BIODEMOGRAPHY AND SOCIAL BIOLOGY
2017, VOL. 63, NO. 2, 87–103
http://dx.doi.org/10.1080/19485565.2017.1279536

Routledge
Taylor & Francis Group

Early-Life Socioeconomic Status and Adult Physiological Functioning: A Life Course Examination of Biosocial Mechanisms

Yang Claire Yang[a,b], Karen Gerken [a], Kristen Schorpp[a], Courtney Boen[a], and Kathleen Mullan Harris[a]

[a]Department of Sociology, and Carolina Population Center, University of North Carolina at Chapel Hill, Chapel Hill, North Carolina, USA; [b]Lineberger Comprehensive Cancer Center, University of North Carolina at Chapel Hill, Chapel Hill, North Carolina, USA

ABSTRACT

A growing literature has demonstrated a link between early-life socioeconomic conditions and adult health at a singular point in life. No research exists, however, that specifies the life course patterns of socioeconomic status (SES) in relation to the underlying biological processes that determine health. Using an innovative life course research design consisting of four nationally representative longitudinal datasets that collectively cover the human life span from early adolescence to old age (Add Health, MIDUS, NSHAP, and HRS), we address this scientific gap and assess how SES pathways from childhood into adulthood are associated with biophysiological outcomes in different adult life stages. For each dataset, we constructed standardized composite measures of early-life SES and adult SES and harmonized biophysiological measurements of immune and metabolic functioning. We found that the relative importance of early-life SES and adult SES varied across young, mid, and late adulthood, such that early-life SES sets a life course trajectory of socioeconomic well-being and operates through adult SES to influence health as adults age. We also documented evidence of the detrimental health effects of downward mobility and persistent socioeconomic disadvantage. These findings are the first to specify the life course patterns of SES that matter for underlying biophysiological functioning in different stages of adulthood. The study thus contributes new knowledge critical for improving population health by identifying the particular points in the life course at which interventions might be most effective in preventing disease and premature mortality.

Introduction

The impacts of social status on physiological, cellular, and molecular processes have been widely documented across species from rodents to nonhuman primates (McClintock et al. 2005; Sapolsky 2005; Tung et al. 2012). For humans, a large body of work indicates the detrimental consequences of adverse social conditions and social stress for disease susceptibility and survival. Socioeconomic status (SES) is among the most important determinants of the quality of the social environment that strongly condition one's exposures to

CONTACT Yang Claire Yang yangy@unc.edu

Color versions of one or more of the figures in the article can be found online at www.tandfonline.com/HSBI.

chronic stress and other risk factors for health (Link and Phelan 1995; Pearlin et al. 1981). Research across disciplines consistently documents an SES gradient in health, with individuals of lower SES having higher rates of disability, disease, and death than higher SES individuals (Adler and Ostrove 1999; Marmot 2006). The ubiquity of SES influences on morbidity and mortality brings into sharp focus the need to reduce status-based health disparities.

SES-related disadvantage is not an isolated point in time experience. It reaches across time in individuals' lives. There has been growing interest in examining the early-life origins of SES disparities in health using a life course perspective (Gluckman and Hanson 2004). A dominant paradigm to explain the early- and later-life connection is the *sensitive period model*, which posits that exposures during sensitive periods of development (e.g., gestation, birth, childhood, and adolescence) induce enduring structural and functional changes in organisms through biological programming that are difficult to reverse and, in turn, affect later disease risk (Barker 1998; Ben-Shlomo and Kuh 2002; Guo and Harris 2000). Such a model suggests that early-life conditions have stronger effects on individual outcomes than conditions experienced at subsequent time points. Empirical evidence would support this model if low early-life SES increases the risk of disease in later life, independent of adult SES and other risk factors.

Although the sensitive period model has gained wide recognition and stimulated much research, the causal relationship it implies (i.e., early life exerts direct and permanent impacts on later-life adult health) has never been properly tested or firmly established. Critical gaps remain in our knowledge about mechanisms underlying the early- and later-life links that are critical for understanding the pathogenesis of diseases of aging. First, it is not clear that the sensitive period model accounts for any observed associations between early SES exposures and later health because most studies have been heavily biased towards examining *only* early-life periods without consideration of social conditions during the intervening adult years between early- and late-life periods. Second, most studies rely on single indicators of SES (typically occupation) that fail to capture the complex and multidimensional nature of socioeconomic well-being and its potentially varying health impacts across the life course. Third, studies of associations between life course SES and later life morbidity and mortality risks lack examination of underlying biological mechanisms. While evidence has emerged on links between social disadvantage and markers of physiological stress response such as immune compromise (McDade, Lindau, and Wroblewski 2011; Yang, McClintock, et al. 2013) and metabolic dysregulation (Sweet et al. 2013; Yang, Li, and Ji 2013), few extant studies move beyond static snapshots of biomarkers in relation to SES at a point in time and cannot inform the dynamics of their interplay as individuals age. The temporal properties as well as specificity of biological processes involved in the multidimensional SES-health link are important for identifying points of intervention but are largely unknown.

This study addresses these fundamental deficits in past research and provides new data on how early-life SES is related to later-life health. We move beyond the notion of early-life sensitive periods and towards a more comprehensive and integrative framework that considers alternative models and biosocial mechanisms that operate across the human life span.

Besides the sensitive period model, three additional conceptual models have been proposed to explain the early SES and later health link. The *accumulation of risks model* holds that deleterious prior exposures associated with low SES accumulate to compound

the adverse effects over time (Ferraro and Shippee 2009; O'Rand 2009; Willson, Shuey, and Elder 2007). Statistical evidence for this model is based on the additive effects of early- and later-life SES factors (Hallqvist et al. 2004). The *pathway model* posits that childhood circumstances affect adult health risk indirectly, such that early experiences set individuals on divergent socioeconomic trajectories that differentially expose them to stressors that impinge on biological functioning and health (Hayward and Gorman 2004; Marmot et al. 2001; Pudrovska and Anikputa 2014). Statistically, this model predicts that the effects of early-life adversity on later-life disease risk is mediated or transmitted through SES at each subsequent life stage. Lastly, the *social mobility model* posits that movement across levels of SES may affect disease risk (Hallqvist et al. 2004; Luo and Waite 2005). This model predicts that the health effects of early-life exposures can be modified by later-life SES such that upward mobility may mitigate negative impacts of early-life adversity, while downward mobility from prosperity to hardship can be a perniciously stressful experience.

