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Original Article

Socioeconomic Status and Biological Risks for Health and Illness Across the Life Course

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Abstract

Objectives: We assess the temporal properties and biosocial mechanisms underlying the associations between early-life socioeconomic status (SES) and later health. Using a life-course design spanning adolescence to older adulthood, we assess how early life and various dimensions of adult SES are associated with immune and metabolic function in different life stages and examine possible bio-behavioral and psychosocial mechanisms underlying these associations.

Method: Data for this study come from 3 national studies that collectively cover multiple stages of the life course (Add Health, MIDUS, and HRS). We estimated generalized linear models to examine the prospective associations between early-life SES, adult SES, and biomarkers of chronic inflammation and metabolic disorder assessed at follow-up. We further conducted formal tests of mediation to assess the role of adult SES in linking early SES to biological functions.

Results: We found that early-life SES exerted consistent protective effects for metabolic disorder across the life span, but waned with time for CRP. The protective effect of respondent education remained persistent for CRP but declined with age for metabolic disorder. Adult income and assets primarily protected respondents against physiological dysregulation in middle and old ages, but not in early adulthood.

Discussion: These findings are the first to elucidate the life-course patterns of SES that matter for underlying physiological functioning during the aging process to produce social gradients in health.

Keywords: Aging, Inflammation, Life course, Metabolic disorder, Physiological functioning, SES

Research documents the detrimental health consequences of adverse social conditions (Pearlin, Schieman, Fazio, & Meersman, 2005). Despite strong evidence for the socioeconomic status (SES) gradient in health, substantial gaps remain in understanding the social and biological mechanisms underlying these disparities. Physiological stress response across major regulatory systems contributes to the initiation and progression of a wide range of age-related conditions and may be strongly conditioned by SES-related exposures. However, few studies examine *how* socioeconomic exposures "get under the skin" to affect health, leaving questions about biological plausibility unanswered. Further, the life course acts as an important—yet understudied—environmental context within which such links exist to produce SES disparities in health and aging.

Although recent epidemiological and biomedical studies have begun to examine the early-life origins of adult disease, major limitations exist in their abilities to inform aging research. Health and aging is a life course process that arguably begins before birth, and SES gradients in health have been documented in all stages of the life span. SES also operates in different contexts, includes multiple components, and affects health through multiple pathways. Further, the importance of these aspects and the mechanisms through which they operate likely vary across life stages. Still, few studies consider how diverse measures of SES relate to health across the life course. Using an innovative life-course research design that draws on data from multiple prospective cohort studies from childhood to older adulthood, this study addresses these fundamental gaps in the current literature by examining pathways and mechanisms linking early-life and adult SES to later-life physiological functioning. Findings from this study refine scientific understandings of the social determinants of population health disparities and aging processes and contribute new knowledge of the particular life stages and socioeconomic factors to target with preventative policy and programmatic efforts.

Early SES and Later-Life Health: Life-Course Theoretical Frameworks

SES strongly conditions one's exposures to chronic stress and a host of psychosocial and bio-behavioral risk factors for health (Link & Phelan, 1995). Indicators of SES, including educational attainment, family income, occupational prestige, and wealth, have been linked to a wide variety of health outcomes (Clark, DesMeules, Luo, Duncan, & Wielgosz, 2009; Herd, Goesling, & House, 2007; Pollack et al., 2007).

The *sensitive period model* provides the dominant paradigm to explain how early life factors influence adult health risk (Ferraro, 2016; Warren, 2016). The sensitive period model posits that exposures during early life (gestation through adolescence) produce stronger effects on health and human development than they would at other life stages (Ben-Shlomo & Kuh, 2002). This model hypothesizes that low early-life SES increases the risk of poor health in later life, independent of adult SES and other intervening risk factors.

Early life conditions may not always exert a direct causal effect on adult health. The *accumulation of risks model* holds that deleterious exposures associated with low SES accumulate over time (O'Rand, 2009; Willson, Shuey, & Elder, 2007). In contrast to the sensitive period model, this model emphasizes the total burden of damage or insults across the life course, demonstrated through the additive effects of early- and later-life SES factors (Hallqvist, Lynch, Bartley, Lang, & Blane, 2004). The *pathway model* posits that childhood circumstances affect adult health risk indirectly by putting individuals on paths differentiated by types and levels of stress exposures (Hayward & Gorman, 2004; Pudrovska & Anikputa, 2014). This model predicts that SES at each subsequent life stage mediates the effects of early-life adversity on later-life health risk.

