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Early-life social origins of later-life body weight: The role of socioeconomic status and health behaviors over the life course

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ABSTRACT

Using the 1957–2004 data from the Wisconsin Longitudinal Study, we apply structural equation modeling to examine gender-specific effects of family socioeconomic status (SES) at age 18 on body weight at age 65. We further explore SES and health behaviors over the life course as mechanisms linking family background and later-life body weight. We find that early-life socioeconomic disadvantage is related to higher body weight at age 65 and a steeper weight increase between midlife and late life. These adverse effects are stronger among women than men. Significant mediators of the effect of parents' SES include adolescent body mass (especially among women) as well as exercise and SES in midlife. Yet, consistent with the critical period mechanism, the effect of early-life SES on late-life body weight persists net of all mediating variables. This study expands current understanding of life-course mechanisms that contribute to obesity and increase biological vulnerability to social disadvantage.

1. Introduction

Research consistently documents the enduring consequences of social environment in childhood and adolescence for life-course trajectories of body weight (Baltrus et al., 2005; James et al., 2006; Langenberg et al., 2003). Socioeconomic status (SES) of the family of origin is a particularly important influence, with children from disadvantaged socioeconomic backgrounds having higher body mass index (BMI) and a greater risk of overweight and obesity in adulthood than children from higher-SES families (Giskes et al., 2008; Khlat et al., 2009; Parsons et al., 1999). Although the long-term effects of early-life socioeconomic resources on body weight in adulthood have been studied extensively, previous research is limited in several important ways. Most longitudinal studies are characterized by a relatively short follow-up, with participants being followed only into young adulthood (Chandola et al., 2006; Parsons et al., 1999) and, rarely, into midlife (Langenberg et al., 2003). Therefore, it is not known whether the reach of early-life SES extends to body weight in later life. When studies do focus on middle-aged and older adults, participants are typically not recruited in childhood but entered the study at midlife, which can obscure differential survival by SES and obesity. Further, in most studies, parents' SES is assessed with one measure, mostly the father's occupation, which does not fully capture the multidimensional nature of socioeconomic environment and does not incorporate measurement error (Baltrus et al., 2005; Langenberg et al., 2003). Another methodological

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limitation is an overwhelming reliance on retrospective reports of early-life SES that may be subject to recall bias and lead to underestimation of the true effect (Giskes et al., 2008; James et al., 2006). Moreover, few studies have explicitly examined gender differences in the life-course mechanisms conveying the effect of early-life SES on body weight in adulthood and later life.

We use the 1957–2004 data from 5778 participants in the Wisconsin Longitudinal Study to examine how family SES at age 18 (in 1957) affects men's and women's body mass index (BMI) in 2004 at age 65. We explore SES and health behaviors over the life course as mechanisms linking socioeconomic family background and later-life body weight. Using structural equation modeling, we decompose the effect of early-life SES into direct and indirect (mediated) effects and compare the relative importance of each hypothesized mechanism in conveying the effect of parents' SES on offspring's BMI in later life. Moreover, we use multiple-group analysis to explore gender differences in the effect of early-life SES and life-course mechanisms underlying this effect. Another methodological contribution of our study is modeling SES at each life-course stage as a latent variable to incorporate multiple indicators and measurement error. Finally, the prospective longitudinal nature of the WLS allows us to address the issues of recall bias and selection bias.

2. Background

Body weight is a multi-faceted phenomenon with a contested cultural meaning and complex biosocial antecedents and consequences. The medical and public health discourse emphasizes individual-level health risks and society-wide costs of overweight and obesity (Roos et al., 2012). Yet, the findings on the health consequences of heavier body weight are equivocal and complicated. Zheng and Yang (2012) show pronounced population heterogeneity in the effect of overweight and obesity on mortality depending on the combination of race, gender, and SES. Although heavier body weight is detrimental for some population subgroups, it is protective for others (Zheng and Yang, 2012). Moreover, the health implications of body weight vary across the life course such that the association between obesity and mortality is weaker in later life than in adulthood (Zheng and Yang, 2012). Being overweight and even obese at later ages protects against mortality controlling for health behaviors and health status (Lantz et al., 2010).

Whereas findings about the links between health and body weight are varied, research has consistently documented *social* inequality in overweight and obesity that are more prevalent among socially disadvantaged individuals compared to persons of higher SES (Sobal and Stunkard, 1989). Thus, socioeconomic resources are an important social influence on body weight (Drewnowski, 2009). The American Medical Association has recently classified obesity as a disease, which can lead to even greater medicalization of body weight and underestimation of its *social* antecedents consequences. In this study, we explore the social influences on body weight and apply a life-course perspective to explore the complex mechanisms generating divergent trajectories of body weight among different groups. We focus on the social aspects of body weight while also acknowledging the close link of social factors with intertwined behavioral, psychological, and biological processes.

2.1. Mechanisms linking early-life SES and body weight in later life

A life-course perspective focuses on long-term trajectories of individual development and enduring influences of past experiences. We adopt a dynamic view of SES and emphasize a lifelong approach to the gendered processes underlying socioeconomic disparities in body weight. In this study, SES is considered as a trajectory characterized by long-term patterns of stability and change (Pearlin et al., 2005). Moreover, the life-course approach underscores that health in later life cannot be explained solely by temporally proximate conditions because earlier experiences and characteristics have long-term implications for later well-being (Pearlin et al., 2005). Within the life-course perspective, three major conceptual mechanisms are proposed to explain the relationship between socioeconomic circumstances and health: the critical period model, the accumulation of risks model, and the pathway model.

2.1.1. The critical period model

The critical period model (Ben-Shlomo and Kuh, 2002) reflects a biological imprinting mechanism and posits that early-life SES has long-lasting effects on biological and behavioral systems, and these effects are irreversible and permanent. Research suggests that low childhood social class is related to total obesity and central adiposity in adulthood among both men and women, independent of adult SES (Blane et al., 1996; Langenberg et al., 2003). Moreover, the most direct support for the critical period model comes from studies showing that the effect of childhood SES is stronger than the effect of social class in adulthood (Blane et al., 1996; James et al., 2006). A potential mechanism through which early-life environment can become embodied and exert direct enduring effects on later-life body weight is early-life stress. Stress in childhood resulting from low SES can lead to a chronic elevation of cortisol levels, which in turn is associated with metabolic irregularities promoting excess weight over the life course (Björntorp and Rosmond, 2000). Moreover, a heightened risk of obesity can be programmed during inadequate prenatal development or via postnatal biochemical disruptions (James et al., 2006). Importantly, the critical period mechanism suggests that early disadvantage can increase body weight decades later by launching long-term physiological changes; thus, early-life stress does not necessarily operate through an immediate increase in childhood weight. Following the critical period model, we hypothesize that early-life SES is inversely associated