These models are often positioned as conceptually distinct, but research documents that the life course processes underlying these models are likely to be complementary rather than mutually exclusive (Hallqvist et al. 2004). Extant studies show mixed support for different conceptual models and highlight the complexity of interrelated life-course processes (Luo and Waite 2005; Lynch et al. 1994; Lynch, Kaplan, and Shema 1997; Osler et al. 2003; Power, Manor, and Matthews 1999). Because prior studies often use a cross-sectional design in which early-life SES (typically reported retrospectively) is related to adult health at a point in time, the timing and pattern of SES across the life course in relation to underlying biological mechanisms that determine health cannot be examined. As a result, previous research cannot adjudicate among different models or explicate their interrelations, and our current understanding of the early-life impact on adult health remains largely speculative. It is unknown whether early or contemporaneous SES is more important or whether there are windows of particular vulnerability throughout life. Studies have generally failed to consider simultaneously the length of exposures to social conditions, timing of the manifestation of their health impact, and change in social exposures in relation to disease risk measured in multiple life stages as aging progresses. There has been no research we are aware of that offers a robust test of the early- and later-life link by accounting for all these problems.

This study breaks new ground by examining the SES-health linkages as they unfold across the life span from young adulthood, to midlife, to old age. Combining four nationally representative longitudinal data sets, we assess the extent to which the associations between early and adult SES and biomarkers of inflammation and metabolic syndrome are consistent with the sensitive period, accumulation of risks, pathway, and/or social mobility models at various stages in the life course. We make several unique contributions to research on social disparities in health. First, the use of an innovative longitudinal research design consisting of multiple large, diverse, population-based samples (Yang et al. 2016) enables us to empirically examine early-life SES health impacts over a far more extensive period of life than any single dataset can allow. The comprehensive longitudinal analyses help to better adjudicate among several life course models. Second, this study utilizes multidimensional measures of SES that are harmonized across data sources to provide a more complete view of socioeconomic impacts on health. These measures include 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. Third, we use objectively measured biomarkers of immune and metabolic functions that are fundamental in physiological regulation underlying the aging processes leading to morbidity and mortality (Finch 2010). This study thus provides a mechanistic view of how early life conditions "get under the skin" to influence disease susceptibility over time. Finally, our study design provides new knowledge about whether and how biological pathways linking SES and disease phenotypes may be specific to each life course stage.

Data and Methods

Data for this study come from four nationally representative U.S. National Insitutes of Health studies that collectively span multiple stages of the life course. Data for young adulthood come from 12,237 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 ages 24–32 in Wave IV (2008–2009). Data for mid adulthood come from 908 respondents aged 25–74 in the National Survey of 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 10,165 participants aged 50 and older in the Health and Retirement Study (HRS) at baseline in 1998 and followed every two years through 2006, as well as 1,026 respondents aged 57–85 in the National Social Life, Health, and Aging Project (NSHAP) at Wave I (2005–2006) and followed up at Wave II (2010–2011). More information about the studies used in this analysis can be found in the Appendix.

Outcome measures represent two key biological pathways underlying the physiological stress process (Finch 2010). For Add Health, HRS, and NSHAP, immune function was measured using C-reactive protein (CRP), an acute phase protein whose elevation in circulating level indicates *systemic inflammation*. Additional measures of immune function were available in MIDUS, including fibrinogen, interleukin 6 (IL-6), E-selectin, and intracellular adhesion molecule 1 (ICAM-1), and were used to create a composite score of *inflammation burden* (Yang, Schorpp, and Harris 2014). The cut points for CRP reflect clinical reference ranges for health risk, while the top quartile was used as a cut point for the other markers. Metabolic function was assessed by a composite measure that includes seven biomarkers used in the clinical definition of *metabolic syndrome*: diastolic blood pressure, systolic blood pressure, glycosylated hemoglobin (HbA_{1c}), waist circumference, high-density lipoprotein (HDL cholesterol), total cholesterol, and triglycerides. For each measure, the cut points for high risk were defined by clinical practice or defined empirically as the top or bottom decile (see Table 1 for cut points used for each data source). We constructed the index of metabolic syndrome as the sum of the positive indicators. Details of biomarker data collection and assays are provided in the Appendix.

For each data set, measures of both early-life and adult SES were used to examine life course patterns of SES. *Early-life SES* measures include parental education, household income, welfare receipt, and subjective financial well-being. Parental education was coded as 1 = less than high school, 2 = high school graduate, 3 = some college, and 4 = college graduate or more. For Add Health, MIDUS, and HRS, parent education was measured as the maximum of mother and father education in two-parent households. For NSHAP, mother and father education were incorporated as separate SES indicators. Parent-

Table 1. Sample characteristics: Weighted descriptive statistics.

