel A shows the decomposition results for cognitive score, a continuous variable ranging from 0 to 27; Panel B shows the decomposition results for cognitive impairment, a dichotomous variable indicating whether individuals were classified as cognitive impaired or not (0/1). For each cognitive outcome, Y axis denotes the individual factors or domains being examined; X axis denotes the association of racial differences in individual factors or domains of early-life circumstances with the racial disparities between White and Black participants. A positive value signifies that equalizing racial differences in individual early-life circumstances was positively associated with the reduction of racial disparities in cognitive outcomes. In Panel A, the unit of cognitive score is point; and in Panel B, the unit of cognitive impairment is percentage point (pp). The point estimates are plotted as circles and their 95% confidence interval are plotted as horizontal lines. The relative magnitude of racial disparities, expressed as percentage (%), is displayed alongside the horizontal lines. These percentages indicate the extent to which racial differences in individual early-life circumstances were associated with the total racial disparities in cognition between White and Black participants. For each domain of factors (e.g., financial factors), its association with racial disparities were estimated by summing the racial disparities linked to each individual factor within that domain, thereby capturing the total racial disparities associated with all the factors in that domain. This additive calculation was also applied when assessing the total racial disparities associated with early-life traditional factors, as well as educational experience. Linear models were used for cognitive score, and logit models were used for cognitive impairment. The pooled estimates were obtained using multiple imputation with 20 imputed datasets. The early-life circumstances were self-reported. Asterisks denote the statistical significance of the association: *** $P < 0.001$, ** $P < 0.01$, * $P < 0.05$.

eFigure 9. Sensitivity Analysis: Using 2016 Wave of Cognitive Assessment with Survey Weight (N=8334)



