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Research Article

Life Course Pathways From Childhood Socioeconomic Status to Later-Life Cognition: Evidence From the Wisconsin Longitudinal Study

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Abstract

Objectives: A growing body of research indicates that older adults are at greater risk for poorer cognition if they experienced low socioeconomic status (SES) as children. Guided by life course epidemiology, this study aimed to advance understanding of processes through which childhood SES influences cognition decades later, with attention to the role of scholastic performance in adolescence and SES in midlife.

Method: We used data from the Wisconsin Longitudinal Study (WLS), which has followed a cohort of high school graduates since they were 18 years old in 1957. Childhood SES was measured prospectively in adolescence, and measures of memory and language/executive functioning were based on neurocognitive assessments at age 72. We used participants' scores on a statewide standardized test in high school as an indicator of scholastic performance in adolescence. The measure of SES in midlife included years of postsecondary education, income, and occupation status at age 53.

Results: Findings from structural equation models indicated that scholastic performance in adolescence and midlife status attainment together fully mediated associations between childhood SES and both memory and language/executive functioning at age 72. Adolescent scholastic performance was directly associated with later-life cognition, as well as indirectly through midlife status attainment.

Discussion: Findings provide support for both latency and social pathway processes when considering how SES in childhood influences later-life cognition. Results contribute to growing calls for social policies and programs to support optimal brain health at multiple phases throughout the life course, especially among individuals with lower SES as children.

Keywords: Cognitive aging, Life course, Social determinants, Social inequalities, Socioeconomic status

Understanding biopsychosocial processes that contribute to later-life cognition is both a U.S. national and international priority for health and aging research. The importance of research in this area is predicated on the value of preventing the onset and progression of Alzheimer's disease and related dementias (ADRD) for individuals, families, and society (El-Hayek et al., 2019). Discourse specifically on the prevention of ADRD orients, in part, to risk and protective factors that predate later life. A growing body of social and epidemiological research has drawn attention to childhood socioeconomic status (SES), in particular. SES is a multifaceted phenomenon referring to people's social standing along intersecting dimensions, such as educational attainment, occupational status, and income (CDC, 2014). Studies drawing on data from diverse national contexts have found that older adults perform better on

neurocognitive tests, on average, when they experienced higher levels of SES in childhood, as conferred by the SES of their parents or guardians (see Greenfield & Moorman, 2019, for a review).

A primary question within research on childhood SES and later-life cognition concerns the life course processes through which these long-term associations emerge (Beck et al., 2018; Lyu & Burr, 2016; Oi & Haas, 2019; Zhang, Gu, & Hayward, 2008). Understanding such processes is important for informing policy and practice that relates conditions of childhood and the life course more generally to the cognitive health of aging populations (e.g., Leggett et al., 2019). It also is important for guiding efforts to optimize the cognitive health of future cohorts of older adults, especially given that children in the United States today face historically high levels of socioeconomic inequality (Warren, 2015).

Drawing on theoretical models from life course epidemiology (Ben-Shlomo & Kuh, 2002), we used data from the Wisconsin Longitudinal Study (WLS), one of the longest-running cohort studies in the United States, to further the empirical study of life course processes through which childhood SES is associated with cognition in later life. The WLS is uniquely positioned to advance research in this area given its prospective measures of adolescent scholastic performance, as well as SES in midlife, within a single birth cohort. These two factors are both highly plausible mediators that are associated with one another, but rarely have been examined simultaneously. These potential mediators also imply distinct leverage points for policy and practice directions to optimize later-life cognition within the population at large.

Mechanisms Through Which Childhood SES Influences Cognition

Researchers have theorized broadly on processes through which SES affects cognition and neurophysiological functioning throughout the life span (Hackman & Farah, 2009). Theoretical accounts of how SES affects child development in general have distinguished mechanisms of risk from mechanisms of protection (Conger, Conger, & Martin, 2010). The former refers to ways in which low SES introduces risk factors that undermine neurocognitive development, whereas the latter addresses ways in which high SES yields resources or assets that optimize neurocognitive development. Examples of risk mechanisms include bioenvironmental toxins, life stress, material deprivation, and perinatal complications; assets include better nutrition and more cognitively stimulating home and community environments (Glymour & Manly, 2008). Theorizing on SES and health more broadly (see Phelan, Link, & Tehranuifar, 2010) suggests that there is unlikely to be a single mechanism that accounts for associations between SES and cognition. Because SES encompasses a variety of resources—including financial resources, knowledge, social connections, and prestige—individuals in childhood families with greater SES are more likely to avoid a variety of risk factors, as well as to access a range of resources, in various ways that benefit their cognitive development over time.

Life Course Epidemiological Models of Pathways From Childhood SES to Later-Life Cognition

Research on the processes through which childhood SES influences cognition decades later generally has drawn on concepts from life course epidemiology. Life course epidemiology broadly orients attention to how adult health and illness are rooted in social and physical exposures occurring earlier in life (Ben-Shlomo & Kuh, 2002). Theoretical developments within life course epidemiology have posited sequences of associations among risk and protective factors that emerge throughout the life course and influence later-life health.