Extant research shows mixed support for the different conceptual models discussed above and highlights the complexities of interrelated life-course processes underlying the associations between early-life SES and health risk in later life (Ferraro, Schafer, & Wilkinson, 2016; Luo & Waite, 2005). Because prior research typically rely on cross-sectional data with retrospective information on early-life SES and measures of adult health during one life stage, these studies implicitly assume that early-life SES has uniform impacts on adult health. However, the associations of early-life SES with health may vary across stages of adulthood as aging progresses. Questions also remain as to whether early or contemporaneous SES affects adult health more strongly or whether the impacts of specific life course socioeconomic factors wax and wane at different periods in the aging process. Although the aforementioned life-course models are often positioned as conceptually distinct, methodological research shows that the life-course processes underlying these models are likely to be complimentary rather than mutually exclusive (Hallqvist et al., 2004). As a result, previous research cannot adjudicate among different models or explicate their interrelations, and our current understanding of the impact of early-life factors on health during different life stages in the aging process remains largely tentative.

Life-Course SES and Biological Mechanisms Underlying Aging and Health

The wide range of age-related health conditions affected by SES suggests common biological mechanisms at work. Evidence increasingly links SES disadvantage to biomarkers of stress-related activation of physiological pathways to chronic diseases of aging. Lower levels of education, income, and occupational status are associated with elevations in circulating levels of acute phase proteins such as C-reactive protein (CRP) and other markers of inflammation (Danese et al., 2009; Koster et al., 2006) and infection (Dowd & Aiello, 2009), as well as cardiovascular disease risk (Cundiff, Uchino, Smith, & Birmingham, 2015; Gustafsson, Persson, & Hammarström, 2011), and metabolic dysregulation such as increased incidence of diabetes, reduced high-density (HDL) and raised low-density lipoprotein (LDL), increased waist circumference, and higher fasting glucose and insulin and glycosylated hemoglobin (Danese et al., 2009; Feldman & Steptoe, 2003).

Though a growing body of studies on SES gradients in health considers biosocial interactions, few of these studies incorporate an aging and life-course framework. Adverse changes in immune and metabolic function may represent pathways linking disadvantaged childhood social experiences to health later in life. For example, childhood

adversity indexed by socioeconomic disadvantage has been associated with adult health conditions such as heightened inflammatory response and clustering of metabolic risk markers (Danese et al., 2009). Tamayo, Koenen, and Kubzansky (2010) found that low parental SES was associated with metabolic abnormalities in later life. However, only limited research considers life course measures of SES simultaneously to examine whether early-life adversity and current socioeconomic circumstances differentially impact immune or metabolic function into old age. As a result, it remains unclear whether exposure to socioeconomic-related adversity may lead to more pronounced effects on adult physiological functioning relative to other life stage periods.

Components, Dynamics, and Mediating Mechanisms of SES

Research indicates that the individual components of SES have varying associations with health, with some components being better predictors of health outcomes than others. However, findings from these studies are mixed (Clark et al., 2009). Further, studies suggest that the relative importance of different SES components for health may vary across the life course (Boen & Yang, 2016; Friedman & Herd, 2010; House et al., 1994; Willson et al., 2007). In addition to its multidimensionality, SES is a dynamic construct in which prior socioeconomic conditions shape present and future SES. The accumulation of wealth in late adulthood often depends upon early-life socioeconomic environment, educational attainment, and income, operating through a life-course process spanning decades. Because so few studies utilize longitudinal data incorporating multiple indicators of SES over time, an integrative biosocial approach would enhance knowledge regarding how specific components of SES differentially impact biomarkers of health and aging dynamically across the life span (Harris, 2010).

Lower SES leads to greater exposure of psychosocial and bio-behavioral risk factors for health and well-being (House et al., 1994; Pearlin, Menaghan, Lieberman, & Mullan, 1981) that conceivably mediate the associations between SES and biological functioning (Danese et al., 2009). Chronic psychosocial stress links low SES with physiological dysregulation (Adler & Snibbe, 2003; McEwen & Gianaros, 2010). A lack of social integration and support to buffer such stress predicts higher CRP, systolic BP, and central and overall obesity in both young and late adulthood (Y. C. Yang et al., 2016). Stressful life events and perceived stress relate to heightened inflammatory response in children (Chen et al., 2006). Health behaviors may mediate SES effects on biological function. Obesity increases the likelihood of inflammation as well as metabolic impairments (Finch, 2010; Y. C. Yang, Johnson, Schorpp, Boen, & Harris, 2017) and declines with lower levels of SES and childhood adversities (Tamayo et al., 2010; Y. Yang & Land, 2013). Cigarette smoking and depression have both been linked with childhood adversity and compromised immune-related processes (Slopen, Koenen, & Kubzansky, 2012). Still, studies have found that accounting for various behavioral and psychosocial risk factors only moderately mediates the association between SES and markers of inflammation (Koster et al., 2006) and physiological dysregulation (Gruenewald et al., 2012).

