Original Article

AMERICAN SOCIOLOGICAL ASSOCIATION

Journal of Health and Social Behavior
2019, Vol. 60(3) 326–343

DOI: 10.1177/0022146519867924
jhsb.sagepub.com
SAGE

Cardiometabolic Risk and Cognitive Decline: The Role of Socioeconomic Status in Childhood and Adulthood

Katsuya Oi[1] and Steven Haas[2]

Abstract

Socioeconomic conditions in childhood predict cognitive functioning in later life. It is unclear whether poor childhood socioeconomic status (SES) also predicts the acceleration of cognitive decline. One proposed pathway is via cardiometabolic risk, which has been linked to both childhood SES and earlier onset of cognitive impairment. Using data from the Health and Retirement Study, we examine the impact of childhood SES on cognitive trajectories over six years and test whether it operates through increased cardiometabolic risk and adult SES. We find that higher childhood SES leads to slower cognitive decline, partially due to lower levels of cardiometabolic risk. However, these pathways operate entirely through adult socioeconomic attainment. The results have important implications for future trends in cognitive population health within the context of growing social inequality and reduced social mobility.

Keywords

cardiometabolic risk, cognitive aging, Health and Retirement Study, life course, socioeconomic status

Over the past two decades, the life course perspective has become the dominant theoretical paradigm with which to understand health. Childhood social conditions have been central to life course health research as important forces shaping trajectories of well-being. Sociological studies of aging and the life course consistently show that early life circumstances predict the timing of chronic disease onset (Hayward and Gorman 2004), the accumulation of physical limitations (Haas 2008), and death (Montez and Hayward 2014).

Likewise, childhood socioeconomic status (SES) shapes children's trajectories of cognitive development and ultimately, cognitive aging (Institute of Medicine 2000). However, the exact pathways through which childhood social conditions may influence later life cognitive trajectories remain unclear. The present study utilizes a life course approach to examine the processes that underlie childhood socioeconomic influences on later life cognitive trajectories. Specifically, we investigate whether childhood SES influences the rate of cognitive decline and how this association may be shaped by adult SES and cardiometabolic risk.

BACKGROUND

Understanding childhood socioeconomic influences on later life cognitive functioning first requires drawing a distinction between two socioeconomic patterns of cognition across the life course—*preserved differentiation* and *differential preservation* (Finkel et al. 2009). Preserved differentiation refers to the phenomenon in which those from socioeconomically disadvantaged childhoods experience

[1]Northern Arizona University, Flagstaff, AZ, USA
[2]Pennsylvania State University, Tempe, AZ, USA

Corresponding Author:
Katsuya Oi, Northern Arizona University, SBS Castro 308 5 E McConnell Dr, Flagstaff, AZ 86011, USA.
Email: Katsuya.oi@nau.edu

worse cognitive function in early life relative to their more advantaged peers—a gap that persists in similar magnitude throughout life. Alternately, differential preservation implies that greater socioeconomic resources provide recurring opportunities for cognitive growth and maintenance throughout the life course. As a result, cognitive performance between the more and less advantaged widens with age. This distinction is crucial in theoretically positioning childhood and adult SES as determinants of cognitive aging.

Life Course Processes and Later-Life Cognitive Functioning

The life course perspective provides a theoretical framework with which to understand the etiologic implications of socially embedded developmental and biological changes experienced by individuals embedded within socio-historical contexts (Elder, Johnson, and Crosnoe 2003). Or according to Bartley (2016:169), life course theory frames health trajectories as emerging at the "social–biological interface." In the case of cognition, life course theory offers three distinct yet non–mutually exclusive processes linking childhood and adult SES and their contribution to preserved differentiation and differential preservation.

The first process, *critical/sensitive periods*, is grounded in the life course principle that the consequences that events/exposures/transitions have is contingent on their timing within the life course (Elder et al. 2003). Critical/sensitive periods posit that early childhood is a period of life characterized by fundamental developmental change. During this period, adverse exposures such as socioeconomic deprivation may irreversibly affect developmental outcomes, permanently altering the structure and function of important body systems (e.g., hippocampal size) in ways that similar exposures occurring outside of this period would not (Ben-Shlomo, Cooper, and Kuh 2016; Cynader and Frost 1999; Daviglus et al. 2010). As such, a substantial "long arm of childhood" literature has emerged documenting critical/sensitive period effects on late-life physical (Hayward and Gorman 2004) and cognitive health (Zhang, Hayward, and Yu 2016).

As a critical period of exposure, childhood SES may create an initial gap in cognition between the more and less advantaged that persists from childhood onward (i.e., preserved differentiation). Childhood SES shapes the material and psychosocial conditions under which early physical, neurocognitive, and psychosocial development occurs, which molds the central nervous system in ways that are likely to persist over the life course. An early childhood environment unconducive to healthy development may result in permanent physiological and neurocognitive developmental deficits (Cynader and Frost 1999).

The second process focuses on the *accumulation of (dis)advantage* across the life course. First used to describe scientific status hierarchies, cumulative (dis)advantage emphasized the role of feedback loops (i.e., Merton's Matthew effect) in which early advantages beget even greater advantages (i.e., multiplicative effects; Merton 1968). Since Merton, multiple conceptualizations of the cumulative (dis)advantage concept have emerged in the sociological literature (DiPrete and Eirich 2006).

Medical sociologists have invoked the concept to describe socioeconomic inequalities in health expanding with age (Dannefer 1987; Ross and Wu 1996). These conceptualizations have further incorporated the life course principle that development and aging are life-long processes and that adaptations and outcomes can only be understood within the context of the cumulative history of prior experience beginning in utero (Elder et al. 2003). Elements consisting of that cumulative history, such as early exposure to health hazard, may be additive or multiplacative with each other, while healthy/risk behavior that individuals develop likely offsets some of those elements (Ferraro and Kelley-Moore 2003).

Differential preservation refers to socioeconomic inequalities in cognitive functioning widening with advancing age. This pattern strongly implies cumulative (dis)advantage processes at work. Childhood socioeconomic resources improve early cognitive development and facilitate the acquisition of cognitive skills via educational attainment. This improves adult socioeconomic position via increased occupational attainment, which in turn provides for the buildup of cognitive reserve, a mental faculty that constantly mitigates neurological damage (Stern 2012; Whalley et al. 2004). Thus, socioeconomically advantaged adults are better able to preserve their cognitive functioning than their less advantaged peers, creating a widening gap over time as childhood cognitive advantages become increasingly larger in late adulthood. Recent sociological work has elaborated similar dynamic life course feedback processes in which childhood health acts as a determinant of adult socioeconomic attainment (health selection), which in turn shapes later life health (social causation; Haas, Glymour, and Berkman 2011).

Critical periods highlight an independent role for childhood and adult SES while cumulative (dis) advantage implies their independent (additive) or, in the case of Merton's formulation, joint (multiplicative) effects. Conversely, the third process, the *pathways* mechanism, conceptualizes childhood and adult SES as forming a single *chain of risk*. A key distinction between cumulative (dis)advantage and pathways processes is that in the latter, childhood SES matters only for its role as a primary determinant of adult SES. The life course principle of linked lives provides a useful way of theorizing the pathways process connecting childhood SES to cognitive aging. Linked lives asserts that each life is lived embedded within a constellation of social relationships (Elder et al. 2003). As such, children's development is intrinsically linked to their parents and others within their social network and shapes the resources, relationships, difficulties, and constraints they experience. Thus, social conditions in the parental/familial environment establish the context within which cognitive and socioeconomic development occurs—initiating a chain of risk. For example, low educated parents are able to provide fewer opportunities for their offspring to develop their cognitive abilities and robust cognitive health (Harrison et al. 2015). As the classic Wisconsin social psychological model of status attainment demonstrates, familial resources also shape early life cognition while also fundamentally shaping socioeconomic aspirations and expectations, which facilitate educational and occupational attainment (Sewell, Haller, and Portes 1969). The material, psychosocial, and occupational resources that individuals gain as returns to human capital investment then serve as buffers against stress and cognitive decline. Lower occupational statuses, for instance, tend to involve less cognitive stimulation and greater stress exposure due to limited autonomy and control (Finkel et al. 2009).

Cardiometabolic Risk and Late-Life Cognitive Functioning

As cognitive trajectories unfold at the sociobiological interface, the life course processes discussed previously can also help theorize the biological pathways connecting childhood and adult SES to preserved differentiation and differential preservation. Specifically, we focus on cardiometabolic risk. Cardiometabolic dysregulation creates "wear and tear" on the body, manifesting in a variety of cardiovascular and metabolic morbidities, including hyperglycemia, chronic inflammation, and atherosclerosis (Elovainio et al. 2011; McEwen and Stellar 1993). Cardiometabolic risk and hyperactivity of the hypothalamic-pituitary-adrenal (HPA) axis are known predictors of cognitive impairment (O'brien et al. 1996) and impair the retention of cognitive functioning in later life (Schmitz et al. 2018). Cardiometabolic risk is one of several potential biological pathways linking socioeconomic deprivation and cognitive aging (Greenfield and Moorman 2018). However, this focus is well grounded in the emerging literature linking childhood adversity to biomarkers of cardiometabolic risk in adulthood (Suglia et al. 2018).