One such sequence comprises a "pathways" model. This model suggests an indirect effect whereby childhood SES influences "social and economic trajectories throughout young adulthood and middle age" (Lyu & Burr, 2016, p. 44), which subsequently affect later-life outcomes. For example, individuals with higher SES in childhood are more likely to obtain a postsecondary degree, and postsecondary degree completion in young adulthood is associated with subsequent advantages throughout adulthood (e.g., higher status occupation, healthier behaviors) (Lawrence, 2017). Such advantages in adulthood potentially contribute to lower risk for poor cognition in later life.

An alternative sequence is reflected in a "latency" model, which suggests that childhood exposures have direct and immediate biological effects that influence later-life health outcomes "independent of other experiences and exposure to risk factors during adulthood" (Lyu & Burr, 2016, p. 43). This model is consistent with theorizing on sensitive periods, which posits that there are specific stages of the life course during which organisms are highly susceptible to contextual conditions. Prenatal, early childhood, and adolescence have been identified as especially sensitive periods for neurological development; the organization of the brain at these developmental stages might have long-lasting effects on cognitive outcomes, regardless of subsequent exposure to other risk and protective factors (Seifan, Schelke, Obeng-Aduasare, & Isaacson, 2015).

Within descriptions of these life course processes, scholars generally acknowledge that latency and pathway processes can both be operational. When considering cognitive outcomes specifically, simultaneous casual processes are especially likely because of reciprocal influences among the brain, environments, and human behavior. Neuroscientific theorizing on SES has characterized the brain as "a major locus of integration and influence for the multitude of environmental factors that shape our lives"

(Farah, 2018, p. 57), with environments encompassing both physical and social factors that are highly correlated with SES (e.g., environmental toxins, life event stress). At the same time, neurophysiological functioning underlies the development of fundamental cognitive skills—including both academic and socioemotional—which can influence one's future socioeconomic position (Spengler et al., 2015). This theorizing suggests the importance of considering both earlier-life cognition, as well as subsequent SES, when examining life course processes from childhood SES to later-life cognition.

Prior Studies on Life Course Pathways From Childhood SES to Later-Life Cognition

Research on mediators of the association between childhood SES and later-life cognition largely has focused on SES in adulthood. Most of these studies, which collectively have drawn on population data sets across different regional contexts, have found evidence for partial mediation, whereby adult SES accounts for some, but not all, of the association (Fors, Lennartsson, & Lundberg, 2009; Horvat et al., 2014; Kaplan et al., 2001; Richards & Wadsworth, 2004; Zhang et al., 2008; Zhang, Liu, Li, & Xu, 2017). Other studies have found evidence of full mediation, indicating that adult SES entirely accounts for linkages between childhood SES and later-life cognition (González, Tarraf, Bowen, Johnson-Jennings, & Fisher, 2013; Greenfield & Moorman, 2019; Lyu & Burr, 2016; Oi & Haas, 2019; Wen & Gu, 2011; Zhang, Hayward, & Yu, 2016).

The aforementioned studies, however, have not incorporated measures of earlier-life cognition, which could confound the mediating effects of adult SES. In other words, the mediating effect of adult SES could be an artifact of individuals with greater SES in childhood having better cognition at earlier periods of the life course. This cognitive ability might persist into later life regardless of subsequent SES in adulthood, and might account for any linkages between subsequent life course factors and later-life cognition through latency processes (Kremen et al., 2019). To our knowledge, only three studies—all based on longitudinal cohort data—have accounted for earlier-life cognition within investigations of childhood SES, adult SES, and later-life cognition.

Two of these studies use data from the British 1946 birth cohort study to examine life course pathways from childhood SES to cognition at ages 53 (Richards & Sacker, 2003) and 69 (Richards et al., 2019). The first study found that participants' cognitive ability at age 8, educational attainment at age 26, and occupational status at age 43 each were associated with cognitive ability at age 53. The pathways from father's occupation through childhood cognitive ability were the strongest, followed by educational attainment, and then occupational status. After accounting for these three life course factors, no statistically significant association remained between father's occupation and memory or visual search, and the remaining significant association between father's occupation and vocabulary was greatly reduced in size. The follow-up study at age 69 analyzed results of a screener test for Alzheimer's disease and frontotemporal dementia. Effects of maternal education and paternal occupational class on participants' screener scores were entirely mediated by participants' cognitive ability at age 8 and educational attainment, occupational complexity, and vocabulary at age 53. Findings highlighted the stability of general cognitive ability across multiple points of the life course (i.e., ages 8, 53, and 69), emphasizing latency explanations, with more modest mediational effects of adult SES.