	Young Adulthood	Young to Mid Adulthood	Late Adulthood	
	Add Health (Age 24–32)	MIDUS (Age 34–74)	HRS (Age 50–98)	NSHAP (Age 57–91)
Wave: Survey Year	IV: 2008–2009	II: 2004–2006	VIII/IX: 2006/2008	II: 2010–2011
Biomarkers				
Inflammation				
*C-Reactive Protein (CRP, mg/dL), Mean (*SD)	3.8 (5.2)		4.5 (8.3)	4.1 (8.4)
< 1 (normal)	32.7%		25.6%	23.1%
1–3 (low chronic inflammation)	29.2%		36.4%	39.7%
3–10 (high chronic inflammation)	29.2%		28.4%	29.1%
> 10 (very high inflammation)	9.0%		9.6%	8.1%
N	12,252		11,251	1,026
Inflammation burden (MIDUS only)				
0		38.3%		
1		30.1%		
2		16.8%		
3		9.6%		
4		3.8%		
5		1.3%		
N		856		
Metabolic syndrome				
0	25.5%	10.1%	1.0%	10.0%
1	34.8%	25.6%	8.3%	24.7%
2	24.9%	26.8%	18.2%	33.4%
3	11.1%	25.6%	25.1%	31.9%
4	3.4%	8.0%	26.9%	
5	0.3%	4.0%	20.5%	
N	12,133	852	10,793	988
Hypertension[a]	26.9%	49.7%	67.9%	70.7%
Abdominal obesity[b]	49.1%	50.9%	63.1%	59.3%
HbA_{1c}[c]	30.1%	72.3%	52.4%	57.2%
HDL cholesterol[d]	14.3%	19.7%	59.8%	
Total cholesterol[e]			88.0%	
Triglycerides[f]	10.2%	15.1%		
Socioeconomic Status				
Early-life SES (Mean [SD] composite of standardized SES items)	−0.003 (0.62)	−0.001 (0.57)	0.04 (0.55)	−0.05 (0.71)
Parent educational attainment				
Less than high school	12.1%	19.2%	45.2%	
High school graduate	32.3%	35.5%	33.2%	
Some college	21.8%	17.9%	9.47%	
College graduate or more	33.8%	27.4%	12.1%	
Mother's educational attainment				
Less than high school				44.3%
High school graduate				35.5%
Some college				10.9%
College graduate or more				9.3%
Father's educational attainment				
Less than high school				44.3%
High school graduate				28.3%
Some college				13.6%
College graduate or more				13.9%
Parent socioeconomic index	46.3 (47.1)	73.2 (29.7)		
Ever received welfare in childhood	17.3%	5.1%		
Rate childhood SES				
Poor			31.6%	41.2%
Average			61.9%	46.8%
Pretty well off			6.5%	12.0%
Father unemployed			21.5%	
Adult SES (Mean [SD] composite of standardized SES items)	−0.003 (0.52)	0.003 (0.53)	0.07 (0.72)	0.15 (0.80)

(*Continued*)

Table 1. (Continued).

	Young Adulthood	Young to Mid Adulthood	Late Adulthood	
	Add Health (Age 24–32)	MIDUS (Age 34–74)	HRS (Age 50–98)	NSHAP (Age 57–91)
Wave: Survey Year	IV: 2008–2009	II: 2004–2006	VIII/IX: 2006/2008	II: 2010–2011
Educational attainment				
Less than high school	8.4%	3.1%	16.5%	8.0%
High school graduate	17.7%	23.5%	53.1%	56.4%
Some college	43.7%	28.4%	5.4%	
College graduate or more	30.1%	45.0%	24.9%	35.6%
Household income (thousands)	63.8 (44.4)	84.2 (61.7)	71.42 (106.64)	64.7 (88.6)
Ever received welfare in adulthood	23.6%	10.2%		
Household assets (thousands)			439.5 (1,003.3)	815.3 (2,082.9)
Covariates				
Age	28.3 (1.9)	44.2 (10.0)	63.2 (9.5)	66.6 (7.0)
Female	51.1%	55.4%	53.9%	48.8%
Race/ethnicity				
White	68.6%	93.6%	87.2%	85.7%
Black	15.0%	2.9%	8.2%	6.4%
Hispanic	11.9%	3.5%	4.6%	5.3%
Other	4.5%			2.6%
Marital status	50.2%		67.3%	65.9%
Self-rated childhood health	3.9 (0.9)		4.5 (0.8)	4.1 (0.9)
Social isolation	25.4%		0.3 (0.2)	17.4%
CES-D[g]	5.3 (4.2)		1.3 (1.8)	4.1 (3.9)
Ever cigarette smoker	25.3%		56.4%	59.5%
Physical activity	14.5%		1.0 (1.3)	69.0%
Obese	37.8%		39.4%	37.7%

Note. [a]Hypertension = 1 if systolic ≥ 140 mmHg or diastolic ≥ 9 0 mmHg or on antihypertensive medication, or ever diagnosed hypertensive.
[b]Abdominal obesity = 1 if waist circumference > 102 cm in males, > 88 cm in females.
[c]HbA_{1c} = 1 if glucose > 5.6 % or ever diagnosed diabetic.
[d]HDL cholesterol = 1 if ≤ 40 mg/dL or on cholesterol medication. For Add Health, = 1 if in the lowest decile for females or lowest two deciles for males.
[e]Total cholesterol = 1 if ≥ 180 mg/dL or on cholesterol medication.
[f]Triglycerides = 1 if ≥ 200 mg/dL. For Add Health, = 1 if in the highest decile.
[g]CES-D = Center for Epidemiologic Studies Depression Scale.

reported household income was included as an indicator of early-life SES for Add Health and was coded as a continuous measure. While household income in early life was not available for the other data sources, MIDUS included a continuous parent socioeconomic index (SEI) that was based on parent education and occupation, which we used as a proxy for household income. In addition, HRS includes an item that asked respondents if their father was unemployed for a period of several months or more when the respondent was under the age of 16, which was reverse coded to reflect father employment. Welfare receipt was used as an SES indicator for Add Health and MIDUS and was reverse coded to indicate higher SES. Finally, both HRS and NSHAP included a measure of perceived financial well-being, in which respondents were asked whether their family was "pretty well off," "about average," or "poor" when the respondent was before the age of 16. Items were recoded so higher values reflect higher SES.

Adult SES measures include respondent education, household income, welfare receipt, and household assets. Respondent education was included in all four data sources and was coded similarly to parent education, with 1 = less than high school, 2 = high school

graduate, 3 = some college, and 4 = college graduate or more. Household income was also included for all four data sources and was coded as a continuous measure. For Add Health and MIDUS, welfare receipt was used as an indicator of adult SES and was recoded to reflect higher SES. Finally, NSHAP and HRS included a continuous measure of total household assets, which is an especially important indicator of SES in late adulthood.

Previous research suggests that multivariate scales of SES are more reliable than single measures (Goosby and Walsemann 2012; Walsemann, Bell, and Goosby 2011) and reduce problems related to missing data on the SES variables. We constructed composite measures of early-life SES and adult SES that are composed of several indicators of SES available within each data source, resulting in eight composite SES scores (one early-life SES measure and one adult SES measure for all four data sources). For each composite SES score, three individual SES items were first standardized by calculating the z scores for each item. The mean of the three z scores was then calculated to produce a continuous composite SES score. Positive values for the SES scores indicate higher levels of SES. To maximize sample size, composite measures of early-life and adult SES were calculated for all respondents for whom there were data on at least two of the variables used in each scale.

