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

Yang Claire Yang*

Karen Gerken**

Kristen Schorpp**

Courtney Boen**

Kathleen Mullan Harris**

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*Department of Sociology, Lineberger Comprehensive Cancer Center, and Carolina Population Center, University of North Carolina at Chapel Hill.

**Department of Sociology and Carolina Population Center, University of North Carolina at Chapel Hill.

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Short Abstract (150 word limit)

A growing literature has demonstrated a linkage between early-life socioeconomic conditions and adult disease measured at one stage in the life course. Understanding this link at any point in time, however, demands comparisons across life stages to more clearly specify timing, duration, and the intermediate mechanisms of SES gradients. Using three national, longitudinal datasets (Add Health, MIDUS, NSHAP), we assess the extent to which the associations between SES and biophysiological outcomes are consistent with the sensitive period, accumulation of risks, pathway, and/or social mobility models at various stages in the life course. For each dataset, we constructed and standardized composite measures of early-life SES and adult SES and harmonized biophysiological measurements of immune, cardiovascular, and metabolic functioning. We find that the relative importance of early-life and adult SES varied across young-, mid-, and late-adulthood, with accumulation of risks operating in young adulthood and pathway and mobility models in later adulthood.

Extended Abstract

Introduction

Research across disciplines consistently documents a social gradient in health, with individuals of lower socioeconomic status (SES) having higher rates of disease, disability, and death than higher SES individuals. Along this line of research, there has been growing interest in examining the early-life origins of SES disparities in health using a life course perspective, and recent studies have revealed the critical importance of early-life exposures in shaping later life outcomes. Findings from these studies have proposed four mechanisms to explain the association between early-life SES and adult health outcomes: 1) the sensitive period model, which suggests that early-life SES has persistent, irreversible effects on later life physical functioning; 2) the accumulation of risks model, which holds that the risks and exposures associated with economic conditions at various points in the life course accumulate and compound to affect health and mortality; 3) the pathway model, which holds that early-life SES relates to later life health through its effects on later-life SES; and 4) the social mobility model, which suggests that the health effects of early-life exposures can be modified by later-life SES. Though these life course processes are often positioned as conceptually distinct, research documents that the four proposed mechanisms are complimentary and interconnected.

While research on the early life origins of adult health outcomes has surged, critical gaps in the literature remain. First, the temporality of this association has not been properly tested. It is not clear the extant models actually account for the associations between early and later life conditions because studies have not examined the intervening years. It remains a question whether early or contemporaneous SES are more important or whether there are windows of 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, or the trajectory of change in social exposures as one ages in relation to disease risk measured at various points in time over the life course. Second, most studies rely on single indicators of SES that fail to capture the complex and multidimensional nature of socioeconomic well-being across the life course. Third, few studies have examined the biological mechanisms underlying the association between life course SES and later life morbidity and mortality risks.

In sum, an important observation emanating from diverse threads of research on social adversity and health is that understanding this link at any point in time demands comparisons across life stages to more clearly specify timing, duration, and the intermediate mechanisms. In this study, we examine how socioeconomic status in early life relates to biophysiological functioning in early-, mid-, and late-adulthood using three nationally representative, longitudinal data sets. In particular, we assess the extent to which the associations between SES and four biomarker outcomes are consistent with the critical period, accumulation of risks, pathway, and/or social mobility models at various stages in the life course. By tracing the physiological consequences of life course exposures to unfavorable social environment, , this study contributes to a life course understanding of how early life conditions "get under the skin" to influence disease susceptibility as individuals age.

Data and Methods

Data for this study come from three nationally representative NIH studies that collectively span multiple stages of the life course. Data for young adulthood come from 14,023 participants in the National Longitudinal Study of Adolescent Health (Add Health) aged 12-18 at Wave I (1994-95) and followed up at aged 24-32 in Wave IV (2008-09). Data for mid adulthood come from 909 respondents aged 25-74 in the National Survey of the Midlife Development in

the United States (MIDUS) surveyed at Wave I (1995-96) and followed up at Wave II (2004-09). Data for late adulthood come from 1,101 participants aged 57-85 in the National Social Life, Health, and Aging Project (NSHAP) at Wave I (2005-06) and followed up at Wave II (2010-11).