In a different sample, Beck and colleagues (2018) used data from the men in the Vietnam Era Twin Study of Aging (VETSA) to examine general cognitive ability at age 20, adult SES at age 56, and degree of engagement in cognitive leisure activities at age 56 as potential mediators of associations between childhood SES and cognitive abilities at age 62. The assessment of cognition included measures of seven individual domains, in addition to an aggregate measure. Findings indicated an indirect path from childhood SES to all cognitive outcomes through general cognitive ability at age 20. The indirect paths involving adult SES and cognitive leisure activities were smaller and less consistent than the effect of general cognitive ability across the different domains of cognition. Overall, these results suggest that earlier-life cognition primarily mediates associations between childhood SES and later-life cognition, thereby providing stronger evidence for latency processes relative to social pathways.

Focus of the Current Study

Our study aims to advance understanding of life course processes through which childhood SES influences later-life cognition by further orienting to earlier-life cognition and midlife SES, similar to the studies reviewed above. We used data from the WLS, which is well suited to advance this area of research given its prospective measurement of childhood SES, its measure of scholastic performance in adolescence (as an earlier-life indicator of cognition), and its prospective assessments of various SES components in adulthood. The WLS also includes a measure of polygenic propensity for general cognitive ability. Controlling for this measure helps to address, in part, the possibility that any associations among childhood SES, adolescent cognitive performance, midlife status attainment, and later-life cognition are purely on account of parents' genetic propensity for cognitive performance passed onto their children (for a discussion, see Belsky et al., 2018). Accordingly, statistically controlling for polygenic propensity allows for stronger empirical evidence (i.e., relative to models without any statistical control for polygenic differences) regarding the social aspects of pathways through which childhood SES influences later-life cognition.

Guided by concepts from life course epidemiology, we employ structural equation models (SEMs) to test a pathways model in contrast to, and in combination with, a latency model. If parental SES affects later-life cognition entirely through a pathways model, then we would expect the association between childhood SES and later-life cognition to be fully accounted for by subsequent social and economic conditions in adulthood regardless of adolescent scholastic performance. In contrast, a latency model would suggest that threats to neurophysiological development in adolescence from low SES—at least some of which are likely to be reflected in relative levels of adolescent scholastic performance—would lead to greater susceptibility to poorer cognition in later life, irrespective of SES in adulthood. Accordingly, we would anticipate that linkages between childhood SES and later-life cognition would be largely accounted for by adolescent scholastic performance and/or that a direct link from childhood SES to later-life cognition would remain, regardless of SES in adulthood. In summary, our study aimed to examine the extent to which adolescent scholastic performance and midlife status attainment mediate associations between childhood SES and later-life cognition, as well as whether a latency, pathways, or combined model best characterizes the nature of mediational associations.

Method

Data

The WLS began in 1957 as a random sample of one third of the students graduating from Wisconsin high schools that year (N = 10,317). It was initially a study of social stratification and intergenerational mobility; as the participants have progressed in age, it has become a study of aging and health. Data were collected in adolescence by paper-and-pencil questionnaires, and participants have been followed primarily via telephone, mail, and in-person surveys. WLS participants of racial/ethnic backgrounds other than White were too few in number to include race/ethnicity as a variable in the data set, both for statistical and ethical reasons (Herd, Carr, & Roan, 2014). Therefore, the data analytic sample includes only non-Hispanic White participants, and this racial/ethnic homogeneity precludes broader population inferences.

Our analytic sample excludes participants without valid scores on several key variables. First, of the original sample of 10,317 participants, 4,380 of the remaining participants left the study before age 72 because of death (N = 2,049), loss to follow-up, or refusal. Second, we excluded an additional 1,415 participants who did not have sufficient measures of cognitive function at age 72, two thirds of whom were not randomly selected for any neurocognitive testing within the study protocol. The remaining third was missing because they completed fewer than three of the cognitive tests. Third, 816 participants did not agree to provide genetic data. We only include participants with genetic data (refer to *Covariates* below). Therefore, our analytic sample included 3,706 participants who provided both valid cognitive and genetic data. We conducted sensitivity analyses to assess the potential effect of selection bias on our findings, as reported in the results below.

Measures

Later-life cognition

We used scores from six cognitive tests that participants completed at the age of 72, including a **similarities test** (abstract reasoning), a **letter fluency** task (phonemic verbal fluency), a **category fluency** task (semantic verbal fluency), a **digit ordering** test (working memory), and an **immediate** and **delayed word recall** test (episodic memory). (Information about these measures is located in Supplementary Appendix A.) All participants took the Wechsler Adult Intelligence Scale (WAIS) test, and 50% of participants were randomly selected to take the category fluency test. The other tests were randomly assigned to 80% of study participants.

