Journals of Gerontology: Social Sciences
cite as: *J Gerontol B Psychol Sci Soc Sci*, 2016, Vol. 71, No. 6, 1097–1107
doi:10.1093/geronb/gbw014
Advance Access publication February 28, 2016

THE GERONTOLOGICAL SOCIETY OF AMERICA®

OXFORD

Original Article

Life-Course Socioeconomic Status and Metabolic Syndrome Among Midlife Women

Jennifer Karas Montez,[1] Joyce T. Bromberger,[2,3] Siobán D. Harlow,[4] Howard M. Kravitz,[5,6] and Karen A. Matthews[2,7]

[1]Department of Sociology and Aging Studies Institute, Syracuse University, New York. [2]Department of Psychiatry and [3]Department of Epidemiology, University of Pittsburgh, Pennsylvania.[4]Department of Epidemiology, University of Michigan School of Public Health, Ann Arbor. [5]Department of Psychiatry and [6]Department of Preventive Medicine, Rush University Medical Center, Chicago, Illinois. [7]Department of Psychology, University of Pittsburgh, Pennsylvania.

Correspondence should be addressed to Jennifer Karas Montez, PhD, Department of Sociology and Aging Studies Institute, 314 Lyman Hall, Syracuse University, Syracuse, NY 13244. E-mail: jmontez@maxwell.syr.edu.

Received August 17, 2015; Accepted February 5, 2016

Decision Editor: Deborah Carr, PhD

Abstract

Objectives: We examine whether women's risks of having metabolic syndrome (MetS) at pre/early-menopausal baseline, and of developing MetS after baseline, are associated with childhood and adult socioeconomic statuses (SESs); and whether the associations are mediated by adult reproductive, economic, behavioral, and psychosocial factors.
Method: Using data on white and black women collected prospectively for 12 years in the Study of Women's Health Across the Nation, we estimated odds of MetS at pre/early-menopausal baseline with logistic regression, and incidence of MetS after baseline with Cox proportional hazards models.
Results: Women raised in "adverse" childhood SES had marginally greater odds of MetS at baseline than did women raised in "good" SES, and women with a high school credential or less had significantly greater odds than college-educated women, in mutually adjusted models. The elevated odds partly reflected SES-related differences in exercise and alcohol consumption. Incidence after baseline was associated with education, not childhood SES, and partly mediated by health behaviors. Differences in the probability of surviving without MetS between the most and least socioeconomically advantaged women nearly doubled between ages 50 and 60.
Discussion: Childhood and adult SES predict women's risks of MetS as they approach the menopause transition; adult SES is primarily important afterwards.

Keywords: Childhood—Education—Health—Inequality

More than one in five U.S. adults has metabolic syndrome (MetS; Beltrán-Sánchez, Harhay, Harhay, & McElligott, 2013), a cluster of cardiometabolic risk factors for chronic illnesses, such as diabetes and cardiovascular disease, as well as death (Ford, 2005). The risk of having MetS increases substantially during midlife (Beltrán-Sánchez et al., 2013), and the health consequences of MetS are especially pronounced among postmenopausal women (Lin, Caffrey, Chang, & Lin, 2010). Better knowledge about the predictors of MetS could help individuals, families, and providers prevent its development and improve the health of U.S. adults, especially older women, who are one of the largest and fastest growing segments of the population.

A well-established predictor of MetS among women is their socioeconomic status (SES), particularly education level (Loucks, Rehkopf, Thurston, & Kawachi, 2007; Lucove, Kaufman, & James, 2007; Matthews, Räikkönen, Gallo, & Kuller, 2008; Ramsay, Whincup, Morris, Lennon,