Research Goals

Our study makes several unique contributions to the literature. First, most extant longitudinal studies collected information on participants during one period of adulthood. We use a recently developed innovative research design (Y. C. Yang et al., 2016) to draw inference from three national population-based longitudinal data sets. This design includes multiple cohorts that collectively span young adulthood, midlife, and older adulthood. It provides an augmented age range, longer periods of stress exposures, and health outcome measures across multiple life stages that enhance our abilities to evaluate associations between early-life to adult SES pathways and biological risk factors over a far more extensive period of life than any single data set can allow and may provide the opportunity to observe various life course variations not possible otherwise. Given the lack of long-running studies with comprehensive, longitudinal SES and biomarker data, the use of three nationally representative samples spanning young through older adulthood represents a novel contribution to the existing literature in this area. Second, this study utilizes multiple measures of life-course SES, including parent education, family income and welfare receipt in early life, and individual education, earnings, and wealth in adulthood. Use of multiple SES indicators allows for a more comprehensive assessment of SES within each life stage and facilitates the comparison of SES-health links across the life span. To the extent that adult SES mediates the association between early life SES and biomarkers in a pathway model, we identify the individual components of adult SES through which early-life SES operates. Furthermore, we explore how psychosocial and bio-behavioral mechanisms mediate the observed associations between SES pathways and our biomarker outcomes at different life-course stages. In sum, the present study investigates three overarching research questions:

1. What is the relationship between early-life SES and later-life physiological functioning in young, mid, and late adulthood?
2. Which components of adult SES—namely, education, income, and wealth—provide SES pathways that link early-life SES to later-life physiological functioning at different life course stages?
3. What are the psychosocial and bio-behavioral factors that mediate the associations between SES and biomarkers of physiological functioning at different life-course stages?

Data and Methods

We use the life-course research design developed by Y. C. Yang and colleagues (2016) to harmonize data analyses using three National Institutes of Health national longitudinal cohort studies. We harmonized outcome, exposure, and covariate measures across data sets, conducted parallel analyses with the three life-course samples, and compared findings regarding the same associations across the life-course samples within the integrated conceptual and analytic framework.

Data

Data for young adulthood come from 10,597 participants in the National Longitudinal Study of Adolescent to Adult Health (Add Health) aged 12–18 at Wave I (1994–1995) and followed up at aged 24–34 in Wave IV (2008–2009). Data for mid adulthood come from 735 respondents aged 35–64 in the National Survey of the Midlife Development in the United States (MIDUS) surveyed at Wave I (1995–1996) and followed up at Wave II (2004–2009). Data for late adulthood come from 6,381 participants aged 65 and older in the Health and Retirement Study (HRS) at baseline in 1998 and followed every 2 years through 2006. The analytic samples included respondents who had complete data on the variables used in the analyses. More information about the studies used in this analysis can be found in Supplement 1.

Measures

Supplement 2 describes all variables used in the analyses. *Chronic inflammation* was measured using CRP. The cut points for CRP reflect clinical reference ranges for health risk. Metabolic function was assessed by a composite measure that includes seven biomarkers used in the clinical definition of *metabolic disorder*: diastolic blood pressure, systolic blood pressure, HbA1c, waist circumference, high-density lipoprotein (HDL cholesterol), total cholesterol, and triglycerides (see Supplement 2a for cut points used for each data source). The index of metabolic disorder is the sum of the positive indicators (Yang, McClintock, Kozloski, & Li, 2013). Details of biomarker data collection and assays are provided in Supplement 1. The Add Health biological measures come from Wave IV (2008–2009); the MIDUS biological measures come from Wave II (2004–2006); and the HRS biological measures come from waves VIII/IX (2006–2008).

We used measures of early-life and adult SES to examine life course patterns of SES. *Early-life SES* measures include parental education, household income, welfare receipt, and subjective financial wellbeing. We constructed composite measures of early-life SES, where each individual SES item was first standardized by calculating the *z*-score. The mean of the *z*-scores was then calculated to produce a continuous, composite SES score. Positive values for the SES scores indicate higher levels of SES. For the Add Health respondents, the index of early-life SES items was measured during adolescence at Wave I of the sample (1994–1995) and hence provides unique prospective measures of SES. See Supplement 2b for more detailed information on early SES items for each data source.

Adult SES measures include respondent education, household income, and household assets. Respondent education was coded as 1 = less than high school, 2 = high school graduate, 3 = some college, and 4 = college graduate or more. Household income and total household net assets were categorical, with the bottom quartile = 1, the middle two quartiles = 2, and the top quartile = 3.

Psychosocial mechanisms include social isolation and the Center for Epidemiologic Studies Depression (CES-D) scale for Add Health and HRS, as well as the Cohen Perceived Stress Scale (PSS) for Add Health. For each data source, those in the bottom quartile of the social isolation index were coded as socially isolated (Y. C. Yang et al., 2016). CES-D was a continuous measure of the items available in each data source, with higher scores indicating more depressive symptoms. The PSS was a continuous index of items related to lack of personal control and ability to overcome and handle problems, with higher scores indicating higher perceived stress.

Bio-behavioral measures and mechanisms include cigarette smoking, physical activity, and body mass index (BMI). Cigarette smoking is binary indicator for whether the respondent is currently a regular smoker. Physical activity is coded as a continuous indicator, with higher values indicating higher physical activity. BMI was assessed by field interviewers during biomarker collection in all data sources and was included as a continuous measure.