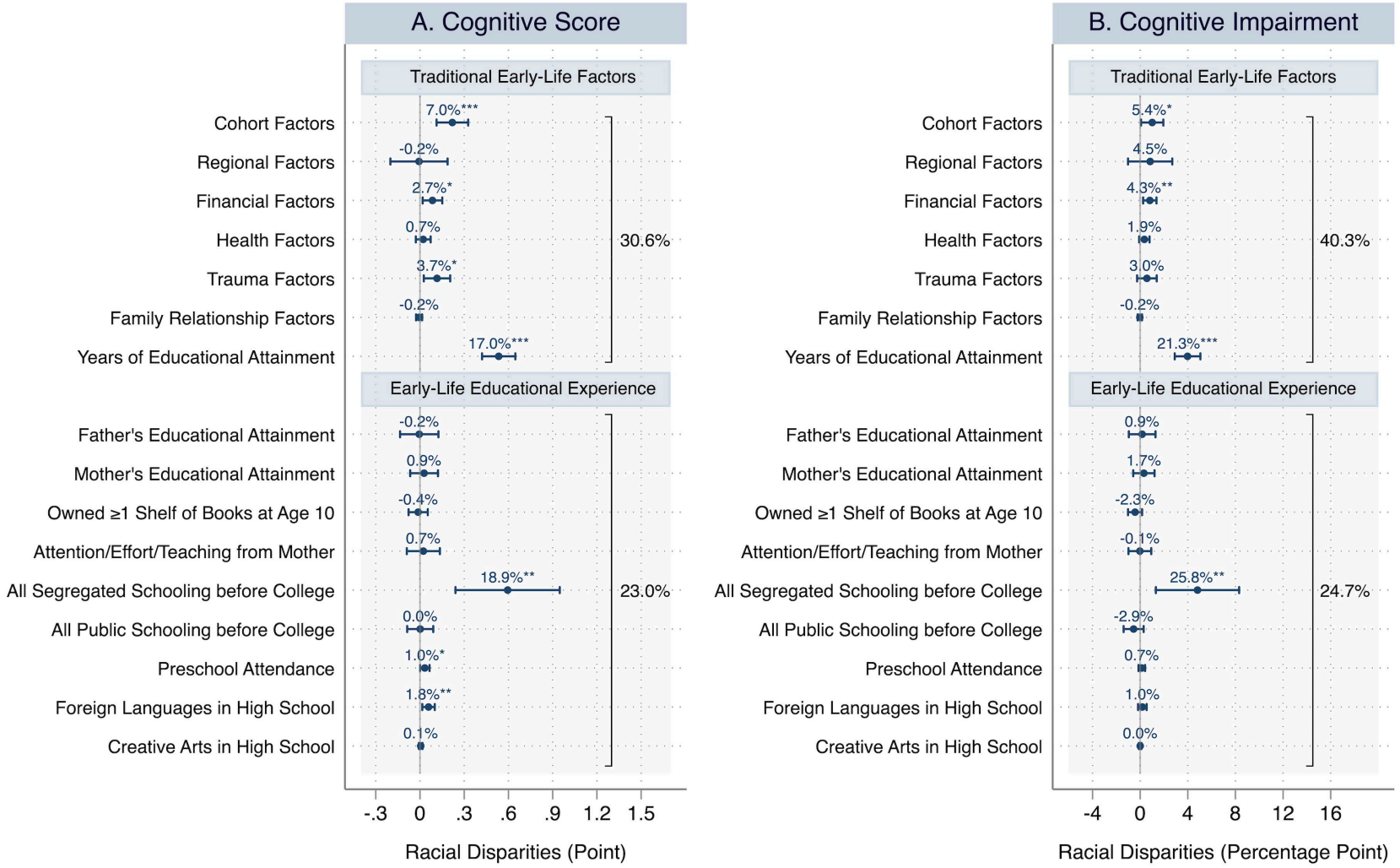
***Notes*:** This figure presents the weighted estimates of sensitivity analysis that use participants' 2016 wave of cognitive assessment and covariates (N=8334). The analysis was restricted to participants who participated in the 2016 core survey with valid survey weight (N=8334). We adjusted for survey weights using the same wave of cognitive assessment in 2016 to account for non-response and sampling and survey design. In this figure, Panel A shows the decomposition results for cognitive score, a continuous variable ranging from 0 to 27; Panel B shows the decomposition results for cognitive impairment, a dichotomous variable indicating whether individuals were classified as cognitive impaired or not (0/1). For each cognitive outcome, Y axis denotes the individual factors or domains being examined; X axis denotes the association of racial differences in individual factors or domains of early-life circumstances with the racial disparities between White and Black participants. A positive value signifies that equalizing racial differences in individual early-life circumstances was positively associated with the reduction of racial disparities in cognitive outcomes. In Panel A, the unit of cognitive score is point; and in Panel B, the unit of cognitive impairment is percentage point (pp). The point estimates are plotted as circles and their 95% confidence interval are plotted as horizontal lines. The relative magnitude of racial disparities, expressed as percentage (%), is displayed alongside the horizontal lines. These percentages indicate the extent to which racial differences in individual early-life circumstances were associated with the total racial disparities in cognition between White and Black participants. For each domain of factors (e.g., financial factors), its association with racial disparities were estimated by summing the racial disparities linked to each individual factor within that domain, thereby capturing the total racial disparities associated with all the factors in that domain. This additive calculation was also applied when assessing the total racial disparities associated with early-life traditional factors, as well as educational experience. Linear models were used for cognitive score, and logit models were used for cognitive impairment. The pooled estimates were obtained using multiple imputation with 20 imputed datasets. The early-life circumstances were self-reported. Asterisks denote the statistical significance of the association: *** $P < 0.001$, ** $P < 0.01$, * $P < 0.05$.

eFigure 10. Sensitivity Analysis: Using 2018 Wave of Cognitive Assessment with Survey Weight (N=7338)