In addition to continuous measures of SES, we also included categorical measures of SES, which indicate social status trajectories using the lowest quartile of each SES composite measure to indicate socioeconomic disadvantage (1 = disadvantaged, 0 = not disadvantaged). Based on the dichotomous SES measure, we constructed measures of change from early to current status that include four categories: persistent disadvantage (disadvantaged–disadvantaged), upward mobility (disadvantaged–not disadvantaged), downwardly mobile (not disadvantaged–disadvantaged), and never disadvantaged (not disadvantaged–not disadvantaged). We included a measure of childhood health in all models to control for health selection early in life. For all four data sources, childhood health was measured using a single item of self-rated health, with response categories of poor, fair, good, very good, and excellent. Information on Add Health childhood health was collected during Wave I of the study, when respondents were adolescents. Childhood health for MIDUS, NSHAP, and HRS was retrospective. Childhood health was operationalized as continuous, with higher values indicating better health. Descriptive statistics on the outcome, SES measures, childhood health, and covariates used in analyses for each of the four data sets are shown in Table 1.

For each outcome, we conducted two sets of analyses. First, we examined the associations between early-life SES, adult SES, and biomarkers assessed at follow-up using ordinal logit models for both inflammation and metabolic syndrome. We estimated models in a stepwise fashion: (1) Model I adjusts for early-life SES, (2) Model II adjusts for adult SES, and (3) Model III simultaneously adjusts for both early-life and adult SES. We controlled for childhood health in all models to account for potential selection of individuals of varying health status early in life into SES classes in adulthood. Second, to assess the utility of the social mobility model, we modeled the biomarker outcomes as a function of changes across SES categories or movement into and out of SES disadvantage. All models adjust for survey design effects and nonresponse using sampling weights and control for age, sex, race, and marital status. Analyses were conducted in Stata 13.

Results

We found strong associations of SES measures with both indices 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 models. As seen in Model I in Table 2, higher early-life SES is associated with a 15% lower odds of elevated CRP (OR = 0.84, CI = [0.78, 0.91]) and a 20% lower odds of metabolic syndrome (OR = 0.80, CI = [0.74, 0.87]) in the Add Health study. These associations are highly significant and were replicated in the MIDUS, NSHAP, and HRS samples with similar magnitude. In Model II, adult SES showed inverse and significant associations with both physiological outcomes across all life stages, with the effect sizes for inflammation increasing from young to old age.

The simultaneous adjustment for both early-life and adult SES in Model III yields different results across life stage samples. In young and mid adulthood (Add Health and MIDUS), both early-life and adult SES had significant negative associations with CRP/inflammation burden and metabolic syndrome, suggesting an accumulation of risks model whereby health risks or benefits associated with SES accumulate over the lifetime to additively affect physiological functioning. In late adulthood (NSHAP and HRS), current SES completely mediated the associations between early-life SES and both biomarker outcomes, supporting a pathway model whereby the influence of childhood SES on adult physiology operates through adult SES. Supplementary analyses utilizing structural equation models (SEM) were consistent with the results of the regression analyses presented here and offered additional evidence that adult SES fully mediated the association between early-life SES and the biomarkers in the NSHAP and HRS samples (results available upon request). Results from the SEM models showed that, in both older adult samples, early-life SES indirectly affected the biomarker outcomes through its impact on adult SES. We note that the results using the NSHAP study and the HRS are highly consistent, which shows the robustness of the strong and dominant effects of adult SES in late life. Childhood general health status, while significant in predicting biomarkers in young adulthood, is not consistently associated with biomarkers in other later periods of life. The adjustment of childhood health did not affect the SES-biomarker associations assessed at any point in life. It is thus unlikely that health selection explains any of the above findings.

Analysis of SES change over time provides tentative evidence for the social mobility model across life stages. Figure 1 summarizes the results regarding the associations between different mobility categories and biomarker outcomes. Downward mobility was associated with lower risk of metabolic syndrome in young and middle adulthood than was persistent disadvantage. In young adulthood, for example, we find a 25% lower risk of metabolic syndrome for downwardly mobile individuals, compared to individuals in persistent disadvantage. Upward mobility was also associated with lower risk of inflammation or metabolic syndrome than persistent disadvantage, except in late adulthood. For example, in midlife, upward mobility was associated with a 39% lower risk of metabolic syndrome. The suggestion of adverse health effects associated with upward mobility documented in late adulthood has emerged in other recent research (Miller et al. 2015). We note, however, the never disadvantaged had lower health risks than all mobility

Table 2. Associations of early-life and current SES with markers of physical health across the life span, odds ratios (95% confidence intervals).

	Young Adulthood (Add Health)			Middle Adulthood (MIDUS)			Old Age (NSHAP)			Old Age (HRS)		
Model	I	II	III	I	II	III	I	II	III	I	II	III
Inflammation												
Early-life SES	0.84***		0.86***	0.71**		0.78*	0.83*		0.88	0.86***		0.94
	(0.78, 0.91)		(0.79, 0.93)	(0.58, 0.88)		(0.63, 0.97)	(0.70, 0.98)		(0.75, 1.04)	(0.80, 0.93)		(0.87, 1.02)
Current SES		0.87**	0.90*		0.68***	0.72**		0.77**	0.80**		0.69***	0.70***
		(0.79, 0.96)	(0.82, 0.99)		(0.56, 0.82)	(0.58, 0.88)		(0.66, 0.89)	(0.68, 0.93)		(0.63, 0.76)	(0.64, 0.77)
N	12,237	12,237	12,237	856	856	856	1,026	1,026	1,026	10165	10165	10165
Metabolic syndrome												
Early life SES	0.80***		0.83***	0.68***		0.74**	0.80*		0.88	0.87***		0.94
	(0.74, 0.87)		(0.77, 0.91)	(0.56, 0.84)		(0.60, 0.91)	(0.66, 0.97)		(0.70, 1.09)	(0.81, 0.94)		(0.86, 1.01)
Current SES		0.78***	0.80***		0.71***	0.76**		0.71*	0.73*		0.78***	0.79***
		(0.71, 0.85)	(0.73, 0.88)		(0.59, 0.86)	(0.63, 0.93)		(0.54, 0.92)	(0.54, 0.99)		(0.71, 0.84)	(0.72, 0.86)
N	12,133	12,133	12,133	852	852	852	988	988	988	9412	9412	9412

Note. All models adjusted for age, sex, race, marital status, and self-rated health in childhood.
***$p < 0.001$; **$p < 0.01$; *$p < 0.05$.

