Published in final edited form as:
Soc Sci Res. 2014 July ; 46: 59–71. doi:10.1016/j.ssresearch.2014.02.007.

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.

Keywords

socioeconomic status; body mass index; life course; gender

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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 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 5,778 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 multiplegroup 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 and 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

(Bjorntorp 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 with BMI in later life, and this association persists net of exposures and behaviors in adulthood. Further, the effect of low early-life SES on higher BMI can be even stronger than the effect of socioeconomic disadvantage in adulthood.

2.1.2. The Accumulation of Risks Model—The accumulation of risks model suggests that deleterious exposures at different life-course stages inflict a cumulative damage on biological systems and, thus, have compounding effects on later-life health (Ben-Shlomo and Kuh, 2002). It is the overall burden of low SES over the life course that contributes to poorer late-life outcomes rather than low SES at a particular life-course stage. Research indicates that socioeconomic circumstances at each life stage have additive effects on body weight (Langenberg et al., 2003; Salonen et al., 2009). Specifically, socioeconomic disadvantage in childhood and adulthood contributes independently to an increased risk of overweight and obesity (Heraclides and Brunner, 2010; James et al., 2006). Consistent with the accumulation of risks model, we hypothesize that SES in adolescence and adulthood are each related to heavier weight independent of other periods.

Related to the accumulation of risks model is the cumulative advantage (CA) mechanism. According to the CA model, the advantage of some individuals or groups expands over time, which leads to increasing within-cohort inequality with advancing age (DiPrete and Eirich, 2006). The status-dependent CA model suggests that socioeconomic gap in body weight is magnified as cohorts grow older. Research provides support for the CA process, especially among women, indicating that individuals from disadvantaged socioeconomic background gain more weight in adulthood than persons from higher-SES families of origin (Clarke et al., 2009; Giskes et al., 2008). Based on the CA mechanism, we hypothesize that the effect of early-life SES on body weight becomes stronger with age such that BMI grows steeper for persons with low early-life SES compared to those from higher-SES family backgrounds.

2.1.2. The Pathway Model—The pathway model suggests that early-life environment is consequential mainly because it shapes beneficial or harmful life-course trajectories of opportunities and constraints (Ben-Shlomo and Kuh, 2002). Of particular interest is not the direct effect of early-life SES but the mechanisms linking socioeconomic family background to health disparities in adulthood and later life. Research indicates that life-course trajectories of health behaviors and socioeconomic achievement can be important pathways linking early-life SES and later-life body weight.

Health behaviors in early life: Studies consistently document that parents' higher SES is associated with lower BMI and a lower risk of obesity among children and adolescents (Rossen and Schoendorf, 2012). In turn, early-life adiposity has enduring consequences for life-course trajectories of body weight, and children with a higher BMI have a greater risk of

obesity in adulthood and later life (Dietz, 1998; Parsons et al., 1999; Salonen et al., 2009). Because childhood body weight is affected by parent's SES and, in turn, affects weight in adulthood, we hypothesize that the effect of socioeconomic family background on BMI in later life is partly mediated by early-life adiposity.

In addition, physical activity (PA) in childhood and adolescence is likely to be a potential pathway linking parents' SES and offspring's BMI in later life. Socioeconomic advantage of the family of origin is associated with higher levels of PA in early life (Gordon-Larsen et al., 2006). In contrast, children from low-SES families have fewer opportunities to participate in sports due to poor availability of local physical activity resources (Gordon-Larsen et al., 2006). Moreover, research shows that children are more likely to participate in organized sports activities in school than exercise independently (Poobalan et al., 2012). Lower participation in school sports among socioeconomically disadvantaged youth also decreases their overall involvement in exercise (Johnston, Delva, and O'Malley, 2007). Therefore, low-SES children are less likely than their high-SES peers to achieve PA level necessary to prevent obesity. Because higher parents' SES increases children's PA, and regular exercise in childhood and adolescence reduces the risk of obesity in adulthood (Parsons et al., 1999), we hypothesize the association between parents' SES and later-life BMI is partially mediated by sports involvement in adolescence.

Health behaviors in adulthood: Early-life SES affects not only contemporaneous PA but also may have a direct effect on exercise in adulthood. The link between PA and weight is complex. To prevent weight gain, 45-60 minutes of regular daily exercise is recommended, whereas for weight loss PA alone is insufficient and needs to be combined with calorie restriction (Goldberg and King, 2007). With respect to the type, PA is typically assessed as moderate (e.g., walking, golfing) and vigorous (e.g., running, bicycling). Both moderate and vigorous activities are effective for weight goals if the duration is long enough to ensure sufficient energy expenditure (Jakicic et al., 1999). These findings suggest that overall higher levels of PA are related to lower weight (Bensimhon et al., 2006; Sherwood and Jeffery, 2000). In turn, higher childhood SES is associated with higher levels of exercise in adulthood and midlife (Blane et al., 1996; Kuh and Cooper, 1992). Thus, we hypothesize that the effect of early-life SES on body weight in later life is partly explained by adult PA.

In addition, early-life SES is linked to tobacco and alcohol use. Parents' social class is inversely related to sons' and daughters' smoking in adulthood and midlife (Blane et al., 1996; Brunner et al., 1999). Because smoking is associated with lower BMI and lower risk of obesity (Kasteridis and Yen, 2012), we expect that smoking *suppresses* the effect of early-life SES. With respect to alcohol use, some studies report no association between alcohol intake and BMI (Fortier, Katzmarzyk, and Bouchard, 2002; Sammel et al., 2003). Others studies show that heavy drinking increases the likelihood of obesity, whereas moderate alcohol intake is associated with lower weight and waist circumference compared to abstaining and heavy drinking (MacInnis et al., 2013; Wannamethee et al., 2004). Moderate drinking can reduce weight by increasing resting energy expenditure (Klesges, Mealer, and Klesges, 1994) and heat release associated with basic metabolism (Westerterp, 2004). With respect to SES, higher-SES individuals are more likely to be moderate drinkers than abstainers and heavy drinkers (Cerdá, Johnson-Lawrence, and Galea, 2011). Thus,

alcohol use can mediate the effect of early-life SES on BMI because higher parents' SES is related to moderate drinking in adulthood, which in turn is related to lower weight.