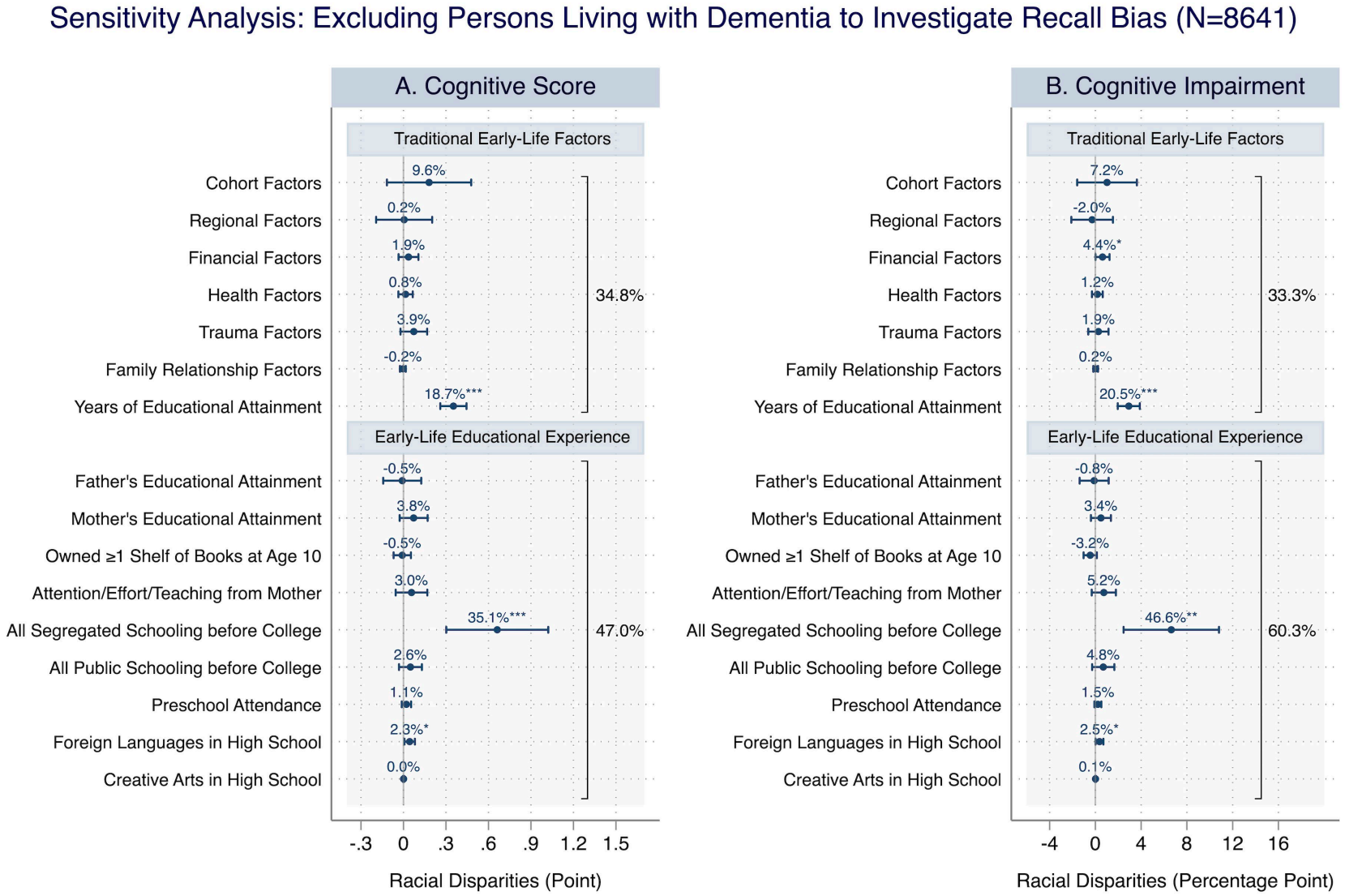
***Notes*:** This figure presents the weighted estimates of sensitivity analysis that use participants' 2018 wave of cognitive assessment and covariates (N=7338). The analysis was restricted to participants who participated in the 2018 core survey with valid survey weight (N=7338). We adjusted for survey weights using the same wave of cognitive assessment in 2018 to account for non-response and sampling and survey design. In this figure, Panel A shows the decomposition results for cognitive score, a continuous variable ranging from 0 to 27; Panel B shows the decomposition results for cognitive impairment, a dichotomous variable indicating whether individuals were classified as cognitive impaired or not (0/1). For each cognitive outcome, Y axis denotes the individual factors or domains being examined; X axis denotes the association of racial differences in individual factors or domains of early-life circumstances with the racial disparities between White and Black participants. A positive value signifies that equalizing racial differences in individual early-life circumstances was positively associated with the reduction of racial disparities in cognitive outcomes. In Panel A, the unit of cognitive score is point; and in Panel B, the unit of cognitive impairment is percentage point (pp). The point estimates are plotted as circles and their 95% confidence interval are plotted as horizontal lines. The relative magnitude of racial disparities, expressed as percentage (%), is displayed alongside the horizontal lines. These percentages indicate the extent to which racial differences in individual early-life circumstances were associated with the total racial disparities in cognition between White and Black participants. For each domain of factors (e.g., financial factors), its association with racial disparities were estimated by summing the racial disparities linked to each individual factor within that domain, thereby capturing the total racial disparities associated with all the factors in that domain. This additive calculation was also applied when assessing the total racial disparities associated with early-life traditional factors, as well as educational experience. Linear models were used for cognitive score, and logit models were used for cognitive impairment. The pooled estimates were obtained using multiple imputation with 20 imputed datasets. The early-life circumstances were self-reported. Asterisks denote the statistical significance of the association: *** $P < 0.001$, ** $P < 0.01$, * $P < 0.05$.