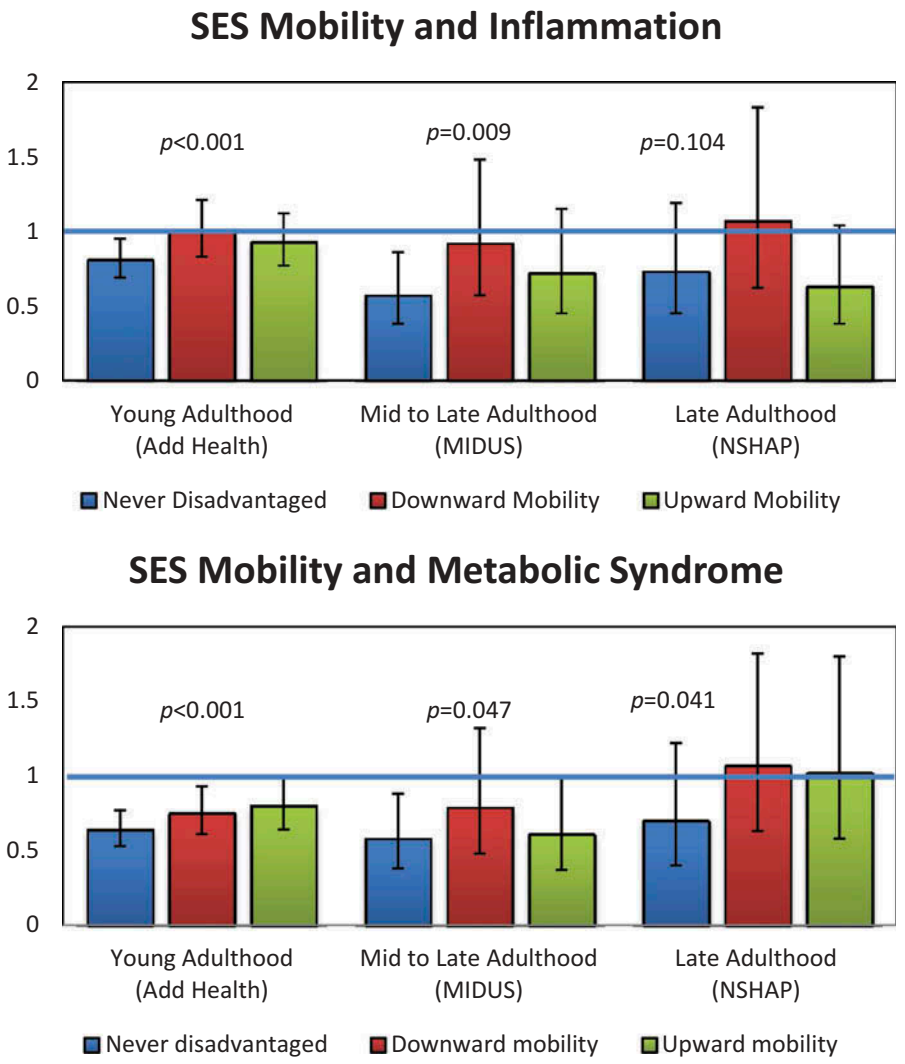

Figure 1. Associations of socioeconomic mobility with markers of physical health. *p* values indicate overall significance of SES mobility variables within each data set. Reference category is persistent disadvantage in early life and adulthood.

categories compared. Taken together, we find that SES disadvantage at any point in life put individuals at higher physiological risks for health.

Discussion

Our study contributes new knowledge on the complex temporal dynamics through which SES impacts physiological indicators of health across the life span. Our results demonstrate how SES operates through several life course and biological mechanisms to affect

health, and how these mechanisms evolve across young, mid, and late adulthood. In particular, we find that the direct effect of early-life SES on physical health wanes as individuals age, though its indirect effect remains important through its influence on the SES trajectory into adulthood.

In early and middle adulthood, we find evidence of an accumulation model, as there are additive and independent associations of both early-life SES and young adult SES with health risks. In old age, early-life SES operates through adult SES in its association with health risks later in the life course, which supports the pathway model. Early-life SES remains salient for health in young to mid adulthood, over and above current adult SES, perhaps because the resources and support that early-life SES provides continue to play an independent role in adult lifestyles and access to health care. Once adults reach old age, the SES they have established as older adults has been fairly stable over many years and early-life SES is important only through its linkage to this longer-term SES. We also find that social mobility matters across the life course. Interestingly, mobility effects do not exist independently from one's location on the SES hierarchy. Regardless of the direction of the movement over time from childhood to adulthood, being in the lowest SES quartile at any point is detrimental to physical health.

Our extensive longitudinal life course study design has offered clear advantages in its ability to adjudicate among different life course models, in contrast to previous research. By examining whether and how early-life SES continues to matter in the presence of adult SES across multiple stages of the adult life course, we are less likely to overlook patterns of associations in the data than previous studies, which evaluated these associations in only one particular period of the life course. Findings that have emerged from this study indicate temporal specificity of exposures to social adversity that can better inform disease intervention and control strategies aimed at minimizing socioeconomic disparities in health. We have thus provided an initial framework for future analysis to incorporate additional datasets and explore the behavioral and psychosocial mechanisms that might explain some of the observed linkages between life course SES and health.

Despite these advances, we acknowledge our study has a number of limitations. First, we lack multiple repeated measures of biomarkers and SES across the early life course, beyond childhood and adolescence, to allow for a closer tracking of SES trajectories across time in relation to subsequent changes in physical indicators. In additional NSHAP analysis where we used two waves of biomarker measures to estimate longitudinal residual change models, we did not find early-life SES to be significantly associated with change in biomarkers over a five-year period in late life. While this is consistent with and strengthens the current finding, it is not known whether the association under question varies by interval length of biomarker measurements within and across life course stages. Second, we cannot exclude the possibility that mortality selection may be occurring with older or frailer adults. The results are thus likely conservative regarding the extent to which early-life and adult SES influence physiological functioning in the older adult population in that those most deprived of SES-related resources earlier in life for whom the SES-biomarker associations tend to be strongest were less likely to have survived to be observed in old age. Third, while the results presented here offer preliminary evidence of the social mobility model, more research incorporating multiple repeated measures of SES is needed. For each of the samples included in the analysis, we have measures of SES at two time points, which limits our ability to fully understand how changes in SES across the life course affect health trajectories, especially among the older aged

cohorts for whom childhood occurred 40–60 years ago. Though this study provides initial evidence of an association between social mobility and health risk, more research incorporating longitudinal SES data is needed.

