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Article

Gender differences in the pathways from childhood disadvantage to metabolic syndrome in adulthood: An examination of health lifestyles

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ABSTRACT

We investigate whether socioeconomic status (SES) in childhood shapes adult health lifestyles in domains of physical activity (leisure, work, chores) and diet (servings of healthy [i.e., nutrient-dense] vs. unhealthy [energy-dense] foods). Physical activity and food choices vary by gender and are key factors in the development of metabolic syndrome (MetS). Thus, we examined gender differences in the intervening role of these behaviors in linking early-life SES and MetS in adulthood. We used survey data (n = 1054) from two waves of the Midlife in the U.S. Study (MIDUS 1 and 2) and biomarker data collected at MIDUS 2. Results show that individuals who were disadvantaged in early life are more likely to participate in physical activity related to work or chores, but less likely to participate in leisure-time physical activity, the domain most consistently linked with health benefits. Women from low SES families were exceedingly less likely to complete recommended amounts of physical activity through leisure. Men from low SES consumed more servings of unhealthy foods and fewer servings of healthy foods. The observed associations between childhood SES and health lifestyles in adulthood persist even after controlling for adult SES. For men, lack of leisure-time physical activity and unhealthy food consumption largely explained the association between early-life disadvantage and MetS. For women, leisure-time physical activity partially accounted for the association, with the direct effect of childhood SES remaining significant. Evidence that material deprivation in early life compromises metabolic health in adulthood calls for policy attention to improve economic conditions for disadvantaged families with young children where behavioral pathways (including gender differences therein) may be shaped. The findings also underscore the need to develop gender-specific interventions in adulthood.

1. Introduction

1.1. Social disadvantage, health lifestyles, and metabolic health

Nearly one third of U.S. adults meet the criteria for metabolic syndrome (MetS), a combination of abdominal obesity, insulin resistance, dyslipidemia, and elevated blood pressure. The risk of developing MetS substantially increases in later life, from around 20% of those between 20 and 39 years of age to half of those aged 60 or older (Aguilar, Bhuket, Torres, Liu, & Wong, 2015). MetS is a known risk factor for several leading causes of death in the U.S., including cardiovascular disease and type 2 diabetes mellitus (Cornier et al., 2008). The biological, behavioral, and social determinants of MetS have been studied extensively (Kaur, 2014). Health-related risk behaviors, in particular, contribute to the development of chronic disease and adult mortality in the U.S. Around 40% of deaths in the U.S. in 2000 were related to health behaviors, including smoking, poor diet, physical activity, and alcohol use (Mokdad, Marks, Stroup, & Gerberding, 2004). Regular physical activity and healthy diet—two potentially modifiable lifestyle behaviors—have been recommended for the prevention and treatment of MetS (Grave *et al.* 2010). Such lifestyle behaviors are embedded in social, cultural, and economic contexts that influence individuals' daily health practices (Bourdieu, 1984).

According to health lifestyle theory (Cockerham, 2005), structural characteristics such as gender, race/ethnicity, and SES shape the availability and appropriateness of health behaviors. Though individuals may have personal preferences in health practices, their resources or environments constrain the type, quantity, and quality of foods or physical activities that are available. Specifically, there is an inverse association between SES and consumption of unhealthy foods (e.g., high in refined grains, added fats, and added sugars), which are energy-dense and nutrient-poor (Darmon & Drewnowski, 2008). Some

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low SES individuals may be aware that reducing their consumption of unhealthy (i.e., energy-dense) foods is important for health, but reside in "food deserts" with limited access to healthy (i.e., nutrient-dense) foods. Financial limitations may further prevent regular consumption of healthy foods (e.g., lean meats, fish, fresh fruits and vegetables) that are more costly per calorie than unhealthy foods (Drewnowski & Darmon, 2005). Indeed, the inverse association between SES and obesity is partially explained by higher consumption of low-cost, energy-dense foods among low SES individuals (Drewnowski & Specter, 2004). In addition, socioeconomic disadvantage is associated with more occupation-related physical activity, but less leisure-time physical activity (Beenackers et al., 2012). Recent studies have shown that only leisure-time physical activity (and not occupational or household physical activity) is associated with better glucoregulation and non-diabetic status (Tsenkova, Lee, & Boylan, 2017). Some low SES individuals may wish to be more active, but are unable to do so due to extended working hours and unsafe exercise environments.

1.2. Childhood disadvantage and health lifestyles

Many practices related to diet and physical activity originate in childhood. Specifically, the social norms and environments that determine appropriate meal etiquette and the characteristics of preferred foods are largely formed during childhood (Winter Falk, Bisogni, & Sobal, 1996). Parents typically have high control over the dietary environments and choices of young children (Scaglioni, Arrizza, Vecchi, & Tedeschi, 2011). Parental SES likely shapes children's consumption of healthy vs. unhealthy foods (Darmon & Drewnowski, 2008). Children from low SES families are likely to consume more foods high in fat and sugar than children from high SES families (Xie, Gilliland, Li, & Rockett, 2003). Such early familiarity with unhealthy foods may influence nutrition patterns throughout the life course. Similarly, children in disadvantaged environments have lower levels of physical activity and are more likely to engage in sedentary behaviors (Drenowatz et al., 2010). Low SES families may not be able to pay for children's sports activities outside of school, and insecure neighborhoods may limit opportunities for physical activity (Crossman, Anne Sullivan, & Benin, 2006). Parents' exercise habits and attitudes about exercise are also positively associated with children's physical activity (Vander Ploeg, Maximova, Kuhle, Simen-Kapeu, & Veugelers, 2012). Low SES parents who themselves are physically inactive may be less supportive of children's engagement in leisure-time physical activity. Such sedentary habits, ingrained in childhood, may persist throughout adulthood and affect metabolic health in later life.