eFigure 11. Sensitivity Analysis: Using the Baseline (Earliest) Wave of Cognitive Assessment with Limited Selection Biases (N=9015)



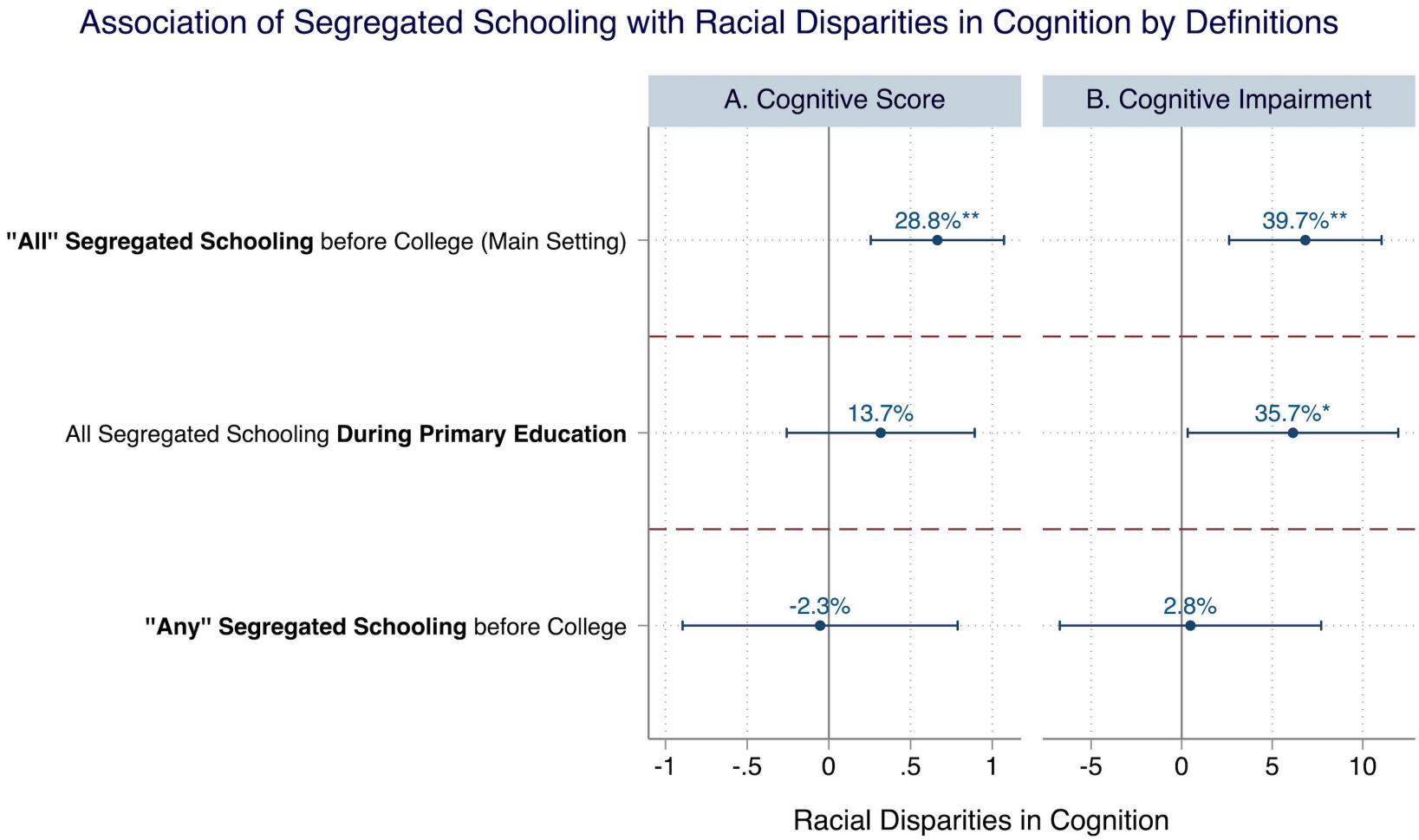
***Notes*:** This figure presents the estimates of sensitivity analysis that use participants' baseline (earliest) wave of cognitive assessment and covariates (N=9015). The baseline wave was the time when the participants first entered the HRS survey and completed the cognitive assessment, which have very limited selection biases (i.e., no selection related to loss to follow up, drop-out, and death). In this figure, Panel A shows the decomposition results for cognitive score, a continuous variable ranging from 0 to 27; Panel B shows the decomposition results for cognitive impairment, a dichotomous variable indicating whether individuals were classified as cognitive impaired or not (0/1). For each cognitive outcome, Y axis denotes the individual factors or domains being examined; X axis denotes the association of racial differences in individual factors or domains of early-life circumstances with the racial disparities between White and Black participants. A positive value signifies that equalizing racial differences in individual early-life circumstances was positively associated with the reduction of racial disparities in cognitive outcomes. In Panel A, the unit of cognitive score is point; and in Panel B, the unit of cognitive impairment is percentage point (pp). The point estimates are plotted as circles and their 95% confidence interval are plotted as horizontal lines. The relative magnitude of racial disparities, expressed as percentage (%), is displayed alongside the horizontal lines. These percentages indicate the extent to which racial differences in individual early-life circumstances were associated with the total racial disparities in cognition between White and Black participants. For each domain of factors (e.g., financial factors), its association with racial disparities were estimated by summing the racial disparities linked to each individual factor within that domain, thereby capturing the total racial disparities associated with all the factors in that domain. This additive calculation was also applied when assessing the total racial disparities associated with early-life traditional factors, as well as educational experience. Linear models were used for cognitive score, and logit models were used for cognitive impairment. The pooled estimates were obtained using multiple imputation with 20 imputed datasets. The early-life circumstances were self-reported. Asterisks denote the statistical significance of the association: *** $P < 0.001$, ** $P < 0.01$, * $P < 0.05$.

eFigure 12. Sensitivity Analysis: Excluding Persons Living with Dementia to Investigate Recall Bias (N=8641)