All analyses adjust for age, sex, race/ethnicity, and childhood self-rated health. Descriptive statistics of all variables used in analyses are shown in Supplement 3.

Analytic Methods

We examined the prospective associations between early-life SES, adult SES, and biomarkers assessed at follow-up using ordinal logit models for inflammation and Poisson regression models for metabolic disorder. We fit models in a stepwise fashion. We first estimated models that (a) adjust for early-life SES only; and then incorporate (b) adult educational attainment; (c) adult household income; and (d) household wealth. We controlled for childhood health in all models to account for potential selection of individuals into SES classes in adulthood. The second set of models builds on the results of the first to further examine additional intervening psychosocial and bio-behavioral mechanisms. For each data set, we included measures of social isolation, perceived stress, CES-D, smoking, physical activity, and obesity in the models of early-SES and adult SES in relation to inflammation and metabolic disorder. All models adjust for survey design effects and nonresponse using

sampling weights. Postestimation tests to compare model coefficients between models were performed using the Stata (suest) command. By testing the equality of the coefficient for early-life SES between models, we assessed the statistical significance of both full and partial mediation of early SES by adult SES and psychosocial factors (Clogg, Petkova, & Haritou, 1995). Analyses were conducted in Stata 14.

Results

We found strong associations of SES measures with both indicators of physiological functioning across all life stages; however, the relative importance of early-life and adult SES varied across young-, mid-, and late-adulthood, providing evidence consistent with different life course hypotheses for each outcome.

As seen in Model I of Table 1, higher early-life SES, as indicated by the standardized SES index score, is associated with a 16% lower odds of elevated CRP (odds ratio [OR] = 0.84, confidence interval [CI] = [0.77–0.91], $p < .001$) in the Add Health study. This association is replicated in the MIDUS and HRS samples with similar magnitude. Model II adds adult educational attainment to Model I. Compared with those with high school degrees, college-educated individuals in all life stages had significantly reduced risk of inflammation, with the negative association between college education and inflammation being particularly pronounced for the middle-aged adults in the MIDUS sample (OR = 0.53, CI = [0.36–0.80], $p < .001$). In contrast, less than high school education increased the risk of inflammation by about 45% in older adulthood (OR = 1.45, CI = [1.27–1.65], $p < .001$). Model III includes adult income, which shows a significant negative association with risk of inflammation independent of adult education in mid adulthood. Being in the highest income quartile, in particular, is associated with a 30% lower risk of elevated CRP in mid adulthood, compared with those in the middle two quartiles. Model IV further adjusts for adult assets. Although assets do not show a significant association with inflammation in young or middle adulthood, they are associated with inflammation in late adulthood. Individuals in the lowest quartile with the fewest financial assets experienced a 19% higher odds of inflammation on average, whereas those in the highest quartile had a 10% lower odds than those in the middle quartiles. In Model IV, the mediation test shows that the associations between early-life SES and inflammation are partially mediated by adult education in all samples ($p < .001$), by adult income in middle ($p = .015$) and older adulthood ($p < .001$), and by adult assets in older adulthood ($p < .001$). Adjusting for the full set of adult SES variables, the association of early-life SES with inflammation remains statistically significant in young adults in the Add Health study. In contrast, the association between early-life SES and inflammation does not hold and is fully attenuated with the inclusion of the adult SES indicators in middle-aged or older adults in the MIDUS or HRS studies.

Table 2 presents the results on the associations of life-course SES measures with metabolic disorder. Model I shows a significant negative association of early-life SES with the risk of metabolic disorder for all three life-stage samples. Higher levels of education are protective against metabolic risks across all adulthood stages, whereas less than high school education is detrimental to metabolic health in late life. Higher income is associated with reduced metabolic risks in both early- and late-adulthood. Across the life stages, we document significant associations between assets and metabolic risks. In older adults, financial assets override income and education in the association with metabolic risks. Adjusting for the adult SES measures does little to explain the associations between early-life SES and metabolic risk across life stages. Formal mediation analyses revealed that the association between early SES and metabolic risks is partially mediated by adult education for all study samples ($p = .04–<.001$), by adult income ($p < .001$) and adult assets in older adulthood ($p < .001$). Still, the coefficients of early-life SES are slightly attenuated and remain statistically significant, suggesting that early-life SES influences metabolic risk largely directly, rather than indirectly through adult SES, particularly later in life.

Adjustment for childhood health did not affect the SES–biomarker associations assessed at any point in the adult life span.