1.3. Gender differences

Experiencing socioeconomic deprivation in early life is linked with risk of developing symptoms and a diagnosis of MetS (Hostinar, Ross, Chen, & Miller, 2017). Most studies use gender as a control variable, thereby neglecting the possibility of differential consequences of early-life disadvantage for women vs. men. Nonetheless, a few studies have suggested that the inverse association between childhood SES and adult MetS is stronger and more consistent for women than men. Low childhood SES, for example, is associated with accelerated trajectories of high blood pressure from young adulthood to early midlife for women, but not men (Janicki-Deverts, Cohen, Matthews, & Jacobs, 2012). The adverse impacts of childhood SES on adult BMI are stronger among women than men (Giskes et al. 2008). Similarly, women, but not men, with low SES parents are at greater risk of developing MetS (Gustafsson, Persson, & Hammarström, 2011). Gender inequality and cumulative SES disadvantage may partially explain the association, yet the impact of early-life SES remains significant, particularly for women, even after controlling for adult SES.

Several studies have found that the mediating role of adult health behaviors (smoking, physical activity, alcohol use) in the association between childhood SES and Mets is relatively small (Chichlowska et al. 2009; Gustafsson et al., 2011; Schooling et al. 2008). Most studies have used dichotomous indicators or single domain assessments of physical activity and have rarely considered diet as a mediator, possibly due to data limitations. Distinct patterns of food preferences and physical activity may vary by gender and socioeconomic background. During childhood, girls like fruits and vegetables more than boys, whereas boys like fatty and sugary foods and processed meat products more than girls (Cooke & Wardle, 2005). In contrast, girls are less physically active than boys (Telford, Telford, Olive, Cochrane, & Davey, 2016). During adulthood men from low SES backgrounds are more likely to consume high calorie diets, while women across social classes are more concerned with healthy eating (Øygard, 2000). However, low SES women are exceedingly less likely than high SES women to participate in physical activity across different domains, except household chores (Ford et al., 1991). These findings suggest that gender is an important moderator, yet few studies have directly investigated how early-life SES affects health lifestyles for men vs. women.

1.4. Aims of the current study

Potentially modifiable lifestyle factors, ingrained in early life, influence the risk of developing MetS. The multifaceted nature of lifestyle behaviors varies by social class and gender; thus, single domains or dichotomous measures of health behaviors may not capture lifestyle behaviors rooted in childhood SES. Using comprehensive measures of health behaviors, with a focus on diet and physical activity, the first aim of this study is to investigate whether disadvantage in early life shapes health lifestyles in adulthood, in terms of physical activity in multiple domains (leisure, work, chores) and healthy (nutrient-dense) vs. unhealthy (energy-dense) foods. Second, we examine whether the effects of childhood disadvantage on comprehensive measures of diet and physical activity vary by gender. Finally, we investigate whether the intervening role of these behaviors in the association between childhood SES and MetS differs by gender.

2. Data and methods

2.1. Sample

Data were drawn from the Midlife in the U.S. Study (MIDUS), a national study of health and well-being. MIDUS began in 1995/96 with a sample of non-institutionalized, English speaking residents of the contiguous United States, aged 25–78 (n = 7108), recruited via random digital dialing (RDD) from the 48 contiguous states, siblings of the RDD sample, and a large sample of twins. Between 2004 and 2006, 4963 of the original respondents completed a follow-up telephone survey and self-assessment questionnaire (see Radler & Ryff, 2010 for retention details). Between 2004 and 2009, around 39% of those eligible for biomarker participation completed a two-day visit to a general clinical research center (GCRC) for a physical exam that included a fasting blood sample, and measurements of height, weight, waist–hip circumference, and blood pressure. Respondents participating in the biomarker project were more likely to have higher levels of education than those from the full sample in wave 2, but other sociodemographic characteristics (e.g., age, gender, race, marital status) were similar to respondents from the full sample (Love, Seeman, Weinstein, & Ryff, 2010). The study was approved by Institutional Review Boards at Georgetown University, University of California, Los Angeles, and University of Wisconsin–Madison. All participants provided written informed consent.

Our analytic sample includes 1054 respondents who participated in two waves of the MIDUS survey (MIDUS 1 and 2) and the biomarker project in MIDUS 2. Approximately 16% of respondents had missing data for at least one variable of interest. We implemented ten imputations to predict missing variables by generating imputed values,

including dependent variables, but then deleted observations with imputed dependent variables (Von Hippel, 2007).