Socioeconomic achievement in adulthood: An important mediator of the association between early-life SES and adult body weight is SES in adulthood (Gustafsson and Hammarström, 2012; Salonen et al., 2009). Parents' SES is positively associated with individuals' own SES (Sewell and Hauser, 1975), and higher socioeconomic resources in adulthood are related to lower body weight (Salonen et al., 2009). Men and women who experience educational and income disadvantage have higher levels of BMI and an elevated risk of obesity compared to their socioeconomically advantaged counterparts (Beydoun et al., 2009). We hypothesize that the effect of parents' SES on BMI in old age is conveyed via offspring's socioeconomic achievement in adulthood.

2.2 Gender Differences

Women have higher levels of obesity than men and experience a disproportionate burden of diseases and functional limitations attributable to excess weight (Muennig et al., 2006; Wang and Beydoun, 2007). Overweight and obesity have a much greater adverse effect on health-related quality of life and mortality among women than men after age 45, although obese women younger than 45 years have lower mortality than their male peers (Muennig et al., 2006). The relationship between visceral fat and risk factors for cardiovascular disease and diabetes, including increased blood pressure and impaired fasting glucose, is stronger for women relative to men (Fox et al., 2007; Tanaka et al., 2004). Moreover, the adverse *social* consequences of overweight and obesity are substantially more pronounced among women than men. Heavier weight among women is associated with lower education, lower occupational standing and lower earnings, while this effect is inconsistent or absent among men (Glass et al., 2010; Judge and Cable, 2011).

Given gender inequality in the consequences of overweight and obesity, it is important to obtain a more nuanced understanding of gender differences in the social origins of body weight, which will facilitate the development of effective gender-specific prevention approaches. Research exploring the gendered association between body weight in adulthood and early-life SES (measured as fathers' occupation) yields mixed findings. Some studies suggest that early-life SES is more consequential for adult body weight among men (Langenberg et al., 2003), whereas others indicate that parents' SES exerts a greater influence on daughters' than sons' BMI and obesity in adulthood (Giskes et al., 2008; Khlat et al., 2009). Further, more research is needed on gender differences in life-course mechanisms explaining the effect of early-life SES. Although it is well-documented that women have lower SES than men, engage in PA less frequently, and are less likely to smoke and consume alcohol than men (Denney et al., 2010; Ham and Ainsworth, 2010; Ross and Mirowsky, 2002), little is known about gender-specific ways in which each mediator operates. Given the potential importance of gender as a source of heterogeneity in the link between parental socioeconomic resources and offspring's body weight in later life, this study investigates whether and how the effect of early-life SES and life-course mechanisms conveying this effect differ between men and women.

3. Material and methods

3.1. Analytic Sample

The Wisconsin Longitudinal Study (WLS) is a long-term cohort study of 10,317 White men and women who graduated from Wisconsin high schools in 1957. Participants were interviewed at ages 17-18 (in 1957), 36 (in 1975), 53-54 (in 1993), and 64-65 (in 2004). This study's analytic sample contains 2,615 men and 3,163 women who participated in the 1957 baseline survey and in the 1993 and 2004 interviews.

3.1.1. Sample attrition—The most important predictors of sample attrition are gender (women are more likely to participate than men), marital status (married persons are more likely to participate than the unmarried), children (parents with more children are less likely to participate), and health (individuals with more chronic illnesses have a higher probability of attrition). Higher BMI is also a significant predictor of attrition because of its positive association with mortality. To take into account potential selection bias, we use the Heckman two-step procedure to create a selection instrument for each of the three reasons for attrition: death, refusal, and inability to locate. Including three selection instruments does not significantly improve model fit; therefore, one variable all reasons for attrition is used in all models.

3.2. Variables

The focal outcome is *body mass index* measured as weight in kilograms divided by height in meters squared. Following recent studies (Clarke et al., 2009; Umberson et al., 2011), we use a continuous measure of BMI rather than discrete categories of overweight and obesity to retain a full scope of variability in BMI. The mean BMI in 2004 equals 28.3 for men and 27.5 for women, with BMI values ranging from 18 to 43. Because height and weight are self-reported in the WLS, we use data from the 1999-2004 National Health and Nutrition Examination Survey (NHANES) to estimate the extent of discrepancy between self-reported and measured indicators of BMI, both of which are available in NHANES (Chang et al., 2009). Among non-Hispanic White adults ages 53-65, we regress measured values of weight and height on self-reported linear and squared values of weight and height, separately for men and women. Then we multiply the gender-specific slopes from these regression models by self-reported measures of height and weight in the WLS to adjust for reporting bias.

Socioeconomic characteristics of the family of origin in 1957 include father's and mother's education measured in years, family income measured in $100's, and father's occupation represented with five categories (unskilled worker, farmer, skilled worker, white-collar worker, and professional/managerial). In addition to individual-level characteristics, we include measures reflecting father's entire occupation: occupational education (log transformed) indicating the proportion of individuals in a given occupation who completed one or more years of college, and occupational income (log transformed) representing the proportion of individuals in a given occupation who earned more than $10,000. To create these measures, proportions of workers with a given threshold of education and income is calculated for each 3-digit Census 1970 occupational code. It should be noted that for

families headed by single mothers we include *mothers*' occupation and occupational characteristics.