Table 3 presents the results that adjusted for the psychosocial and bio-behavioral covariates. In young adulthood, physical activity and BMI are strong risk factors for inflammation and their inclusion fully attenuated the associations of both early-life SES and adult education with CRP. In mid and older adulthood, depressive symptoms, perceived social stress, current smoking status, physical activity, and BMI are all significantly associated with CRP. Their presence substantially attenuated the association of adult education with CRP in middle adulthood but not in late adulthood. The results for metabolic disorder are similar. Cigarette smoking and physical activity are significant risk factors that partially explain the associations of early-life SES and young adult income and wealth with metabolic disorders ($p < .001$). Mediation analyses revealed that many of the psychosocial and behavioral risk factors for metabolic disorder in mid or older adulthood, including perceived stress, depressive symptoms, smoking, physical activity, and BMI, partially mediated the associations of early SES and metabolic risks ($p < .001$), though early-life SES continued to operate directly to influence metabolic risk rather than indirectly through the mechanisms. We did not find evidence that the psychosocial or bio-behavioral mechanisms attenuated the associations of adult assets with metabolic disorders.

Discussion

Contribution

Our study contributes new knowledge on the temporal dynamics through which SES relates to physiological

Table 1. Associations of Early-Life and Adult SES With Inflammation Across Life Course Studies

Independent Variables	Young adulthood: Add Health (*N* = 10,586)				Middle age: MIDUS (*N* = 735)				Older adulthood: HRS (*N* = 6,381)			
	I	II	III	IV	I	II	III	IV	I	II	III	IV
Early-life SES	0.84***	0.89**	0.89**	0.88**	0.84†	1.00	1.04	1.04	0.88***	0.95	0.95	0.95
	(0.77–0.91)	(0.82–0.96)	(0.81–0.96)	(0.80–0.95)	(0.69–1.02)	(0.80–1.25)	(0.82–1.31)	(0.82–1.31)	(0.81–0.95)	(0.87–1.02)	(0.88–1.03)	(0.88–1.03)
Education (ref. HS)												
Less than HS		1.03	1.05	1.02		1.24	1.23	1.18		1.45***	1.40***	1.35***
		(0.82–1.29)	(0.82–1.33)	(0.80–1.31)		(0.36–4.29)	(0.37–4.03)	(0.36–3.90)		(1.27–1.65)	(1.22–1.60)	(1.18–1.55)
Some college		0.96	1.01	0.96		0.98	1.01	1.01		1.44**	1.47**	1.50***
		(0.85–1.10)	(0.87–1.14)	(0.84–1.10)		(0.66–1.46)	(0.68–1.51)	(0.68–1.50)		(1.13–1.83)	(1.16–1.87)	(1.18–1.91)
College +		0.73***	0.77**	0.77**		0.53**	0.56**	0.57**		0.79***	0.82**	0.84*
		(0.62–0.85)	(0.66–0.90)	(0.66–0.90)		(0.36–0.80)	(0.37–0.84)	(0.38–0.86)		(0.69–0.90)	(0.71–0.94)	(0.73–0.97)
Income (ref. 2nd–3rd quartile)												
1st quartile			0.98	0.97			1.14	1.11			1.14*	1.06
			(0.86–1.11)	(0.85–1.12)			(0.82–1.60)	(0.79–1.57)			(1.00–1.29)	(0.93–1.21)
4th quartile			0.90	0.92			0.70*	0.73†			0.92	0.95
			(0.78–1.03)	(0.80–1.06)			(0.50–1.00)	(0.51–1.05)			(0.80–1.05)	(0.83–1.10)
Assets (ref. 2nd–3rd quartile)												
1st quartile				1.07				1.10				1.19*
				(0.96–1.19)				(0.76–1.58)				(1.03–1.38)
4th quartile				0.98				0.86				0.90†
				(0.95–1.01)				(0.60–1.23)				(0.80–1.01)

Note. Results are odds ratios from ordinal logistic regression models. All models adjust for age, race/ethnicity, sex, and self-rated childhood health. Standard errors in parentheses.
***$p < .001$. **$p < .01$. *$p < .05$. †$p < .1$.

Table 2. Associations of Early-Life and Adult SES With Metabolic Disorder Across Life Course Studies