2.2. Measures

Life-course SES. Childhood disadvantage is a summary score with a range of 0 through 10. Consistent with prior work (Lee & Ryff, 2016), we used four indicators from MIDUS 1 to create the score: parental education (0 = some college, 1 = high school graduate, 2 = less than high school), occupational prestige of parent using Duncan's Socioeconomic Index (0 = top 40%, 1 = 30–60%, 2 = no job or bottom 30%) (Hauser & Warren, 1997), welfare status (0 = never, 2 = ever), and financial level growing up (0 = better off than others, 1 = about the same as others, 2 = worse off than others). Following Gruenewald et al. (2012), we created an adult disadvantage summary score with a range of 0 through 10 using five indicators obtained in MIDUS 1: education level (0 = bachelor's degree or higher, 1 = some college, 2 = high school/GED or less), ratio of income to poverty line adjusted for family size (0 = 600% or more, 1 = 300–599%, 2 = less than 300%), current financial situation (0 = best possible, 1 = average, 2 = worst possible), availability of money to meet basic needs (0 = more than enough, 1 = just enough, 2 = not enough), and level of difficulty paying bills (0 = not at all difficult, 1 = not very difficult, 2 = very or somewhat difficult). The correlation between two SES summary scores is modest (r = .23).

Metabolic Syndrome (MetS) was measured during biological assessments in MIDUS 2. The National Cholesterol Education Program: Adult Treatment Panel III (2004) defines MetS by counting one's total number of the following symptoms: (1) abdominal obesity (waist circumference > 102 cm in men and > 88 cm in women), (2) hypertension (systolic pressure ≥ 130 mm Hg, diastolic pressure ≥ 85 mm Hg, or Rx to treat hypertension) (3) hyperglycemia (> 100 mg/dL, Rx to treat diabetes), (4) elevated triglyceride (TG) cholesterol levels (≥ 150 mg/dl, Rx to dyslipidemia) and (5) low high-density lipoprotein (HDL) cholesterol (< 40 mg/dl in men and < 50 mg/dL in women, Rx to dyslipidemia). Individuals who did not meet the criteria for both TG and HDL but take medications to treat dyslipidemia were coded as having an additional MetS component. We created two outcome variables for MetS: the number of MetS components (range of 0–5) and MetS diagnosis (whether respondents have 3+ MetS components).

Physical activity domains were included as part of medical history during the MIDUS 2 GCRC visit. Respondents were first asked if they engaged in any type of physical activity for 20+ minutes at least three times a week. Those who indicated "no" were classified as inactive. Those answering "yes" then provided up to seven types of physical activity, stating the duration, frequency, and intensity (light, moderate, or vigorous) of each type of physical activity. Uniform definitions of what constituted light (e.g., easy walking), moderate (e.g., light tennis), and vigorous activity (e.g., vigorous swimming) were provided to respondents. Data were converted to metabolic equivalent minutes per week (MMW) following established criteria: minutes per week of activity was multiplied by an intensity factor (Ainsworth et al., 2011). For example, MMW equals 1080 for an individual who participates in vigorous swimming (6 MMW) 3 days for week for 60 minutes per occasion.

The domain of physical activity was determined by referencing major activity categories within the Compendium of Physical Activity (Ainsworth et al., 2011). Specifically, work was determined by cross-referencing respondents' occupation indicated in MIDUS 2. Activities that fit in the major categories of home activities and home repair were classified as chores. All other activities were considered leisure. We created dichotomous measures (yes/no) to identify *presence of moderate or vigorous activity* in each domain and continuous measures (MMW) to identify *amount of activity* in each domain with a 3 SD winsorization. Following the Physical Activity Guidelines for Americans (USDHHS, 2008), we also created a categorical variable in each domain: 1 = inactive/light activity, 2 = low activity (< 500 MMW), 3 = medium activity (500–1000 MMW), and 4 = high activity (> 1000 MMW).

Diet. During the GCRC visit, respondents reported their frequency of consuming the following three items in an average day: fruits/vegetables, whole grains, and sugary beverages. They also reported their consumption of the following five items in an average week: ocean fish, lean meat (e.g., white meat chicken, lean beef, pork), high-fat meat (e.g., fried chicken, ribs, sausage), non-meat protein (e.g., eggs, tofu, nuts), and fast food. The scale of measurement varies by item; for example, a scale of 1 = none to 5 = five or more servings/day for fruits and vegetables, while a scale of 1 = none to 5 = five or more servings per week was used for ocean fish, beef, high-fat meat, or non-meat protein foods. We created two diet indexes. Healthy foods represent summed servings (range of 5 to 25) of five nutrient-dense items: fruits/vegetables, fish, whole grains, lean meat, and non-meat protein. Unhealthy foods were the summed servings (range of 3 to 15) of three energy-dense items: sugary beverages, high-fat meat, and fast food. The correlation between the two indexes was moderate (.16 for women and .20 for men), indicating different dimensions of eating behaviors in an individual's overall diet.

Covariates. We included confounding variables at MIDUS 2. First, poor sleep quality and mood disorders can elevate the risk of MetS (Goldbacher, Bromberger, & Matthews, 2009). As such, we included sleep problems measured by the Pittsburgh Sleep Quality Index (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989) and depressive symptoms measured by a seven-item scale (Wang, Berglund, & Kessler, 2000). Second, health risk behaviors include smoking (never, formerly smoked, or current smoking) and heavy drinking (4+ drinks per day). Finally, biodemographic controls include age, race/ethnicity, marital status, parental history of diabetes or high blood pressure, and menopause status (yes/no for women only).

