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Gender and Reinforcing Associations between Socioeconomic Disadvantage and Body Mass over the Life Course

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Abstract

Using the 1957–1993 data from the Wisconsin Longitudinal Study, we explore reciprocal associations between socioeconomic status (SES) and body mass in this 1939 birth cohort of non-Hispanic white men and women. We integrate the fundamental cause theory, the gender relations theory, and the life-course perspective to analyze gender differences in (a) the ways that early socioeconomic disadvantage launches bidirectional associations of body mass and SES, and (b) the extent to which these mutually-reinforcing effects generate socioeconomic disparities in midlife body mass. Using structural equation modeling, we find that socioeconomic disadvantage at age 18 is related to higher body mass index and a greater risk of obesity at age 54, and that this relationship is significantly stronger for women than men. Moreover, women are more adversely affected by two mechanisms underlying the focal association: the obesogenic effect of socioeconomic disadvantage and the SES-impeding effect of obesity. These patterns were also replicated in propensity score matching models. Gender and SES act synergistically over the life course to shape reciprocal chains of two disadvantaged statuses: heavier body mass and lower SES.

The past three decades in the United States have been characterized by an increasing prevalence of overweight and obesity, often referred to as the “obesity epidemic” (Reither, Olshansky, and Yang 2011). Despite the fact that the obesity increase levelled off in the 2000s (Rokholm, Baker, and Sorensen 2010), new models forecasting cohort trends suggest that obesity prevalence could rise to record-high levels as cohorts born in the 1980s grow older and enter the peak obesity ages (Reither et al. 2011; Robinson et al. 2013a). Although overweight and obesity have increased for all social groups since 1980, research consistently documents that socioeconomic disparities in body mass persist across periods and cohorts (Fowler-Brown et al. 2010; Wang and Zhang 2006). Low education, low income, and

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poverty are all associated with higher body mass index (BMI) and a greater risk of obesity (Drewnowski 2009; Sobal and Stunkard 1989). Moreover, the future growth of overweight and obesity may be more pronounced among lower-SES groups (Rossen and Schoendorf 2012).

The inverse association between socioeconomic status (SES) and body mass has been attributed to two mechanisms (Sobal 1991): (1) low SES increases the risk of overweight and obesity, and (2) elevated body mass impedes socioeconomic attainment (Drewnowski 2009; Glass, Haas, and Reither 2010; Parsons et al. 1999). Each of these mechanisms has been studied in two disparate strands of research. Some studies explored the short- and long-term effects of overweight and obesity on status attainment (Crosnoe 2007; Glass et al. 2010), whereas others examined the impact of SES on body mass (Drewnowski 2009; Giskes et al. 2008). Reciprocal relationships between body mass and SES over the life course have not received sufficient empirical attention.

The social determinants of overweight and obesity originate early in life (Parsons et al. 1999). Children from disadvantaged socioeconomic backgrounds are more likely than higher-SES children to be overweight and obese in childhood, adolescence, adulthood, and later life (Giskes et al. 2008; Langenberg et al. 2003). Because early-life SES also has a strong effect on socioeconomic standing in adulthood (Erikson and Goldthorpe 1992), socioeconomic family background may be a powerful force in launching mutually-reinforcing pathways of lower SES and higher body mass. Although the consequences of parents' socioeconomic resources for offspring's body mass are well-documented, existing research has notable limitations.

First, most studies of the effect of early-life SES on obesity – whether based on recent or earlier birth cohorts – follow individuals for a short period of time, typically into childhood, adolescence, or young adulthood (Hearst et al. 2012; Howe, Lawlor, and Propper 2013; Wang and Zhang 2006). Moreover, some studies begin in midlife and use retrospective reports of parents' SES (Giskes et al. 2008). Even studies that begin in childhood and use contemporaneous information on parents' SES overwhelmingly rely on one measure – father's occupational class (Langenberg et al. 2003; Salonen et al. 2009). In this investigation, we will explore the effect of early-life SES measured in adolescence with multiple indicators of parental socioeconomic resources in an older cohort that came of age before the obesity epidemic. We will also analyze whether the implications of childhood SES for body mass extend into midlife – a life-course stage when obesity tends to peak (Robinson et al. 2013a).

Second, the mechanisms underlying the association between early-life SES and later-life body mass are not well understood. Specifically, it is not known whether and how the reciprocal relationship between obesity and socioeconomic disadvantage mediates the effect of childhood socioeconomic environment on body mass in adulthood and later life. Although some studies exploring the effect of early-life body mass on individuals' own SES control for parents' SES (Clarke et al. 2010; Crosnoe 2007; Glass et al. 2010), existing research (a) does not specifically focus on early-life SES as a precursor launching reciprocal chains of disadvantage and (b) does not examine the bidirectional relationship between body

mass and SES over the life course as conveying the effect of socioeconomic family background. Our current study seeks to elucidate the complex chain of intertwined relationships.

Third, the observed associations between SES and body mass found in previous research may partly reflect unmeasured endowments of study participants. These endowments include genetic and epigenetic predispositions that are transmitted through families and may affect both SES and BMI, thus creating a spurious relationship. For example, socioeconomically disadvantaged individuals might also have genetic characteristics that lead to low basal metabolism (Baum and Ruhm 2009). In our study, we address unobserved heterogeneity by using propensity score matching and simulating the effects of unobserved covariates.

Using the 1957–1993 data from the Wisconsin Longitudinal Study (WLS), we explore reciprocal associations between SES and body mass over the life course in a cohort of 10,317 non-Hispanic white men and women born in 1939. Our study will shed light on the ways in which these life-course chains of adversity unfold differently for men and women. Specifically, we analyze gender differences in (a) the ways that early-life disadvantage gives rise to bidirectional life-course associations between body mass and status attainment, and (b) the extent to which these mutually-reinforcing associations generate socioeconomic disparities in body mass in midlife. We further estimate whether the focal and mediating relationships are robust to unobserved heterogeneity. Our empirical examination of these questions is guided by a theoretical framework integrating the fundamental cause theory, the gender relations theory, and the life-course perspective.

THEORETICAL FRAMEWORK AND EMPIRICAL BACKGROUND

According to the fundamental cause theory (Phelan, Link, and Tehranifar 2010), SES is a fundamental cause of population health disparities because socioeconomic position affects multiple diseases through a host of risk factors (Phelan et al. 2010). The effect of SES cannot be reduced to more proximal individual-level risks and behaviors because socioeconomic inequalities in health reproduce themselves even when intervening mechanisms change (Phelan et al. 2010). Moreover, SES facilitates or blocks access to a wide range of flexible resources that can be used strategically to maximize health under widely divergent conditions (Phelan et al. 2010). From the fundamental cause perspective, it is important to consider obesity at the individual level as an outcome of macrosocial stratification processes. The debate about the obesity epidemic in the United States has emphasized individual behaviors but downplayed issues of socioeconomic inequality (Drewnowski 2009). Yet, lower-SES groups have fewer resources to maintain healthy weight because of powerful social and “economic forces that are largely beyond their control” (Drewnowski 2009:S36). Thus, consistent with the fundamental cause theory, we emphasize the social origins of body mass and the interplay between individual outcomes at the micro-level and social and cultural forces at the macro-level (Sobal 1991).