Independent Variables	Young adulthood: Add Health (N = 10,597)				Middle age: MIDUS (N = 709)				Older adulthood: HRS (N = 5,946)			
	I	II	III	IV	I	II	III	IV	I	II	III	IV
Early-life SES	0.89***	0.93***	0.93**	0.93***	0.85***	0.89*	0.90*	0.90*	0.95***	0.97**	0.97**	0.97**
	(0.86–0.93)	(0.90–0.96)	(0.89–0.96)	(0.89–0.97)	(0.78–0.93)	(0.81–0.98)	(0.82–0.99)	(0.82–0.99)	(0.93–0.97)	(0.94–0.99)	(0.95–0.99)	(0.95–0.99)
Education (ref. HS)												
Less than HS		0.94	0.95	0.93		0.84	0.83	0.82		1.04*	1.03†	1.02
		(0.86–1.03)	(0.87–1.04)	(0.84–1.03)		(0.49–1.42)	(0.50–1.39)	(0.49–1.37)		(1.00–1.07)	(1.00–1.07)	(0.99–1.06)
Some college		0.93	0.94	0.95		1.01	1.02	1.02		0.94	0.95	0.96
		(0.88–1.0)	(0.89–1.00)	(0.88–1.03)		(0.86–1.18)	(0.87–1.19)	(0.87–1.19)		(0.88–1.01)	(0.88–1.02)	(0.89–1.03)
College +		0.76***	0.79***	0.79***		0.81*	0.83†	0.83†		0.95**	0.96*	0.97
		(0.71–0.82)	(0.73–0.84)	(0.74–0.85)		(0.68–0.98)	(0.69–1.00)	(0.69–1.00)		(0.91–0.99)	(0.92–1.00)	(0.94–1.01)
Income (ref. 2nd–3rd quartile)												
1st quartile			1.00	0.99			1.11	1.11			1.00	0.98
			(0.95–1.05)	(0.950–1.07)			(0.96–1.28)	(0.96–1.29)			(0.97–1.04)	(0.95–1.02)
4th quartile			0.89**	0.89**			0.90	0.93			0.95*	0.97
			(0.83–0.95)	(0.82–0.98)			(0.77–1.05)	(0.80–1.08)			(0.91–0.99)	(0.93–1.01)
Assets (ref. 2nd–3rd quartile)												
1st quartile				1.05*				0.93				1.01
				(1.00–1.10)				(0.80–1.09)				(0.98–1.06)
4th quartile				0.97				0.85*				0.92***
				(0.91–1.47)				(0.72–0.99)				(0.89–0.96)

Note. Results are incidence rate ratios from Poisson regression models. All models adjust for age, race/ethnicity, sex, and self-rated childhood health. Standard errors in parentheses.
***p < .001. **p < .01. *p < .05. †p < .1.

Table 3. Life-Course SES and Biological Markers: Psychosocial and Bio-behavioral Mechanisms

	C-reactive protein			Metabolic disorder		
Independent Variables	Add Health	MIDUS	HRS	Add Health	MIDUS	HRS
Early-life SES	0.93	1.17	0.98	0.97*	0.94	0.98†
	(0.85–1.02)	(0.92–1.50)	(0.90–1.06)	(0.93–0.99)	(0.87–1.03)	(0.96–1.00)
Education (ref. HS)						
Less than HS	1.06	1.29	1.30***	0.94	0.99	1.01
	(0.82–1.37)	(0.38–4.43)	(1.13–1.49)	(0.87–1.02)	(0.69–1.42)	(0.98–1.04)
Some college	0.91	1.18	1.41**	0.91***	1.05	0.94†
	(0.79–1.06)	(0.76–1.81)	(1.12–1.79)	(0.87–0.96)	(0.91–1.19)	(0.88–1.01)
College +	0.91	0.74	0.93	0.85***	0.94	1.00
	(0.77–1.08)	(0.48–1.14)	(0.81–1.07)	(0.80–0.90)	(0.80–1.11)	(0.96–1.04)
Income (ref. 2nd–3rd quartile)						
1st quartile	1.01	1.00	1.04	1.01	1.04	0.99
	(0.87–1.17)	(0.69–1.43)	(0.91–1.19)	(0.95–1.06)	(0.91–1.17)	(0.96–1.02)
4th quartile	0.99	0.80	0.97	0.95	1.00	0.97
	(0.86–1.15)	(0.55–1.17)	(0.84–1.12)	(0.89–1.01)	(0.88–1.14)	(0.93–1.01)
Assets (ref. 2nd–3rd quartile)						
1st quartile	1.01	1.17	1.06	1.01	0.99	0.98
	(0.90–1.14)	(0.81–1.69)	(0.91–1.23)	(0.97–1.06)	(0.87–1.13)	(0.95–1.02)
4th quartile	1.00	0.98	0.98	1.00	0.94	0.95***
	(0.87–1.16)	(0.66–1.46)	(0.86–1.10)	(0.94–1.07)	(0.82–1.08)	(0.92–0.98)
Female	2.35***	2.41***	1.24***	0.92***	0.77***	0.95**
	(2.12–2.61)	(1.78–3.26)	(1.11–1.38)	(0.88–0.95)	(0.70–0.86)	(0.93–0.98)
Age	1.01	1.00	1.00	1.03***	1.02***	1.01***
	(0.98–1.04)	(0.98–1.02)	(0.99–1.01)	(1.02–1.04)	(1.01–1.02)	(1.00–1.01)
Race (ref. white)						
Black	0.93	0.65	1.25*	1.12***	0.84	1.05*
	(0.80–1.09)	(0.27–1.58)	(1.04–1.50)	(1.07–1.17)	(0.59–1.20)	(1.01–1.10)
Hispanic	1.08	0.98	0.70*	1.07**	0.81	1.05
	(0.94–1.24)	(0.50–1.92)	(0.52–0.92)	(1.02–1.13)	(0.60–1.08)	(0.97–1.13)
Early life self-rated health	0.99	1.09	1.00	0.99	1.01	1.01
	(0.93–1.05)	(0.93–1.29)	(0.94–1.07)	(0.97–1.00)	(0.95–1.07)	(0.99–1.03)
Social isolation	1.12	0.74	1.10	1.01	0.93	0.98
	(0.99–1.28)	(0.49–1.13)	(0.98–1.23)	(0.97–1.06)	(0.80–1.08)	(0.96–1.01)
CES-D	0.99	1.04*	1.05**	1.00	0.99	1.01**
	(0.98–1.01)	(1.01–1.07)	(1.02–1.09)	(0.99–1.00)	(0.98–1.01)	(1.00–1.02)
PSS	1.00	0.95**		0.99	1.01†	
	(0.98–1.03)	(0.92–0.98)		(0.98–1.00)	(1.00–1.03)	
Current smoker	1.11	1.37	1.32***	1.07**	1.22*	1.02
	(0.97–1.25)	(0.86–2.18)	(1.19–1.46)	(1.01–1.13)	(1.05–1.42)	(0.99–1.05)
Physical activity	0.93***	0.96*	0.89***	0.96***	0.99†	0.98**
	(0.90–0.96)	(0.93–1.00)	(0.86–0.93)	(0.96–0.97)	(0.98–1.00)	(0.97–0.99)
BMI	1.14***	1.17***	1.08***	1.05***	1.07***	1.03***
	(1.13–1.15)	(1.14–1.20)	(1.07–1.09)	(1.04–1.05)	(1.06–1.08)	(1.03–1.04)