2.3. Analytic approaches

We performed three sets of analyses. First, we conducted bivariate analyses to evaluate gender differences in all variables (Table 1). Second, we used multivariate regression models to investigate the extent to which childhood disadvantage was associated with the two lifestyle behaviors. Approximately 30% of respondents did not participate in any domain of physical activity at moderate or vigorous levels. Using a two-part model approach (Belotti, Deb, Manning, & Norton, 2015), we analyzed two mixed discrete–continuous outcomes: (a) participation vs. non-participation and (b) amount of moderate or vigorous activity among participants (Table 2). We fit a logit model in the binary part and an ordinary least square (OLS) regression model with a log-transformed dependent variable in the second part. For diet, we used OLS regression models to predict effects of childhood disadvantage on servings of healthy and unhealthy foods (Table 3). For both health lifestyle variables, we investigated whether the effects of childhood disadvantage remain statistically significant even after adjusting for adult disadvantage (Model 1 vs. Model 2, Tables 2 and 3). To better compare effect size, variables for disadvantage in childhood and adulthood were standardized (to mean = 0, SD = 1) based on the pooled distribution (both genders combined).

Consistent with prior work (e.g., Lee, Tsenkova, & Carr, 2014), we used a set of OLS regression models to test the extent to which health lifestyles mediate the relationship between childhood disadvantage and the number of MetS components in adulthood (Table 4). The crude models estimated each predictor separately while the adjusted models included those mediators that were significant in the crude models to investigate the change in coefficients of childhood disadvantage. The contribution of mediators to MetS was evaluated by (a) the percentage of total effects explained by each mediator and (b) the significance of mediation effects using bootstrapping (95% CI). We used the KHB-method for nonlinear probability models (for MetS diagnosis) to compare the estimated coefficients of nested models (Kohler, Karlson, &

Table 1
Descriptive statistics by gender, means (SD) or proportions from the Midlife in the U.S. Study (MIDUS).

Variables	Women (n = 577)	Men (n = 477)	Gender Difference
Life-course SES			
Childhood disadvantage	2.96 (1.96)	2.88 (1.80)	$p = .52$
Adult disadvantage	4.94 (2.64)	4.16 (2.50)	$p < .001$
Metabolic syndrome			
Number of MetS components	2.08 (1.45)	2.63 (1.36)	$p < .001$
MetS diagnosis	.38	.56	$p < .001$
Health Lifestyles: physical activity and diet			
Participation in physical activity			
Leisure	.59	.54	$p = .11$
Work	.05	.16	$p < .001$
Chores	.17	.19	$p = .41$
Amount (MMW) of physical activity			
Leisure activity	951 (788)	1,114 (878)	$p = .02$
Work activity	2,253 (1,726)	2,816 (1,938)	$p = .18$
Chore activity	837 (732)	873 (802)	$p = .75$
Diet index			
Healthy foods[a]	.09 (1.00)	-.10 (.99)	$p < .01$
Unhealthy foods[a]	-.21 (.94)	.25 (1.02)	$p < .001$
Life-course confounders			
Harmful stress response			
Sleep problems[a]	.14 (1.05)	-.16 (.91)	$p < .001$
Depressive symptoms[a]	.13 (1.15)	-.16 (.75)	$p < .001$
Behavioral factors			
Never smoked	.34	.19	$p < .001$
Formerly smoked	.56	.70	$p < .001$
Current smoking	.10	.11	$p = .63$
Heavy drinking	.18	.39	$p < .001$
Biodemographic controls			
Age	54.64 (11.57)	56.00 (11.20)	$p = .06$
White	.93	.95	$p = .27$
Married/cohabitating	.68	.76	$p < .001$
Parental diabetes or hypertension	.67	.59	$p < .01$
Menopause	.70	–	–

Note. MetS = metabolic syndrome; MMW = metabolic equivalent minutes per week, calculated by multiplying three components of physical activity (intensity, frequency, and duration).

[a] Standardized at mean = 0 and SD = 1 based on the pooled distribution for both genders.

Holm, 2011). In supplementary analysis, we confirmed that the findings from OLS regression models were similar to those from Poisson regression (Table S1). Results using MetS diagnosis (Table S2) were also consistent with findings from Table 4. Due to space limitations, we present only results from MetS components. Table S3 summarizes the results of mediation analyses for both the number of MetS components and MetS diagnosis. Given that biological risk factors and the development of MetS differ by gender (Cornier et al., 2008), we created gender-stratified models. Gender differences were tested by pooling data from both genders and testing gender interaction terms.

3. Results

3.1. Descriptive statistics

Gender differences in each variable are presented in Table 1. Women have a lower number of MetS symptoms than men (2.1 vs. 2.6) and are less likely to be diagnosed as having MetS (38% vs. 56%). This gender difference is wider than that found in prior work (Cornier et al., 2008; Yang & Kozloski, 2011), which have indicated that the gap between men and women narrows and even reverses in later life. We also observed a reduced gender gap among older adults (not shown in Table 1). While men and women do not differ in their probability of participating in physical activity related to leisure or chores, men are more likely than women to engage in physical activity at work (16% vs. 5%). Among individuals engaging in moderate or vigorous levels of physical activity, men have higher levels of leisure-time activity than women (1114 MMW vs. 951 MMW). Relative to men, women consume more servings of healthy foods and fewer servings of unhealthy foods. Gender differences appear in various confounding factors; women have more sleep problems and depressive symptoms while men have a greater likelihood of smoking and heavy drinking.