As a critical/sensitive period effect, childhood SES may contribute to preserved differentiation via the alteration of cardiometabolic function. In fact, most early research on critical/sensitive period health effects examined the impact of early life material and nutritional deprivation on late-life cardiometabolic outcomes (Barker 2007). This work documented that early life insults can "program" the body's physiology in ways that elevate heart rate, blood pressure, blood glucose, and lipids, manifesting in disease decades later (Ben-Shlomo et al. 2016). A broad array of adverse childhood exposures are associated with risk of obesity, diabetes, and cardiovascular disease (Hemmingsson, Johansson, and Reynisdottir 2014; McLaughlin et al. 2015). Childhood deprivation and adverse events have also been linked to chronic activation of the HPA axis and sympathetic nervous system (Barker 2007; Hackman, Farah, and Meaney 2010), which leave cardiovascular markers through the elevation of immunological and endocrine responses (McLaughlin et al. 2015).

Cardiometabolic function is also subject to processes of cumulative (dis)advantage. The stress-buffering effect of socioeconomic resources manifests physiologically among both children/adolescents and adults (Lupien et al. 2000; McEwen and Stellar 1993). Accordingly, the concept of allostatic load emerged in the 1990s to describe the cumulative wear and tear due to multisystem dysregulation resulting from a lifetime of adaptation to the stressors of life (McEwen and Stellar 1993). However, the extent to which childhood SES is an independent predictor of later life cardiometabolic risk net of adult SES remains unclear. Both childhood and adult SES have been linked to multisystem dysregulation (Evans and English 2002; Singer and Ryff 1999), suggesting they additively or multiplicatively influence later cardiometabolic risk, in which case, we would expect each to exert independent or joint effects on cognitive functioning.

Alternatively, the influence of childhood SES on later-life cardiometabolic risk may be completely mediated by adult SES. In that case, childhood disadvantage is simply a pathway to adult disadvantage, which initiates the chain of cardiometabolic risk, hastening cognitive decline.

Prior Evidence

Suggestive of critical/sensitive periods effects that contribute to preserved differentiation research across international contexts finds a positive correlation between childhood SES and the level of cognitive functioning in later life (Greenfield and Moorman 2018; Lyu 2015; Zhang et al., 2017). Some studies report a pathway process by which a substantial portion of childhood SES effects is accounted for by adult SES (Forstmeier et al. 2012; Horvat et al. 2014; Zhang et al. 2017). Consistent with cumulative (dis)advantage, other studies find finds that those who occupy low social positions in both childhood and adulthood are at particularly high risk (Lynch, Kaplan, and Shema 1997; Lyu 2015).

Evidence for the contribution of childhood SES to differential preservation remains conflicted. The NIH's consensus statement reports that childhood SES does not influence the rate of decline of cognitive function after baseline (Daviglus et al. 2010). This has largely been confirmed by subsequent studies (Greenfield and Moorman 2018; Lyu 2015; Staff et al. 2012). However, some evidence suggests that those from advantaged childhoods experience slower declines in cognitive functioning (Lyu and Burr 2016).

The inconsistent evidence for childhood SES and differential preservation is surprising given the consensus that childhood adversity threatens the integrity of cardiometabolic systems. One possible explanation is that cardiometabolic mechanisms of cognitive decline may be driven by more proximal adult SES. Research has identified a consistent link between adult SES and cardiometabolic risk (Kelli, Kassas, and Lattouf 2015). Nevertheless, we are not aware of any existing research investigating cardiometabolic risk as a mechanism connecting childhood SES and cognitive decline while simultaneously accounting for adult SES.

The Present Study and Hypotheses

The current study examines the extent to which childhood SES contributes to the differential preservation of late-life cognition through processes of cardiometabolic risk while accounting for adult SES. In doing so, we aim first to confirm the impact of childhood SES on cognitive aging. Do those with higher childhood SES experience slower rates of cognitive decline. We then examine if that association is mediated by cardiometabolic biomarkers. Finally, we test whether childhood SES determines the rate of cognitive decline independently of or jointly with adult SES. Substantively, empirical support for the independent roles of childhood SES implies differential preservation has its roots in childhood and critical/sensitive period processes, while an interactive role with adult SES indicates cumulative (dis)advantage—compounding an amplification of childhood advantages and disadvantages, depending on adulthood socioeconomic attainment. Conversely, substantial/complete mediation of the effect of childhood SES by adult SES would suggest a pathways or chains of risk process.

Based on the previous discussion, we offer four hypotheses.

Hypothesis 1: Higher childhood SES is associated with a slower rate of cognitive decline.

Hypothesis 2: Cardiometabolic risk mediates the effects of childhood SES.

Hypothesis 3: The direct pathway between childhood SES and the rate of cognitive decline and the indirect pathway through cardiometabolic risk remain after adjustment for adult SES.

Hypothesis 4: The association between adult SES and cognitive decline is amplified by childhood SES (i.e., a significant interaction between childhood and adult SES).

DATA AND METHODS

This study drew on data from multiple waves of the Health and Retirement Study (HRS), one of the longest running longitudinal studies of aging in the United States. The first wave was fielded in 1992, with follow-up surveys conducted biannually. Since 2006, the HRS has collected blood and saliva samples from a representative subsample of 16,438 respondents and assayed for cholesterol, blood glucose, C-reactive protein (CRP), and cystatin C (Crimmins et al. 2013). Participants also had their blood pressure, waist circumference, and physical performance (i.e., grip strength and timed walk) measured. The analytic sample was drawn from this subsample, who had biomarkers collected in 2006 and subsequently in 2010 and 2014. We further constrained our analysis to 9,449 individuals whose

cognitive functioning was assessed at least once since 2008. This restriction ensured that for every sample member, the baseline assessment of cognitive functioning was taken following the observation of their cardiometabolic risk.[1] We pooled 23,893 person-year observations of cognitive performance from 9,449 respondents. Each sample member contributed as many observations as were available. As the most recent available wave of HRS was 2014, respondents had a maximum of four observations for the Telephone Interview for Cognitive Status (TICS) score.

Cognitive Function

The HRS uses a modified version of the (TICS-M; Brandt, Spencer, and Folstein 1988). The TICS questionnaire has been used extensively by large surveys as a composite measure of general cognitive functioning (Bugliari et al. 2016). Respondents were administered 10-word immediate and delayed recall tests, a serial sevens subtraction test, a counting backwards test, an object naming test, and recall of the date, president, and vice president to assess working memory, attention and processing speed, language, and temporal orientation, respectively (Crimmins et al. 2013). For a review of the validity, reliability, and methodological limitations of TICS, see Lachman and Spiro (2002). The number of correct answers to TICS items quantifies individual cognitive performance on a scale from 0 to 35. All TICS items were standardized, and the scoring was harmonized across waves.

Cardiometabolic Risk Index (CM Index)

The analysis utilized nine markers of cardiometabolic risk: glycated hemoglobin (A1C), low density lipoproteins (LDL), high density lipoproteins (HDL), CRP, cystatin C, systolic and diastolic blood pressure, pulse, and waist circumference. High levels of A1C, cystatin C, blood pressure, pulse, and central adiposity collectively represent dysregulation in cardiometabolism (Schmitz et al. 2018). High CRP is a marker of inflammation, and its chronic elevation indicates vascular morbidities such as atherosclerosis and is an independent predictor of cardiometabolic dysfunction (Ridker, Wilson, and Grundy 2004). High LDL cholesterol is reflective of cardiovascular risk in the combination of low HDL cholesterol (Rizzo et al. 2008).[2]

Using these biomarkers, we constructed an index of cardiometabolic risk. For each biomarker, we determined a threshold by estimating the 75th percentile value based on the total HRS sample to ensure representativeness to the U.S. population. One exception is HDL, which utilizes the 25th percentile. The threshold values for all biomarkers are listed in Table 1. For a given biomarker, an individual was assigned a value of 1 if his or her value exceeded the threshold (or lower for HDL) and 0 if not. The index then summed across the markers and ranged between 0 and 9.[3]

Childhood SES

Three measures of childhood SES were used: mother's education, father's education, and the main occupation of the father/main breadwinner. Parental education was measured as years of completed schooling. Our measure of occupation focused on cognitive demands and thus taps into the concept of cognitive reserve. Three-digit census occupation codes for respondents and their father/main breadwinner were matched to the level of cognitive demands associated with each occupation using the Occupational Information Network (O*Net). O*Net data are widely used in cognitive research to assess the cognitive demands of occupations (Andel et al. 2015; Forstmeier et al. 2012). O*Net assesses a number of abilities, including oral expression, written expression, oral comprehension, written comprehension, deductive reasoning, inductive reasoning, fluency of ideas, originality, problem sensitivity, information ordering, and category flexibility. Scores for each ability domain range from 0 to 100—the occupation requires the highest possible level of a given ability. We constructed a composite rating for each occupation by averaging the 12 ability domain scores. The composite ratings were matched to respondent and parent occupation. Following prior research, we then transformed the composite rating into a *z*-score (based on the total HRS sample; Andel et al. 2015).