Gender Relations Theory and Intersectionality

We emphasize the gendered nature of the relationship between socioeconomic disadvantage and body mass. Because the notion of fundamental causality focuses primarily on *between-SES* differences and generic SES processes, little attention has been devoted to *within-SES* heterogeneity (Miech et al. 2011). Yet, within SES, body mass varies substantially by gender and race/ethnicity, presenting a provocative challenge to the FCT (Miech et al. 2011). Therefore, it would be beneficial to integrate the FCT with research on intersectionality and multiple social dimensions of health inequality (Miech et al. 2011; Springer and Mouzon 2011). In this study, we elaborate the FCT by considering SES and gender as intersecting contexts.

According to gender relations theory, gender is a relational, ongoing, and negotiated process that is simultaneously emergent and institutionalized as a system of practices (Ferree 2010). As a pervasive social institution, gender is intertwined with other social realms. The intersectionality perspective emphasizes that gender is inseparable from other structural inequalities, including social class (Ferree 2010). Gender and SES define each other and synergistically affect individual outcomes, including health and health behaviors. To explore intersecting gender and SES inequalities, we apply a categorical approach that “focuses on the complexity of relationships among multiple social groups within and across analytical categories” and, thus, allows a joint and simultaneous examinations of both advantages and disadvantages (McCall 2005:1786).

Because of the intersection of gender and SES, socioeconomic disadvantage may have different implications for women’s and men’s body mass. Moreover, the cultural meanings of body weight are gendered, and the stigma of overweight and obesity is more severe among women than men (Khlat, Jusot, and Ville 2009). Women are also more likely to internalize negative attitudes with adverse consequences for their well-being (Sobal and Stunkard 1989). The bodies of women are scrutinized more than men’s because appearance is a central dimension of women’s social valuation. Obese women are evaluated more negatively and discriminated against more severely than obese men largely because the standards of attractiveness are considerably slimmer for women than men (Judge and Cable 2011). Thus, gender inequality can exacerbate the health risks of low SES as well as the social risks of obesity (Crosnoe 2007; Giskes et al. 2008; Glass et al. 2010; Langenberg et al. 2003).

Life Course: Historical Context and Long-Term Influences

By integrating the fundamental cause theory with the life-course perspective, we adopt a dynamic approach to SES and explore its development over the life course as well as the relative power of SES at different stages as a predictor of body mass. In this study, SES is considered as a trajectory characterized by long-term patterns of stability and change (Pearlin et al. 2005). The life-course perspective emphasizes that individual lives unfold within historical and sociocultural contexts reflected in the experiences of birth cohorts (Gee, Pavalko, and Long 2007). Cohort effects encompass cultural and social processes through which individuals sharing a birth year move together at a particular life-course stage

(Gee et al. 2007). Participants in this study were born in 1939 and entered adulthood in the 1950s and early 1960s.

The “obesity epidemic” in the US began in the 1980s (Robinson et al. 2013a). Our participants were already in midlife in the 1980s; thus, their formative years unfolded before the dramatic rise in obesity prevalence. Interestingly, among cohorts who came of age before the obesity epidemic, the WLS cohort born in 1939 had a greater prevalence of obesity than subsequent birth cohorts, such as Baby Boomers (Robinson et al. 2013b). This relatively high prevalence of obesity was true regardless of gender, but was especially pronounced among women (Robinson et al. 2013b). With respect to trends in the *relationship* between SES and obesity, the effect of SES on obesity was *stronger* in 1970s when our participants were young adults than after the start of the obesity epidemic. The SES gap narrowed but persisted when our cohort was middle-aged (Wang and Zhang 2006). In the 1960s and 1970s, the inverse relationship between SES and obesity was remarkably strong, especially among women (Sobal and Stunkard 1989). The risk of obesity was six times greater for low-SES women than their higher-SES counterparts (Goldblatt, Moore, and Stunkard 1965). Moreover, the social costs and stigma of obesity were already evident in the 1960s. Obese students, and particularly obese women, were more often denied college acceptance than average-weight peers. One mechanism responsible for this disparity in college admittance was discrimination against obese students by college admissions officials during in-person interviews (Canning and Mayer 1966). This research suggests that associations between SES and obesity have been persistent for several decades and are therefore relevant to both newer and older generations of Americans.

In addition to historical contexts, the life-course perspective focuses on long-term trajectories of individual development and enduring influences of past experiences. Moreover, the life-course approach underscores that health in later life cannot be explained solely by temporally proximate conditions because earlier conditions and characteristics have long-term implications for later well-being (Pearlin et al. 2005). Our theoretical framework suggests that there is a complex gendered mechanism linking socioeconomic family background to body mass in later life. Low SES early in life can increase the risk of overweight and obesity decades later by launching a reciprocal chain of low SES and heavy body mass over the life course. Below we specify each link of this chain with particular attention to gender differences.

Early-Life SES → Early-Life Body Mass → Adult Body Mass

The focal relationship between early-life SES and body mass in adulthood can be partly mediated by body mass in childhood and adolescence. Parents’ socioeconomic resources affect children’s body mass. Children from low-SES families are significantly more likely to be overweight and obese than their higher-SES peers as shown by poverty-income ratio (Wang and Zhang 2006). In turn, early-life body mass has enduring consequences for later body mass. Children with higher BMI have greater risks of overweight and obesity in adulthood and later life (Parsons et al. 1999). Highlighting the importance of gender, research documents that the adverse effect of socioeconomic disadvantage on body mass of children and adolescents is often (but not always) stronger among girls than boys, whether

measured by father's occupation (Giskes et al. 2008; Lagenberg et al. 2003) or parental education (Glass et al. 2010). The strong effect of socioeconomic disadvantage on girls' obesity was already evident in the 1960s and 1970s when our participants were young adults (Sobal and Stunkard 1989).