3.2. Childhood SES and physical activity in adulthood

Fig. 1 illustrates that both men and women from high SES families are more likely to participate in moderate or vigorous physical activity than those from low SES families (see pie charts). Among those who are moderately or vigorously active, those from high SES families perform a greater percentage of physical activity during leisure, but not work or chores, than those from low SES families (see bar charts). Table 2 shows that the associations between childhood SES and physical activity in adulthood are statistically significant for the odds and the amount of participation in leisure-time activity, but not work or chores. For women, each SD increase in childhood disadvantage is associated with 31% lower odds of participating in leisure activity (OR = .69) and a 16% decrease in the amount of moderate or vigorous leisure activity (Model 1). The effects of childhood disadvantage on both the odds and amount of participation in leisure activity remain statistically significant after adjusting for adult SES. For men, childhood disadvantage is significantly associated with lower odds of participation in leisure activity (OR = .67), but not amount of leisure activity. Fig. 2 shows the association between childhood SES and a categorical measure of leisure-time activity. Among individuals who engage in physical activity during leisure, women from low SES families are notably less likely to complete recommended amounts of physical activity through leisure (500+ MMW) than women from high SES families (43% vs. 25%). No difference was observed for men.

3.3. Childhood SES and diet in adulthood

Table 3 shows the results of OLS regression models for diet. For men, childhood disadvantage is negatively associated with consumption of healthy foods ($\beta = -.17$) and positively associated with consumption of unhealthy foods ($\beta = .18$). Both coefficients remain statistically significant after adjusting for adult SES. For women, the effects of childhood disadvantage on the two diet indexes are attenuated and no longer significant when adult SES is added to the model. The effects of childhood disadvantage on both diet variables appear stronger for men than women (Model 2). Tests of gender interaction were only marginally significant. Fig. 3 illustrates how patterns of diet vary by childhood disadvantage and gender. Men from high SES families tend to consume more servings of healthy foods (.16 SD above the mean) while those from low SES families tend to consume fewer servings of such foods (.18 SD below the mean). Similarly, compared to the average individual, consumption of unhealthy foods is higher for men from low SES families (.35 SD above the mean) than from high SES families (.04 SD above the mean). Significant differences across childhood SES were not observed for women.

3.4. Health Lifestyles as intervening processes between childhood SES and MetS in adulthood

Table 4 displays a series of nested models to investigate mediating roles of health lifestyles by comparing the percentage changes in the association between childhood SES and MetS that were explained by

Table 2
Two-part model predicting the odds and the amount of participation in physical activity among US adults, by domain and gender.

	Leisure		Work		Chores	
	Probability Logit OR	**Amount** OLS Coef.	**Probability** Logit OR	**Amount** OLS Coef.	**Probability** Logit OR	**Amount** OLS Coef.
Women (n = 577)						
Model 1:						
Childhood disadvantage	.69***	-.16**	1.30	-.10	1.12	-.07
Model 2:						
Childhood disadvantage	.78**	-.15**	1.15	-.14	1.18	-.04
Adult disadvantage	.65***	-.07	1.63*	-.16	.86	.13
Men (n = 477)						
Model 1:						
Childhood disadvantage	.67***	-.10	1.19	-.24	1.11	.05
Model 2:						
Childhood disadvantage	.72**	.08	1.06	-.28	1.09	.04
Adult disadvantage	.70**	-.07	1.62**	.21	1.07	.04

Note. All models were adjusted for age, race, and marital status. We fit a logit model in the first part (probability of engaging in moderate or vigorous physical activity) and an OLS regression model in the second part (amount of moderate or vigorous physical activity). OR = odds ratio; Coef = coefficient.
* p < .05.
** p < .01.
*** p < .001.

Table 3
OLS regression models predicting effects of life-course SES on servings of healthy vs. unhealthy foods among US adults.

	Healthy foods Standardized Coef.	**Unhealthy foods** Standardized Coef.
Women (n = 577)		
Model 1:		
Childhood disadvantage	-.10*	.07*
Model 2:		
Childhood disadvantage	.05	.02
Adult disadvantage	-.15***	.17***
Men (n = 477)		
Model 1:		
Childhood disadvantage	-.17**	.18***
Model 2:		
Childhood disadvantage	-.15**	.18***
Adult disadvantage	-.04*	-.00

Note. All models were adjusted for age, race, and marital status.
* p < .05.
** p < .01.
*** p < .001.

mediators. For women, for each additional SD increase of childhood disadvantage, the number of MetS components increases by .24 in the crude model. In models that control for adult disadvantage and confounders, introducing leisure-time physical activity decreases the coefficient for childhood disadvantage by 24% (from .19 in Model 2a to .15 in Model 2b). However, there is little change in the coefficient of childhood disadvantage when a diet indicator is included (Model 2c). The direct effect of childhood disadvantage on MetS remains statistically significant even after controlling for both mediators, while the effect of adult disadvantage is no longer statistically significant (Model 2d). The indirect pathway from childhood disadvantage to MetS through leisure activity is statistically significant (see Table S3).

For men, for each additional SD increase of childhood disadvantage, the number of MetS components increases by .19. The coefficient remains marginally significant after adjusting for adult disadvantage and life-course confounders in Model 2a (p = .07). After including either lifestyle mediator, the coefficient decreases dramatically and is not statistically significant. Specifically, leisure activity explains 36% of the association between childhood disadvantage and MetS (Model 2a vs. Model 2b) while diet accounts for 44% of the association (Model 2a vs. Model 2c). Results of mediation analyses for the indirect effects via both mediators are statistically significant (see Table S3). After controlling for both health lifestyle mediators (in Model 2d), the effect of childhood disadvantage is close to zero and not statistically significant for men, while adult disadvantage maintains a modest effect on MetS components.