Mediators in 1957—*Physical activity in adolescence* is reflected as sports participation in high school obtained from participants' high school yearbooks. This measure is the sum of the number of varsity sports, club sports, and intramural sports. *Body weight in adolescence* is measured as relative body mass (RBM) coded from facial adiposity in high school yearbook pictures in 1957 (Reither et al., 2009). Six coders assigned RBM scores separately for boys and girls on scale ranging from 1 to 11. Among individuals who participated in the 1957, 1993 and 2004 waves, yearbook pictures are not available for 839 (15%) of participants. These missing values are handled using multiple imputation based on Bayesian estimation.

Mediators in 1993 and 2004—*Physical activity* is measured as the frequency of light or moderate exercise (such as walking, dancing, gardening, golfing, bowling) and vigorous exercise (such as aerobics, running, swimming, bicycling) with four response categories: 1 = less than once per month, 2 = one to three times per month, 3 = once or twice per week, 4 = three or more times per week. *Smoking* is coded as current smoker, former smoker, and nonsmoker. *Alcohol use* reflects moderate drinking defined based on the 2010 Federal Dietary Guidelines as 1 daily drink for women and 1-2 daily drinks for men.

Socioeconomic status in 1993 and 2004 includes education (the total completed years of schooling), occupation of the current or last job (professional/managerial, clerical/sales/service, and crafts/operatives/laborers), a natural log of household wealth, occupational education (log transformed) reflecting the percentage of persons in a given occupation who completed one year of college or more and occupational income (log transformed) representing the percentage of persons who earned at least $14.30 per hour in 1989. Occupational education and income are created based on the 1990 Census occupational codes using the same procedure as for fathers' measures. Each variable is used as an indicator in a measurement model shown in Table 1.

Control variables in 2004—We control for several potential mediators to emphasize that the effects of our focal variables are not driven by other important pathways. Marriage, parenthood, and depression are related to higher BMI (Needham et al., 2010; Umberson et al., 2009; Umberson et al., 2011), and individuals from higher-SES background are less likely to be married, and have fewer children and lower depression than those from lower-SES families (McLaughlin et al., 2010; Murphy and Wang, 2001; Torr, 2011). Marital status equals 1 for the married and 0 for the unmarried. Parental status reflects the number of children. Depressive symptoms are based on the Center for Epidemiologic Studies Depression Scale (α = .88).

All variables have 2 to 3% of missing values on average. To impute missing values, we carry out multiple imputation procedure using Bayesian estimation in *Mplus*.

3.3. Analytic Approach

Structural equation modeling (SEM) is applied to examine life-course mechanisms linking early-life SES to BMI in later life. The structural model estimates a direct path from early-life SES to BMI at age 65, and potential indirect pathways via health behaviors in 1957, 1993 and 2004 and socioeconomic resources in 1993 and 2004. We use multiple group analysis to examine whether path coefficients differ significantly for men and women. We estimate a series of models for each hypothesized mediator. The initial model in each series imposes equality constraints such that all structural paths are constrained to be equal for men and women. Then a number of nested models are tested relaxing one equality constraint at a time and comparing the model fit by subtracting χ^2 of the less restrictive model from χ^2 of the more restrictive model. A significant improvement in model fit ($\Delta\chi^2 \geq 3.841$ with one degree of freedom) is interpreted as evidence of a significant gender difference in a given path. Parameters in structural equation models are estimated in *Mplus* 7 using weighted least squares with standard errors and mean- and variance-adjusted χ^2 test statistic.

4. Results

The measurement part of the structural equation model is shown in Table 1. Standardized factor loadings and fit indices suggest that the indicators measure each factor well.

4.1. Decomposition of the Total Effect of Early-Life SES into Direct and Indirect Effects

The total effect of early-life SES indicates that higher parental SES is related to lower offspring's BMI in 2004 among both men (β = -.330, p < .001) and women (β = -.740, p < .001), although the effect of early-life SES is significantly stronger among women, as indicated by a highly significant improvement in model fit (p < .001) when this path is allowed to vary by gender. To illustrate, this effect size means an extra five pounds for a woman who is 5′5″ tall and weighs 150 lb. and an extra three pounds for a man who is 6 feet tall and weighs 190 lb. This SES difference in body weight, although modest, can have meaningful implications for health, especially in midlife and early old age when most individuals are already overweight or obese (Mirowsky, 2011).

Panel A in Table 2 shows the mediating effects of early-life health behaviors. RBM in adolescence is a significant mediator of the effect of early-life SES among women. The ratio of the indirect (β = -.199, p < .001) to total effects indicates that RBM mediates 27% of the effect of parents' SES on women's BMI in old age. The mediating effect of RBM is not significant among men because parents' SES is unrelated to sons' facial adiposity in adolescence. Sports participation in high school *suppresses* the effect of early-life SES on late-life BMI among men ($\beta_{indirect}$ = .041, p < .01). Although boys of higher-SES families are more likely to participate in sports than boys from low-SES families, greater involvement in high school sports is related to *higher* BMI among men. The ratio of indirect to total effects suggests that the beneficial effect of higher early-life SES on men's BMI would be 12.4% larger if not for the positive relationship between high school sports and late-life BMI. Both 1957 mediators explain 37.6% of the total effect of parents' SES on late-life BMI among women, whereas among men there is no significant mediating effect of early-life variables. The direct effect of early-life SES remains large in magnitude and

statistically significant net of the 1957 variables among men (β_{direct} = -.286, p < .01) and women (β_{direct} = -.462, p < .001).