Childhood SES

Parents' educational attainment was measured in years of schooling, based on the participant's reports from the first survey in 1957. Given the strong correlation between mother's and father's educational attainment in the sample, we selected the larger of the two scores to create the measure. **Fathers' occupational status** was used as a proxy for parental occupational status since the majority of mothers (two thirds) did not work for pay. The data were compiled from Wisconsin state tax filings from 1957 to 1960 and then recoded based on the 1950 Duncan Socioeconomic Index (SEI). The SEI is a weighted average of occupational education and occupational income with scores ranging from 1 to 100 (Hauser & Warren, 1997). We used an averaged measure of SEI, combining the 4-year period into one score. Averages over the same 4-year period were used to measure **household income**, also obtained from state tax filings. Because the values were skewed, the variable was further transformed by dividing the values into quartiles, creating an ordinal measure.

Adolescent scholastic performance

This measure was based on a raw score from a standardized test that all Wisconsin high school students were required to take in their freshman or junior year of high school: The Henmon–Nelson Test of Mental Ability (Henmon & Nelson, 1954). Administration of this 30-min test presented 90 problems of increasing difficulty, including anagrams, proverb interpretation, vocabulary, completing sentences, spatial reasoning, and quantitative reasoning, with more than half of the items emphasizing verbal components (WLS Documentation, 1996). The WLS obtained these scores from the school districts' administrative records, and the researchers converted the scores into percentile rank scores equivalent to junior-year scores.

Midlife status attainment

We used three measures from self-reports when respondents were 53 years old (in 1993). We chose this time point because it preceded the time at which the cognitive tests were administered (age 72), at which point many were retired from the full-time labor force. The first measure was an indicator of occupational education to represent **occupational status**. Occupational education was a measure created using the 1970 U.S. Census, based on the percentage of participants within each occupation who had completed at least 1 year of college. The WLS researchers coded participants' reports of their occupation using this percentage, creating a normally distributed measure (Hauser & Warren, 1997). **Household income** was compiled based on every available source of income, including businesses; annuities; retirement accounts; and government programs (e.g., disability benefits). This measure was divided into quartiles, creating an ordinal measure, to address skew. Third, we created a continuous measure of **educational attainment**. The participants reported information about degrees attained and years spent as a student.

Covariates

Gender indicated whether the participant identified as male or female. We included three additional dichotomous variables to control for potentially spurious effects between childhood SES and later-life cognition. These variables included **family structure**, which indicated whether participants reported that until 1957 they lived with both parents most of the time; **number of living siblings**; and **adolescent geographic setting**, which indicated whether respondents attended a high school in a locality with 9,999 or fewer, or 10,000 or more, residents. We also included a **polygenic score for general cognitive ability**, which was based on the genome-wide association study (GWAS) of Lee and colleagues (2018). The polygenic score was the sum total of genes identified as related to cognitive ability that each participant carried, weighted for the strength of the association between the gene and the trait. (In the analytic sample, the polygenic measure was associated in the anticipated direction with each indicator of childhood SES—except for father's occupational prestige—as well as adolescent scholastic performance, the indicators of educational attainment, and the measures of cognitive function. Refer to Supplementary Appendix B.) Finally, to account for population stratification by ancestry, we included five principal components in the analyses.

Analytic Strategy

We used SEMs to analyze the data. Childhood SES was a latent construct as measured by highest parental educational attainment, father's occupational prestige, and household income. Adolescent scholastic performance was a manifest variable. Midlife status attainment was a latent construct indicated by educational attainment, income, and occupational prestige. Confirmatory factor analysis indicated two dimensions of later-life cognition measured by the six cognitive tasks: (a) memory, and (b) language/executive function (refer to Supplementary Appendix C). Memory comprised immediate and delayed recall and the digit ordering task, and language/executive function comprised the WAIS similarities task, the letter fluency task, and the category fluency task. We modeled each outcome separately. All models controlled for family structure, number of living siblings, gender, geographic setting in adolescence, polygenic score for cognitive ability, and five principle components. Each measure was standardized in the models.

Regarding the data analytic sequence, first, we estimated a simple model to establish that, net of covariates, there were significant associations between childhood SES and the two domains of later-life cognition (i.e., memory and language/executive function). Then, to test the extent to which adolescent scholastic performance and midlife status attainment mediated associations between childhood SES and later-life cognition, we estimated three models for each outcome. The first tested the social pathways model, as displayed in Figure 1A. The second tested the latency model, as displayed in Figure 1B. The third tested a combined model including both social pathways and latency, as displayed in Figure 1C. Finally, to test for complete mediation,