Nonetheless, we provide new scientific data on when and how SES matters for underlying biophysiological functioning across the human life span, which has important implications for interventions to improve population health. It is clear that early-life SES matters for later life health. This finding suggests that policies and interventions aimed at improving the socioeconomic conditions of children in disadvantaged families will reduce SES disparities in health in all stages of the life span—from early adulthood through middle and late adulthood. 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 an SES trajectory that will continue to benefit their health into old age. Our findings also suggest directions for further research on SES and health. More in-depth study of the timing and patterns of SES across the life course and specific biological response to SES-related stress are needed to better understand the lifetime health impacts of SES. The varying life stage relationships between SES and physical health that our study has shown invites follow-up studies to provide deeper mechanistic explanations for the life course development of social disparities in health.

Acknowledgments

This research uses data from Add Health, a program project directed by Kathleen Mullan Harris and designed by J. Richard Udry, Peter S. Bearman, and Kathleen Mullan Harris at the University of North Carolina at Chapel Hill, and funded by grant P01-HD31921 from the Eunice Kennedy Shriver National Institute of Child Health and Human Development, with cooperative funding from 23 other federal agencies and foundations. We are grateful to our colleagues, especially Robert Hummer, for helpful suggestions and comments.

Funding

This research is supported by the National Institute of Aging grant K01AG036745 and University Cancer Research Funds at the Lineberger Cancer Center (to the first author), and by the Eunice Kennedy Shriver National Institute of Child Health and Human Development grant P01HD31921 (to the last author). We are grateful for training support (T32 HD007168) and for general research support (R24 HD050924) from the Carolina Population Center, University of North Carolina at Chapel Hill.

ORCID

Karen Gerken http://orcid.org/0000-0002-3612-3278

References

Adler, N. E., and J. M. Ostrove. 1999. Socioeconomic status and health: What we know and what we don't. *Annals of the New York Academy of Sciences* 896:3–15. doi:10.1111/nyas.1999.896.issue-1.

Barker, D. J. 1998. In utero programming of chronic disease. *Clinical Science* 95 (2):115–28. doi:10.1042/cs0950115.

Ben-Shlomo, Y., and D. Kuh. 2002. A life course approach to chronic disease epidemiology: Conceptual models, empirical challenges and interdisciplinary perspectives. *International Journal of Epidemiology* 31 (2):285–93. doi:10.1093/ije/31.2.285.

Brim, O. G., C. D. Ryff, and R. C. Kessler. 2004. *How healthy are we?: A national study of well-being at midlife*. Chicago: University of Chicago Press.

Crimmins, E., J. Faul, J. K. Kim, H. Guyer, K. Langa, M. B. Ofstedal, A. Sonnega, R. Wallace, and D. Weir. 2013. *Documentation of biomarkers in the 2006 and 2008 Health and Retirement Study*. Ann Arbor, MI: Survey Reseach Center, University of Michigan. http://hrsonline.isr.umich.edu/sitedocs/userg/Biomarker2006and2008.pdf.

Dienberg Love, G., T. E. Seeman, M. Weinstein, and C. D. Ryff. 2010. Bioindicators in the MIDUS national study: Protocol, measures, sample, and comparative context. *Journal of Aging and Health* 22 (8):1059–80. doi:10.1177/0898264310374355.

Entzel, P., E. A. Whitsel, A. Richardson, J. Tabor, S. Hallquist, J. Hussey, C. T, and K. M. Harris. 2009. *Add Health Wave IV documentation: Cardiovascular and anthropometric measures*. Chapel Hill, NC: Carolina Population Center, University of North Carolina at Chapel Hill. http://www.cpc.unc.edu/projects/addhealth/data/guides/Wave%20IV%20cardiovascular%20and%20anthropometric%20documentation%20110209.pdf.

Ferraro, K. F., and T. P. Shippee. 2009. Aging and cumulative inequality: How does inequality get under the skin? *Gerontologist* 49 (3):333–43. doi:10.1093/geront/gnp034.

Finch, C. E. 2010. *The biology of human longevity: Inflammation, nutrition, and aging in the evolution of lifespans*. Burlington, MA: Academic Press.

Gluckman, P. D., and M. A. Hanson. 2004. Living with the past: Evolution, development, and patterns of disease. *Science* 305 (5691):1733–36. doi:10.1126/science.1095292.

Goosby, B. J., and K. M. Walsemann. 2012. School racial composition and race/ethnic differences in early adulthood health. *Health & Place* 18 (2):296–304. doi:10.1016/j.healthplace.2011.10.002.

Guo, G., and K. M. Harris. 2000. The mechanisms mediating the effects of poverty on children's intellectual development. *Demography* 37 (4):431–47. doi:10.1353/dem.2000.0005.

Hallqvist, J., J. Lynch, M. Bartley, T. Lang, and D. Blane. 2004. Can we disentangle life course processes of accumulation, critical period and social mobility? An analysis of disadvantaged socio-economic positions and myocardial infarction in the Stockholm Heart Epidemiology Program. *Social Science & Medicine* 58 (8):1555–62. doi:10.1016/s0277-9536(03)00344-7.

Harris, K. M. 2010. An integrative approach to health. *Demography* 47 (1):1–22.

Hayward, M. D., and B. K. Gorman. 2004. The long arm of childhood: The influence of early-life social conditions on men's mortality. *Demography* 41 (1):87–107.

Heeringa, S. G., and J. H. Connor. 1995. *Technical description of the Health and Retirement Study survey sample design*. Ann Arbor, MI: Institute for Social Research, University of Michigan. http://hrsonline.isr.umich.edu/sitedocs/userg/HRSSAMP.pdf.

Link, B. G., and J. Phelan. 1995. Social conditions as fundamental causes of disease. *Journal of Health and Social Behavior* (Spec No):80–94.