Outcome measures were harmonized across the three studies and include four biomarker measurements that represent key biological pathways underlying stress process. Our outcomes were collected in all the above studies at the follow-up surveys and span a range of biological systems. The measure of immune function and inflammation was assessed by C-reactive protein (CRP). Cardiovascular function was assessed by diastolic and systolic blood pressure (BP). The metabolic function was assessed by measures of waist circumference (WC) and body mass index (BMI) based on measured weight and height. Analyses employed both continuous outcome measures that directly indicate physiological functioning and categorical measures based on clinical cut points that correspond to disease outcomes such as chronic inflammation (CRP <1 mg/DL = normal, 1-3 mg/DL = low inflammation, 3-10 mg/DL = high inflammation, >10 mg/DL = very high inflammation), hypertension (SBP $\geq$ 140 or DBP $\geq$ 90), abdominal obesity (WC > 102 cm for males, > 88 cm for females), and obesity (BMI $\geq$ 30 kg/m^2).

For each data set, we constructed composite measures of early-life SES and adult SES, as multivariate scales of SES are more reliable than single measures. Early-life SES was calculated as the mean of standardized (*z*-score) measures of SES including parental education, household income, welfare receipt, and subjective financial well-being. Adult or current SES was operationalized similarly to early-life SES and included measures of respondent education, household income, welfare receipt, and household assets. Composite measures of early-life and adult SES were calculated for all respondents who had data on at least two of the variables used in each scale. In addition to continuous measures of SES, we also include categorical measures

of SES, which indicate long-term social status using the lowest quartiles of each SES measures. Based on the dichotomous SES measures, we constructed measures of change from early to current status that include four categories: (1=high-high-; 2=high-low-; 3=low-high; 4=low-low).

All models presented here control for age, sex, and race. Future analyses will further adjust for behavioral and psychosocial factors that may mediate the associations between SES and the biomarker outcomes, including smoking, physical activity, drinking alcohol, depressive symptoms, perceived social stress, chronic conditions, and medication use.

We examined the associations between early-life SES, adult SES, and biomarkers assessed at the follow-up surveys. We modeled continuous biomarker outcomes using the OLS regression models and categorical outcomes of biomarkers using generalized linear models, including ordinal logit models for inflammation and logistic models for the hypertension, abdominal obesity, and overall obesity. For each outcome, we estimate two sets of models. First, we estimate models in a stepwise fashion using continuous measures of SES: 1) adjusting for early life SES; 2) adjusting for adult SES; 3) simultaneously adjusting for both early life and adult SES; 4) adjusting for all other covariates. Second, we model the biomarker outcomes as a function of the categorical measures of SES mobility. All models adjust for survey design effects and nonresponse using sampling weights. Analyses were conducted in Stata 13.

Results

Models utilizing continuous measures of early life and adult SES show a strong association of SES with physiological functioning and dysregulation across all life stages; however, the relative importance of early life and adult SES varied across young-, mid-, and late-adulthood, providing evidence for age variation in life course mechanisms. Adjusting only for early life SES showed significant, linear associations of childhood SES with inflammation, blood

pressure, waist circumference, and BMI in young adulthood, and modest associations of early life SES with these markers in mid- and late-adulthood. Adult SES, when modeled separately from early life SES, had robust and inverse associations with all physiological outcomes across all life stages. Mutual adjustment for both early life and adult SES showed that the linkages of SES to physiological functioning followed different life course mechanisms depending on the life stage. In young adulthood, both early life and adult SES had significant independent associations with physiological functioning, suggesting an accumulation of risks model. However, in mid- to late-adulthood, associations of early life SES with physiological outcomes were mostly attenuated by adult SES, suggesting a pathway model whereby the influence of childhood SES on adult physiology operates through socioeconomic pathways (support for this model is especially strong in NSHAP results). All models of physiological dysregulation (clinically elevated inflammation, hypertension, abdominal obesity, and overall obesity) were consistent with models of linear effects.