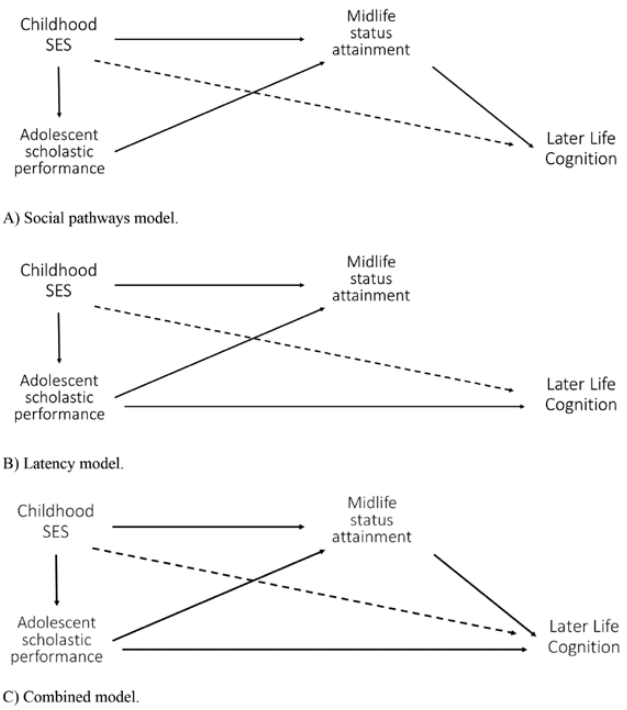

Figure 1. Three theoretical models of the relationship between childhood socioeconomic status (SES) and later-life cognition. The social pathways model (A) indicates that childhood SES influences later-life cognition through midlife status attainment, irrespective of adolescent scholastic performance. The latency model (B) indicates that childhood SES influences later-life cognition through adolescent scholastic performance, irrespective of midlife status attainment. The combined model (C) indicates that childhood SES influences later-life cognition through the association between adolescent scholastic performance and midlife status attainment, as well as through midlife status attainment and adolescent scholastic performance independently.

Table 1. Descriptive Statistics, Participants in the Wisconsin Longitudinal Study at Age 72, 2011 (N = 3,706)

	Mean or %	*SD*	Min	Max
Childhood SES				
Highest parental education (in years)	11.51	3.04	7	18
Father's occupational prestige (SEI)[a]	30.41	22.44	2	96
Household income (in quartiles)	2.55	1.12	1	4
Adolescent scholastic performance[b] (range: 61–145)	102.45	14.64	61	145
Midlife status attainment				
Educational attainment (in years)	13.80	2.38	12	21
Occupational prestige (SEI)[a]	49.42	22.74	2	96
Income (in quartiles)	2.65	1.10	1	4
Covariates				
Female	0.53	—	0	1
Rural residence in adolescence	0.52	—	0	1
Number of siblings	2.99	2.26	0	10
Family structure (1 = lived with both parents)	0.09	—	0	1
Polygenic score: cognitive ability	−0.34	0.22	−1.05	0.38
Later-life cognition				
Immediate recall (number of words)	5.47	1.45	0	10
Delayed recall (number of words)	3.43	1.78	0	10
Digit order (out of 12)	6.76	2.63	0	12
Letter fluency (number of words)	11.29	4.18	0	31
Category fluency (number of words)	19.64	5.97	0	47
WAIS[c] similarities (out of 12)	6.35	2.32	0	12

Note. Summary statistics from unstandardized, unimputed data. SES = socioeconomic status.
[a]SEI = 1950 Duncan Socioeconomic Index (Hauser & Warren, 1997).
[b]Adolescent scholastic performance = Henmon–Nelson Test of Mental Ability (Henmon & Nelson, 1954).
[c]WAIS = Wechsler Adult Intelligence Scale Revised (Wechsler, 1981).

we estimated the combined model excluding the direct path between childhood SES and adult cognition.

We evaluated two indicators of fit—Bayesian Inference Criterion (BIC) and Akaike Inference Criterion (AIC)—for each model. Both fit statistics use model comparisons and favor parsimony: The difference lies in the penalties subtracted from the log-likelihood for additional parameters (K) and sample size (n). For AIC the penalty is $2K + (2K(K+1))/(n - K - 1)$, and for BIC the penalty is $K\log(n)$ (Burnham & Anderson, 2004). The model with the smallest BIC value has the best fit, with the difference between the model BICs indicating the strength of the evidence for improved fit. A difference of 0–2 indicates weak evidence, 2–6 indicates positive evidence, 6–10 indicates strong evidence, and a difference of 10 and greater is very strong evidence (Raftery, 1995). Model fit using AIC is also determined by model comparison, where the model with the smallest value is favored (Akaike, 1987).

Missing Data

Of the analytic sample of 3,706 participants, 79.6% had complete data and an additional 16.9% were missing data on only one or two variables. The variables missing the most data were the two derived from tax returns (childhood household income and father's occupational status), each missing 12.5% of observations. Analyses suggested that the data were, at worst, missing at random (MAR). Therefore, we used multiple imputation by chained equations (MICE) to create five imputed data sets. Post-imputation, we used Rubin's (1987) rules to estimate the models presented below by combining model estimates across the five data sets.

Results

Table 1 displays descriptive statistics for all analytic variables. The first SEM model that we estimated (not shown) established that a 1-SD increase in childhood SES was associated with better memory and better language/executive function at age 72. A 1-*SD* increase in childhood SES was associated with an 8 percentage point higher score on memory ($p < .001$) and a 34 percentage point higher score on language/executive function ($p < .001$).