***Notes*:** This figure presents the estimates of sensitivity analysis that exclude persons living with dementia (N=8641). Persons living with dementia were most likely to suffer from recall biases and excluding them may provide insights on the potential influence of recall bias on our main estimates. As shown in the figure, when we excluded dementia patients from the analysis to reduce recall bias, our observed associations were more pronounced and robust, suggesting that our main estimates (shown in Figures 3 & 4) may have been somewhat conservative due to the presence of recall bias. In this figure, Panel A shows the decomposition results for cognitive score, a continuous variable ranging from 0 to 27; Panel B shows the decomposition results for cognitive impairment, a dichotomous variable indicating whether individuals were classified as cognitive impaired or not (0/1). For each cognitive outcome, Y axis denotes the individual factors or domains being examined; X axis denotes the association of racial differences in individual factors or domains of early-life circumstances with the racial disparities between White and Black participants. A positive value signifies that equalizing racial differences in individual early-life circumstances was positively associated with the reduction of racial disparities in cognitive outcomes. In Panel A, the unit of cognitive score is point; and in Panel B, the unit of cognitive impairment is percentage point (pp). The point estimates are plotted as circles and their 95% confidence interval are plotted as horizontal lines. The relative magnitude of racial disparities, expressed as percentage (%), is displayed alongside the horizontal lines. These percentages indicate the extent to which racial differences in individual early-life circumstances were associated with the total racial disparities in cognition between White and Black participants. For each domain of factors (e.g., financial factors), its association with racial disparities were estimated by summing the racial disparities linked to each individual factor within that domain, thereby capturing the total racial disparities associated with all the factors in that domain. This additive calculation was also applied when assessing the total racial disparities associated with early-life traditional factors, as well as educational experience. Linear models were used for cognitive score, and logit models were used for cognitive impairment. The pooled estimates were obtained using multiple imputation with 20 imputed datasets. The early-life circumstances were self-reported. Asterisks denote the statistical significance of the association: *** $P < 0.001$, ** $P < 0.01$, * $P < 0.05$.