Luo, Y., and L. J. Waite. 2005. The impact of childhood and adult SES on physical, mental, and cognitive well-being in later life. *Journals of Gerontology. Series B, Psychological Sciences and Social Sciences* 60 (2):S93–S101.

Lynch, J. W., G. A. Kaplan, R. D. Cohen, J. Kauhanen, T. W. Wilson, N. L. Smith, and J. T. Salonen. 1994. Childhood and adult socioeconomic status as predictors of mortality in Finland. *Lancet* 343 (8896):524–27.

Lynch, J. W., G. A. Kaplan, and S. J. Shema. 1997. Cumulative impact of sustained economic hardship on physical, cognitive, psychological, and social functioning. *New England Journal of Medicine* 337 (26):1889–95. doi:10.1056/nejm199712253372606.

Marmot, M., M. Shipley, E. Brunner, and H. Hemingway. 2001. Relative contribution of early life and adult socioeconomic factors to adult morbidity in the Whitehall II study. *Journal of Epidemiology and Community Health* 55 (5):301–07.

Marmot, M. G. 2006. Status syndrome: A challenge to medicine. *JAMA* 295 (11):1304–07. doi:10.1001/jama.295.11.1304.

McClintock, M. K., S. D. Conzen, S. Gehlert, C. Masi, and F. Olopade. 2005. Mammary cancer and social interactions: Identifying multiple environments that regulate gene expression throughout the life span. *Journals of Gerontology. Series B, Psychological Sciences and Social Sciences* 60 (Spec No 1):32–41.

McDade, T. W., S. T. Lindau, and K. Wroblewski. 2011. Predictors of C-reactive protein in the National Social Life, Health, and Aging Project. *Journals of Gerontology. Series B, Psychological Sciences and Social Sciences* 66 (1):129–36. doi:10.1093/geronb/gbq008.

Miller, G. E., T. Yu, E. Chen, and G. H. Brody. 2015. Self-control forecasts better psychosocial outcomes but faster epigenetic aging in low-SES youth. *Proceedings of the National Academy of Sciences of the United States of America* 112 (33):10325–30. doi:10.1073/pnas.1505063112.

O'Muircheartaigh, C., S. Eckman, and S. Smith. 2009. Statistical design and estimation for the National Social Life, Health, and Aging Project. *Journals of Gerontology. Series B, Psychological Sciences and Social Sciences* 64 (Suppl 1):i12–9. doi:10.1093/geronb/gbp045.

O'Rand, A. M. 2009. Cumulative processes in the life course. In *The craft of life course research*, ed. G. H. Elder, Jr., and J. Z. Giele. New York: Guilford Press.

Osler, M., A. M. Andersen, P. Due, R. Lund, M. T. Damsgaard, and B. E. Holstein. 2003. Socioeconomic position in early life, birth weight, childhood cognitive function, and adult mortality. A longitudinal study of Danish men born in 1953. *Journal of Epidemiology and Community Health* 57 (9):681–86.

Pearlin, L. I., M. A. Lieberman, E. G. Menaghan, and J. T. Mullan. 1981. The stress process. *Journal of Health and Social Behavior* 22 (4):337–56.

Power, C., O. Manor, and S. Matthews. 1999. The duration and timing of exposure: Effects of socioeconomic environment on adult health. *American Journal of Public Health* 89 (7):1059–65.

Pudrovska, T., and B. Anikputa. 2014. Early-life socioeconomic status and mortality in later life: An integration of four life-course mechanisms. *Journals of Gerontology. Series B, Psychological Sciences and Social Sciences* 69 (3):451–60. doi:10.1093/geronb/gbt122.

Radler, B. T., and C. D. Ryff. 2010. Who participates? Accounting for longitudinal retention in the MIDUS national study of health and well-being. *Journal of Aging and Health* 22 (3):307–31. doi:10.1177/0898264309358617.

Sapolsky, R. M. 2005. The influence of social hierarchy on primate health. *Science* 308 (5722):648–52. doi:10.1126/science.1106477.

Sweet, E., A. Nandi, E. K. Adam, and T. W. McDade. 2013. The high price of debt: Household financial debt and its impact on mental and physical health. *Social Science & Medicine* 91:94–100. doi:10.1016/j.socscimed.2013.05.009.

Tung, J., L. B. Barreiro, Z. P. Johnson, K. D. Hansen, V. Michopoulos, D. Toufexis, K. Michelini, M. E. Wilson, and Y. Gilad. 2012. Social environment is associated with gene regulatory variation in the rhesus macaque immune system. *Proceedings of the National Academy of Sciences of the United States of America* 109 (17):6490–95. doi:10.1073/pnas.1202734109.

Walsemann, K. M., B. A. Bell, and B. J. Goosby. 2011. Effect of school racial composition on trajectories of depressive symptoms from adolescence through early adulthood. *Race and Social Problems* 3 (3):131–45.

Williams, S. R., and T. W. McDade. 2009. The use of dried blood spot sampling in the National Social Life, Health, and Aging Project. *Journals of Gerontology. Series B, Psychological Sciences and Social Sciences* 64 (Suppl 1):i131–6. doi:10.1093/geronb/gbn022.

Willson, A. E., K. M. Shuey, and G. H. Elder, Jr. 2007. Cumulative advantage processes as mechanisms of inequality in life course health. *American Journal of Sociology* 112 (6):1886–924.

Yang, Y. C., C. Boen, K. Gerken, T. Li, K. Schorpp, and K. M. Harris. 2016. Social relationships and physiological determinants of longevity across the human life span. *Proceedings of the National Academy of Sciences of the United States of America* 113 (3):578–83. doi:10.1073/pnas.1511085112.

Yang, Y. C., T. Li, and Y. Ji. 2013. Impact of social integration on metabolic functions: Evidence from a nationally representative longitudinal study of US older adults. *BMC Public Health* 13:1210. doi:10.1186/1471-2458-13-1210.

Yang, Y. C., M. K. McClintock, M. Kozloski, and T. Li. 2013. Social isolation and adult mortality: The role of chronic inflammation and sex differences. *Journal of Health and Social Behavior* 54 (2):183–203. doi:10.1177/0022146513485244.