Early-Life Body Mass → Life-Course Status Attainment

Parents' SES is positively related to children's academic performance (Howe, Lawlor, and Propper 2013). Children's body mass is a potentially important mediator of this relationship. In the 1960s when many WLS participants sought higher education, obese students were significantly less likely to be accepted into college because of discrimination by admission officers during in-person interviews, which was particularly pronounced for women (Canning and Mayer 1966). This pattern has persisted across decades. Recent studies show that obesity in adolescence adversely affects academic performance in secondary school as well as the odds of high school graduation and college matriculation, especially among girls (Crosnoe 2007; Sabia 2007). In turn, high school academic performance can influence adult body mass both directly and – even more likely – indirectly through status attainment in adulthood. Higher achievement in secondary school is related positively to college attendance and entry into more selective colleges (Moller et al. 2010), which is a crucial step to socioeconomic advantage.

Moreover, being heavier in adolescence and young adulthood increases socioeconomic disadvantage in adulthood, including lower occupational standing and lower earnings (Conley and Glauber 2007). This effect is observed whether body mass is measured as continuous BMI (Cawley 2003; Conley and Glauber 2007; Judge and Cable 2011; Sabia 2007) or with categories for overweight and obesity (Cawley 2003; Crosnoe 2007; Glass et al. 2010; Sabia 2007). The adverse implications of obesity for socioeconomic achievement are particularly pronounced among women while being inconsistent or absent among men (Glass et al. 2010; Judge and Cable 2011). A recent study based on the WLS showed that women who were heavier in adolescence obtained less post-secondary education than thinner women, which had an adverse effect on occupational standing in adulthood (Glass et al. 2010). In contrast, early-life underweight rather than overweight was detrimental to occupational attainment among men (Glass et al. 2010).

Early-life SES → Adult SES

Socioeconomic disadvantage of the family of origin is associated with lower SES in midlife. Because of intergenerational transmission of unequal resources, opportunities, and constraints, parents' SES is positively associated with individuals' own SES, such as occupational status and income (Erickson and Goldthorpe 1992).

Adult SES ↔ Adult Body Mass

Whether early-life SES affects adult SES directly or indirectly via mechanisms such as early-life body mass or high school grades, individuals from disadvantaged social backgrounds tend to have lower SES themselves. In turn, adult SES has a consistently documented influence on obesity. Socioeconomic resources in adulthood are strongly and negatively related to body mass (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 (Sobal and Stunkard 1989). Midlife BMI can also affect midlife SES (Clarke et al. 2010); thus, BMI and SES mutually reinforce one another during this phase of the life course. Moreover, lower education and lower income in adulthood are more closely linked to higher BMI and obesity among women than men (Drewnowski 2009; Salonen et al. 2009).

SUMMARY

We evaluate a complex chain of adversity reflecting how (1) early socioeconomic disadvantage increases body mass in adolescence, (2) higher body mass in early life impedes status attainment over the life course, and (3) socioeconomic disadvantage in adulthood further increases the risk of overweight and obesity, with a possible nonrecursive effect of BMI on SES in midlife. Using structural equation modeling, we simultaneously estimate all links in this chain and decompose the effect of early-life SES into direct and indirect effects via each hypothesized mechanism. In addition, we apply propensity score matching on observed variables with simulation of unobserved heterogeneity to improve causal inference with respect to the reciprocal effects of body weight and SES. Compared to prior research, the strengths of our study also include its longitudinal design with over 50 years of follow-up, early-life characteristics assessed prospectively when participants were adolescents, an innovative measure of adolescent body mass based on high school yearbooks, and a detailed assessment of multiple dimensions of SES at two life stages.

DATA AND METHODS

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. Study participants were interviewed at ages 18 (in 1957), 36 (in 1975), 54 (in 1993), and 65 (in 2004). Our analytic sample contains 3,192 men and 3,683 women who participated in the 1957 baseline survey and in the 1993 phone and mail interviews. Retention of study participants is exceptionally high in the WLS. About 82% of the 1957 sample participated in the 1993 survey. Because of the regular links to the National Death Index, the vital status is known for 94% of the original participants as of 2011. Table 1 in Appendix A shows summary statistics comparing early-life variables in our analytic sample with the baseline 1957 sample; most variables are very similar in the two samples. Nevertheless, we conducted a detailed analysis to estimate how modest sample attrition could potentially influence our findings. Overall, early-life socioeconomic disadvantage is associated with a greater likelihood of sample attrition. Elevated body mass in high school is also a significant predictor of attrition because of its positive association with mortality. To take into account potential selection bias, we apply a propensity score matching approach. Using separate logistic regression equations for men and women, each person's observed early-life characteristics (parents' SES, high school grades, and adolescent body mass) were summarized into a composite propensity score reflecting a probability of sample attrition by 1993 due to mortality, refusal, or inability to locate. Gender-specific attrition propensity scores are included in each structural equation model.

Measures

In 1993 participants reported weight in pounds and height in inches. Our focal outcome *body mass index* (BMI) was calculated as $(\text{weight}/\text{height}^2) \times 703$ and was treated as a continuous variable with values ranging from 18 to 43. In addition to treating BMI as a continuous outcome, we use a binary indicator of obesity, coded 1 if BMI ≥ 30 . To assess possible differences between our sample and national estimates, we compared BMI at age 54 in the 1993 WLS to the BMI of non-Hispanic whites aged 50–60 in the 1988–1994 National Health and Nutrition Examination Surveys (NHANES) III. The measured mean BMI in NHANES III is 27.59 for men and 27.34 for women, which is very similar to the self-reported mean BMI in the WLS: 27.52 for men and 27.16 for women (Table 2, Appendix A).

The main predictors in our study are body mass and SES during adolescence. *Body mass in adolescence* is measured via the relative body mass (RBM) scale, coded from facial adiposity in high school yearbook pictures in 1957 (Reither, Hauser, and Swallen 2009). Six coders assigned RBM scores separately for boys and girls on scale ranging from 1 (the lowest) to 11 (the highest). Previous research has shown that the RBM measure is a valid and reliable indicator of body mass (Glass et al. 2010; Reither et al. 2009). The RBM scale fares well with respect to interrater reliability (Cronbach's $\alpha = 0.91$) and intrarater reliability, where test-retest correlations are 0.8 or higher for a majority of coders (Reither et al. 2009). RBM in adolescence is also a significant predictor of BMI, obesity, and health outcomes in midlife (e.g., hypertension, diabetes and premature mortality), demonstrating excellent criterion-related validity. In fact, adolescent RBM performs much the same as adolescent BMI in terms of its ability to predict body mass and health outcomes in later life. To incorporate measurement error, we use separate coder scores as indicators in all structural equation models. The measurement model for RBM is shown in Table 2, Appendix A. In addition to the continuous variable, we also use a binary indicator as a proxy for adolescent obesity, coded 1 if RBM $\geq$ the 75th percentile.

The measure of *academic performance* in high school is based on high school grades in 1957. *High school rank* is calculated as $100 - (\text{rank in class}/\text{number of students in class} \times 100)$.