Adult SES

Adult SES was captured using four measures: household income, wealth, cognitive demands of longest held occupation, and educational attainment. Total household income and wealth represent the sum of the respondent's and spouse's total income or assets from all sources. These were then adjusted for inflation and household size and log transformed. Cognitive demands of longest held occupation was derived as previously described. Educational attainment was measured as years of completed schooling. We utilized the earliest

Table 1. Descriptive Statistics for Demographic and Cardiometabolic Variables (Health and Retirement Study 2006–2014).

Variable	Mean/Proportion	SD	Minimum	Maximum	Threshold[a]
Baseline age	70.72	10.31	50.00	100.32	
Non-Hispanic white	.70				
Hispanic	.11				
Non-Hispanic black	.16				
Other	.03				
Male	.44				
A1C (%)	5.89	1.00	3.01	17.26	6.14
C-reactive protein (ug/mL)	4.18	8.29	.02	219.12	4.51
Cystatin C (mg/L)	1.12	.57	.07	10.17	1.20
Low density cholesterol (mg/dL)	144.64	39.41	33.02	378.62	171.27
High density cholesterol (mg/dL)	53.96	15.84	12.11	130.04	42.74
Systolic blood pressure	134.70	22.16	68	240	149
Diastolic blood pressure	80.33	12.78	23	146	91
Pulse	69.86	11.34	25	133	78
Waist circumference (inches)	39.85	5.88	23	74.25	43.51
Cardiometabolic risk index	1.85	1.37	0	9	

Note: $N = 9{,}449$. SD = standard deviation.
[a]Threshold = 75th percentile value for all biomarkers except high density cholesterol (25th percentile).

available financial information, if possible, prior to first biomarker assessment.

Controls

The analysis included controls for age at first observation, race-ethnicity, and sex. Age was centered on the sample mean. Race-ethnicity was measured in four categories: non-Hispanic white, non-Hispanic black, Hispanic, and other. Descriptive statistics for the sample are provided in Table 1.

Analytic Plan

We estimated latent growth curves of cognitive function within a structural equation framework (SEM; Muthén 2004:347). A SEM approach to growth curve modeling is distinct from the multilevel modeling (MLM) approach. While both approaches yield identical estimates of trajectories with the same model specification, depending on specific applications, each has practical advantages and disadvantages (Hox and Stoel 2014). For our purposes, SEM has three advantages over MLM. The first is its ease in incorporating latent constructs allowing multiple indicators and a complex measurement structure for childhood and adult SES. Second, SEM offers a more flexible approach to specifying time scores and the functional form of change over time (elaborated on in the following). Finally, the MLM approach is inherently univariate while the SEM approach is inherently multivariate. The multivariate nature of SEM provides a straightforward approach to modeling the complex pathways between constructs as well as ease of decomposing total, direct, and indirect pathways of mediation. This was particularly advantageous in testing our hypotheses as it enabled us to estimate the total effect of childhood SES on the rate of cognitive decline and decompose its potential direct and indirect effects via adult SES and cardiometabolic risk while also accounting for between-individual differences in cognitive performance at the baseline.

The model consists of a measurement component, which statistically links the observed variables to the latent constructs that they measure, and a structural component, composed of multiple equations that relate the latent constructs to other variables of interest (Kline 2004). Figure 1 presents a path diagram of the model. Four variables in ovals represent latent constructs, childhood SES, adult SES, and the intercept and slope of cognitive trajectories, based on the corresponding set of observed measures.

The unconditional (without covariates) latent growth curve model for the TICS score (Y) for individual i at time t can be expressed as

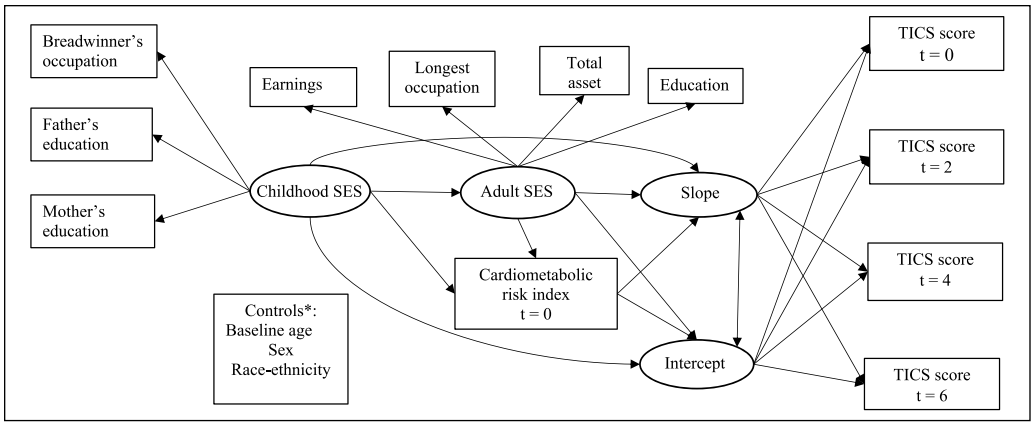

Figure 1. Latent Growth Curve Model Fitted to the TICS Score Observed at Four Time Points, with Parental SES, Adult SES, and Cardiometabolic Risk Index as Covariates.

Note: t represents the number of years elapsed since the baseline. SES = socioeconomic status; TICS = Telephone Interview for Cognitive Status.

*Adult SES, childhood SES, biomarkers, slope, and intercept are regressed on controls.

$$Y_{it} = \eta\alpha i + \lambda_t \eta\beta i + \varepsilon_{it},$$

where $\eta\alpha_i$ and $\eta\beta_i$, respectively, represent the intercept and slope for individual i and ε_{it} are individual and time-specific random errors. The observed TICS score at the baseline is Y_{i0}, with subsequent time points denoted as Y_{i2}, Y_{i4}, and Y_{i6}. The individual intercepts are latent components expressed as the sum of the overall mean score at baseline ($\mu\alpha$) and their individual deviation from it ($\zeta\alpha_i$) ($\eta\alpha_i = \mu\alpha + \zeta\alpha_i$). Individual slopes can be expressed as a function of the average change in the TICS score over time ($\mu\beta$) and the individual deviation ($\zeta\beta_i$) ($\eta\beta_i = \mu\beta + \zeta\beta_i$). In a linear growth model, λ_t would be fixed to [0, 2, 4, 6], corresponding to time score factor loadings for four observation periods starting at time 0 and then at subsequent two-year intervals. However, rather than a linear model, we estimated a freely specified model in which $\lambda_t = [0, *, *, *, 1]$. In this model, * represent estimated (i.e., free rather than fixed) factor loadings corresponding to the proportion of total change occurring by each observation point (i.e., t = 0, 2, 4, and 6), and $\eta\beta_1$ represents a general shape factor of total change over the period rather than an annual rate of change. The advantage of this specification is that it does not impose a particular functional form on trajectories of cognition at the individual or population level. This flexibility is especially useful in the HRS, which covers a wide range of birth cohorts observed at different spans of later life, across which the underlying functional form of cognitive trajectories is likely to vary substantially. In addition, the model fit was superior with the freely estimated model compared to linear/curvilinear specifications. We further allowed the latent intercept and slope to correlate.

In conditional models with covariates (observed and latent), the latent intercept and slope can be further expressed as:

$$\begin{aligned}\eta\alpha i &= \mu_{\alpha 1} + \mu_{\alpha 2}\eta_{ASESi} + \mu_{\alpha 3}\eta_{CSESi} + \mu_{\alpha x}X\ldots + \zeta_{\alpha i}\\ \eta\beta i &= \mu_{\beta 1} + \mu_{\beta 2}\eta_{ASESi} + \mu_{\beta 3}\eta_{CSESi} + \mu_{\beta x}X\ldots + \zeta_{\beta i}\end{aligned},$$

where η_{ASESi} and η_{CSESi} represent the latent adult SES and childhood SES factor scores for individual i, respectively, and X are other observed covariates. The latent constructs, childhood SES and adult SES, were each estimated with a one-factor confirmatory factor model. The measurement model for η_{ASES} and η_{CSES} is

$$\boldsymbol{Y} = \boldsymbol{\lambda\eta} + \boldsymbol{\delta}$$

$$\begin{bmatrix} Y_{1income} \\ Y_{2assets} \\ Y_{3Education} \\ Y_{4Occupation} \\ Y_{5Mother\ Ed.} \\ Y_{6Father\ Ed.} \\ Y_{7Parental\ Occ.} \end{bmatrix} = \begin{bmatrix} \lambda_{11} & 0 \\ \lambda_{12} & 0 \\ \lambda_{13} & 0 \\ \lambda_{14} & 0 \\ 0 & \lambda_{21} \\ 0 & \lambda_{22} \\ 0 & \lambda_{23} \end{bmatrix} \begin{bmatrix} \eta_{ASES} \\ \eta_{CSES} \end{bmatrix} + \begin{bmatrix} \delta_1 \\ \delta_2 \\ \delta_3 \\ \delta_4 \\ \delta_5 \\ \delta_6 \\ \delta_7 \end{bmatrix}.$$

Estimation was accomplished via full information maximum likelihood (FIML) using Mplus 6.12. As not all individuals had cognition measured at all observation points, FIML takes advantage of all available information, allowing those individuals with missing information in any particular wave to contribute to the estimation. Similar to the logic of imputation (Oi 2017), the FIML estimation for missing cases was also aided by variables that predict missing values but are not included in the main analysis. These variables included time-varying measures of widowhood, retirement/employment status, activities of daily living, whether their mother/father was alive, and family caregiving obligations, all of which are linked to late-life cognitive functioning (Insler 2014). The results were robust to the selection of auxiliary variables.

