Early Life Socioeconomic Disadvantage and Young Adult Cardiometabolic Risk:
Sex Differences and Underlying Mechanisms

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Short Abstract

Extensive evidence links early life socioeconomic disadvantage to adult health, but the importance of longitudinal socioeconomic conditions across contexts and the physiological processes that underlie these links remain unclear. Using the National Longitudinal Study of Adolescent to Adult Health, this study examines the longitudinal associations of household and neighborhood disadvantage across the transition to adulthood with young adult cardiometabolic risk. Results indicate that both early life and adult disadvantage contribute to young adult cardiometabolic risk. Further, both household and neighborhood disadvantage are associated with cardiometabolic risk, though effects of neighborhood disadvantage are mostly explained by household disadvantage. We also identify significant sex and race differences in longitudinal and multilevel associations, such that disadvantage experienced by females and white respondents is more strongly predictive of cardiometabolic risk compared to males and black respondents. These findings have important implications for understanding the early life determinants of health disparities among U.S. adults.
Extended Abstract

An extensive body of literature has linked early life socioeconomic disadvantage to adult morbidity and mortality (Hayward & Gorman 2004; Cohen et al. 2010; O’Rand & Hamil-Luker 2005). While recent explorations have begun to shed light on the underlying physiological processes that potentially contribute to poorer health outcomes among those with a history of disadvantage, including cardiovascular, metabolic, immune functioning (Loucks et al. 2010; Pollitt et al. 2008), several gaps in the literature persist.

First, most studies use point-in-time measures of disadvantage, therefore neglecting to capture how longitudinal dynamics of disadvantage across life stages are uniquely tied to physiological outcomes. Indeed, persistent disadvantage, movement into disadvantage, or movement out of disadvantage across the transition to adulthood are unique experiences that could have distinct implications for physiological functioning in young adulthood.

Second, most studies have limited measures of disadvantage, using one or two indicators such as low parent educational attainment or low household income. However, disadvantage spans across multiple domains within the household, including the additional strain experienced by families who have difficulty paying bills, rely on welfare, or are headed by a single parent. The experience of disadvantage is also not limited to the household, but is also experienced through interactions with the surrounding neighborhood. Neighborhood disadvantage in the form of low neighborhood educational attainment, a high proportion of household poverty, a high proportion of single parent households, etc. has the potential to negatively impact the health of those
within those neighborhoods, net of individual socioeconomic status (Ross & Mirowsky 2001; Yao & Robert 2007; Morenoff et al. 2007).

Finally, studies of the relation between early life disadvantage and adult health tend to rely on older adult samples and retrospective accounts of childhood socioeconomic conditions. Although aging-related chronic disease is not likely to emerge by young adulthood, the physiological imprints of disadvantage might already be present, which would have useful implications for health interventions. Further, retrospective accounts of childhood conditions are potentially biased or inaccurate in older samples, motivating further research that uses longitudinal studies that include interviews in both early life and adulthood.

To fill these gaps in the literature, the current study examines the associations of household and neighborhood disadvantage across the transition to adulthood with young adult cardiometabolic risk. We use measures of disadvantage in both adolescence and young adulthood to capture the timing and duration of disadvantage across the transition to adulthood. Second, we use indexes that aggregate multiple indicators of disadvantage to capture how the magnitude of disadvantage across domains is related to cardiometabolic risk. Indexes are also created for both household and neighborhood disadvantage, thus providing a more comprehensive assessment of disadvantage across both households and neighborhoods. Third, we use a national sample that includes multiple measures of cardiometabolic functioning in young adulthood, allowing us to observe physiological outcomes often long before the emergence of chronic illness.
Data and Methods:

The data come from 9,446 participants in the National Longitudinal Study of Adolescent to Adult Health, a nationally representative, school-based sample of adolescents that were first interviewed in grades 7-12 during the 1994-95 academic year. Respondents were followed for four survey waves, with the most recent survey conducted in 2008. Our analysis uses data from Wave I (1994-95) when respondents were age 12-18, and Wave IV (2008-09) when respondents were 24-32. Biospecimens were collected by trained interviewers during Wave IV. In addition, tract-level data on neighborhood characteristics and composition was gathered from the US Census around the time of data collection for both survey waves.