Socioeconomic characteristics of the family of origin in 1957 include father's and mother's education 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). We also include measures reflecting father's occupational status: logged occupational education indicating the proportion of individuals in a given occupation who completed one or more years of college, and logged occupational income representing the proportion of individuals in a given occupation who earned more than \$10,000.

Socioeconomic status in 1993 includes education (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 of the current or last job, and occupational income of the current or last job, which in 1989 represented the proportion of persons in that occupation who earned at least \$14.30 per hour.

All SES measures in 1957 and 1993 were reverse-coded (while preserving their original scale) such that higher values correspond to greater socioeconomic disadvantage. Each inversely coded SES variable was used as an indicator in the measurement models (shown in Table 2, Appendix A) reflecting latent disadvantage constructs in 1957 and 1993. We also create binary measures of disadvantage in 1957 and 1993 coded 1 for individuals in the lowest SES quartile.

Control variables in 1957—We control for early-life variables that can affect both SES and health and, thus, potentially confound the relationship between parents' SES and offspring's body mass (Hayward and Gorman 2004; Hearst et al. 2012): whether a participant *grew up with both parents*, whether a participant lived in a *rural area*, whether a participant's *mother was employed outside the home*, the number of *siblings*, *religion* of parents (Catholic, Lutheran, other Protestant, other religion), father's *nationality* (German, Scandinavian, British, Eastern-Central Europe, other nationality), and father's *age* in 1957.

Control variables in 1993—We control for family variables that can confound the relationship between SES and BMI (Umberson et al. 2009; Umberson et al. 2011): marital status coded 1 for the *married* and 0 for the *unmarried*, and parental status measured as the total *number of children*.

Analytic Approach

We apply structural equation modeling (SEM) to examine reciprocal life-course effects of body mass (BM) and SES. The structural model estimates a direct path from early-life SES to BM at age 53. Further, we estimate potential indirect pathways via BM in adolescence, high school grades, and socioeconomic disadvantage in adulthood to formally test life-course mechanisms linking socioeconomic family background to midlife body mass. We decompose the total effect of early-life disadvantage into direct and indirect effects and calculate the proportion of the effect of early-life SES due to each mediator and more complex mediating pathways. We also use multiple-group analysis to examine whether path coefficients differ significantly for men and women. 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 statistics. To evaluate model fit, we use χ^2 , the comparative fit index (CFI), and the root mean square error of approximation (RMSEA).

Further, we apply propensity score matching (PSM) to take into account potential bias due to confounding factors that can affect both socioeconomic disadvantage and BM. PSM estimates the effect of disadvantage on BM by comparing low-SES individuals (the "treatment group") to higher-SES individuals (the "control group") who are as similar as possible on all observable characteristics, with the exception of SES. PSM is a two-step procedure. The first step is to estimate a propensity score – each person's propensity to be socioeconomically disadvantaged based on observed characteristics. The second step is to match the treatment and control groups on their propensity scores. Matched individuals are then compared in terms of BM to estimate the average treatment effect for the treated (ATT), i.e. group differences in body mass between the socioeconomically disadvantaged and matched controls that have similar propensities of being in the low-SES group on the

basis of observable characteristics. Following the same algorithm, we estimate the effect of obesity in adolescence on socioeconomic disadvantage in midlife. Variables used for matching are described in the note to Table 3.

A hidden bias arising from unobserved variables is a potential weakness of propensity score matching estimators. We address this problem with a sensitivity analysis based on the bounding approach proposed by Rosenbaum (2002). The sensitivity analysis simulates how strongly an unmeasured variable must influence selection into treatment in order to undermine conclusions of propensity score matching. An unobserved binary confounder (U) is simulated in the data based on its joint distribution with the treatment variable. U is used as any other covariate and is included in the set of matching variables used to estimate the propensity score and the ATT. A comparison of the simulated ATT and the actual ATT indicates the degree of robustness of the latter (Ichino, Mealli, and Nannicini 2008). To simulate the unobserved confounder U , we use the distributions of two variables that are strongly related both to early-life SES and early-life body mass: whether a person grew up with both parents or one parent (Parsons et al. 1999) and whether a person grew up in a rural or urban area (Bailey-Davis et al. 2012).

RESULTS

Summary statistics for all variables are shown by gender in Table 2, Appendix A. Factor loadings for each indicator reflecting the measurement part of the structural equation model are also presented in Table 2, Appendix A. Standardized factor loadings and fit indices suggest that the indicators measure each factor well. In our analysis, measurement models are specified to be identical for men and women because there was no significant improvement in model fit indices when the constraints of gender invariance in measurement models were relaxed.

Table 1 shows how the effect of early-life SES on midlife BMI changes with the sequential addition of each mediator. The residual total effect of socioeconomic family background net of early-life characteristics, marital status, and the number of children (Model 1) indicates that parental socioeconomic disadvantage is related to higher BMI in 1993 at age 54 among both men ($\beta = .338$, $p < .001$) and women ($\beta = .510$, $p < .001$). To illustrate, this effect size means that a standard-deviation increase in early-life SES disadvantage is associated with extra 15 pounds for a woman who is 5'5" tall and weighs 150 lb. and extra 10 pounds for a man who is 6 feet tall and weighs 190 lb. The effect of early-life SES is significantly stronger among women, as indicated by a substantial improvement in model fit ($p < .001$) for the less restrictive model with gender difference compared to the more restrictive model with gender invariance constraints.

Model 2 indicates that the direct effect of early-life SES on midlife BMI declines but remains statistically significant when adolescent relative body mass (RBM) is added to the model. The gender difference in the effect of early disadvantage disappears in Model 2 because adjustment for adolescent RBM substantially reduces the effect of early-life SES among women while changing this effect only modestly among men. Notably, RBM in adolescence is strongly and positively related to BMI in 1993 among both men and women.

Adjustment for high school grades in Model 3 further reduces the direct effect of early-life SES, although it remains statistically significant and large in magnitude. High school percentile rank is associated negatively with midlife BMI. Finally, with the inclusion of socioeconomic disadvantage in midlife, the direct effect of early-life disadvantage becomes not significant among women but still significant (although diminished) among men. The gender difference reemerges because midlife SES explains a larger proportion of the association between early-life disadvantage and midlife BMI among women than men.

Model 4 in Table 1 includes pathways that reflect complex chains of mediation. These pathways are presented in Table 2 and Figure 1 along with the decomposition of the total effect of early-life SES into direct and indirect effects via each mediator and chain of mediators.