Analysis using categorical SES disadvantage over time provides evidence for both accumulation of risks and social mobility life course models. Figure 1 presents some illustrative findings. Among young adults, the experience of disadvantage in early life, young adulthood, or both was significantly associated with poorer physiological outcomes, providing further evidence for the accumulation of risks model. Socioeconomic disadvantage had limited associations with physiological functioning in mid adulthood, though persistent disadvantage (low-low) was significantly associated with elevated blood pressure and higher odds of hypertension. Finally, moving from high SES in early life to low SES in late adulthood was associated with increases in inflammation, waist circumference, and BMI, suggesting adverse effects of downward mobility on physiological functioning. Interestingly, upward mobility from low to high SES also showed

some adverse effects in late adulthood using the NSHAP data. This suggests that low SES at origin or any point prior to late life could be harmful.

These results illuminate the complex temporal dynamics through which SES impacts physiological indicators of health across the life span. They suggest that SES operates through several life course and biological mechanisms to affect health, and that these mechanisms evolve across young-, mid-, and late-adulthood. Future analysis may incorporate additional datasets and explore the behavioral and psychosocial mechanisms that might explain some of the observed linkages between life course SES and health. Our innovative study design allows for a direct comparison of how different conceptual models fit the data at the same and different life course stages. Such study is much less likely to overlook patterns of associations in the data than previous studies which only evaluate these associations in one particular (sensitive) period of the life course. Findings that emerge thus can indicate specificity of life course adversity exposures that can better inform disease intervention and control strategies aimed at minimizing socioeconomic disparities in health.