*Cardiometabolic risk* was measured using seven physiological indicators that capture metabolic, inflammatory, and cardiovascular systems (Wickrama et al. 2015). These include waist circumference, triglycerides, HDL and LDL cholesterol, glycosylated hemoglobin, hypertension, and C-reactive protein. A risk score was constructed by creating binary measures for each biomarker based on risk cutpoints and summing binary measures, which produced an index ranging from 0-7.

We constructed four disadvantage indexes that separately capture adolescent and adult household and neighborhood disadvantage. *Household disadvantage indexes* were created for adolescence (Wave I) and young adulthood (Wave IV) using five binary indicators. For Wave I, we used the sum of low parent educational attainment, parent-reported difficulty paying bills, parent welfare receipt, single parent household, and parent unemployment. Wave IV household disadvantage was measured using disadvantage indicators that are appropriate for young adulthood. Each disadvantage
scale ranges from 0-5 with higher numbers indicating higher disadvantage. *Neighborhood disadvantage indexes* in Wave I and Wave IV were constructed based on five disadvantage indicators that were available at all waves and capture tract-level prevalence of poverty, low educational attainment, utilization of welfare, female-headed households, and unemployment. Consistent with the household disadvantage indices, the neighborhood disadvantage indexes range from 0-5.

We estimated OLS models that assess the associations of disadvantage with cardiometabolic risk. We first assessed binary associations of each disadvantage index separately, and next simultaneously included disadvantage indexes in models to assess relative significance of the timing and context of disadvantage. We also tested interactions between disadvantage indexes to determine whether combinations of household and neighborhood disadvantage from adolescence to young adulthood predict cardiometabolic risk differently. Finally, we interacted the disadvantage indexes with sex to determine whether there are significant sex differences in these associations. All models adjust for sex, race/ethnicity, and age.

**Results:**

As shown in Models 1-4, we find significant bivariate associations of disadvantage with cardiometabolic risk for all disadvantage indexes (adolescent and adult household and neighborhood disadvantage). Models 5-9 demonstrate that associations of household disadvantage with cardiometabolic risk are stronger than associations of neighborhood disadvantage, as effects of neighborhood disadvantage are largely attenuated when simultaneously including household disadvantage indicators. However, models interacting disadvantage indexes across time and context (Models 10-13) indicate
that household and neighborhood disadvantage across time multiplicatively affect cardiometabolic risk. In particular, we find a significant interaction of adolescent household disadvantage with adult household disadvantage, and a significant interaction of adolescent household disadvantage with adult neighborhood disadvantage. These interactions will be further explored in additional analysis. Finally, we find that associations of household and neighborhood disadvantage with cardiometabolic risk differ by sex (Figures 1 and 2), such that females appear to have lower cardiometabolic risk in less disadvantaged settings, but females also have significantly greater cardiometabolic risk in higher disadvantage settings. Further analysis will explore whether that impacts of longitudinal and multidimensional experiences of disadvantage on cardiometabolic risk also differ by sex. In addition, we will incorporate measures of social integration, neighborhood and household disorder, psychological conditions, and health behaviors as potential mediators in the associations between disadvantage and cardiometabolic risk.

References


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*** p<0.001, ** p<0.01, * p<0.05, + p<0.1

HDI=Household disadvantage index
NDI=Neighborhood disadvantage index
All models adjust for sex, race/ethnicity, and age.
Figure 1. Sex Differences in Associations of Disadvantage with Cardiometabolic Risk

1a. Adult Household Disadvantage

1b. Adolescent Neighborhood Disadvantage