As shown in Figure 1, socioeconomic disadvantage in 1957 significantly increases girls' body mass in adolescence ($\beta = .143, p < .001$). In contrast, early-life disadvantage is unrelated to boys' body mass. Therefore, body mass in adolescence is a strong mediator of the effect of early-life SES on women's BMI in midlife ($\beta_{\text{indirect}} = .187, p < .001$) and not a significant mediator for men. Table 2 indicates that adolescent RBM explains 35% of the effect of early-life SES among women and only 10% among men. Further, early-life disadvantage is associated with lower high school grades. In turn, academic performance in adolescence is related inversely to BMI in midlife. These pathways are similar for men and women; the indirect effect of early-life SES on adult BMI via high school performance does not differ significantly by gender: $\beta_{\text{women}} = .041$ and $\beta_{\text{men}} = .036$ ($p < .01$ for both). Not only does disadvantaged family background increase girls' RBM, but also higher body mass in adolescence has a large adverse effect on high school grades among girls ($\beta = -2.315, p < .001$) but not boys ($\beta = .701, \text{ns}$). Therefore, the indirect effect of early-life SES on midlife BMI via adolescent body mass and grades in high school is significant only among women ($\beta = .002, p < .05$). This indirect effect is modest in magnitude despite the strong adverse influence of girls' RBM on their grades because the direct effect of high school grades on midlife BMI is relatively small.

Moreover, early-life SES affects midlife BMI through the perpetuation of socioeconomic disadvantage over life course, although this effect is evident among women only. Figure 1 shows that socioeconomic disadvantage in 1957 is positively related to disadvantage in 1993 similarly for men and women, yet midlife SES has a strong effect on women's ($\beta = .325, p < .01$) but not men's ($\beta = .116, \text{ns}$) contemporaneous BMI. Among women, a one standard-deviation increase in socioeconomic disadvantage in 1993 increases adult BMI by one-third of a standard deviation. Thus, as indicated in Table 3, midlife disadvantage mediates 21.3% of the effect of early-life SES among women and only 11.7% among men. In addition, to explore reciprocal effects in midlife, we tested a pathway from BMI to SES. The model fit slightly (although not significantly) deteriorates when we specify a causal path from BMI to contemporaneous SES ($\chi^2 = 2, p > .05$). The effect itself is statistically significant ($\beta_{\text{women}} = .018, p < .01$, and $\beta_{\text{men}} = .013, p < .05$) but relatively small in magnitude. The effect is significantly stronger among women than men because the model fit improves from 2818 to 2812 ($\chi^2 = 6, p < .05$) when we allow this path to vary by gender.

Figure 1 also illustrates other ways that social disadvantage is perpetuated. RBM in adolescence is associated positively with socioeconomic disadvantage in midlife among women ($\beta = .074$, $p < .001$), and the indirect effect of early-life disadvantage on midlife SES via adolescent body mass is significant among women ($\beta_{\text{indirect}} = .003$, $p < .05$). This indirect pathway is relatively small in magnitude because the adverse effect of women's adolescent body mass on status attainment in adulthood is modest although highly statistically significant. Yet another chain reflecting the accumulation of socioeconomic disadvantage among women shows how low parental SES reduces high school academic performance, which in turn impedes status attainment—and lower SES in midlife has a direct adverse effect on women's contemporaneous BMI. While this chain mediates only 5.4% of the effect of early-life disadvantage among women, it is significant at the .01 level. Finally, the most complex mediating chain (disadvantaged background $\rightarrow$ higher adolescent body mass $\rightarrow$ lower high school grades $\rightarrow$ lower midlife SES $\rightarrow$ higher midlife BMI) is also significant among women ($\beta_{\text{indirect}} = .002$, $p < .05$). The longer the chain of indirect effects, the weaker those effects tend to be due to their multiplicative nature. The small coefficients simply reflect longer causal chains but do not undermine the importance of early-life SES.

Although the total effect of early-life disadvantage on midlife BMI is greater among women than men, it is the effect of early-life SES among men that is largely unexplained by all variables in the model ($\beta = .213$, $p < .05$) whereas the direct effect among women becomes not significant. This pattern reflects the fact that most mediating effects are stronger among women and, thus, all mediators combined explain a greater proportion of the focal association for women than men.

In addition to the models with continuous variables, we estimated reciprocal effects of SES and body mass using binary measures of socioeconomic disadvantage and obesity (Figure 2). A model with these binary variables produces very similar results to models with the continuous BMI and SES indicators. The effects of early-life disadvantage on adolescent and midlife obesity as well as the effect of early-life obesity on midlife socioeconomic disadvantage are observed among women but not among men. The effect of midlife disadvantage on midlife obesity is stronger for women than men. This consistency of patterns between models with continuous and binary variables provides evidence that our main findings are robust to different measurement choices for body mass and SES.

Propensity Score Matching

We use PSM with sensitivity analysis to take into account potential bias resulting from unobserved heterogeneity and to improve causal inference with respect to the reciprocal effects of SES and body mass. PSM estimates are shown in Table 3. Model 1 indicates that the effect of socioeconomic disadvantage in adolescence (SES $\geq 25^{\text{th}}$ percentile) has a strong effect on women's midlife BMI (ATT = .665, $t = 3.627$, $p < .001$). In contrast, there is no significant effect among men (ATT = .101, $t = .639$, $p > .05$). If there are unobserved covariates that affect selection into treatment and are distributed as family structure and rural residence, then ATT among women becomes .663 – very similar to the original ATT. These results provide no evidence of hidden bias due to unobserved heterogeneity.

Model 2 indicates that early-life socioeconomic disadvantage strongly and significantly increases women's body mass in adolescence, whereas the effect is negative, albeit not significant, among men. The original ATT among women ($ATT_{original} = .129$) is very similar to ATT with simulated unobserved covariates that have the same effects of early-life disadvantage as family structure and rural residence ($ATT_{simulated} = .124$).

Finally, Model 3 shows that obesity in adolescence (relative body mass 75th percentile) dramatically increases the risk of women's socioeconomic disadvantage in midlife ($ATT = .096$, $t = 5.703$, $p < .011$) but has no significant effect on men's SES ($ATT = -.006$, $t = -.275$, $p > .05$). The sensitivity analysis shows that the effect of adolescent obesity among women is robust to unobserved heterogeneity: $ATT_{original} = .096$ vs. $ATT_{simulated} = .102$.

DISCUSSION

Using the 1957–1993 data from the Wisconsin Longitudinal Study (WLS), we analyzed life-course associations between SES and body mass from socioeconomic disadvantage at age 18 to BMI and obesity at age 54. Our study combined two mechanisms explaining the negative relationship between SES and body mass: the obesogenic effect of socioeconomic disadvantage and the SES-impeding effect of obesity. We further explored and uncovered vast gender differences in both mechanisms. By integrating the fundamental cause theory with the life course perspective and the gender relations theory, we adopted a dynamic approach to the gendered reciprocal associations between SES and body mass.