Arrows shown in Figure 1 are estimated as coefficients, with the origin as a predictor and the destination as a dependent variable. Arrows connecting from childhood SES, adult SES, and the CM index to the intercept and slope are of interest here. Hypothesis 1 predicts a positive path coefficient of childhood SES predicting the slope (childhood SES→slope): A higher level of childhood SES leads to a smaller loss of cognitive functioning over time. Hypothesis 2 predicts a chain that begins with the negative association between childhood SES and the CM index and then a negative association between the CM index and the slope (childhood SES→CM index→slope). These specifications imply that the direct association between childhood SES and the slope is at least partially mediated by the CM index. Hypothesis 3 was evaluated by examining if the associations specified in Hypotheses 1 and 2 hold after including adult SES. We formerly tested the mediation effects of adult SES and the CM index using a built-in routine in Mplus 6.12 (Muthén, Muthén, and Asparouhov 2017). Finally, Hypothesis 4 was evaluated by testing for a positive interaction term between childhood SES and adult SES.

RESULTS

Table 2 presents the estimates for measurement models for childhood SES, adult SES, and the latent growth parameter (slope). The far-right column presents the number of observations for each measure. Missing data are particularly severe for third and fourth cognitive assessments. Less than half of the sample had their cognitive functioning assessed more than twice. All fit indices indicate good model fit to the data (Comparative Fit Index/Tucker-Lewis Index > .9; root mean square error of approximation < .05; Kline 2014). Both the latent adult SES and childhood SES constructs are strongly correlated with their observed measures. The bottom pane presents the measurement model of the TICS growth curve. In the estimation of the intercept, the unstandardized factor loading is set to 1 for each measurement because the baseline is fixed across time. The unstandardized factor loadings for the slope indicate cumulative proportional change at each observation point, so that $\lambda_t = [0, .331, .725, 1]$.

Table 3 presents coefficients for the structural component of the model, including the mean estimates of the intercept and slope, with the upper and lower panes containing estimates predicting the intercept and slope, respectively. Recall that the intercept represents the average baseline score ($\mu\alpha$) and the slope is the average total change in the TICS score over the six-year period ($\mu\beta$). We estimated five models, with the fourth model presented in Figure 1. All models control for baseline age, sex, race-ethnicity, and childhood SES. Model 2 adds the CM index, while Model 3 instead includes adult SES. Model 4 includes childhood and adult SES and the CM index. Finally, Model 5 adds an interaction term between childhood and adult SES. Across the five models, the variables included explain 30.6 percent to 49.6 percent of the variance in the intercept and 46.7 percent to 49.7 percent of variation in the slope.[4]

Beginning with Model 1, the estimates from the slope equation show that when all the covariates are at the value of 0 (e.g., a baseline age of 70), the average cognitive decline over six years is –1.701. The intercept is largely dependent on individual age at baseline. Individuals who were older at the baseline experience greater cognitive decline over the period, by .178 per one-year increase in age. Childhood SES positively influences the level of cognitive performance at the first observation; a one standard deviation increase in childhood SES increases the baseline score by .407. In addition, supporting Hypothesis 1, childhood SES is also associated with cognitive decline; a one standard deviation increase in childhood SES lowers the rate of decline by .097. None of the other variables significantly predicts the slope.

Model 2 presents the estimates after including the CM index. The CM risk itself is associated with both the intercept and slope in the expected direction: Higher cardiometabolic dysregulation is associated with a lower baseline TICS score and a larger decline over time. Consistent with Hypothesis 2, the positive coefficient of childhood SES predicting

Table 2. Descriptive Statistics and Measurement Model Estimates for Parental SES, SES, and TICS Score (Health and Retirement Study 2006–2014).

	Mean	Unstandardized Factor Loading	Standardized Factor Loading	R^2	Observed *N*
Childhood SES					
Mother's education	9.378	1.000	.805	.649	8,494
	(.039)		(.018)		
Father's education	9.117	1.063***	.792***	.627	7,976
	(.043)	(.019)	(.015)		
Father's occupation	−.524	.096***	.521***	.272	6,189
	(.006)	(.004)	(.013)		
Adult SES					
Income (log)	10.340	1.000	.487	.238	9,449
	(.011)		(.012)		
Total assets (log)	11.097	.137***	.431***	.186	9,449
	(.022)	(.004)	(.011)		
Years of education	12.373	.335***	.871***	.758	9,449
	(.023)	(.010)	(.008)		
Longest held occupation	−.220	.188***	.561***	.272	5,920
	(.009)	(.006)	(.011)		
	Latent growth curve model of TICS score				
	Slope Coefficients				
	Mean	Unstandardized	Standardized	R^2	Observed *N*
Baseline (t = 0)	21.717	.000	.000	.751	9,449
	(.050)				
Second observation (t = 2)	21.255	.331***	.136***	.713	6,392
	(.067)	(.021)	(.014)		
Third observation (t = 4)	20.754	.725***	.290***	.798	4,812
	(.076)	(.030)	(.022)		
Fourth observation (t = 6)	20.710	1.000	0372***	.793	3,240
	(.121)		(.031)		
RMSEA			.017		
CFI/TLI			.996/.987		

Note: *N* = 9,449. Standard errors in parentheses. SES = socioeconomic status; TICS = Telephone Interview for Cognitive Status; RMSEA = root mean square error of approximation; CFI = Comparative Fit Index; TLI = Tucker-Lewis Index; TICS = Telephone Interview Cognitive Status.
***$p < .001$.

the slope is reduced from .097 in Model 1 to .086 in Model 2 (a nearly 10 percent reduction), though remains statistically significant.

In contrast to Hypothesis 3, when adult SES is alternately added to the model (Model 3), the effect of childhood SES on the slope is reduced substantially from .097 to .027 and is no longer statistically significant. Similarly, childhood SES is no longer predictive of baseline cognitive score. Adult SES is significantly associated with both the intercept and slope; one standard deviation increase in adult SES increases the baseline cognitive score by 1.002 and reduces the extent of cognitive decline by .114. In the presence of both the CM index and adult SES (Model 4), childhood SES is not significantly associated with the intercept or slope. Finally, we do not find support for Hypothesis 4 as there is no significant interaction between childhood SES and adult SES (Model 5).

To visualize how childhood SES contributes to the differentiation of cognitive trajectories over time, Figure 2 plots the predicted age-specific TICS

Table 3. Structural Coefficients (β) Predicting the Intercept/Slope of the Latent Growth Model of TICS Score (Health and Retirement Study 2006–2014).