4. Discussion

Life-course models have been posed to explain pathways through which early exposures increase an individual's risk of MetS. Based on health lifestyle theory (Cockerham, 2005), we investigated behavioral pathway models with a focus on diet and physical activity. To our knowledge, this is the first study to investigate gender-specific life-course pathways from childhood SES to adult MetS via comprehensive measures of diet and physical activity. Our study yields five main findings. First, early-life environments influence participation in various domains of physical activity in adulthood as well as amount of leisure-time physical activity. Individuals from low SES families are more likely than those from high SES families to be sedentary or to engage only in light physical activity. Among those who are moderately or vigorously active, those from low SES families are more likely to be physically active through work or chores than leisure. Regular physical activity improves health in general (USDHHS, 2008), but our findings show no significant effect of work-related physical activity on MetS. This finding converges with prior evidence that physical activity during work was not associated with a reduced risk of diabetes (Tsenkova et al., 2017). Manual labor is typically highly repetitive and of long duration, with little control over the amount of work that must be done, and the stress that accompanies physically demanding jobs may offset some of the health benefits (Johnson & Hall, 1988). These findings suggest that health interventions need to monitor the domain of physical activity for best results, particularly for individuals from disadvantaged backgrounds.

Second, women from low SES families are much less likely to meet physical activity requirements. Structural factors pertaining to different life opportunities and options may play roles in daily exercise practices.

Table 4
OLS regression models predicting effects of life-course SES and health lifestyle on number of MetS components among US adults, by gender.

	Women					Men				
	Crude models		Adjusted models			Crude models		Adjusted models		
	Model 1	Model 2a	Model 2b	Model 2c	Model 2d	Model 1	Model 2a	Model 2b	Model 2c	Model 2d
Life-course SES										
Childhood disadvantage	.24***	.19**	.15**	.19**	.15*	.19**	.13[b]	.08	.07	.05
Adult disadvantage	.25***	.14**	.10	.12[b]	.07	.26***	.22**	.18**	.22**	.19**
Physical activity										
Leisure[a]										
Low (< 500 MMW)	-.46**		-.36*		-.31	-.30		-.25		-.22
Medium (500–1000 MMW)	-.55**		-.35*		-.33**	-.62***		-.60**		-.51**
High (< 1000 MMW)	-.93***		-.72**		-.65*	-.78***		-.69***		-.56***
Work[a]										
Low (< 500 MMW)	.54					.79				
Medium (500–1000 MMW)	.54					.30				
High (< 1000 MMW)	-.18					-.13				
Chores[a]										
Low (< 500 MMW)	.18					.06				
Medium (500–1000 MMW)	-.07					.17				
High (< 1000 MMW)	-.23					-.09				
Diet										
Healthy foods	.04					-.13*			-.03	
Unhealthy foods	.27***			.22**	.18**	.29***			.28***	.23***

Note. Life-course SES and diet variables were both standardized (mean = 0, SD = 1) based on the pooled distribution. Crude models, which are separate models for each predictor, include biodemographic controls. In all adjusted models, we also added life-course confounding variables.
[a] Inactivity or light activity in each domain is the reference group. Model 2d includes potential mediators which were statistically significant in crude models.
[b] $p < .10$.
* $p < .05$.
** $p < .01$.
*** $p < .001$.