Panel B in Table 2 shows the indirect effects of early-life SES via midlife SES and health behaviors. SES in 1993 is a significantly stronger mediator of the effect of parents' SES on sons' ($\beta_{indirect}$ = -.171, p < .001) than daughters' ($\beta_{indirect}$ = -.106, p < .05) BMI in late life. Socioeconomic resources at midlife mediate 52% and 14% of the total effect of early-life SES on BMI among men and women, respectively. Light PA is a significant mediator of the effect of early-life SES among women only, whereas vigorous PA mediates the association between parents' SES and late-life BMI similarly among men and women. Smoking suppresses the effect of early-life SES on men's late-life BMI ($\beta_{indirect}$ = .034, p < .01). Men with higher-SES backgrounds are less likely to smoke than their lower-SES peers, and smoking is related to lower BMI. Among women, smoking is not a significant mediator because parents' SES is unrelated to daughters' smoking at midlife. Finally, being a moderate drinker explains 3.5% of the effect of early-life SES on BMI among women ($\beta_{indirect}$ = -.026, p < .01), whereas there is no mediating effect of moderate drinking among men. All 1993 mediators explain 35% of the effect of early-life SES among women and 34% among men. The direct effect of early-life SES net of all 1993 variables and controls equals -.218 (p < .05) among men and -.483 (p < .001) among women.

Panel C in Table 2 decomposes the total effect of early-life SES into direct and indirect effects via mediators in 2004. SES at age 65 mediates 33% of the effect of early-life SES among men ($\beta_{indirect}$ = -.100, p < .01), while not being a significant mediator among women. Light PA and smoking in 2004 do not mediate the effect of early-life SES on BMI at age 65. Vigorous PA explains a small but significant proportion of the total effect of early-life SES among men (3.6%) and women (2.7%). Finally, moderate drinking plays a similar mediating role among men and women ($\beta_{indirect}$ = -.032, p < .01). All 2004 mediators explain 39.5% of the total effect of early-life SES among women and 42% among men, and the direct effect of early-life SES remains statistically significant among men (β_{direct} = -.191, p < .05) and especially among women (β_{direct} = -.448, p < .001). We could not estimate a structural model in which all mediators (1957, 1993, and 2004) were included simultaneously because the model did not converge. A path analytic model that does not incorporate the measurement model and includes all indicators (available upon request) indicates that the effect of early-life SES on BMI in 2004 is statistically significant net of all mediating and control variables (β_{men} = -0.179, p < .05; β_{women} = -0.305, p < .01).

4.2. Life-Course Trajectories of SES and Body Weight

Table 3 presents the results from models analyzing the effect of early-life SES and mediators in 1957 and 1993 on change in BMI between 1993 (age 54) and 2004 (age 65). Parents' SES predicts daughters' change in BMI between midlife and old age. Although BMI increases for everyone during this period, the increase in BMI is less pronounced among women from higher-SES family backgrounds than their peers from low-SES families (β = -.163, p < .01). Further, women's higher RBM in adolescence is associated with a steeper increase in BMI between 1993 and 2004 (β = .136, p < .05). In contrast, early-life SES and RBM are unrelated to BMI change in men. The only significant predictor among men is

midlife SES, which is associated negatively with an increase in BMI from midlife to old age. The findings for BMI change are consistent with patterns for BMI in 2004 and suggest that early-life SES is more consequential for women's adult BMI, whereas own SES is more important for men's BMI.

Figure 1 shows estimates from a structural equation model including body mass at three time points to explore the extent to which adiposity at age 18 and 53 mediates the effect of early-life SES on BMI at age 64. Parental SES has a significant direct effect on daughters' BMI in old age (β = -.119, p < .05), whereas the direct effect of early-life SES on men's late-life BMI is not significant. The indirect effect of SES in 1957 on BMI in 2004 via midlife BMI equals -.241 for men and -.401 for women (p < .001 for both). Given that parents' SES is unrelated to sons' body mass in yearbook pictures, the indirect effect of SES in 1957 via adiposity in adolescence is significant among women only ($\beta_{indirect}$ = -.021, p < .05). Finally, among women only, early-life SES is linked to late-life BMI by affecting body mass in adolescence, which in turn influences BMI in midlife ($\beta_{indirect}$ = -.199, p < .001).

Figure 2 illustrates a structural equation model that estimates the effects of SES at three life-course periods on BMI in old age. SES in 1957 and 1993 are related significantly and negatively to BMI in 2004 independent of each other, whereas SES in 2004 is unrelated to contemporaneous BMI. Moreover, among women, the effect of early-life SES (β = -.563, p < .001) is stronger than the effect of midlife SES (β = -.289, p < .001). In contrast, among men, SES in 1993 has a more pronounced effect on BMI (β = -.436, p < .001) than SES in adolescence (β = -.180, p < .05). Finally, SES in 1993 is a significant mediator of the effect of early-life SES for men ($\beta_{indirect}$ = -.155, p < .001) and women ($\beta_{indirect}$ = -.107, p < .05).

5. Discussion

This study documents an enduring effect of socioeconomic family background on body weight in midlife and later life among men and women. Our central finding that lower SES in adolescence is related to higher BMI decades later contributes to a growing body of evidence documenting the long reach of childhood socioeconomic environment (Gustafsson and Hammarström, 2012; James et al., 2006; Langenberg et al., 2003). It is important to emphasize that unequal trajectories of high- and low-SES individuals originate in early life and are “anchored in conditions that long antecede the decades in which these trajectories take shape” (Pearlin et al., 2005, p. 207).

Our findings indicate that childhood socioeconomic disadvantage has a stronger adverse effect on women's than men's body weight in adulthood and later life. Men and women in our study came of age in 1950s and 1960s, experiencing a traditional gender-typed division of labor. Health management of family members and competence in health-related matters are commonly viewed as women's domain (Reczek and Umberson, 2012). Thus, higher-SES parents may have emphasized healthy weight management more in the upbringing of daughters than sons because girls as future gatekeepers of their families' health are socialized to be attentive to health matters (Giskes et al., 2008; Reczek and Umberson, 2012). Moreover, childhood SES may be overall more important for women than men in this

traditional cohort in which women were expected to focus on familial obligations and had limited opportunities for status attainment (Carr, 2004).