Next, key results from the social pathways model, latency model, and combined model are presented in Table 2 (for memory) and Table 3 (for language/executive function). Table 4 displays the goodness-of-fit statistics for social pathways, latency, and combined models in addition to a full mediation model, which includes the combined model without a direct path from childhood SES to later-life cognition.

The direct association between childhood SES and memory remained significant in both the social pathways model and the latency model, although in the social pathways model, the coefficient became negative (−0.05, $p < .05$). This path was not significant and was near zero (−0.02) in the combined model, which had better fit than both the latency model and the social pathways model. The complete results of the combined model are presented in Supplementary Appendix B. The final model for memory that eliminated the direct path further improved fit, indicating full mediation.

The direct association between childhood SES and language/executive function remained significant in both the social pathways model and the latency model, although,

Table 2. Three Models of the Relationship Between Childhood SES and Memory

Social pathways model	Standardized estimate	*SE*
Childhood SES → Adolescent scholastic performance	0.51***	0.04
Childhood SES → Midlife status attainment	0.60***	0.04
Adolescent scholastic performance → Midlife status attainment	0.38***	0.02
Childhood SES → Memory	−0.05*	0.02
Midlife status attainment → Memory	0.17***	0.02
Latency model	**Standardized estimate**	***SE***
Childhood SES → Adolescent scholastic performance	0.52***	0.04
Childhood SES → Midlife status attainment	0.61***	0.04
Adolescent scholastic performance → Midlife status attainment	0.36***	0.02
Childhood SES → Memory	0.05**	0.02
Adolescent scholastic performance → Memory	0.11***	0.01
Combined model	**Standardized estimate**	***SE***
Childhood SES → Adolescent scholastic performance	0.52***	0.04
Childhood SES → Midlife status attainment	0.60***	0.04
Adolescent scholastic performance → Midlife status attainment	0.36***	0.02
Childhood SES → Memory	−0.02	0.02
Adolescent scholastic performance → Memory	0.08***	0.01
Midlife status attainment → Memory	0.08***	0.02

Note. SE = standard error; SES = socioeconomic status.
*$p < .05$. **$p < .01$. ***$p < .001$.

Table 3. Three Models of the Relationship Between Childhood SES and Language/Executive Function

Social pathways model	Standardized estimate	*SE*
Childhood SES → Adolescent scholastic performance	0.50***	0.04
Childhood SES → Midlife status attainment	0.61***	0.04
Adolescent scholastic performance → Midlife status attainment	0.40***	0.01
Childhood SES → Language/executive function	−0.22***	0.05
Midlife status attainment → Language/executive function	0.67***	0.04
Latency model	**Standardized estimate**	***SE***
Childhood SES → Adolescent scholastic performance	0.54***	0.05
Childhood SES → Midlife status attainment	0.68***	0.05
Adolescent scholastic performance → Midlife status attainment	0.35***	0.02
Childhood SES → Language/executive function	0.26***	0.03
Adolescent scholastic performance → Language/executive function	0.32***	0.01
Combined model	**Standardized estimate**	***SE***
Childhood SES → Adolescent scholastic performance	0.52***	0.04
Childhood SES → Midlife status attainment	0.60***	0.04
Adolescent scholastic performance → Midlife status attainment	0.36***	0.02
Childhood SES → Language/executive function	−0.07	0.04
Adolescent scholastic performance → Language/executive function	0.20***	0.02
Midlife status attainment → Language/executive function	0.37***	0.03

Note. SE = standard error; SES = socioeconomic status.
***$p < .001$.

again, in the social pathways model, the coefficient became negative (−0.22, $p < .001$). As with memory, this path was not significant and was near zero (−0.07) in the combined model, which had better fit than both the latency model and the social pathways model. The final test for full mediation was more ambiguous for language/executive function than for memory, with BIC favoring the more parsimonious model without the direct path, and AIC favoring the complete combined model.

Sensitivity Tests

We conducted a series of sensitivity tests to check the robustness of our results. The first set addressed potential subgroup differences in the results reported above. First, we tested the social pathways model, latency model, and combined model separately for men and women. As in the pooled results, the combined model had the best fit for both genders, with the exception of women's memory scores. There was positive evidence (BIC difference of 4) to prefer the latency model over the combined model for women's memory scores. Second, we tested the social pathways model, latency model, and combined model separately for participants with above-average and below-average polygenic scores for cognitive performance. We split the sample at the mean standardized polygenic score of zero. As in the pooled results, the combined model had the best fit for both persons with above-average and below-average polygenic scores. Finally, we divided the sample between participants who were working for pay at age 72 (approximately 40% of the analytic sample) and

Table 4. Goodness-of-Fit Statistics for Successive Nested Structural Equation Models

Model	*df*	BIC[a]	BIC difference	AIC[b]	Log-likelihood
Memory					
Social pathways model	85	53,900	—	53,465	995
Latency model	85	53,844	56	53,409	938
Combined model	84	53,822	78	53,381	909
Full mediation model	85	53,815	85	53,380	910
Language/executive function					
Social pathways model	85	54,530	—	54,094	1,045
Latency model	85	54,605	−75	54,170	1,121
Combined model	84	54,398	132	53,957	906
Full mediation model	85	54,394	136	53,959	910

Note. [a]BIC = Bayesian Inference Criterion.
[b]AIC = Akaike Information Criterion.

those who were not. There was no difference in the results for the two groups.