& Wannamethee, 2008; Scuteri et al., 2008; Wamala et al., 1999). Indeed, educational attainment is generally considered a “fundamental cause” of disparities in health (Link & Phelan, 1995). Education is thought to enhance health largely through indirect pathways such as economic well-being, health-related behaviors, and psychosocial resources (Mirowsky & Ross, 2003). Compared with lower-educated peers, higher-educated adults are more likely to be employed and have an adequate income for purchasing nutritious foods, gym memberships, and other salubrious resources. Higher-educated adults are less likely to engage in risky behaviors such as smoking and are more likely to engage in preventive behaviors. Higher-educated women have fewer children (Monte & Ellis, 2014) and later age of natural menopause (Gold et al., 2001), which may lower the risk of MetS. Schooling also enhances psychosocial resources. For instance, higher-educated adults are more likely to have salubrious social ties (Umberson & Montez, 2010) and less likely to experience depression (Miech & Shanahan, 2000). In addition to indirect pathways, schooling may enhance health through direct physiological benefits (e.g., Rosenberg-Lee, Barth, & Menon, 2011).

A handful of studies have examined whether socioeconomic circumstances from childhood might also influence the risk of MetS in adulthood (e.g., Langenberg, Kuh, Wadsworth, Brunner, & Hardy, 2006; Lehman, Taylor, Kiefe, & Seeman, 2005; Lucove et al., 2007; Ramsay et al., 2008). Given that childhood SES predicts several health consequences of MetS in adulthood, such as diabetes (Blackwell, Hayward, & Crimmins, 2001), cardiovascular disease (Galobardes, Smith, & Lynch, 2006), and mortality (Montez & Hayward, 2011), it is reasonable to conjecture that it also predicts MetS. However, findings across the handful of studies are inconsistent. It remains poorly understood whether MetS in adulthood is primarily associated with adult SES, or whether SES in childhood is also important.

The main aim of this study is to examine whether SES exposures across the life course shape women’s odds of MetS during midlife. To conceptualize how these exposures might shape MetS, we draw mainly from Cumulative Inequality Theory (CIT: Ferraro, Shippee, & Schafer, 2009), but also from life-course epidemiology (Ben-Shlomo & Kuh, 2002). CIT is a social science theory of aging. It utilizes basic life stages (e.g., childhood, adulthood) as a framework for elucidating how socially structured risks and resources accumulate across life stages to shape well-being and health throughout adulthood.

Five components of CIT frame our study. The first is that childhood conditions are important for adult health. Here, we assess whether childhood SES is associated with women’s odds of MetS before and after adjusting for women’s education level. The importance of timing of SES exposure can be gleaned from noting the extent to which the odds associated with childhood SES are attenuated after adjustment. A second component asserts that inequality is multidimensional. Here, we develop a multidimensional measure of childhood SES using latent classes. Third, CIT asserts that early disadvantage increases exposure to risks and resources across adult domains. Here, we assess the extent to which associations between childhood SES, women’s education, and MetS are mediated by four types of adult factors. Fourth, CIT proposes that turning points in the life course may alter the consequences of earlier exposures. Here, we examine whether childhood SES predicts MetS at baseline when women were pre/early menopausal and the subsequent risk of MetS as women traverse the menopause transition. In addition, because education may be a turning point (particularly in the context of intergenerational mobility), we assess whether the association between childhood SES and adult MetS is moderated by education. A fifth component underscores the importance of assessing interindividual differences. Here, we examine how disparities in MetS between life-course SES groups change as women age.

In general, extant studies find that three types of adult mediators—health behaviors, psychosocial resources, and socioeconomic achievement processes—are important in accounting for the association between childhood SES and adult health (see Montez & Hayward, 2011). Regarding behaviors, individuals experiencing childhood environments characterized by economic adversity, harsh and unsupportive family interactions, family dysfunction, or abuse are more likely to smoke, abuse alcohol and illicit drugs, engage in sexually promiscuous behavior, be physically inactive, and obese (Felitti et al., 1998). Obesity may be particularly important given its central role in MetS and its strong association with other MetS components such as insulin resistance (Grundy et al., 2005). Poor sleep quality is another behavior that may help explain the associations between adverse childhood environments and adult health risks such as MetS (Lee, Tsenkova, & Carr, 2014). Some behavioral mechanisms may be sex specific. Adverse childhood SES is associated with higher fertility and earlier age of menopause (Lawlor, Ebrahim, & Smith, 2003), which can elevate women’s risk of MetS. In addition to influencing behaviors, childhood SES may influence the development of psychosocial resources, such as mental well-being (Wickrama, Conger, & Abraham, 2005), sense of personal control (Krause, 1993), and supportive social relationships (Poon & Knight, 2011), which are health promoting. Furthermore, childhood SES may have a long reach on adult health through its influence on socioeconomic achievement processes. Educational attainment is particularly important (e.g., Luo & Waite, 2005). Its importance partly reflects the relatively early completion of education in the life span (education generally precedes and influences the attainment of other socioeconomic resources such as occupation and income) and its role in shaping numerous other risks and resources for health including behaviors and psychosocial well-being (Mirowsky & Ross, 2003).