5.1. Mechanisms Underlying the Relationship between Early-life SES and Later-Life BMI

5.1.1. The Critical Period Model—Consistent with the critical period model, the enduring effect of early-life SES on later-life body weight persists among men and especially among women net of all mediating variables. Providing even stronger support for the critical period model, the effect of early-life SES on women's late-life BMI is greater than the effect of women's SES in midlife. It is possible that the direct effect of early-life SES observed in our study creates an illusion of the critical period processes because we cannot capture all aspects of early-life and adult social environment through which parents' SES can operate indirectly. Yet, given that we included many powerful mediators, biological imprinting reflecting the direct material effects of early-life exposures on the body should be considered a plausible mechanism. Early conditions can permanently alter metabolic processes *in utero*, infancy and childhood (Khlat et al., 2009). Moreover, maternal obesity is directly associated with an increased risk of obesity in offspring due to a fetal programming mechanism (Power and Schulkin, 2008), and low-SES mothers are more likely to be obese than higher-SES women (Langenberg et al., 2003; Wang and Beydoun, 2007). It is also possible that girls are more sensitive than boys to obesogenic environment because of biological sex differences in fat storage and fat metabolism related to estrogen (Power and Schulkin, 2008). The stress of early-life disadvantage can increase the risk of obesity directly via dysregulation of the neuroendocrine axis resulting in elevated cortisol (Pasquali, 2012). Chronically elevated cortisol is in turn associated with metabolic irregularities promoting excess weight over the life course (Bjorntorp and Rosmond, 2000), particularly among women (Pasquali, 2012). Because women are metabolically more inclined than men to store fat, environments conducive to weight accumulation (including low SES) are more detrimental to women than men (Power and Schulkin, 2008). In addition, the stress of socioeconomic disadvantage can affect women's body mass indirectly via food intake because women tend to increase food consumption more than men as a mechanism for coping with chronic stressors (Pasquali, 2012).

5.1.2. The Accumulation of Risks Model—Our results support the accumulation of risks model with respect to the additive effects of SES in adolescence and midlife among both men and women. This finding emphasizes the importance of joint effects of socioeconomic resources at different periods of the life course on body weight (Gustafsson and Hammarström, 2012; Murray et al., 2011). Because the lingering effects of earlier socioeconomic position tend to operate in concert with current resources, continuity in low SES over several life-course periods is likely to entail the greatest health burden. Further, consistent with the CA mechanism, we find that the adverse effects of childhood SES and adolescent adiposity among women increase with age such that women with high and low childhood SES become more divergent with respect to body weight during the transition from midlife to old age. In contrast, early-life SES and adolescent body mass are unrelated to BMI change in men. Thus, our findings indicate that early-life SES is a stronger predictor of widening disparities in body weight with advancing age among women, which suggests

that unfavorable body weight trajectories originating early in life may be less amenable to modification in adulthood among women than men.

5.1.3. The Pathway Model—Consistent with the pathway model, we find that early socioeconomic advantage and disadvantage initiate divergent socioeconomic and behavioral life-course sequences affecting body weight in later life. A large proportion of the effect of early-life SES is indirect and conveyed by men's and women's socioeconomic resources and healthy or unhealthy behaviors in adolescence, midlife, and early old age.

Socioeconomic pathway: SES in midlife and later life is a significantly stronger mediator of the effect of parents' SES on sons' than daughters' BMI because own SES is related stronger to men's body weight compared to women. SES in adulthood is more consequential for men's than women's body weight because preferences for body size and adherence to weight management are more polarized by SES among men than women. The symbolic meaning of body size in our culture varies across social groups. A larger body size may be desirable as a symbol of strength and masculinity among low-SES men for whom physical effort and stamina are often a part of daily work routine (Khlat et al., 2009). In contrast, given that a trim body is a salient symbol of higher social status, high-SES men may be willing to employ multiple resources associated with social advantage to maintain lean bodies (Phelan et al. 2010). Among women, the effect of adult SES on body weight is weaker than among men because physical attractiveness standards are more closely tied to thinness and more strictly enforced for women than men (Tovée and Cornelissen, 1999). Thus, women express a preference for thinness across the SES spectrum (Schieman et al., 2006).

Early health behaviors pathway: Early socioeconomic disadvantage increases body weight over the life course such that higher body mass in adolescence leads to higher BMI in midlife which in turn increases BMI in old age. This chain of risk is more significant among women than men. Not only is the effect of early-life SES on RBM stronger for women but also continuity of body weight is more characteristic of women than men. A possible explanation is a sex difference in fat metabolism because women are more efficient than men at energy conservation and its storage as fat (Wu and O'Sullivan, 2011). Another mechanism may be related to women's higher levels of the hormone leptin, which is produced by adipose tissue and helps fat mass to remain relatively constant during adulthood (Wu and O'Sullivan, 2011).

In contrast to early-life adiposity, sports participation in high school does not mediate the effect of early-life SES because greater involvement in high school sports is related to *higher* BMI in later life among men. One potential pathway linking men's exercise in adolescence to greater body weight in adulthood can be high energy intake, which is the greatest for more physically active men (Platz et al., 2003). In turn, higher energy intake is associated with higher circulating insulin and insulin-like growth factor IGF-1 levels (Fontana et al., 2006), which is positively related to BMI (DeLellis et al., 2004).

It is important to note gender differences in high school sports participation. Our measure is based on the number of varsity, intramural, and club sports. Women in our study were the

most likely to participate in intramural sports and the least likely to participate in varsity sports. Women outnumbered men in intramural sports but were barely represented in varsity sports. Although our measure of PA does not capture informal activity outside school, research among current cohorts of adolescents shows that girls are less likely to exercise on weekends than boys, and this difference is particularly pronounced in low-SES communities (Wardle et al., 2012). This finding underscores the importance of school sports; thus, our measure is likely to capture an important dimension of exercise in adolescence, especially among women.