Moreover, we specified models in which we represented childhood SES as three manifest indicators (i.e., parent's educational attainment, father's occupational status, and household income) rather than as a latent variable. The indicators behaved similarly to one another with regard to the size, direction, and significance of coefficients. These results suggest that no single component of childhood SES is accounting for the results based on the latent variable specification of SES, as reported above. We also tested for threshold effects in childhood SES and found that the associations were consistent across the categories of childhood SES levels.

Finally, we estimated simulation models to assess the extent to which study attrition might have biased our results. Prior studies have found that individuals with lower cognitive ability die earlier, on average, within longitudinal studies, and this differential sample selection restricts the range of cognition data at the lower end of the distributions (Luciano, 2014). These analyses are available in Supplementary Appendix D. In sum, the results were consistent with those reported here: The combined model fit best for both domains of cognition, and in that model, the direct path between childhood SES and cognition was not statistically significant.

Discussion

Drawing on data from the WLS, we estimated SEMs to examine life course pathways from childhood SES to later-life cognition. We examined differences in scholastic performance in adolescence, as well as midlife status attainment, as indicators of two potentially interconnected life course pathways from childhood SES to later-life cognition. Overall, findings indicated that adolescent scholastic performance and midlife status attainment fully accounted for associations between childhood SES and later-life memory, as well as later-life language/executive functioning. Overall, we found the models allowing for both latency processes through adolescent scholastic performance, alongside social pathway processes through midlife status attainment, to fit the data best. These results suggest that, overall, for the participants in our study, both latency and social pathway processes simultaneously operate and account for how childhood SES influences later-life cognition.

Our findings in support of the comparable salience of both latency and social pathway processes from childhood SES to later-life cognition are somewhat incongruent with similar analyses using data from other longitudinal cohort samples. Beck and colleagues (2018) concluded that "…at least in late middle age, early adult (general cognitive ability) is a much stronger reserve factor compared with adult SES…" (p. 9). Though Richards and colleagues (Richards & Sacker, 2003; Richards et al., 2019) found more evidence than Beck and colleagues did for the mediational role of adult SES, they too found stronger evidence for general cognitive ability in childhood and midlife as mediators. Our findings, in contrast, generally indicate that adolescent scholastic performance is associated with later-life cognition directly as well as *through* its relationship to adult status attainment—rather than *more than* adult status attainment.

These differences might be because of cohort variation across the studies' samples. Beck and colleagues (2018) used data from the VETSA sample of male twin pairs born between 1943 and 1957 who served in the U.S. military during the Vietnam era. Richards and colleagues (2019) used data from men and women who were born in England, Scotland, or Wales in March of 1946. The WLS study, in contrast, comprises a slightly older cohort of men and women: Those who graduated from Wisconsin high schools in 1957, most of whom were in the birth cohort of 1939. Dominant mediational pathways might differ for people exposed to unique sets of historical conditions at different stages of the life course. Congruent with this idea, using data from the Health and Retirement Study, Hale (2017) found that older adults who were 0–2 years old when the stock market crashed in the 1929 Great Depression demonstrated consistently lower levels of fluid

cognition compared to earlier-born adults, despite higher levels of education and occupational attainment. It is possible that latency pathways similarly might be stronger for men and colleagues; their earliest years of childhood transpired shortly after the end of World War II when social and economic institutions were still recovering from war. Moreover, the VETSA study includes only men who were in the military during the Vietnam era. Many VETSA participants likely benefited from social policies intended to improve the lives of servicemen and women, such as college education subsidies. Such benefits might weaken social pathway processes by muting linkages between earlier-life circumstances and social and economic conditions in adulthood. Fitting with this idea, a study using data from the Health and Retirement Study found that linkages between low maternal education and adult cognition were smaller among veterans of the Korean and Vietnam Wars in contrast to their nonveteran peers (Vable et al., 2018). Overall, these findings suggest continued attention to how intersectional social positions—including cohort—influence life course pathways from childhood SES to later-life cognition.

Our study did not include clinical assessments of cognitive functioning nor measures of rates of decline in cognition. Nevertheless, findings regarding life course antecedents of individuals' performance levels on neurocognitive tests at age 72 remain important for efforts to promote individual and population health. Risk for Alzheimer's disease increases dramatically with advancing age, affecting 17.6% of people age 75–84 in contrast to 17.6% of people age 75–84 (Hebert, Weuve, Scherr, & Evans, 2013). Consistent with a functional threshold model (Tucker-Drob, Johnson, & Jones, 2009), individuals who begin with relatively higher levels of cognition earlier in later life likely have a greater number of years before potential age- and disease-related processes yield clinically significant symptoms and impaired functional capacity.