eFigure 13. Association of Segregated Schooling with Racial Disparities in Cognition by Definitions (N=9015)



***Notes*:** This figure presents the association of segregated schooling with racial disparities in cognition between White and Black participants by different definitions of segregated schooling (N=9015 for all three settings). In this figure, Panel A shows the decomposition results for cognitive score, a continuous variable ranging from 0 to 27; Panel B shows the decomposition results for cognitive impairment, a dichotomous variable indicating whether individuals were classified as cognitive impaired or not (0/1). Each

dotted points represent the decomposition estimate of segregated schooling in a particular variable specification. In all settings, the variable was defined as binary, which has the advantages of estimation accuracy and more direct and straightforward interpretation in our decomposition analysis. The first row represents our main definition of segregated schooling, i.e., experiencing all segregated schooling before college (0/1), or not. The reference group was those who did not experience all their education in segregated schools before college. We used this specification because Black adults who had this "extreme" school segregation experience can be more clearly defined and identified. They constituted a large proportion of Black older adults (>70% among Black adults) and have policy relevance due to their excessive vulnerability. Moreover, they would have been more likely to benefit from policy measures and interventions to reduce racial inequities. In the second and third rows, we present the decomposition estimates for two alternative specification of segregated school attendance with less "extreme" exposures. Specifically, in the second row, we defined the exposure as all segregated schooling during primary education (0/1), or not, which represents the critical periods of cognitive development. In the third row, we defined the exposure as attending any segregated school, regardless of degree and timing of exposure, or not (i.e., any segregated schooling vs. not). We expected the associations would diminish with the loose definition of the exposure. As shown in the figure, all segregated schooling during primary education was still associated with large racial disparities in cognitive impairment but not cognitive score. This finding may imply the potential significance of the critical period (i.e., during primary school education) and the cumulative nature of the association. By contrast, for the least restrictive setting (any vs. no), we observed no association. This finding indicates that the degree of segregated school exposure matters. All the settings included the full list of early-life circumstances and demographic covariates in LifeHistory$_2$; and only the estimates for segregated schooling are shown in this figure (estimates for other factors/domains are available upon request). For each cognitive outcome, Y axis denotes different definitions of segregated schooling; X axis denotes the association of segregated schooling with racial disparities in cognition between White and Black participants. A positive value signifies that equalizing racial differences in individual early-life circumstances was positively associated with the reduction of racial disparities in cognitive outcomes. In Panel A, the unit of cognitive score is point; and in Panel B, the unit of cognitive impairment is percentage point (pp). The point estimates are plotted as circles and their 95% confidence interval are plotted as horizontal lines. The relative magnitude of racial disparities, expressed as percentage (%), is displayed alongside the horizontal lines. These percentages indicate the extent to which racial differences in individual early-life circumstances were associated with the total racial disparities in cognition between White and Black participants. Linear models were used for cognitive score, and logit models were used for cognitive impairment. The pooled estimates were obtained using multiple imputation with 20 imputed datasets. The early-life circumstances were self-reported. Asterisks denote the statistical significance of the association: *** $P < 0.001$, ** $P < 0.01$, * $P < 0.05$.