Adult health behaviors pathway: Of all health behaviors, our findings emphasize the importance of PA, especially, vigorous exercise for conveying the effect of parents' SES on late-life BMI. Vigorous PA in 1993 and 2004 is a stronger mediator than light PA among both men and women. This finding points to the potential importance of interventions aimed at increasing the level of vigorous exercise. Such interventions may the most beneficial for women who are less likely to engage in vigorous PA than men (Hagströmer et al., 2007; Hardy et al., 2008). Interestingly, light PA is a more important mediator among women than men. Women engage less frequently than men in leisure-time exercise (Ham and Ainsworth, 2010) but are more likely to perform housework regularly, which contributes to women's consistent involvement in light PA. Moreover, our findings reveal the enduring benefits of vigorous exercise in midlife for weight reduction in later life. Because health behaviors unfold over the life course, health habits at earlier stages have long-term implications for later health outcomes (Umberson et al., 2010).

In contrast to PA, smoking *suppresses* the effect of parents' SES on offspring's body weight. Smokers have lower BMI than non-smokers but individuals from higher-SES family background are less likely to smoke in adulthood than their low-SES peers. Thus, when smoking is added to the model, the difference in BMI between persons with high- and low-SES parents becomes more pronounced. Finally, moderate drinking in 2004 mediates 10% of the effect of early-life SES on men's weight and 4% on women's weight. Our findings are consistent with studies showing that moderate drinking is related to lower weight compared to abstaining and heavy drinking. In turn, men and women from higher-SES families of origin are more likely to drink moderately than their low-SES peers. Interestingly, alcohol use in 2004 is a stronger mediator of the effect of early-life SES among men than women because parents' SES has a stronger effect on sons' than daughters' alcohol use. In other words, the gap in the likelihood of moderate drinking is greater between high- and low-SES men than between high- and low-SES women. Moreover, men from low-SES families are more prone to heavy drinking than their female counterparts.

5.2. Limitations and Future Research

The WLS sample comprises only White participants who completed at least a high school education, which makes it representative of two-thirds of this generation of Americans. Although there is sufficient variability in early-life SES because participants come from widely diverse socioeconomic family backgrounds, a potential limitation of this study is the absence of individuals who did not graduate from high school. Given that persons who are the most disadvantaged with respect to their own SES are not included, our findings are

likely to underestimate socioeconomic disparities in body weight. Further, we could not explore race and ethnic differences in the effect of early-life SES on adult BMI. This direction is important for future research because minority groups have both lower SES (Williams and Collins, 1995) and higher risk of overweight and obesity (Wang and Beydoun, 2007) compared to White individuals. Moreover, this study is based on one cohort born in 1939. Because the prevalence of obesity is higher in younger than older cohorts (Keyes et al., 2010), future studies should compare the life-course effects of SES on body weight in older and younger generations.

Because the WLS initially was not designed to study health and health behaviors, the measure of adolescent body mass was obtained decades later by coding facial adiposity in high school yearbook pictures taken in 1957. The validity and reliability of this measure is documented in previous research showing, in particular, that it is an excellent predictor of BMI in midlife and late life (Reither et al., 2009). Yet, given that facial characteristics may not always accurately reflect overall body mass, it would be ideal to have measures of participants' height and weight obtained in high school. Thus, the WLS has only limited information about health behaviors in adolescence (body mass and PA from high school yearbooks) and no measures of healthy lifestyle in childhood and young adulthood. For example, it would be advantageous to have such important information as patterns of alcohol use and smoking in adolescence and young adulthood. A detailed assessment of health behaviors is available in the WLS starting only in midlife. Future studies of SES and body weight should include information about health behaviors affecting obesity collected prospectively over the life course. Further, given a positive association of maternal and paternal body weight with their children's obesity (Martin, 2008), the lack of data on parents' BMI in 1957 is a potential limitation of our study.

6. Conclusion

This study elucidates the importance of long-term consequences of early-life SES as a fundamental cause operating across the life course. We expand existing research by incorporating an array of prospective measures of early-life SES, assessing the dynamics of SES, health behaviors and BMI across the life course, and estimating the direct and indirect effects of SES in adolescence on BMI in later life through multiple mediating pathways. Our findings emphasize the complexity of life-course mechanisms underlying the effect of SES in adolescence on late-life body weight. This study expands the current understanding of complex mechanisms generating divergent trajectories of body weight among different socioeconomic groups. This understanding can potentially improve effectiveness of policies and clinical approaches aimed at reducing social inequality in body weight among older adults.

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Research Highlights

- SES in adolescence is related negatively to body weight in midlife and later life.
- Early socioeconomic disadvantage has a stronger effect on women's than men's weight.
- Life-course health behaviors and SES partly mediate the effect of early-life SES.
- The direct effect of socioeconomic background persists net of all mediators.
- Policies addressing obesity in mid- and late life should adopt a life course approach.