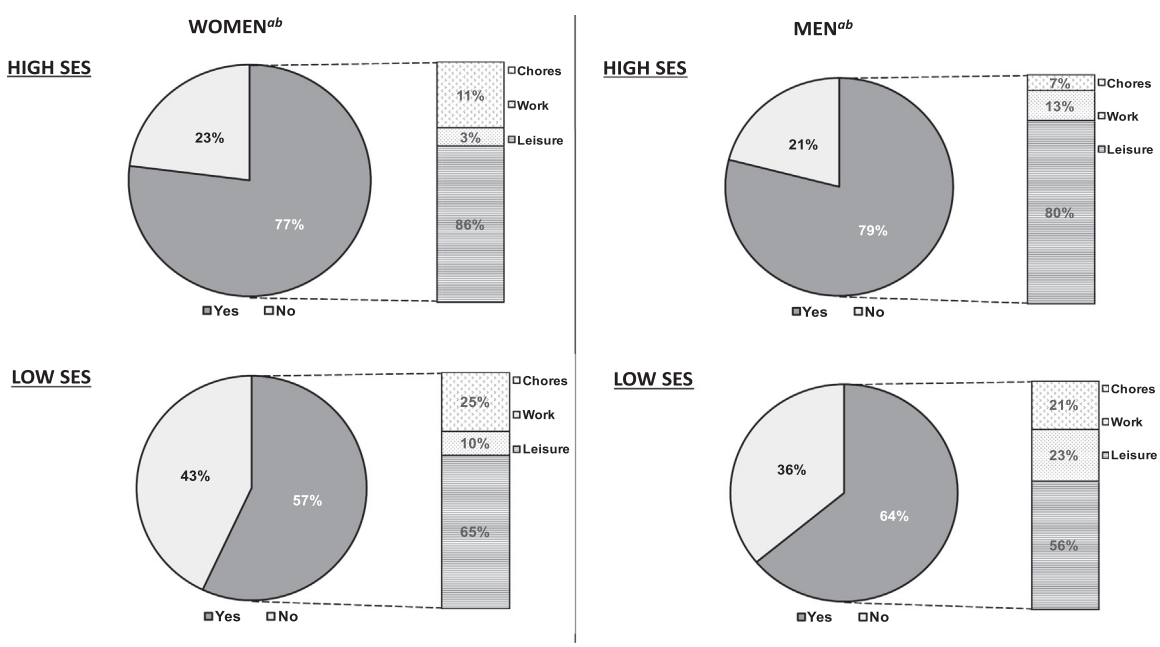

Fig. 1. Association between childhood disadvantage and physical activity among US adults, by domain and gender. *Note*. High SES = 0 or 1 score of childhood disadvantage. Low SES = 4+ score of childhood disadvantage. Pie charts represent percentage of respondents who engaged in moderate or vigorous physical activity in any domain. Bar charts represent the percentage of each domain of physical activity (moderate or vigorous) among those who participated in regular physical activity. [a]Indicates that participation in physical activity differs between high vs. low SES at $p \leq .05$. [b]Indicates that domains in physical activity differs between high vs. low SES at $p \leq .05$.

Women, compared to men, are more likely to have "contaminated" leisure-time, whereby housework and childcare responsibilities intrude on their free time (Craig & Brown, 2017). Indeed, low SES women are more likely than high SES women to be physically active via household chores than via leisure-time activities (McLaren, Godley, & MacNairn, 2009). Thus, our findings may reflect gender/SES inequalities in time for leisure-time physical activity. Further research is needed to understand how patterns of physical activity negatively impact women from low SES families.

Third, socioeconomic position in childhood shapes diet in adulthood

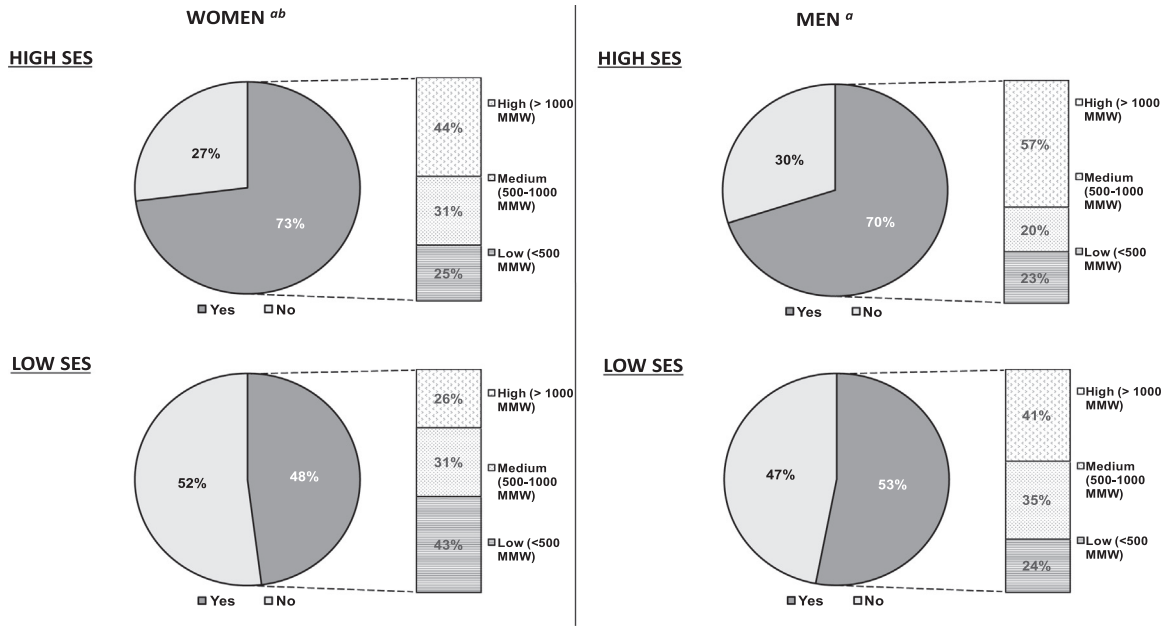

Fig. 2. Association between childhood disadvantage and leisure-time physical activity among US adults, by gender. **Note** High SES = 0 or 1 score of childhood disadvantage. Low SES = 4+ score of childhood disadvantage. Pie charts represent percentage of respondents who engaged in moderate or vigorous leisure-time physical activity. Bar charts represent the percentage of amount of leisure-time physical activity. [a]Indicates that participation in leisure-time physical activity differs between high vs. low SES at $p \leq .05$. [b]Indicates that the amount of leisure-time physical activity differs between high vs. low SES at $p \leq .05$.