Note. Standard errors in parentheses.
***$p < .001$. **$p < .01$. *$p < .05$. †$p < .1$.

indicators of age-related health processes at different stages of the life span. We demonstrate how SES operates through several life course and biological mechanisms in its association with these indicators and how these mechanisms evolve in the aging process. Our data and analytic approach advanced our understandings of the process linking early-life conditions and later health, demonstrating more complex and varied SES patterning of distinct biological stress responses than previous research. In particular, we find that the direct relationships between early-life SES and inflammation wane as individuals age, while its indirect association with health remains important through its influence on the SES trajectories of individuals into adulthood. For metabolic risk, we show that early-life SES influences health directly in an additive fashion with adult SES to influence health across the life course. Further, we find that

the components of adult SES that predict contemporaneous adult health differ across stages of adulthood. While education remains consistently associated with health, the importance of wealth increases in middle to older adulthood.

In young adulthood, we find additive and independent associations of both early-life SES and young adult SES with health risks, providing evidence in support of the accumulation of risk hypothesis. In middle to older adulthood, early-life SES bears different relationships with the two biological indicators. Early SES operates through adult SES in its association with inflammation later in the life course, supporting the pathway hypothesis. These varying results across life samples suggest that, in young adulthood, early-life social conditions continue to independently shape inflammatory risk, net of contemporaneous SES. By mid- and later-adulthood, the direct link between early-life conditions and inflammation erodes, while early-life conditions having indirectly shaped inflammatory risk through their impact on adult SES. Conversely, our metabolic risk findings showed that early SES synergizes with adult SES in its association with metabolic functioning, suggesting the accumulation of risk process that is largely continuous throughout adulthood. Though we find statistical evidence of mediation for both outcomes, results from the inflammation analyses, in particular, revealed that adult SES mediates a substantial part of the association of early-life SES with later-life health.

Our findings reveal how early-life SES relates to later-life physiological function through its connection to the individual components of adult SES. In general, early-life SES to adult educational attainment seems to be the most important SES pathway associated with adult health across all stages of adulthood. The findings consistently show that adult education partially mediates the early-life SES effects for both inflammation and metabolic health outcomes from young to mid to older adulthood. We also find that of all the adult SES dimensions, education matters the most for health. The finding that wealth mediates any remaining direct impact of early-life SES reflects the shifting importance of distinct and different components of SES across the life course and into older adulthood. Our results show that the human capital that evolves from educational attainment is most critical for health early in adulthood, and that this human capital translates to income and wealth gains throughout adulthood. The successful accumulation of wealth provides added protection from health risks in late life. These findings are consistent with previous studies (Boen & Yang, 2016; McDade, Lindau, & Wroblewski, 2011; Robert & House, 1996) showing the critical importance of wealth in shaping later-life trajectories of health.

We further find that key psychosocial and health behavioral factors that have been previously linked to immune and metabolic health outcomes play different roles in explaining the SES–biomarker associations. The covariates jointly account for the residual associations between early SES and biological outcomes, with BMI being the most prominent bio-behavioral mechanism in young adulthood. No covariate individually or jointly mediates the adult SES–biomarker associations in middle and older adulthood, however. This lack of mediation in mid- and late adulthood could be interpreted one of two ways. First, while we have included major psychosocial stressors and bio-behavioral risk factors common to all datasets to facilitate cross-study comparison, there may be other relevant factors stratified by SES that we have not been able to include, such as health care access, neighborhood exposures, and cognitive and noncognitive skills. On the other hand, the persistent SES–biomarker associations we found here align with recent research that shows a lack of mediation of the effects of wealth change on systolic blood pressure in mid to old age by similar bio-behavioral risk factors (Boen & Yang, 2016). In this case, socioeconomic disadvantage may act as a direct stressor that serves to induce changes in physiological function, rather than an indirect stressor that operates through bio-behavioral, emotional, and other psychosocial factors.