Although this study focuses on adult circumstances as potential pathways between childhood SES and adult metabolic health, the association may also reflect biological embedding of childhood exposures. For instance, prenatal conditions have been associated with adult metabolic and cardiovascular functioning (Lehman et al., 2005; McEniry, Palloni, Dávila, & Gurucharri, 2008). In addition, some studies find childhood SES predicts obesity (Heraclides, Witte, & Brunner, 2008), diabetes (Maty, Lynch, Raghunathan, & Kaplan, 2008), and metabolic function (Lehman et al., 2005), even after controlling for adult circumstances.

As we stated earlier, the few studies that have investigated the role of childhood SES on the risk of MetS in adulthood are inconclusive. This may reflect differences in childhood SES measures and respondent ages across the studies. With few exceptions (Non et al., 2014), studies have used either parental education (Lee et al., 2014; Lehman et al., 2005; Miller et al., 2011) or occupation (Gustafsson & Hammarström, 2012; Langenberg et al., 2006; Lucove et al., 2007; Ramsay et al., 2008) as a single indicator of childhood SES, but these indicators can have different implications for a child's physiological and social development. Parental education may reflect cognitive stimulation in the home, parenting styles, and routine meal and bed times. Parental occupation may more closely reflect economic circumstances. Moreover, prior studies have been either cross-sectional or prospective follow-up studies that assessed MetS at a single age. Any potential effect of childhood SES on the risk of MetS may change with age. For instance, the effect may strengthen with age as the prevalence of MetS among women increases after menopause (Carr, 2003) or decline as the proportion of life spent in childhood shrinks.

This longitudinal study examines whether childhood and adult SES predict MetS among women during midlife. It provides new evidence on the life-course origins of MetS by: (a) using a prospective data set containing repeated measures of MetS, (b) assessing how childhood and adult SES predict the odds of MetS at study baseline when women were pre/early menopausal, as well as the subsequent risk of developing MetS, (c) creating a measure of childhood SES that is multidimensional and meaningful, and (d) examining whether the associations are mediated by adult reproductive, economic, behavioral, and psychosocial factors.

Method

Data

The Study of Women's Health Across the Nation (SWAN) is a multisite, community-based, prospective study of aging and the menopause transition. From a cross-sectional study of 16,065 women from seven locations (Boston, Chicago, the Detroit area, Los Angeles, New Jersey, Oakland, and Pittsburgh), 3,302 women who at the 1996 baseline were aged 42–52 years, premenopausal, and self-identified with the site's designated race-ethnic groups were recruited for the prospective study. Details on sampling and recruitment methods are published elsewhere (Sowers et al., 2000). Study visits between baseline and follow-up assessment 13 included interviews, anthropometry, questionnaires, and blood draws. The analytical sample for the present study includes participants from the four SWAN locations (Boston, Chicago, Detroit, and Pittsburgh) that administered an ancillary 10-item questionnaire about childhood circumstances during Visit 13 in 2012/2013. These sites enrolled white and black women. Among these 1,399 participants, 1,109 (79%) returned the ancillary questionnaire.