	Model 1		Model 2		Model 3		Model 4		Model 5	
	β	SE	β	SE	β	SE	β	SE	β	SE
Intercept										
Baseline age	−.132***	.005	−.131***	.005	−.132***	.005	−.132***	.005	−.134***	.005
Male	−.683***	.089	−.627***	.089	−.925***	.086	−.915***	.084	−.938***	.087
Hispanic[a]	−2.265***	.190	−2.281***	.189	−.528***	.191	−.525***	.197	−.472*	.209
Black[a]	−3.388***	.139	−3.319***	.140	−2.555***	.139	−2.539***	.132	−2.535***	.139
Other[a]	−2.221***	.320	−2.218***	.317	−2.602***	.288	−2.602***	.284	−2.577***	.288
Childhood SES	.407***	.022	.386***	.029	−.059	.033	−.052	.032	−.062	.032
Adult SES					1.002***	.047	1.001***	.047	1.015***	.048
Child × adult									−.001	.005
CM index[b]			−.197***	.029			−.024	.029	−.006	.030
Constant	23.339***	.068	23.726***	.087	23.340***	.068	23.612***	.091	23.356***	.090
R^2	.306		.309		.480		.496		.504	
Slope										
Baseline age	−.178***	.013	−.186***	.011	−.180***	.013	−.188***	.015	−.161***	.011
Male	.002	.132	.030	.129	−.055	.132	−.029	.139	−.023	.129
Hispanic[a]	.636	.273	.678*	.284	.932***	.279	.972***	.292	.803***	.273
Black[a]	.089	.229	.146	.266	.221	.231	.269	.241	.277	.225
Other[a]	.877	.527	.887	.506	.905	.522	.934	.545	.884	.486
Childhood SES	.097***	.032	.086***	.031	.027	.045	.034	.047	.027	.044
Adult SES					.114**	.040	.100*	.040	.105*	.054
Child × adult									.003	.008
CM index[b]			−.132***	.046			−.114**	.042	−.099**	.032
Constant	−1.701***	.119	−1.493***	.153	−1.652***	.120	−1.343***	.152	−1.363***	.136
R^2	.467		.473		.495		.497		.493	
$R_{\text{Intercept with Slope}}$	.190**		.179*		.137		.136		.145	
CFI/TLI	.978/.967		.979/.965		.996/.995		.986/.979		.990/.960	

Note: N = 9,449. SE = standard error; SES = socioeconomic status; TICS = Telephone Interview for Cognitive Status; CFI = Comparative Fit Index; TLI = Tucker-Lewis Index.
[a]Non-Hispanic whites = reference.
[b]Cardiometabolic risk index.
*$p < .05$, **$p < .01$, ***$p < .001$.

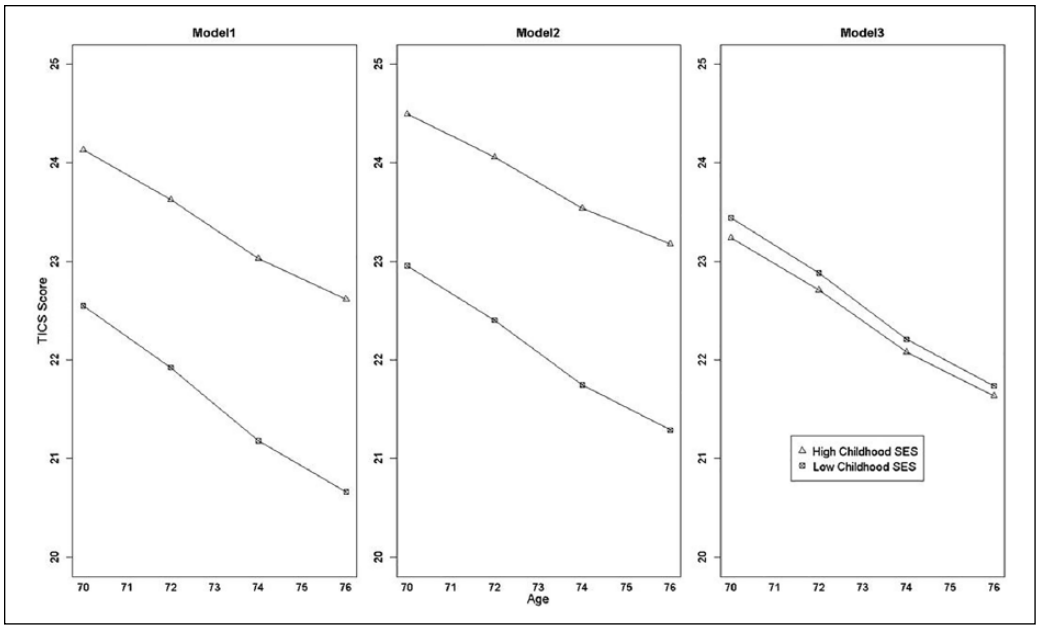

Figure 2. Predicted Trajectories of TICS Scores for Those with High/Low Childhood SES.
Note: Model 1 includes childhood SES and controls. Model 2 holds biomarkers constant. Model 3 holds adulthood SES constant. Age-specific predicted values are based on the sum of the intercept, set at the baseline age of 70, and the slope estimated for each of the three subsequent observations at two-year intervals. SES = socioeconomic status; TICS = Telephone Interview for Cognitive Status.

score over the six-year period based on the estimates from Models 1, 2, and 3. Trajectories are plotted for the mean respondent at baseline (age 70 and all covariates except childhood SES held to their baseline means). As discussed previously, the slope of this latent growth model is freely specified, meaning that change in the TICS score is not linear or curvilinear but rather expressed as the proportion of the overall change having occurred by each observation point. In other words, the model cannot extrapolate change beyond the six-year period. These plotted TICS scores represent aging trajectories between 70 and 76 for two groups: those with high childhood SES (two standard deviations above the mean) and low childhood SES (two standard deviations below the mean). Childhood SES estimated by confirmatory factor analysis follows the normal distribution. Thus, those with high childhood SES are in the 95th percentile of the distribution of the SES factor score, and low childhood SES means that their SES score is located in about the 5th percentile. Models 1 and 2 show that those with high childhood SES have significantly higher cognitive function at baseline and experience a slower rate of cognitive decline than their peers with low childhood SES. However, no such differentiation is observed in Model 3, which holds adult SES constant.

Formal tests of mediation are presented in Table 4, where all coefficients are standardized to ensure comparability and grouped in three types: direct, indirect, and total effects. Direct effects are coefficients attributed solely to childhood SES, while indirect effects involve mediators in question (i.e., adult SES/the CM index). Total effects are the sum of direct and indirect effects. Significant indirect effects via adult SES/CM index in the same sign as direct/total effects is evidence of mediation (Alwin and Hauser 1975).

Starting with Model 2, the total effect of childhood SES on the slope (i.e., the rate of cognitive decline) is estimated to be .089 and significant. The indirect effect involving the CM risk in Model 2 is .006, showing modest but significant mediation that accounts for nearly 7% of the childhood SES effect. In Model 3, the direct effect of childhood SES on the slope is .027 and no longer significant, and the indirect effect via adult SES is .062 ($p < .001$). Given the total effect of childhood SES in this model is .089, this means the mediation of adult

Table 4. Decomposition of the Effect of Childhood SES on the Slope (Health and Retirement Study 2006–2014).

Direct Effect		Indirect Effect		Total Effect
Model 2				
Childhood SES	.083**	→ CM index	.006**	.089**
	(.031)		(.002)	(.031)
Model 3				
Childhood SES	.027	→ Adult SES	.062**	.089**
	(.045)		(.027)	(.022)
Model 4				
Childhood SES	.020	Total	.067**	.087**
	(.045)		(.027)	(.030)
		→ Adult SES	.062**	
			(.028)	
		→ CM index	–.003	
			(.002)	
		→ Adult SES	.007**	
		→ CM index	(.003)	

Note: N = 9,449. All coefficients are standardized. Standard errors in parentheses. CM index = cardiometabolic risk index; SES = socioeconomic status.
** $p < .01$.

SES is nearly 70%. Finally, in Model 4, there are three indirect paths, among which two are statistically significant at the p value below .001: childhood SES→adult SES→slope (.062) and childhood SES→adult SES→CM index→slope (.007). Both mediation paths involve adult SES, meaning that the significant mediation effect of the CM index we observed in Model 2 is ultimately due to adult SES.

To further illustrate this point, Table 5 presents the estimates from the equations predicting the CM index and adult SES from Models 2 and 4. This demonstrates the extent to which childhood SES predicts cardiometabolic risk, with/without adult SES included, as well as the effect of childhood SES on adult SES. In Model 2, higher childhood SES predicts lower cardiometabolic risk (–.040), but that is no longer the case in Model 4 (.020). A one standard deviation increase in childhood SES predicts a .547 standard deviation increase in adult SES.

DISCUSSION

The life course represents the flow of time, beginning from conception to the moment of death, during which individuals are exposed to a variety of risks. Some exposures in early life extend a "long arm" over the life course with irreversible and persistent effects on health (Ben-Shlomo and Kuh 2004). These effects manifest through the intersection of social, developmental, and biological processes (Bartley 2016; Elder et al. 2003). We combine the cognitive science concepts of differential preservation and preserved differentiation with a life course perspective on health to test the relative contribution of childhood SES and adult SES to variation in later-life cognitive trajectories and whether cardiometabolic risk mediates these socioeconomic influences.

We observed a significantly slower cognitive decline associated with higher childhood SES over time and also modest yet significant mediation of that association through later-life cardiometabolic risk. However, we also found that childhood SES does not directly predict cardiometabolic function in later life or the rate of cognitive decline net of adult socioeconomic resources. Mediation analysis revealed that cardiometabolic risk of cognitive decline operates exclusively through adult SES. Further, we found no evidence for an interaction effect of experiencing low SES in both childhood and adulthood on cognitive trajectories.

The results have important implications for understanding life course influences on cognitive aging. Prior research has yielded mixed results on the relationship between childhood SES and later-life cognitive function, with some studies reporting a strong mediation through adult SES (Horvat et al.