Our findings indicate that socioeconomic disadvantage of the family of origin at age 18 is related to higher BMI and a greater risk of obesity at age 54, and the effect of early-life SES is significantly stronger among women than men. Our analysis of the mechanisms underlying this effect revealed that body mass and SES are simultaneously antecedents and consequences of each other over the life course via mutually-reinforcing patterns of effects. Obesity is not just a biological characteristic but also a disadvantaged social status (Carr and Friedman 2005). Therefore, exploring the mutually-reinforcing effects of two social disadvantages – lower SES and higher body mass – can shed further light on social origins of population health disparities.

First, early-life SES affects midlife obesity through the perpetuation of socioeconomic disadvantage among women only. Although low SES in 1957 increases socioeconomic disadvantage in 1993 similarly for men and women, midlife SES has a strong effect on women's but not men's body mass. Thus, socioeconomic disadvantage both in early and later life is more deleterious for women's than men's risk of overweight and obesity. Similarly, we found that the modest but statistically significant effect of higher BMI in 1993 on socioeconomic disadvantage in 1993 was more pronounced among women as well. Second, adolescent body mass is strongly and positively related to BMI in midlife among both men and women. Yet, while low early-life SES significantly increases girls' body mass in adolescence, it is unrelated to boys' body mass. Adolescent RBM explains 35% of the effect of early-life SES for women and only 10% for men. Third, higher body mass in adolescence has a large adverse effect on high school grades among girls but not boys. In turn, heavier weight in adolescence increases women's risk of socioeconomic disadvantage

in midlife both directly and indirectly through reduced academic achievement in high school. The effect of early-life disadvantage among women is significantly mediated by the path from adolescent body mass to academic performance and midlife SES. Overall, most pathways mediating the effect of early-life SES on midlife body mass are stronger among women than men; thus, all mediators combined explain a greater proportion of the focal association for women than men.

Life Course: Long-Term Consequences and Historical Context

Our findings contribute to a growing body of evidence documenting the long reach of the childhood socioeconomic environment for health in adulthood and later life (Hayward and Gorman 2004; Kuh et al. 2002; Pearlin et al. 2005). Because the fundamental cause theory typically focuses on social health disparities in adulthood, it is important to emphasize that unequal health 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. 2005:207). The present study suggests that, in addition to socioeconomic resources in adulthood, parental SES is another fundamental cause of health disparities operating through multiple mechanisms over considerable periods of time.

Given the pronounced society-wide changes in the population landscape of body mass, it is important to contextualize the main findings from our study in terms of the historical period trends in obesity. Our findings uncover the reciprocal effects of SES and body mass across the life course of individuals in the 1939 birth cohort. As our cohort matured from adolescence (late 1950s) to midlife (mid-1990s), *all* SES groups in the U.S. experienced increasing rates of obesity (Zhang and Wang 2004). Moreover, the disparity in obesity across low, medium, and high SES in the U.S. in general was *greater* when the WLS participants were adolescents and young adults and smaller when they were in midlife. The SES gap in obesity has decreased over the past three decades, especially among women (Zhang and Wang 2004). Among white women, the relative difference in obesity prevalence between the highest and lowest SES groups declined from 71% to 27% over the period 1970–2000. Among white men this relative difference also declined since 1970, from 33% to 14% (Zhang and Wang 2004).

The *life-course* trajectories observed in our study and recent *period* trends in obesity overlap to some degree, and both reflect the dynamics of body weight over time. For example, our findings indicate that SES disparities in obesity begin early in life, and persist through midlife. In a similar vein, studies of the U.S. obesity epidemic show persistent SES disparities at all ages in both earlier and later periods of observation (before and after the dramatic increase in obesity). Therefore, while the SES gap in obesity has narrowed in recent decades, this does not necessarily mean that SES inequalities have declined *within* birth cohorts that matured during this period of time. To the contrary, our study reveals an important pattern in our 1939 birth cohort: Despite the obesity epidemic and increase in the prevalence of obesity even among higher-SES individuals, socioeconomic inequality in obesity is just as strong in later life of the WLS cohort as it was in its earlier life course. An important direction for future research will be to explore life-course and period changes in the reciprocal effect of SES and body mass in other cohorts born before and after 1939 – our

participants' birth year. This shows the importance of clearly separating age, period, and cohort effects and understanding their interrelationships (Reither et al. 2011).

Moreover, because of gender inequality, gender and SES act synergistically as fundamental causes increasing women's vulnerability to socioeconomic disadvantage with respect to body mass. Girls born into socioeconomically disadvantaged families are exposed from early life to an unfolding chain of lower SES and higher body mass. This chain comprises many interconnected links and is already in full swing even in adolescence. Thus, our findings provide strong argument against the cultural stigma of obesity and the attribution of obesity to individual traits, such as poor self-restraint, gluttony, or laziness (Carr and Friedman 2005). Moreover, given the adverse impact of obesity on status attainment, obesity should be viewed not only as a health risk but also as a *social risk* and an axis of social disadvantage (Carr and Friedman 2005; Crosnoe 2007).

Our findings further underscore the centrality of the gender dimension by documenting that the reciprocal chain of disadvantage is stronger among women than men. Low-SES family background sets in motion women's life-course patterns of increased body mass, reinforced by persistent socioeconomic disadvantage. Women are more sensitive than men both to adverse effects of low SES on BMI and to adverse effects of obesity on status attainment. Below we discuss the potential mechanisms underlying these gender differences.

Gender Differences in the Effect of SES on Body Mass

Early-life SES can affect body mass through health socialization in the family of origin and intergenerational transmission of health behaviors related to energy balance, including physical activity. The WLS cohort graduated from high school in 1957 before the implementation of Title IX of the Education Amendments requiring equal athletic opportunities for girls and boys in schools. Given the lack of universal opportunities for girls' exercise, well-educated parents may have been more likely than low-SES parents to encourage daughters to exercise and instill in them the value of physical activity (Pudrovska and Anishkin 2012).

Moreover, early-life socioeconomic disadvantage can promote obesity through physiological stress-related processes (Bjorntorp and Rosmond 2000). For example, cohorts that experienced lower levels of economic stress early in life had a reduced risk of central obesity than subsequent cohorts who were subjected to more economic insecurity (Robinson et al. 2013b). 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). In addition to its direct physiological effect, the stress of socioeconomic disadvantage affects women's body mass indirectly via food intake. Women tend to increase food consumption more than men as a mechanism for coping with chronic stressors (Pasquali 2012).