Figure 1a: Predicted LogCRP
by Categorical Measure of SES*

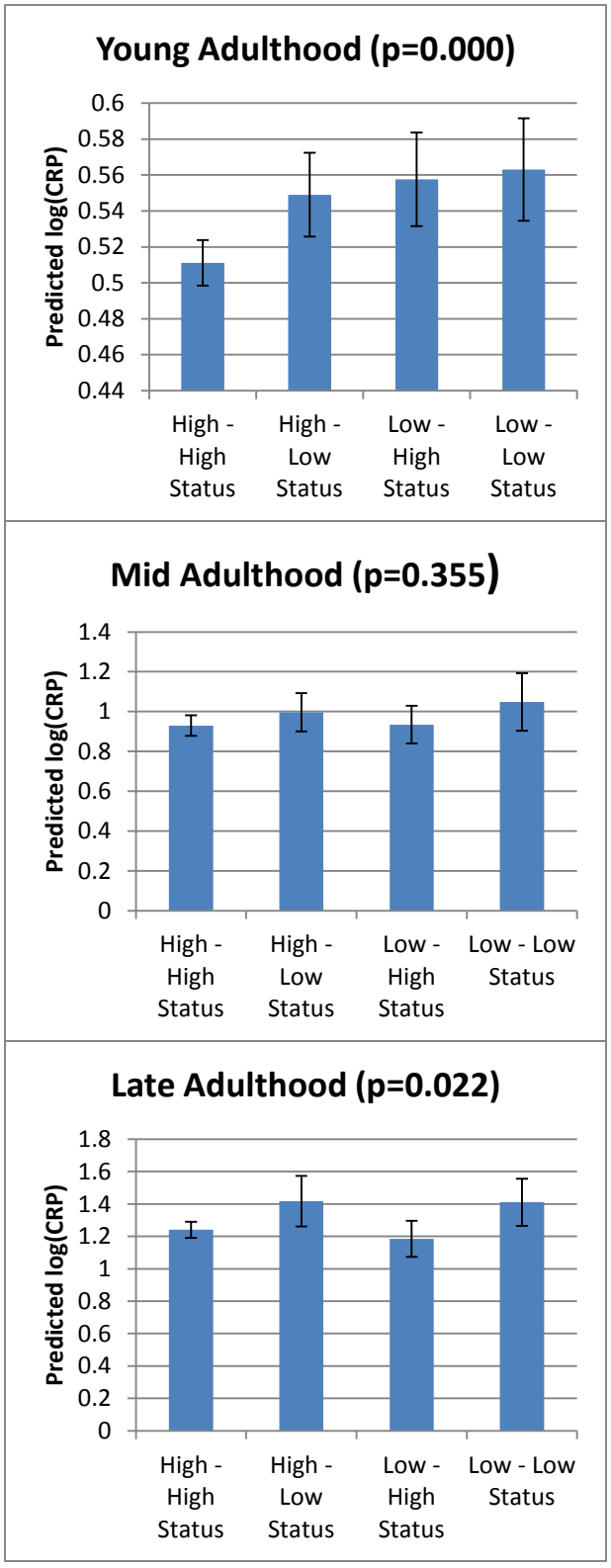
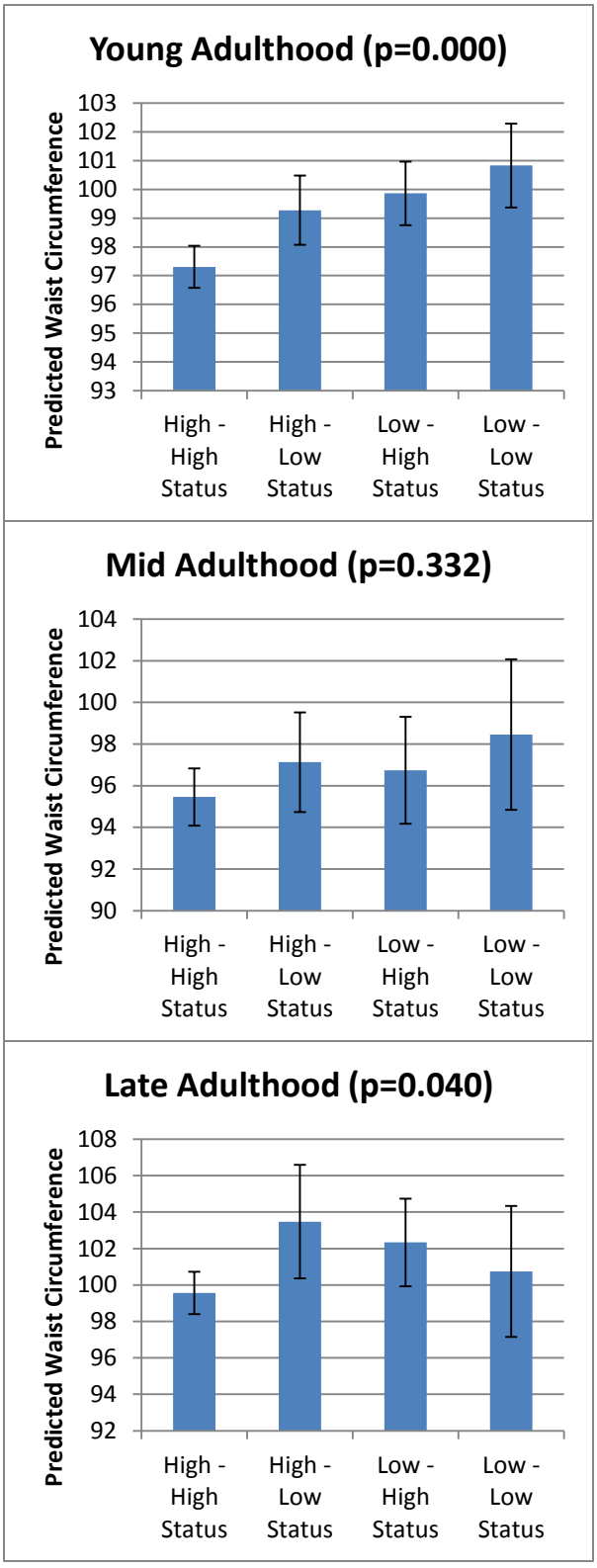


Figure 1b: Predicted Waist Circumference
by Categorical Measure of SES*



*The models presented here adjust for age, sex and race/ethnicity.