Table 5. Structural Coefficients Predicting the Cardiometabolic Risk Index and Adult SES in Models 2 and 4 (Health and Retirement Study 2006–2014).

	Model 2		Model 4	
	β	SE	β	SE
Cardiometabolic risk index				
Baseline age	.012***	.002	.012***	.002
Male	.282*	.032	.310***	.032
Hispanic[a]	.037***	.065	–.168	.067
Black[a]	.506***	.048	.405***	.050
Other[a]	–.056***	.106	–.011	.103
Childhood SES	–.040***	.007	.020	.011
Adult SES			–.115***	.013
R^2	.034		.053	
Adult SES				
Baseline age			.000	.003
Male			.241***	.059
Hispanics[a]			–1.690***	.133
Blacks[a]			–.818***	.094
Others[a]			.428***	.197
Childhood SES			.547***	.016
R^2			.532	

Note: N = 9,449. SE = standard error; SES = socioeconomic status; TICS = Telephone Interview for Cognitive Status.
[a]Non-Hispanic whites = reference.
*$p < .05$, ***$p < .001$.

2014) and others finding a cumulative effect of socioeconomic position across the life course (Lynch et al. 1997). Our results are most consistent with the former. We find that rather than manifesting as critical period effects or through the cumulative effect of socioeconomic conditions, the dominant mechanism by which early life socioeconomic resources influence later-life cognition is through the initiation of a chain of socioeconomic risk. Childhood socioeconomic resources facilitate the movement into advantaged educational and occupational niches, which provide an array of material, cognitive, and psychosocial resources key to the preservation of cognitive function. Such resources protect cognition directly through the development of cognitive reserve (e.g., cognitively demanding occupations) and indirectly through the mitigation of stress processes and other drivers of cardiometabolic risk. As such, our results suggest that any discussion of socioeconomic inequality in cognitive aging must be firmly rooted in an understanding of larger life course processes of social stratification and status attainment. Our results also highlight the need to further disentangle the social and biological processes connecting adult SES and cognitive aging. As part of that agenda, more attention must be paid to the sociobiological interface across a larger swath of the life course rather than focusing exclusively on cognition in late life. It should also investigate a wide array of biological mechanisms linking social context to brain function.

We would also emphasize that other types of early life exposures may manifest adverse impacts on cognition through pathways that are not rooted primarily in the stratification process. Indeed, more research is needed to disentangle the variety of sociobiological mechanisms linking different types of early life exposures/insults to later-life cognitive function. It is likely that some exposures, such as physical or sexual abuse/victimization, will operate in a manner consistent with critical period effects, permanently altering neurocognitive or other biological systems that shape cognitive trajectories in late life. We echo calls for research on the early life influences on health to examine multiple domains of childhood experience simultaneously (Ferraro, Schafer, and Wilkinson 2016).

It would be tempting to view the present results as dismissive of the impact of early-life conditions

on cognitive aging. However, while they do highlight the central role of adult SES, our results confirm that childhood socioeconomic deprivation remains a systematic obstacle to the development and maintenance of cognitive health. Indeed, our estimate of the correlation between childhood and adult SES was nearly .7 (not shown). Constrained opportunities for upward mobility affect many aspects of individual and population health that perpetuate health inequalities (Woolf 2009). This holds for cognitive health as well. With growing public and policy interest in the maintenance of cognitive health in later life, the promotion of individual-level strategies in late adulthood to develop resilience tends to dominate policy discourse. However, the present study illustrates the importance of structural factors and underscores the limitations to individual-level interventions in later life to mitigate cognitive decline. Conversely, it points to policies to reduce socioeconomic deprivation in childhood, promote greater adult socioeconomic attainment, and reduce barriers to upward social mobility as critical targets for improving later-life cognitive health in the long run. Indeed, research demonstrates that investments in early childhood development are more productive and cost-efficient than investments later in life and yield a broad array of social and economic returns (Heckman 2006). Our work suggests such investments would yield long-term cognitive health benefits as well.

Emphasis on socioeconomic inequality and its genesis in early life also raises concerns for future trends in cognitive health. In the past few decades, the United States has witnessed persistently high rates of child poverty (Koball and Jiang 2018), declining social mobility (Chetty et al. 2017), and as a result, divergent destinies of children based on their parental/family characteristics (McLanahan 2004). These trends have negative implications for future population health. Indeed, socioeconomic disparities in health and mortality have been expanding among recent cohorts (Masters, Hummer, and Powers 2012). The evidence presented here portends similar trends in the future for cognitive health. At the very least, these socioeconomic trends are likely to pose significant structural head winds to public health efforts to promote successful cognitive aging.

We urge the readers to consider several caveats when interpreting the results. First, the array of biomarkers included in the study is not comprehensive and is limited by the design of the HRS (Crimmins et al. 2013). A fuller understanding of cumulative damage resulting from childhood adversity would include stress hormones and other biomarkers indicative of cumulative damage over the life course. The future expansion of HRS biomarker data assessing multiple body systems will further that endeavor. Second, we emphasize that the inferences from the findings presented in this study are strictly limited to the linkage between childhood SES and cardiometabolic risk. We also considered early life health conditions and childhood health status, but these were excluded because none significantly predicted cognitive decline independent of adult SES. Finally, there is the issue of mortality selection of the sample before biomarker data collection began in 2006. Even with the "refresher" samples of younger individuals added to HRS, the average age in the sample at first observation is nearly 71. It is likely that those cohort members who are at higher cognitive risk also had higher mortality prior to 2006, making the estimated effects of childhood SES on cognitive decline biased downward.

Despite these caveats, the present study is one of the first to examine childhood SES and the rate of cognitive decline while accounting for cardiometabolic risk and adult SES. It also highlights the value of theorizing the determinants of cognitive aging from a life course perspective. The study of cognitive aging is inherently interdisciplinary. While it is grounded in the sociological tradition, one of the great strengths of the life course approach is that it is flexible, capable of accommodating and illuminating theoretical constructs from other disciplines. Here, life course theory provided a set of processes and orienting principles for understanding the cognitive science constructs of preserved differentiation and differential preservation and the complex sociobiological interface that shapes lifelong cognitive trajectories. Future research on cognitive aging would be well served by an approach grounded in life course theory.

APPENDIX

Structural Coefficients Predicting the Intercept/Slope of the TICS Score Trajectory, with Alternative Model Specifications (Health and Retirement Study 2006–2014).

	Model 3 + Behavioral		Model 4 (Alternative CM Index)		Model 4 (Truncated)	
	β	SE	β	SE	β	SE
Intercept						
Baseline age	–.120***	.005	–.130***	.005	–.041***	.010
Male	–.990***	.084	–.923***	.084	–.863***	.110
Hispanic[a]	–.658***	.180	–.507***	.191	–.563*	.229
Black[a]	–2.511***	.130	–2.478***	.138	–2.473***	.156
Other[a]	–2.377***	.268	–2.592***	.287	–2.751***	.351
Childhood SES	–.068	.032	–.063	.033	–.063	.043
Adult SES	1.032***	.045	1.008***	.049	.952***	.052
Exercise	.172***	.030				
BMI	.038***	.007				
Ever smoked	.046***	.086				
Currently smoke	–.215*	.134				
CM index			–.096***	.029	–.051	.047
Constant	23.006***	.242	23.562***	.094	23.947***	.121
R^2	.513		.500		.523	
Slope						
Baseline age	–.172***	.011	–.159***	.015	–.170***	.037
Male	–.102	.132	–.108	.138	–.061	.173
Hispanic[a]	.764*	.277	.834***	.292	.898***	.331
Black[a]	.177	.219	.318	.241	–.048	.273
Other[a]	.476	.457	.857	.544	.828	.651
Childhood SES	.032	.044	.035	.045	–.001	.056
Adult SES	.085	.052	.093*	.042	.098*	.045
Exercise	–.100***	.047				
BMI	.003	.012				
Ever smoked	.029	.132				
Currently smoke	–.461	.222				
CM index			–.137***	.045	.171***	.052
Constant	–1.366***	.402	–1.375***	.160	1.442***	.176
R^2	.469		.488		.495	
$R_{\text{Intercept with Slope}}$	.167*		.126		.134	

Note: N = 9,449. Model 3 + Behavioral = adding smoking, BMI, and exercise variables to Model 3. Model 4 (Alternative CM Index) = replacing the CM index with an alternative measure that includes forced expiratory volume. Model 4 (Truncated) = Model 4 estimates based on the model that includes the sample members whose TICS score was measured four times (N = 4,869). CM index = cardiometabolic risk index; BMI = body mass index; SES = socioeconomic status. TICS = Telephone Interview Cognitive Status.

[a]Non-Hispanic whites = reference.

*$p < .05$, ***$p < .001$.