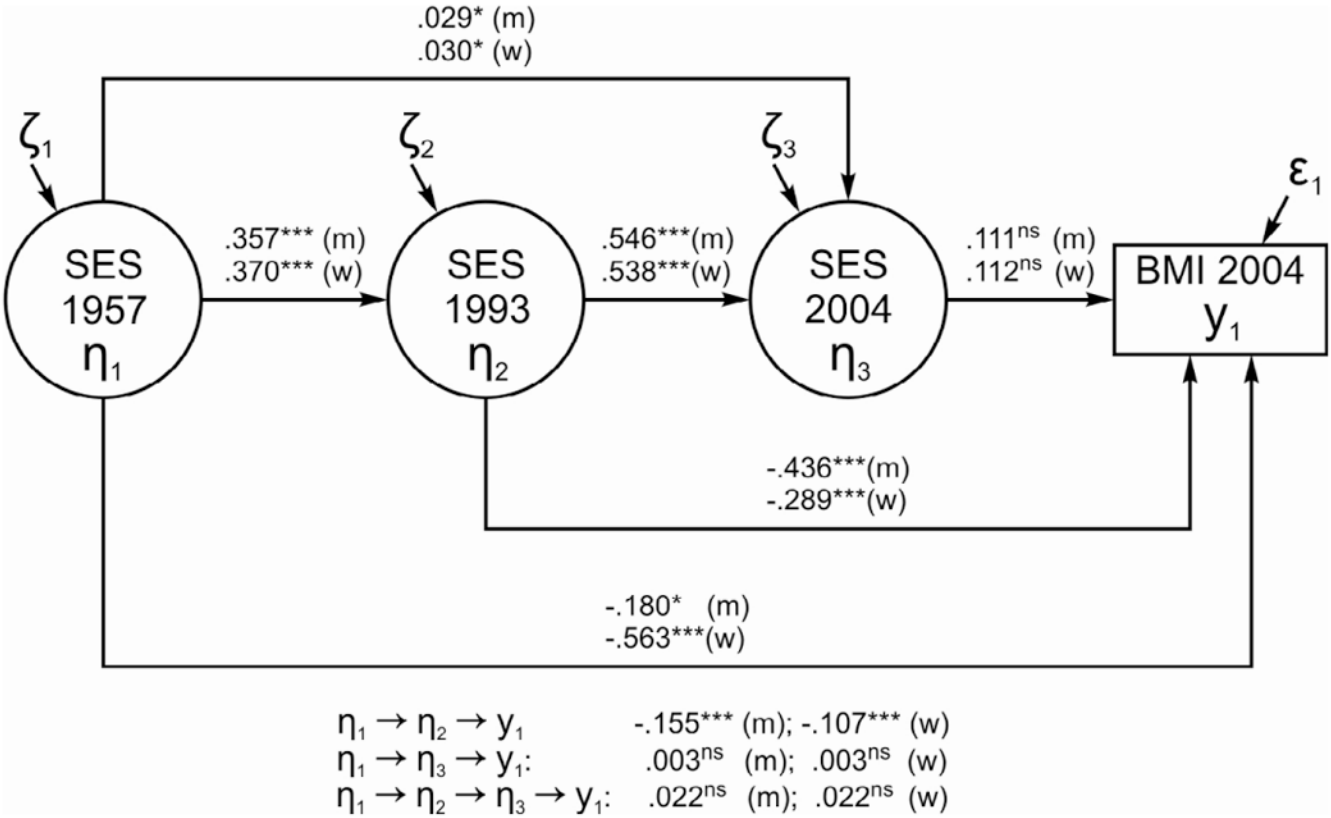

Fig. 1. The Effects of Socioeconomic Status over the Life Course on Body Weight in 2004

Note: m = men; w = women; ns = not significant at the .05 level. SES = socioeconomic status. BMI = body mass index. *p < .05. ***p < .001.

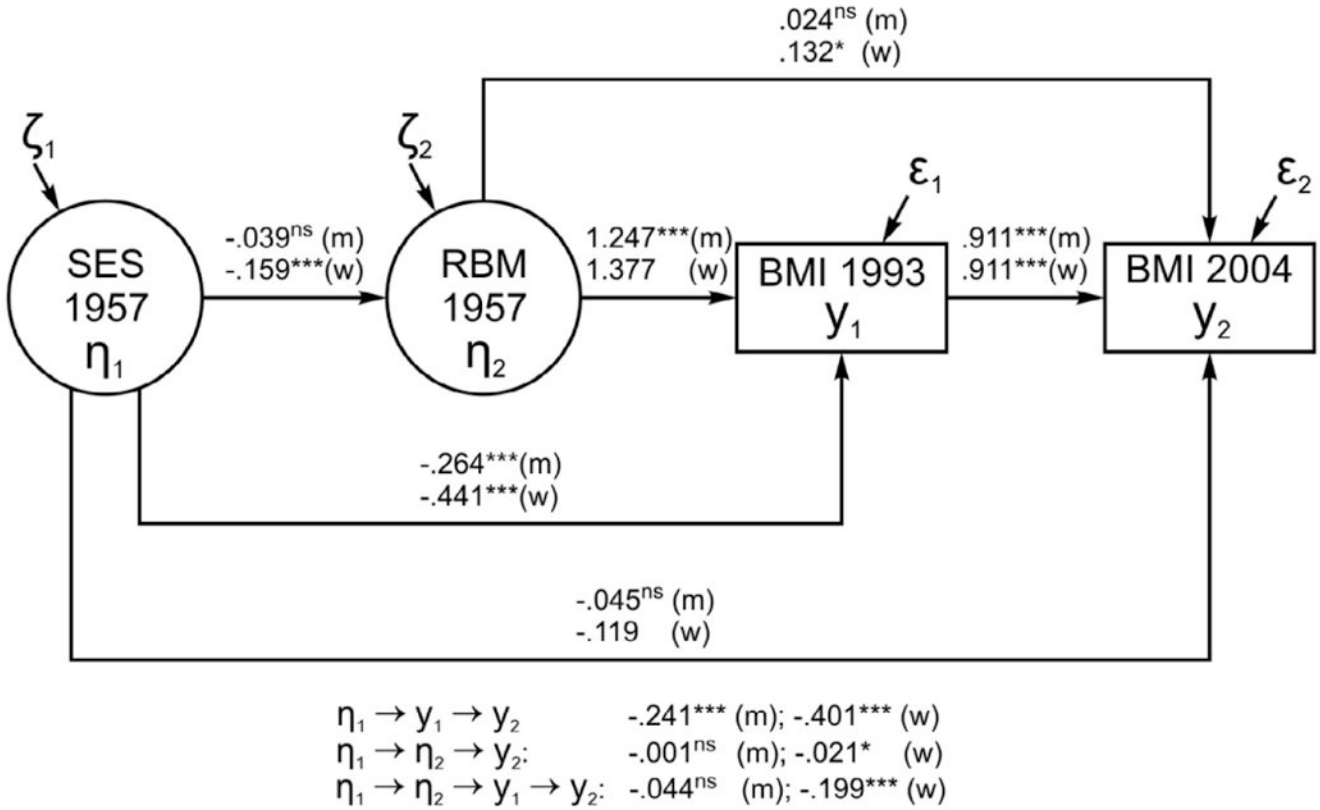

Fig. 2. The Effect of Socioeconomic Status in 1957 on Life-Course Trajectories of Body Weight

Note: m = men; w = women; ns = not significant at the .05 level. SES = socioeconomic status. RBM = relative body mass. BMI = body mass index. *p < .05. ***p < .001.