Metabolic Syndrome

Metabolic syndrome (MetS) is defined using clinical thresholds in the modified National Cholesterol Education Program Adult Treatment Panel III definition (Grundy et al., 2005). Women were identified as having MetS if they had at least three of the following five components: hypertension (systolic blood pressure ≥ 130 mmHg or diastolic blood pressure ≥ 85 mmHg or taking any blood pressure medication), impaired fasting glucose (glucose ≥ 100 mg/dL fasting value or taking medication for elevated glucose), obesity (waist > 88 cm), low high-density lipoprotein (< 50 mg/dL), and high triglycerides (≥150 mg/dL fasting value). MetS was measured at baseline and at each follow-up through Visit 12. For missed visits, the SWAN imputes the score for MetS if the participant was known to have MetS in prior visits (imputed score = 1) or if the participant was known to not have MetS in both prior and subsequent visits (imputed score = 0).

Childhood Circumstances

Information on childhood circumstances was collected during Visit 13 in 2012/2013 with a 10-item questionnaire, the SWAN Childhood Context Ancillary Study. To assess childhood SES specifically, it asked participants about their mother's and father's education and whether, before age 19, their childhood family owned a car, owned a home, ever received public assistance, and ever had difficulty paying for food or rent. In general, extant studies find that certain retrospective measures of childhood SES can be used judiciously. A review of studies assessing retrospective measures found that objective ones (e.g., parental education) are fairly reliable and valid, whereas subjective ones that rely heavily on interpretation or judgment (e.g., quality of relationship with parent) are questionable (Hardt & Rutter, 2004). Recent studies also support the careful use of objective measures. Havari and Mazzonna (2015) examined retrospective reports of childhood health, SES, and living conditions provided by adults aged 50 and older across 13 European countries and found strong internal and external consistency.

Our analyses indicate the SWAN childhood measures are fairly reliable and valid. To assess reliability, we compared the responses in Visits 13 and 7; the latter were collected only at Pittsburgh. The concordance of the questions across visits ranged from 86.1% to 96.9% among these 259 participants. To assess validity, we confirmed the data concur with three expected patterns for SWAN cohorts: childhood SES was less favorable for black than white women; compared with participants' mothers, their fathers were more likely to have 0–11 years of education or a bachelor's degree; and parental high school graduation rates approximated historical records (Goldin, 1998; Heckman & LaFontaine, 2010).

We conducted a latent class analysis to identify distinct subgroups of childhood SES among SWAN participants. This approach is often preferable to alternatives such as including all SES indicators (running the risk of multicollinearity), or creating a summation index of the indicators (ignoring their natural clustering), or using one indicator (potentially overestimating its effect). Following recommended procedures (Wang & Wang, 2012), we found that three latent classes provided the best fit (see also Matthews et al., in press). See Supplementary Material for methodological details. Characteristics of the classes are shown in Table 1. Class 1 contains 26% of women and is the most disadvantaged, with low-educated, poor parents ("adverse SES"). Class 2 contains 50% of women, with low-educated, nonpoor parents ("fair SES"). Class 3 contains 24% and is the most advantaged, with high-educated, nonpoor parents ("good SES").

Adult Circumstances

The adult factor of main interest is women's educational attainment. It is categorized into high school or less; some college; a bachelor's degree or higher. In addition, we include four adult factors (reproductive, economic, psychosocial, and behavioral) which may mediate the associations between childhood SES, women's education, and MetS (our conceptual model is depicted in Supplementary Figure 1). We select several measures of these factors based on prior literature and data availability within SWAN. We exclude women missing education (n = 13) given its importance in our framework. The few missing values for other adult factors are specified by a missing category (for categorical variables) or the average value. Our results are robust to different specifications of missing.

We include three reproductive factors. Number of live births is a 6-category measure, from 0 to 5 or more. We include an indicator of hormone use (including birth control) in the month preceding each visit. At each visit, women are classified into one of four menopausal status categories: pre/early menopausal, late peri menopausal, post/surgical menopausal, and unknown due to hormone therapy.

We include three measures of economic well-being. Employment indicates whether the participant was employed during the prior 2 weeks. Economic hardship identifies participants who stated they experienced "major money problems" during the prior year. Annual family income categories include <\$35,000, \$35,000–74,999, and \$75,000 plus.