NOTES

1. Auxiliary analysis assessing representativeness of the analytic sample against the Health and Retirement Study biomarker subsample found that the two do not differ in terms of education, income, occupation, race, or Hispanic ethnicity. The analytic sample was nearly five years younger.
2. There is a considerable overlap in the array of biomarkers used to assess cardiometabolic risk and allostatic load. For multiple reasons, our measure most closely aligns with the former. First, allostatic load includes measures of stress hormones. Ours does not. The array of biomarkers used in this study is too limited to be considered as a comprehensive measure of cumulative dysregulation. Second, it's not possible to determine whether the level of the biomarkers observed here reflect recent fluctuation, acute responses, or decades of exposures across the life course. Finally, these biomarkers capture cardiometabolic risk, a particular biological pathway linking childhood socioeconomic status and cognitive decline.
3. We followed this conventional practice to construct the cardiometabolic risk index (Howard and Sparks 2016). Auxiliary analysis shows that the predictive power of the index is similar, regardless of how it was constructed.
4. We conducted sensitivity analyses (Appendix), including alternative models with health behaviors. We also ran analyses on a sample constrained to ages 55 to 73 (the interquartile range) to reduce potential confounding from cohort effects. Finally, we examined the cardiometabolic risk index including forced expiratory volume. None altered the substantive conclusions.

REFERENCES

Alwin, Duane F., and Robert M. Hauser. 1975. "The Decomposition of Effects in Path Analysis." *American Sociological Review* 40(1):37–47.

Andel, Ross, Frank J. Infurna, Elizabeth A. Hahn Rickenbach, Michael Crowe, Lisa Marchiondo, and Gwenith G. Fisher. 2015. "Job Strain and Trajectories of Change in Episodic Memory before and after Retirement: Results from the Health and Retirement Study." *Journal Epidemiology and Community Health* 69(5):442–46.

Barker, David J. P. 2007. "The Origins of the Developmental Origins Theory." *Journal of Internal Medicine* 261(5):412–17.

Bartley, Mel. 2016. *Health Inequality: An Introduction to Concepts, Theories and Methods*. Malden MA: Polity Press.

Ben-Shlomo, Yoav, Rachel Cooper, and Diana Kuh. 2016. "The Last Two Decades of Life Course Epidemiology, and Its Relevance for Research on Ageing." *International Journal of Epidemiology* 45(4):973–88.

Ben-Shlomo, Yoav, and Diana Kuh, eds. 2004. *A Life Course Approach to Chronic Disease Epidemiology*. Oxford, UK: Oxford University Press.

Brandt, Jason, Miriam Spencer, and Marshal Folstein. 1988. "The Telephone Interview for Cognitive Status." *Neuropsychiatry Neuropsychology Behavioral Neurology* 1(2):111–17.

Bugliari, Delia, Nancy Campbell, Chris Chan, Orla Hayden, Michael Hurd, R. Main, J. Mallett, C. McCullough, E. Meijer, M. Moldoff, and P. Pantoja. 2016. *RAND HRS Data Documentation, Version P*. Santa Monica, CA: RAND Center for the Study of Aging.

Chetty, Raj, David Grusky, Maximilian Hell, Nathaniel Hendren, Robert Manduca, and Jimmy Narang. 2017. "The Fading American Dream: Trends in Absolute Income Mobility Since 1940." *Science* 356(6336):398–406.

Crimmins, Eileen, Jessica Faul, Jung Ki Kim, Heidi Guyer, Kenneth Langa, Mary Beth Ofstedal, Amanda Sonnega, Robert Wallace, and David Weir. 2013. *Documentation of Biomarkers in the 2006 and 2008 Health and Retirement Study*. Ann Arbor, MI: Survey Research Center, University of Michigan.

Cynader, Max S., and Barrie J. Frost. 1999. "Mechanisms of Brain Development: Neuronal Sculpting by the Physical and Social Environment." Pp. 153–84 in *Developmental Health and the Wealth of Nations*, edited by D. Keating and C. Hertzman. New York: Guilford Press.

Dannefer, Dale. 1987. "Aging as Intracohort Differentiation: Accentuation, the Matthew Effect, and the Life Course." *Sociological Forum* 2(2):211–36.

Daviglus, M. L., C. C. Bell, W. Berrettini, P. E. Bowen, J. E. Connolly, N. J. Cox, J. M. Dunbar-Jacob, E. C. Granieri, G. Hunt, K. McGarry, and D. Patel. 2010. "NIH State-of-the-Science Conference Statement: Preventing Alzheimer's Disease and Cognitive Decline." *NIH Consensus and State-of-the-Science Statements* 27(4):1–30.

DiPrete, Thomas A., and Gregory M. Eirich. 2006. "Cumulative Advantage as a Mechanism for Inequality: A Review of Theoretical and Empirical Developments." *Annual Review of Sociolology* 32:271–97.

Elder, Glen H., Monica Kirkpatrick Johnson, and Robert Crosnoe. 2003. "The Emergence and Development of Life Course Theory." Pp. 3–19 in *Handbook of the Life Course*, edited by J. Mortimer and M. Shanahan. Boston: Springer.

Elovainio, Marko, Jane E. Ferrie, Archana Singh-Manoux, Martin Shipley, G. David Batty, Jenny Head, Mark Hamer, Markus Jokela, Marianna Virtanen, Eric Brunner, Michael Marmot, and Mika Kivimäki. 2011. "Socioeconomic Differences in Cardiometabolic Factors: Social Causation or Health-Related Selection? Evidence from the Whitehall II Cohort Study, 1991–2004." *American Journal of Epidemiology* 174(4):779–89.

Evans, Gary W., and Kimberly English. 2002. "The Environment of Poverty: Multiple Stressor Exposure,

Psychophysiological Stress, and Socioemotional Adjustment." *Child Development* 73(4):1238–48.

Ferraro, Kenneth F., and Jessica A. Kelley-Moore. 2003. "Cumulative Disadvantage and Health: Long-Term Consequences of Obesity?" *American Sociological Review* 68(5):707–29.

Ferraro, Kenneth F., Markus H. Schafer, and Lindsay R. Wilkinson. 2016. "Childhood Disadvantage and Health Problems in Middle and Later Life: Early Imprints on Physical Health?" *American Sociological Review* 81(1):107–33.

Finkel, Deborah, Ross Andel, Margaret Gatz, and Nancy Pedersen. 2009. "The Role of Occupational Complexity in Trajectories of Cognitive Aging before and after Retirement." *Psychology and Aging* 24(3):563–573.

Forstmeier, Simon, Andreas Maercker, Wolfgang Maier, Hendrik Van Den Bussche, Steffi Riedel-Heller, Hanna Kaduszkija, Michael Pentzek, Siegfried Weyerer, Horst Bickel, Franziska Tebarth, Melanie Luppa, Anja Wollny, Birgitt Wiese, and Michael Wagner. 2012. "Motivational Reserve: Motivation-Related Occupational Abilities and Risk of Mild Cognitive Impairment and Alzheimer Disease." *Psychology and Aging* 27(2):353–63.

Greenfield, Emily A., and Sara M. Moorman. 2018. "Childhood Socioeconomic Status and Later Life Cognition: Evidence From the Wisconsin Longitudinal Study." *Journal of Aging and Health*. doi:10.1177/0898264318783489

Haas, Steven A. 2008. "Trajectories of Functional Health: The 'Long Arm'of Childhood Health and Socioeconomic Factors." *Social Science & Medicine* 66(4):849–61.

Haas, Steven A., M. Maria Glymour, and Lisa F. Berkman. 2011. "Childhood Health and Labor Market Inequality over the Life Course." *Journal of Health and Social Behavior* 52(3):298–313.

Hackman, Daniel A., Martha J. Farah, and Michael J. Meaney. 2010. "Socioeconomic Status and the Brain: Mechanistic Insights from Human and Animal Research." *Nature Reviews Neuroscience* 11(9):651–59.

Harrison, Stephanie L., Ayesha Sajjad, Wichor M. Bramer, M. Arfan Ikram, Henning Tiemeier, and Blossom Stephan. 2015. "Exploring Strategies to Operationalize Cognitive Reserve: A Systematic Review of Reviews." *Journal of Clinical and Experimental Neuropsychology* 37(3):253–64.

Hayward, Mark D., and Bridget K. Gorman. 2004. "The Long Arm of Childhood: The Influence of Early-Life Social Conditions on Men's Mortality." *Demography* 41(1):87–107.

Heckman, James J. 2006. "Skill Formation and the Economics of Investing in Disadvantaged Children." *Science* 312(5782):1900–1902.

Hemmingsson, Erik, K. Johansson, and S. Reynisdottir. 2014. "Effects of Childhood Abuse on Adult Obesity: A Systematic Review and Meta-Analysis." *Obesity Reviews* 15(11):882–93.

Horvat, Pia, Marcus Richards, Sofia Malyutina, Andrej Pajak, Ruzena Kubinova, Abdonas Tamosiunas, Hynek Pikhart, Anne Peasey, Michael Marmot, and Martin Bobak. 2014. "Life Course Socioeconomic Position and Mid-Late Life Cognitive Function in Eastern Europe." *Journals of Gerontology Series B: Psychological Sciences and Social Sciences* 69(3):470–81.