Table 1

The Measurement Part of the Structural Equation Model Estimating the Effect of Socioeconomic Status in 1957 on Body Mass Index in 2004

Indicators	Factors			
	SES 1957	RBM 1957	SES 1993	SES 2004
Father's education	.739 (.453)			
Mother's education	.623 (.611)			
Family income	.672 (.547)			
Father's occupation	.756 (.428)			
Father's occupational education	.835 (.302)			
Father's occupational income	.680 (.536)			
Eigenvalue 2.939, χ^2 (df) 136 (9), CFI .985, RMSEA .050				
Coder 1 ranking		.806 (.349)		
Coder 2 ranking		.777 (.396)		
Coder 3 ranking		.732 (.462)		
Coder 4 ranking		.813 (.337)		
Coder 5 ranking		.816 (.332)		
Coder 6 ranking		.653 (.573)		
Eigenvalue 3.950, χ^2 (df) 124 (9), CFI .96, RMSEA .047				
Education			.719 (.482)	.602 (.637)
Occupation			.866 (.249)	.664 (.558)
Occupational education			.862 (.256)	.775 (.398)
Occupational income			.792 (.371)	.652 (.574)
Wealth			.676 (.541)	.723 (.476)
Eigenvalue 2.703, χ^2 (df) 60 (5), CFI .992, RMSEA .044				

Note: Each cell contains factor loadings and residual variances (in parentheses). All factor loadings are significant at the .001 level. RBM = relative body mass; SES = socioeconomic status; df = degrees of freedom; CFI = the comparative fit index; RMSEA = the root mean square error of approximation.

Table 2

Decomposition of the Total Effect of Socioeconomic Status in 1957 on Body Mass Index in 2004 (β_{men} = -0.330, β_{women} = -0.740)

Mediators	Direct effect (β) of SES in 1957		Indirect effect (β) of SES in 1957		Ratio of indirect to total effects		Model fit[a]
	Men	Women	Men	Women	Men	Women	
Panel A: 1957 Variables							
Body mass	-.282**	-.541***	-.048	-.199***	.145	.269	1701 (215), .928, .050
High school sports	-.371***	-.732***	.041**	-.008	-.124	.011	903 (92), .924, .055
All 1957 mediators	-.286**	-.462***	-.044	- 278***	.133	.376	1580 (247), .937, .044
Panel B: 1993 Variables							
SES [b]	-.159*	-.634***	-.171***	-.106*	.518	.143	3137 (190), .929, .051
Light PA [c]	-.328***	-.688***	-.002	-.052***	.006	.070	801 (93), .932, .049
Vigorous PA [c]	-.215***	-.605***	-.79***	-.079***	.239	.107	829 (94), .928, .052
Smoking	-.328***	-.695***	.034**	.011	-.103	-.015	771 (93), .934, .050
Alcohol	-.331***	-.714***	.001	-.026**	-.003	.035	829 (93), .927, .052
All 1993 mediators	-.218*	-.483***	-.112*	-.257***	.339	.347	2857 (292), .918, .055
Panel C: 2004 Variables							
SES [b]	-.220***	-.678***	-.110**	-.062	.333	.084	1587 (184), .923, .051
Light PA [c]	-.339***	-.749***	.009	.009	-.027	-.012	943 (94), .929, .056
Vigorous PA [c]	-.282***	-.664***	-.012**	-.020***	.036	.027	932 (93), .928, .055
Smoking	-.316***	-.727***	.022	.042	-.067	-.057	914 (93), .919, .055
Alcohol use	-.298***	-.708***	-.032**	-.032**	.097	.043	880 (94), .923, .053
All 2004 mediators	-.191*	-.448***	-.139*	-.292***	.421	.395	3039 (296), .918, .056

Note: All models adjust for marital status, the number of children, and depressive symptoms in 2004 and the hazard of attrition.

* p < .05.

** p<.01.

*** p<.001.

[a] Indices of model fit include χ^2 and degrees of freedom in parentheses, the comparative fit index (CFI), and the root mean square error of approximation (RMSEA).

[b] SES = socioeconomic status.

[c] PA = physical activity.

Table 3

The Effects of Early-Life Socioeconomic Status and Mediators on Change in Body Mass Index between 1993 and 2004

	Men	Women	Model fit [a]
1957 Variables:			
SES [b]	-.048	-.163**	729 (86), .935, .055
Body mass	.025	.136*	1609 (246), .939, .045
High school sports	.011	.040	1015 (108), .924, .053
1993 Variables:			
SES [b]	-.222***	.073	1807 (214), .928, .051
Light PA [c]	-.032	-.032	1067 (134), .926, .051
Vigorous PA [c]	-.036	-.036	1120 (134), .931, .050
Smoking	.005	.005	1136 (134), .920, .051
Alcohol use	-.007	-.007	1188 (133), .924, .052

Note: All models adjust for marital status, the number of children, and depressive symptoms in 2004 and the hazard of attrition.

* $p < .05$.

** $p < .01$.

*** $p < .001$.

[a] Indices of model fit include χ^2 and degrees of freedom in parentheses, the comparative fit index (CFI), and the root mean square error of approximation (RMSEA).

[b] SES = socioeconomic status.

[c] PA = physical activity.