Finally, gender differences in the effect of SES on BMI can reflect the cultural meanings of thinness and obesity. Socialization to be attentive to body size is more intensive and begins

earlier in life for girls than boys (McLaren and Kuh 2004). The desire to be thin increases with social class among adolescent girls, so it is unsurprising that actual weight is negatively related to SES (Sobal and Stunkard 1989). This trend continues into adulthood because the cultural norms of thinness as central to attractiveness tend to be accepted and internalized by higher-SES women, but not by lower-SES women or men regardless of SES (Sobal and Stunkard 1989). Women in higher social classes are generally more dissatisfied with their body size and shape than women of lower socioeconomic position, after adjusting for BMI (McLaren and Kuh 2004). Because thinness is a marker of social status, socioeconomically advantaged women are more concerned with weight and invest considerable effort in attaining a lean appearance, whereas socially disadvantaged women are less likely to aspire to thinness (Sobal and Stunkard 1989).

Gender Differences in the Effect of Body Mass on SES

Obesity can affect status attainment through macro-level cultural ideals and micro-level social psychological processes. Culture “allows for much less deviation from aesthetic ideals for women than it does for men” (Fikkan and Rothblum 2012:575). Obesity carries a strong cultural meaning of stigma among women, while being evaluated as a relatively neutral condition among men (Sobal and Stunkard 1989). Physical attractiveness standards are more closely tied to thinness and more strictly enforced for girls and women than boys and men (Crosnoe 2007; Tovée and Cornelissen 1999). Heavier women are also punished in the labor market (Glass et al. 2010) because wages and occupational attainment are positively related to physical attractiveness, and obese women are perceived as less attractive (Glass et al. 2010; Tovée and Cornelissen 1999). Indeed, the social cost of heavier body weight for white women begins below the conventional cutoffs for overweight and obesity. Gregory and Ruhm (2009) showed that wages for white women peaked at a BMI of 21.8, which is about 3 units below the clinical threshold for overweight. Similarly, the most severe wage penalty for women was associated with the transition from “thin” to the “average weight” category (Judge and Cable 2011). In contrast, if the penalty of weight in adulthood exists for men’ wages and occupational outcomes, it is smaller and evident only at the upper tail of BMI distribution (Judge and Cable 2011).

These findings further emphasize the distinction between body weight as a health risk and body weight as a social risk. Importantly, the social risks for women clearly begin at BMI levels that are healthy from a medical standpoint. Members of stigmatized groups, including obese persons, often internalize negative societal attitudes into their perceptions of self, and these negative self-perceptions appear to curtail life chances and impede status attainment (Carr and Friedman 2005; Crosnoe 2007). Women are far more likely than men to internalize the stigma of obesity (Sobal and Stunkard 1989). The stigma of obesity is related to self-rejection, academic failure, truancy, and substance use among high-school girls, and these psychological and behavioral processes in turn lead to lower college enrollment (Crosnoe 2007). Moreover, obese individuals are penalized in employment settings due to unfavorable stereotypes of heavier workers as less productive, which are stronger for women than men (Judge and Cable 2011).

Limitations and Future Directions

The WLS is almost entirely comprised of non-Hispanic white individuals who grew up in Wisconsin. It is conceivable that our findings are not unique to the WLS cohort, but may also apply to a large group of Americans that came of age during this era. The sample is broadly representative of older, white Americans who obtained at least a high school diploma, which is about two-thirds Americans in this generation. Yet, given the nature of the WLS, future research should incorporate other sociodemographic groups and, in particular, address race/ethnic differences in the reciprocal effects of SES and body mass (Cawley 2003).

Given that the prevalence of obesity has increased since the 1980s (Reither et al. 2011), an important direction for future research is to examine how the reciprocal relationship between obesity and SES differs in younger cohorts compared to older cohorts, such as the WLS. The ubiquitous presence of overweight and obesity may have promoted perceptions of larger bodies as more normative and less stigmatized, which could weaken the link between SES and obesity for recent birth cohorts. Alternatively, the stigma of obesity may have intensified, bolstered in part by the upsurge of medical concerns about health risks of overweight and obesity.

Although we document that the total effect of early-life disadvantage on midlife BMI is greater among women than men, it is the effect of early-life SES among men that remains largely unexplained by other variables in our study. This pattern reflects the fact that most variables we considered are stronger mediators among women. Thus, it is important to search for other mechanisms conveying the effect of early-life disadvantage among men. Finally, although we emphasize the reciprocal effects of two disadvantaged statuses—SES and obesity—future research should expand on our approach and incorporate the interplay between social constraints and individual agency. We also require deeper knowledge about conditions and resources that can promote resilience and counteract the adverse effects of a disadvantaged start in life. Understanding how to alter unfavorable life trajectories is essential for the design and implementation of effective policies and intervention programs.

Policy Implications

A central policy implication of our study is that deleterious outcomes associated with obesity could be limited by minimizing the extent of socioeconomic disadvantage and social inequality experienced during childhood. Once established, life course trajectories characterized by poor health and social disadvantage are difficult to modify through subsequent programs. However, research also shows that interventions initiated early in the life course can have long-term benefits for children from low-SES families (Reynolds et al. 2007). Programs and policies designed to reduce socioeconomic disadvantage in childhood promise to reduce rates of obesity in adolescence and midlife, improving the quality of life enjoyed by millions of Americans.

Our study also emphasizes the importance of policies and interventions addressing the socioeconomic penalties of obesity. It is necessary to increase public awareness of weight-based discrimination in the labor market and the ways in which employees' body weight

affects employers' decisions about pay and career advancement (Fikkan and Rothblum 2012; Judge and Cable 2011). Because obesity is not a protected status under federal law, promoting legal protection of overweight and obese persons from unfair treatment in the workplace is important, especially among women. Our study provides compelling evidence that policies and intervention programs should be attentive to gender differences, and incorporate life course perspectives. By combining all three theoretical approaches in programs that target early, malleable stages of the life course, it may be possible to prevent gendered life-course trajectories characterized by persistent socioeconomic and related health disadvantages.

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Figure 1.

The Reciprocal Relationship between Socioeconomic Disadvantage and BMI: The Wisconsin Longitudinal Study, 1957–1993, Women ($N = 3,138$) and Men ($N = 2,677$)^a

Note: Figure 2 is based on Model 4 in Table 2. w = women; m = men; ns = not significant.

*p < .05. **p < .01. ***p < .001.