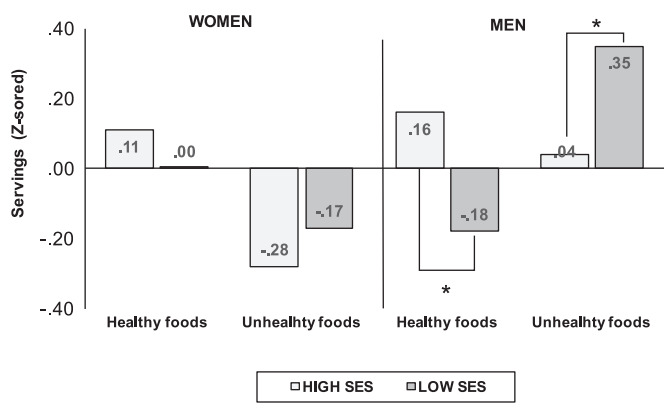

Fig. 3. Association between childhood disadvantage and diet among US adults, by gender. *Note*. High SES = 0 or 1 score of childhood disadvantage. Low SES = 4+ score of childhood disadvantage. The value of zero refers to mean consumption for the full sample; negative values indicate fewer servings than the mean; and positive values refer to more servings than the mean. *Indicates that the distribution differs by SES at $p \leq .05$.

for men. An inverse association between SES and consumption of unhealthy foods is well-known (Darmon & Drewnowski, 2008). Low SES individuals are more likely to consume a high-calorie diet characterized by heavy and fatty foods, while affluent individuals tend to consume a lower-calorie diet high in vitamins, minerals, and other micronutrients. Such differences by social class are rooted in differences in the accessibility of healthy vs. unhealthy foods as well as early socialization regarding foods: while high SES families emphasize healthy eating and food quality, low SES families must attend to the cost of foods, with less expensive foods tending to be energy-dense and nutrient-poor (Fielding-Singh & Wang, 2017). Our findings suggest that men from low SES families consume more servings of sugary drinks, high-fat meat, and fast food than men from high SES families and than women in general.

Fourth, the mediating role of diet and physical activity in the association between childhood SES and MetS varies by gender. For men, both leisure-time activity and servings of unhealthy foods largely explained the association. These findings support the life-course pathway model whereby early-life circumstances may have little direct effect on later-life health, but rather influence subsequent socioeconomic, psychological, and lifestyle trajectories that determine adult health outcomes (Ben-Shlomo & Kuh, 2002). Therefore, intervention strategies seeking to modify the health-risk behaviors of men from low SES families may be particularly effective at reducing the risk of MetS. In contrast, the mediating role of leisure-time physical activity for women was relatively small. The negligible contribution of diet in our study may indicate that adult SES, rather than childhood SES, plays a significant role in food choices.

Finally, the direct effect of childhood SES on MetS remains significant for women even after accounting for potential mediators and confounding factors. Prior studies have also found robust effects of early-life SES on MetS for women, but not for men, after controlling for adult covariates (Chichlowska et al., 2009; Gustafsson et al., 2011; Schooling et al., 2008). Possible explanations are worth considering. First, early life might be a more sensitive period for women than men, in terms of shaping metabolic health. The critical period model suggests that exposure to adverse environments during childhood may have lifelong effects on the structure and function of physiological systems, resulting in elevated risk of chronic diseases (Barker, 1990; Ben-Shlomo & Kuh, 2002). Early-life adversities might be associated with a key biological pathway (chronic inflammation) from obesity to metabolic syndrome (Monteiro & Azevedo, 2010), particularly for women (Senese, Almeida, Fath, Smith, & Loucks, 2009). In addition, women's life transitions, such as childbearing and menopause, may play a role in the association between childhood SES and development of MetS. Recent studies have proposed that reproductive history (early and repeated childbearing), which varies by social class, is involved in the association between childhood SES and women's cardiometabolic health (Gustafsson & Hammarström, 2012; Lee & Ryff, 2016). Using a longitudinal study, Montez, Bromberger, Harlow, Kravitz, and Matthews (2016) found that childhood SES shapes women's risk of MetS during pre/perimenopause but not after. We found a similar pattern in auxiliary analyses (Table S4), albeit with cross-sectional biomarker data.

5. Limitations and conclusions

Several methodological limitations should be noted. First, retrospective reports of childhood SES are susceptible to recall bias, although recall accuracy of childhood SES has been supported in twin studies (Krieger, Okamoto, & Selby, 1998). A review suggests that if retrospective reports of childhood SES on obesity cause bias, it is likely to be underestimation (Senese et al., 2009). Second, although our measures of diet and physical activity are a notable advance relative to prior studies, detailed reports on these variables and outcome were first collected during the GCRC visit. Major life transitions, such as retirement and illnesses, may change food and exercise habits (Helldan, Lallukka, Rahkonen, & Lahelma, 2012). Thus, repeated observations of health lifestyles are needed. To isolate the mediating effects of diet and physical activity from confounding factors, we included extensive covariates. However, omitted variables which are correlated with both the outcome and mediators may bias the estimation of the observed association in this study. Finally, the sample was limited in terms of racial/ethnic diversity (6% non-White); thus, our findings may not be generalizable to other racial/ethnic groups. Growing evidence suggests that SES-driven life course pathways might persist and be reproduced for future cohorts (Link et al., 2017). Such an issue should be tracked with samples collected across generations. Despite such limitations, our results echo prior findings that structural factors, such as income and education, are fundamental drivers of health disparities (Link & Phelan, 1995). Improving such upstream determinants is key to promoting health over the life course for socially disadvantaged children. Designing gender-specific behavioral interventions in adulthood may also be needed for individuals who experienced socioeconomic deprivation in early life.

Appendix A. Supplementary material

Supplementary data associated with this article can be found in the online version at http://dx.doi.org/10.1016/j.ssmph.2018.01.003.

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