Moreover, taking our findings as a whole, results suggest that childhood SES does not guarantee later-life cognition, but rather, is associated with later-life cognition through potentially modifiable sequelae, such as adolescent scholastic performance and midlife status attainment. Overall, understanding of these life course processes encourages conceptualizing lifelong brain health beyond an emphasis on individual behaviors or health statuses in adulthood, such as physical activity, cognitive stimulation, smoking status, or hypertension. Our findings support the importance of incorporating research, policy, and practice on life course social determinants of health within the field of cognitive aging as well, with particular attention to inequalities from childhood. Social determinants of health refer to "conditions or circumstances in which people are born, grow, live, work, and age" (World Health Organization, 2019), which are influenced by political, social, and economic contexts. Congruent with results from our study, there is growing evidence that people's exposure to social dis/advantages in childhood is associated with later exposures in young adulthood, midlife, and later life (Carr, 2019). In this sense, our findings highlight the relevance of targeting efforts to lower risk exposures in adulthood, especially for individuals with low SES as children. This approach constitutes one strategy to reduce socioeconomic gradients in health (see Dow, Schoeni, Adler, & Stewart, 2010). Specifically, our results suggest that programs and policies to promote the scholastic performance of children from lower SES families, as well as to enhance access to postsecondary education, financial well-being, and higher prestige jobs among children with lower SES, are relevant as potential strategies to promote optimal cognitive aging.

Our findings further support the importance of attending to intracohort variation in later-life cognition. Recent research using longitudinal, population health samples have found declining prevalence of dementia among contemporary cohorts of older adults (Langa et al., 2017), with evidence that higher levels of education among more recent cohorts of older adults largely account for these differences (Leggett et al., 2019). Our study's findings encourage continued attention to subgroups of older adults within the same birth cohort who are at differential risk for poor cognition as they age because of intracohort differences by social positions, including life course SES.

It is important to note the correlational nature of this study, which precludes our ability to conclude that adolescent scholastic performance and midlife status attainment alone influence later-life cognition. Linkages between scholastic ability in adolescence and later-life cognition, for example, might be on account of children from lower-SES families having poorer childhood nutrition, lower birthweight, or less advantaged neighborhood environments, on average. Because the WLS does not offer robust measures of such circumstances, our study is unable to investigate them.

Our study is also unable to address the potential neurophysiological processes through which childhood SES, adolescent scholastic performance, and midlife status attainment affect brain development and cognitive performance in later life. For example, contextual models of cognitive aging posit that life course conditions—including SES—influence neurophysiological structure and function (Reuter-Lorenz & Park, 2014). One such neurophysiological process is cognitive scaffolding, which refers to the compensatory use and development of alternative neural circuits to maintain cognitive abilities in the face of neurofunctional decline. Our findings suggest the importance of studies with biomarker measures of neurophysiological functioning to examine whether greater childhood SES and its sequelae (such as adolescent scholastic performance and midlife status attainment) are predictive of better later-life cognition because of mechanisms such as cognitive scaffolding.

Furthermore, because polygenic scores only account for a fraction of the estimated total genetic influence on cognitive abilities, our results might underestimate the extent to which heritability processes explain the observed associations. Also, the WLS measure of early-life cognitive performance

was limited to the Henmon–Nelson Test of general cognitive ability, precluding our ability to test associations for more specific indicators of adolescent cognitive performance. Moreover, as stated in our description of the sample, the WLS study includes only White high school graduates. Therefore, estimates might differ for samples inclusive of other population subgroups. It is especially important for studies that explicitly engage members of underrepresented racial/ethnic groups, many of whom have disproportionately higher rates of Alzheimer's disease and related dementias (Mayeda, Glymour, Quesenberry, & Whitmer, 2016).

Despite these limitations, this study contributes to a growing body of evidence on life course processes through which childhood SES has implications for later-life cognition. Overall, results suggest that the sequelae of childhood SES on later-life cognition are not constrained to childhood alone (independent of adulthood) nor to adulthood alone (independent of childhood), but extend and are interconnected across the life course. By using structural equation modeling and capitalizing on data from one of the longest-running cohort studies in the United States, our study helps to further elucidate the life course origins of interindividual differences in later-life cognition. It also encourages the incorporation of life course perspectives on efforts to promote healthy brain aging as people experience longer, and more socioeconomically unequal, lives.

Supplementary Material

Supplementary data are available at *The Journals of Gerontology, Series B: Psychological Sciences and Social Sciences* online.

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Author Contributions

E. A. Greenfield conceptualized the study, wrote the introductory and discussions sections, and codesigned the analytic sequence. S. Moorman wrote the *Method* and *Results* section, created the statistical tables, and codesigned the analytic sequence. S. Moorman and A. Rieger conducted the statistical analyses, and A. Rieger assisted with the literature review. All authors were involved in editing the paper.

Conflict of Interest

None reported.

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