Figure 2.
Binary Measures of Socioeconomic Disadvantage and Obesity

Table 1
Structural Equation Models Predicting the Effect of Socioeconomic Disadvantage in 1957 on Body Mass Index in 1993

Variables and Factors	Model 1		Model 2		Model 3		Model 4 ^a	
	Women	Men	Women	Men	Women	Men	Women	Men
<i>1957 Predictors:</i>								
Socioeconomic disadvantage	.510***	.338***	.331***	.303***	.268**	.249***	.132	.213*
Relative body mass			1.336***	1.370***	1.320***	1.354***	1.306***	1.342***
High school percentile rank					-.010***	-.010***	-.007***	-.007***
<i>1993 Predictors:</i>								
Socioeconomic disadvantage							.325***	.116
<i>Control Variables in 1957:</i>								
Single-parent family	.038*	-.016	.035*	-.015	.032*	-.015	.033*	-.017
Mother employed	.018	.003	.022	-.001	.021	-.002	.022	-.001
Rural residence	-.002	.011	-.015	.014	-.012	.017	-.015	.013
Number of siblings	.039*	.042*	.024	.043*	.025	.035*	.021	.033*
<i>Religion of parents:</i>								
Roman Catholic								
Lutheran	.025	-.036	.024	-.037	.024	-.028	.024	-.029
Other Protestant	.044*	-.031	.048**	-.024	.048**	-.018	.050**	-.017
Other religion	.001	.007	.001	-.002	.002	.001	.002	.002
<i>Father's nationality:</i>								
German								
Scandinavian	.023	.004	.023	.006	.023	.006	.025	.006
Great Britain	-.009	.012	-.005	.014	-.005	.015	-.002	.017
Eastern-Central Europe	.027	.022	.025	.011	.026	.012	.027	.012
Other nationality	.053**	-.005	.048**	-.006	.049**	-.008	.049**	-.009
Age of head of household	.010	-.011	.010	-.011	.010	-.007	.011	-.007
<i>Control Variables in 1993:</i>								
Married	-.118	-.118	-.202	-.149	-.191	-.141	-.122	-.122

Variables and Factors	Model 1		Model 2		Model 3		Model 4 ^a	
	Women	Men	Women	Men	Women	Men	Women	Men
Number of children	.206***	.206***	.210***	.210***	.206***	.206***	.198***	.198***
χ^2 (degrees of freedom)	495 (62)		900 (191)		1022 (215)		2812 (346)	
CFI ^b	.970		.967		.964		.908	
RMSEA ^c	.049		.036		.036		.050	

Note: Each cell contains standardized path coefficients (β) with asterisks denoting significance levels:

* $p < .05$.

** $p < .01$.

*** $p < .001$.

Highlighted in bold are coefficients that significantly differ for men and women ($\chi^2 = 3.84$).

^a Other paths in Model 4 are shown in Figure 2.

^b Comparative fit index.

^c Root mean square error of approximation.

Table 2

The Decomposition of the Total Effect of Socioeconomic Disadvantage in 1957 on Body Mass Index in 1993 into Direct and Indirect Effects ^a

Mediating Pathways	Indirect effect ^b		Indirect/Total	
	Women	Men	Women	Men
$\eta_1 \rightarrow \eta_2 \rightarrow \text{BMI}$	.187***	.036	.347	.103
$\eta_1 \rightarrow \text{High school rank} \rightarrow \text{BMI}$	.041**	.036**	.076	.103
$\eta_1 \rightarrow \eta_2 \rightarrow \text{High school rank} \rightarrow \text{BMI}$	.002*	.000	.004	.000
$\eta_1 \rightarrow \eta_3 \rightarrow \text{BMI}$	.115**	.041	.213	.117
$\eta_1 \rightarrow \eta_2 \rightarrow \eta_3 \rightarrow \text{BMI}$	.003*	.000	.006	.000
$\eta_1 \rightarrow \text{High school rank} \rightarrow \eta_3 \rightarrow \text{BMI}$	.029**	.009	.054	.026
$\eta_1 \rightarrow \eta_2 \rightarrow \text{High school rank} \rightarrow \eta_3 \rightarrow \text{BMI}$	.002*	.000	.004	.000
All mediating pathways via family statuses	.028***	.014*	.052	.040
Total effect: $\beta_{\text{women}} = .539^{***}$, $\beta_{\text{men}} = .349^{***}$				
Total indirect effect: $\beta_{\text{women}} = .407^{***}$, $\beta_{\text{men}} = .136^{**}$				
Total direct effect: $\beta_{\text{women}} = .132$, $\beta_{\text{men}} = .213^*$				

Note: Each cell contains standardized path coefficients (β) with asterisks denoting statistical significance of indirect effects:

* $p < .05$.

** $p < .01$.

*** $p < .001$.

All indirect effects, except for high school rank, significantly differ by gender at least at the .05 level ($\chi^2 = 3.84$). All models adjust for family structure, maternal employment, and rural residence in 1957 and marital status and the number of children in 1993.

^a Based on Model 4 in Table 2. All paths in Table 3 are illustrated in Figure 2.

^b Direct effect = total effect – indirect effect.

^c η_1 = Socioeconomic disadvantage in 1957.

^d η_2 = Relative body mass in 1957.

^e η_3 = Socioeconomic disadvantage in 1993.

^f BMI = Body mass index.

Propensity Score Matching (PSM) Estimates of the Reciprocal Effects between SES and Body Mass: The Wisconsin Longitudinal Study, 1957–1993, Women

Table 3

	SES disadvantage 1957 → Body mass 1993		SES disadvantage 1957 → Body mass 1957		Obesity 1957 → SES disadvantage 1993	
	Women	Men	Women	Men	Women	Men
Average outcome for matched treated	26.490	27.616	.063	-.005	.648	.396
Average outcome for matched controls	25.825	27.515	-.065	.037	.551	.402
Difference between the treated and controls (ATT ^a)	ATT ^c = 0.665 SE ^d = 0.183 t = 3.627	ATT ^a = 0.101 SE ^b = 0.158 t = .639	ATT ^a = 0.129 SE ^b = 0.034 t = 3.756	ATT ^a = -.044 SE ^b = 0.043 t = -1.024	ATT ^a = 0.096 SE ^b = 0.017 t = 5.703	ATT ^a = -.006 SE ^b = 0.020 t = -.275
N of treatment observations	1,294	1,019	1,294	1,019	1,319	1,001
N of matched treatment observations	1,294	1,019	1,294	1,019	1,319	1,001
N of control observations	1,251	1,056	1,251	1,056	1,228	1,078

Note: The PSM estimates shown in the Table 3 are based on the kernel matching estimators. Results from nearest neighbor and radius matching estimators (available upon request) are very similar. Treatment and control groups in *Model 1* are matched on all the following 1957 variables: body mass, family structure, maternal employment, rural residence, the number of siblings, religion, father's nationality, and father's age. Treatment and control groups in *Model 2* are matched on the same variables as in *Model 1* with the exception of body mass. Treatment and control groups in *Model 3* are matched on BMI in 1993 and the following 1957 variables: family structure, maternal employment, rural residence, the number of siblings, religion, father's nationality, father's age, parents' education, family income, father's occupation, and father's occupational education and income

^aThe lowest SES quartile.

^bRelative body mass 75th percentile.

^cAverage treatment effect for the treated.

^dStandard error.