Howard, Jeffrey T., and P. Johnelle Sparks. 2016. "Does Allostatic Load Calculation Method Matter? Evaluation of Different Methods and Individual Biomarkers Functioning by Race/ethnicity and Educational Level." *American Journal of Human Biology* 28(5):627–35.

Hox, Joop, and Reinoud D. Stoel. 2014. "Multilevel and SEM Approaches to Growth Curve Modeling." *Wiley StatsRef: Statistics Reference Online*. https://onlinelibrary.wiley.com/doi/abs/10.1002/9781118445112.stat06603.

Insler, Michael. 2014. "The Health Consequences of Retirement." *Journal of Human Resources* 49(1):195–233.

Institute of Medicine. 2000. *From Neurons to Neighborhoods: The Science of Early Childhood Development*. Washington, DC: The National Academies Press.

Kelli, Heval M., Ibrahim Kassas, and Omar M. Lattouf. 2015. "Cardio Metabolic Syndrome: a Global Epidemic." *Journal of Diabetes and Metabolism* 6(3):1–14.

Kline, Paul. 2014. *An Easy Guide to Factor Analysis*. Abington, UK: Routledge.

Kline, Rex B. 2004. *Structural Equation Modeling*. New York: Guilford Press.

Koball, Heather, and Yang Jiang. 2018. *Basic Facts about Low-Income Children: Children under 18 Years, 2016*. New York: National Center for Children in Poverty, Columbia University Mailman School of Public Health.

Lachman, Margie, and Avron Spiro. 2002. "Critique of Cognitive Measures in the Health Retirement Study (HRS) and the Asset and Health Dynamics among the Oldest Old (AHEAD) Study." *HRS Data Monitoring Board Report*. https://www.researchgate.net/profile/Avron_Spiro/publication/242096659_.

Lupien, Sonia J., Suzanne King, Michael J. Meaney, and Bruce McEwen. 2000. "Child's Stress Hormone Levels Correlate with Mother's Socioeconomic Status and Depressive State." *Biological Psychiatry* 48(10):976–80.

Lynch, John W., George A. Kaplan, and Sarah J. Shema. 1997. "Cumulative Impact of Sustained Economic Hardship on Physical, Cognitive, Psychological, and Social Functioning." *New England Journal of Medicine* 337(26):1889–95.

Lyu, Jiyoung. 2015. "Gender Differences in the Association between Childhood Socioeconomic Status and Cognitive Function in Later Life." *Journal of Geriatrics* Article ID 896876. doi:10.1155/2015/896876

Lyu, Jiyoung, and Jeffrey A. Burr. 2016. "Socioeconomic Status across the Life Course and Cognitive Function among Older Adults: An Examination of the Latency, Pathways, and Accumulation Hypotheses." *Journal of Aging and Health* 28(1):40–67.

Masters, Ryan K., Robert A. Hummer, and Daniel A. Powers. 2012. "Educational Differences in US Adult Mortality: A Cohort Perspective." *American Sociological Review* 77(4):548–72.

McEwen, Bruce S., and Eliot Stellar. 1993. "Stress and the Individual: Mechanisms Leading to Disease." *Archives of Internal Medicine* 153(18):2093–101.

McLanahan, Sara. 2004. "Diverging Destinies: How Children are Faring Under the Second Demographic Transition." *Demography* 41(4):607–27.

McLaughlin, Katie A., Margaret A. Sheridan, Florin Tibu, Nathan Fox, Charles Zeanah, and Charles Nelson. 2015. "Causal Effects of the Early Caregiving Environment on Development of Stress Response Systems in Children." *Proceedings of the National Academy of Sciences* 112(18):5637–42.

Merton, Robert K. 1968. "The Matthew Effect in Science: The Reward and Communication Systems of Science are Considered." *Science* 159(3810):56–63.

Montez, Jennifer K., and Mark D. Hayward. 2014. "Cumulative Childhood Adversity, Educational Attainment, and Active Life Expectancy among U.S. Adults." *Demography* 51(2):413–35.

Muthén, Bengt. 2004. "Latent Variable Analysis." Pp. 345–68 in *The Sage Handbook of Quantitative Methodology for the Social Sciences*, edited by D. W. Kaplan. Thousand Oaks, CA: Sage.

Muthén, Bengt O., Linda K. Muthén, and Tihomir Asparouhov. Regression and mediation analysis using Mplus. Los Angeles, CA: Muthén & Muthén, 2017.

O'brien, John T., David Ames, Ian Schweitzer, Maree Mastwyk, and Peter Colman. 1996. "Enhanced Adrenal Sensitivity to Adrenocorticotrophic Hormone (ACTH) Is Evidence of HPA Axis Hyperactivity in Alzheimer's Disease." *Psychological Medicine* 26(1):7–14.

Oi, Katsuya. 2017. "Inter-connected Trends in Cognitive Aging and Depression: Evidence from the Health and Retirement Study." *Intelligence* 63:56–65.

Ridker, Paul M., Peter W. F. Wilson, and Scott M. Grundy. 2004. "Should C-reactive Protein be Aadded to Metabolic Syndrome and to Assessment of Global Cardiovascular Risk?" *Circulation* 109(23):2818–25.

Rizzo, Manfredi, Egle Corrado, Giuseppe Coppola, Ida Muratori, Giuseppina Novo, and Salvtore Novo. 2008. "Prediction of Cardio-and Cerebro-Vascular Events in Patients with Subclinical Carotid Atherosclerosis and Low HDL-cholesterol." *Atherosclerosis* 200(2):389–95.

Ross, Catherine E., and Chia-Ling Wu. 1996. "Education, Age, and the Cumulative Advantage in Health." *Journal of Health and Social Behavior* 37(1):104–20.

Schmitz, Norbert, Sonya S. Deschênes, Rachel J. Burns, Sofia Danna, ,Oscar Franco, M. Arfan Ikram, Mika Kivimäki, Archana Singh-Manoux, and Henning Tiemeier. 2018. "Cardiometabolic Dysregulation and Cognitive Decline: Potential Role of Depressive Symptoms." *The British Journal of Psychiatry* 212(2):96–102.

Sewell, William H., Archibald O. Haller, and Alejandro Portes. 1969. "The Educational and Early Occupational Attainment Process." *American Sociological Review* 34(1):82–92.

Singer, Burton, and Carol D. Ryff. 1999. "Hierarchies of Life Histories and Associated Health Risks." *Annals of the New York Academy of Sciences* 896(1):96–115.

Staff, Roger T., Alison D. Murray, Trevor S. Ahearn, Nazahan Mustafa, Helen Fox, and Lawerence Whalley. 2012. "Childhood Socioeconomic Status and Adult Brain Size: Childhood Socioeconomic Status Influences Adult Hippocampal Size." *Annals of Neurology* 71(5):653–60.

Stern, Yaakov. 2012. "Cognitive Reserve in Ageing and Alzheimer's disease." *The Lancet Neurology* 11(11):1006–12.

Suglia, Shakira, Karestan Koenen, Renée Boynton-Jarrett, Paul Chan, Cari Clark, Andrea Danese, Myles Faith, Benjamin Goldstein, Laura Hayman, Carmen Isasi, Charlotte Pratt, Natalie Sloppen, Jennifer Sumner, Aslan Turer, Christy Turer, and Justin Zachariah. 2018. "Childhood and Adolescent Adversity and Cardiometabolic Outcomes: A Scientific Statement from the American Heart Association." *Circulation* 137(5):e15–28.

Whalley, Lawrence J., Ian J. Deary, Charlotte L. Appleton, and John Starr. 2004. "Cognitive Reserve and the Neurobiology of Cognitive Aging." *Ageing Research Reviews* 3(4):369–82.

Woolf, Steven H. 2009. "Social Policy as Health Policy." *Journal of the American Medical Association* 301(11):1166–69.

Zhang, Zhenmei, Mark D. Hayward, and Yan-Liang Yu. 2016. "Life Course Pathways to Racial Disparities in Cognitive Impairment among Older Americans." *Journal of Health and Social Behavior* 57(2):184–99.

Zhang, Zhenmei, Jinyu Liu, Lydia Li, and Hongwei Xu. 2017. "The Long Arm of Childhood in China: Early-Life Conditions and Cognitive Function among Middle-Aged and Older Adults." *Journal of Aging and Health* 30(8):1319–44.

AUTHOR BIOGRAPHIES

Katsuya Oi is an assistant professor of Sociology at Northern Arizona University. His research is dedicated to understand the social risks of cognitive aging and how society can better address aging-related issues. He is often seen typing away manuscripts at his favorite cafe Steep in Flagstaff, Arizona.

Steven A. Haas is an associate professor of Sociology and Demography at Pennsylvania State University. His research examines the social and economic causes and consequences of poor health over the life course. His recent work has appeared in Demography, Journal of Health and Social Behavior, and Social Science & Medicine.