Our extensive life-course study design offers clear advantages in its ability to document various temporal processes leading from social status to biological health compared with previous research. Findings from this study indicate that intervention programs aimed at minimizing socioeconomic disparities in health should focus on educational attainment particularly in young adulthood, but income and assets among middle-aged and older adults. For example, policies that enhance human capital development during the transition to young adulthood through job training and career counseling would be effective in reducing health disparities early in adulthood; whereas savings, wealth, and retirement policies would be more effective combatting later-life health inequites. We have thus provided an initial framework for future analysis to incorporate additional data sets and explore other life-course processes and bio-behavioral and psychosocial mechanisms that might further explain some of the observed linkages between life-course SES and health.

Limitations

Despite these advances, we acknowledge a number of study limitations. First, while our analyses assessed prospective associations between early-life SES and later life health measures using the longitudinal data, we lack multiple repeated measures of biomarkers and SES within each study, particularly during early life (beyond childhood and adolescence), to allow for a closer tracking of how individuals' SES trajectories across time relate to subsequent changes in their physiological indicators. In additional analysis where we used two waves of biomarker measures in the HRS to estimate longitudinal residual change models, we found consistent results that strengthen the current findings. Given the current lack of comprehensive longitudinal biomarker data in national studies, results from our harmonized study

design provide initial evidence of the varying associations between diverse measures of SES and essential markers of physiological functioning. As more longitudinal data become available, future research will be able to build on the results presented here to further test for causal associations between the SES indicators and markers of health.

Second, our study assessed the prospective associations between SES pathways and biological risks using independent samples rather than one sample that combines all observations to allow for the direct test of such associations and their age variation. At present, this study provides qualitative comparisons of associations across life stages and preliminary evidence to be corroborated by additional studies to examine how changing socioeconomic conditions relate to trajectories of physiological risk over time. Third, mortality selection may be occurring with older or frailer adults. Our results may under-estimate the full extent to which early life and adult SES influence physiological functioning in older adulthood because the most deprived adults experience earlier mortality, and would therefore not be included in our old age sample, a limitation common to aging studies.

Fourth, our analyses of metabolic disorder show modest associations of adult SES with biomarker outcomes in middle adulthood compared with the other stages of the life course. This may be due to both methodological issues and alternative substantive reasons. The biomarker sample in MIDUS is homogeneous in race and SES, with the majority of sample members being white and of relatively high SES, therefore truncating the variation in SES over the life course. To test whether sample compositions affected the findings, we ran supplementary analyses restricting the Add Health and HRS samples to similarly high SES respondents. The supplementary analysis produce results substantively similar to the full sample findings, with some loss of statistical significance, likely due to decreased sample size. Alternatively, SES may be less related to metabolic disorder in middle adulthood because of employment-based health insurance or routine health screening prevalent for this life period.

Implications

Findings from our study provide new insights into when and how SES matters for underlying biophysiological functioning integral to the aging process across the adult life span. Our findings from young adulthood clearly suggest the importance of addressing early life disadvantage because it influences health risk both directly and indirectly, though its associations with life-course trajectories of SES. Policies and interventions designed to enhance the education, training, income, job skills, and job security of parents in low-SES families will directly improve the health of their children as they enter adulthood and set them on paths that will continue to benefit their health and socioeconomic attainment later in life. Moreover, the association between SES and health risks early in adulthood suggests interventions to improve the development of socioeconomic resources in young adulthood would be especially effective in reducing health risks and permanent biological damage before disease manifests later in life. The inclusion of midlife in linking early life conditions with later life health highlights its role as a transition period that bridges young and older adulthood. Later in the life course, policies and interventions aimed at improving socioeconomic conditions during old age and retirement would improve health independent of earlier disadvantage.

Supplementary Material

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

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Conflict of Interest

The researchers have no conflicts of interest to report. The design and interpretation of the analysis conducted by the authors were not directly influenced by any of the funding sources.

Author Contributions

Y. C. Yang, K. Schorpp, C. Boen, M. Johnson, and K. M. Harris contributed to this study. Y. C. Yang and K. M. Harris led the conceptual design of the study and Y. C. Yang led the analytic design, drafted, and revised the article. K. M. Schorpp conducted statistical analyses and created tables and figures for the HRS and MIDUS samples. M. P. Johnson conducted statistical analyses and created tables and figures for the Add Health sample. All authors contributed to the study design, write-up of the manuscript, and the revision process.

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