Yang, Y. C., K. Schorpp, and K. M. Harris. 2014. Social support, social strain and inflammation: Evidence from a national longitudinal study of U.S. adults. *Social Science & Medicine* 107:124–35. doi:10.1016/j.socscimed.2014.02.013.

Appendix: Data Description

National Longitudinal Study of Adolescent Health (Add Health)

Add Health is a nationally representative study of over 20,000 adolescents in grades 7–12 the U.S. in 1994–1995 who were followed into adulthood. The scientific purpose of Add Health is to understand the social, behavioral, and biological linkages in health across the life course (Harris 2010). Add Health used a stratified school-based design and selected a nationally representative sample of all high schools and a feeder school in the U.S. An in-school questionnaire was administered to all students who attended the selected schools during 1994–1995 (Wave I). An in-home sample was then selected from the school rosters for more in-depth interviews in the home setting with adolescents and a parent at Wave I. The Add Health cohort was followed up in 1996 (Wave II), 2001–2002 (Wave III), and finally in 2008–2009 (Wave IV), when individuals were 24–32 years old. The independent variables and covariates for the present study are drawn from the in-home interviews at Waves I and IV. Biomarker data are drawn from the Wave IV interviews. High-sensitivity C-reactive protein (hsCRP), glycosylated hemoglobin (HbA_{1c}), triglycerides, and high-density lipoprotein cholesterol (HDL-C) data come from assay of dried blood spots. Field interviewers measured systolic and diastolic blood pressure, height, weight, and waist circumference for the respondents during the in-home interview. For more information about Add Health biomarker collection, see the protocol in Entzel et al. (2009).

We used a separate analytical sample for each biomarker outcome that included respondents for whom there were complete data on the variables used in the analysis and those with valid sampling weights, resulting in two subsamples: one for CRP (N = 12,237) and one for metabolic syndrome (N = 12,133). All analyses used survey weights to account for the unequal chances of selection, and error variances were adjusted for the clustered sampling design.

National Survey of the Midlife Development in the United States (MIDUS)

MIDUS is a national longitudinal study of behavioral, psychological, and social factors that contribute to age-related differences in overall health and well-being. A total of 7,108 participants aged 25–74 were recruited for the original study in 1995–1996 (Wave I) by random-digit dialing. Phone interviews and self-administered questionnaires were completed by MIDUS participants. The Wave II data were collected 9–10 years later (2004–2006), with a mortality-adjusted retention rate of 75% (Brim, Ryff, and Kessler 2004; Radler and Ryff 2010). The independent variables for this study were drawn from self-administered questionnaires from Wave I. Biological data came from the MIDUS II biomarkers study (2004–2009), which assessed key biological parameters indicative of physical health. Eligible participants lived in the continental U.S. and completed the

MIDUS II core phone interview and self-administered questionnaire. Fasting whole-blood samples were collected and serum aliquots were assayed for inflammatory markers (hsCRP, fibrinogen, interleukin-6, E-selectin, and intracellular adhesion molecule-1), as well as metabolic indicators (HbA_{1c}, HDL cholesterol, and triglycerides). Blood pressure and waist circumference were measured by MIDUS staff members during the physical exam. More detailed information about the MIDUS biospecimen collection and protocol can be found in Dienberg Love et al. (2010).

Analytic samples included respondents with complete data on the variables used in the analyses: inflammation (N = 856) and metabolic syndrome (N = 852). Survey weights are not available for the MIDUS II biomarkers study. To account for sampling of family members, we clustered the sample by family membership and reported robust standard errors.

Health and Retirement Study (HRS)

The Health and Retirement Survey (HRS) is a nationally representative longitudinal survey of the U.S. population aged 50 years and older conducted every two years from 1992 to 2008, using a multistage sampling of households design with an oversample of blacks and Hispanics and overall response rate of about 87% (Heeringa and Connor 1995). We used information from noninstitutionalized respondents included in the 2006 and 2008 waves who consented to the biomarker collection and completed the blood test. Respondents' blood pressure and waist circumference were measured by trained interviewers, and CRP, HbA_{1c}, HDL cholesterol, and total cholesterol were collected and assayed using dried blood spots. The physical and blood test consent procedure, laboratory measures, equipment, and protocols are described in Crimmins et al. (2013).

The final samples for the analyses included respondents with complete data on the variables used in the analyses: CRP (N = 10,165) and metabolic syndrome (N = 9,412). The 2006 or 2008 biomarker sample weights were implemented in the analyses (depending on the wave of biomarker collection for the respondent), which were supplemented by the core sample weights of the 2006 and 2008 surveys.

National Social Life, Health, and Aging Project (NSHAP)

The National Social Life, Health, and Aging Project (NSHAP) is a nationally representative longitudinal study of older adults years in 2005–2006 (Wave 1) and followed up in 2010–2011 (Wave 2). NSHAP Wave 1 used a national area probability sample of community-residing adults ages 57 to 85 at the time of the interview and oversampled African-American and Hispanic areas. The original Wave 1 sample included 3,005 respondents, and 2,261 of these Wave 1 respondents were re-interviewed at Wave 2. The overall response rate for Wave 1 was 75.5%, and the response rate for respondents who were re-interviewed at Wave 2 was 87.8%. More information about the survey design can be found in O'Muircheartaigh, Eckman, and Smith (2009). NSHAP data collection consisted of an in-person questionnaire, biomeasure collection, and a paper leave-behind questionnaire. The in-person questionnaire and biomeasures were administered by an NORC field

interviewer in the respondent's home. The independent variables and covariates for the study are drawn from the in-person and leave-behind questionnaires from Waves 1 and 2. Biomarker data are drawn from Wave 2 of the study. To assess levels of hsCRP and HbA_{1c}, NSHAP collected dried blood spots. Detailed information on the collection of dried blood spots by NSHAP can be found in Williams and McDade (2009). Respondents completed two or three seated blood pressure (BP) measures on their left arms. We used the mean of each respondent's BP readings. Finally, respondents were weighed, and their height and waist circumference were also measured by the field interviewers.

Each set of analyses used a different sample size, which included respondents with complete data on the variables used in the analysis: CRP (N = 1,026) and metabolic syndrome (N = 988). All analyses used survey weights to adjust for survey design and account for the probability of selection, with post-stratification adjustments for nonresponse based on age and urbanicity.