We include four health-related behaviors. Smoking indicates the participant smoked cigarettes regularly. Alcohol consumption contains four categories: abstainer, infrequent (<2 servings/week), light to moderate (2–7 servings/week), and heavy (>7 servings/week). Sleep problems is based on three questions regarding the frequency of trouble falling asleep, staying asleep, and waking early in the prior 2 weeks. Responses range from *never* to *five or more times per week* and are summed to a score ranging from 0 to 12. Physical activity measures how often the participant played sports or exercised during the prior year. The categories include never, less than once a month, once a month, 2–3 times a month, once a week, and more than once a week.

We include three psychosocial resources. Marital status includes never married, currently married, and previously married. Social support is based on four questions about having someone to listen to when you need to talk; take you to the doctor; confide in; help with daily chores if you were sick. Responses to each question range from *none of the time* to *all of the time* and are summed to a score ranging from 0 to 16. Depression is measured with the 20-item Center for Epidemiologic Studies—Depression (CES-D) scale (Radloff, 1977), where lower scores indicate fewer resources.

Analytic Strategy

Prevalence of MetS at baseline

We first assess the extent to which childhood SES and women's education predict MetS at baseline when all women were pre/early menopausal. Of the 1,109 participants in the Childhood Context Ancillary Study, 1,064 are included in the prevalence analysis (we excluded 32 women missing baseline MetS and 13 missing education). We examine the odds of MetS at baseline using logistic regression. Model 1 estimates the odds from childhood SES. Model 2 adds education to Model 1. Models 3–6 include adult factors at baseline. The models do not adjust for menopausal status or hormone use, because at baseline participants were pre/early menopausal and not using hormones. All models adjust for age, race, and SWAN location and were estimated with SAS 9.4.

Incidence of MetS after baseline

We next examine the degree to which childhood SES and women's education predict the incidence of MetS after baseline. This analysis includes participants without MetS at baseline, plus participants who were missing MetS at baseline and did not have MetS in the first visit it was assessed (total n = 826). At the time of this study, MetS information for Visit 13 was not available. We estimate Cox

Table 1. Distribution of Demographics and Childhood Socioeconomic Indicators at Baseline (*N* = 1,109)

		Childhood SES latent class		
		Adverse	Fair	Good
	Full sample	Low-educated, poor parents	Low-educated, not poor parents	High-educated, not poor parents
Father's education (%)				
Less than high school	28.5	37.9	37.4	3.2
High school credential	27.9	22.2	43.3	6.2
Technical or vocational school	5.1	1.8	8.3	3.0
Some college	10.3	6.5	8.0	18.0
Bachelor's degree	10.8	3.8	0.0	37.0
Postgraduate degree	8.7	2.4	0.0	30.6
Don't know	8.7	25.4	3.0	2.0
Mother's education (%)				
Less than high school	26.4	41.2	31.1	3.3
High school credential	39.0	32.0	50.0	26.7
Technical or vocational school	7.2	4.9	8.3	7.7
Some college	11.0	5.4	8.3	21.4
Bachelor's degree	7.6	2.7	1.4	23.4
Postgraduate degree	4.6	1.1	0.0	16.2
Don't know	4.1	12.6	0.9	1.4
Childhood family . . . (%)				
. . . did not own a car	13.5	38.6	5.4	2.7
. . . did not own a home	20.8	61.2	7.4	4.4
. . . ever received public assistance	19.1	59.2	6.8	1.0
. . . ever had difficulty paying for food or rent	35.7	76.1	23.6	16.9
Race (%)				
White	56.1	31.0	58.9	77.3
Black	43.9	69.0	41.1	22.7
SWAN location (%)				
Boston	27.0	27.0	21.9	37.5
Chicago	14.0	12.7	13.5	16.4
Detroit	31.1	39.3	34.0	16.3
Pittsburgh	27.9	21.0	30.6	29.8
Participants' educational attainment (%)				
High school or less	20.9	31.5	22.7	5.8
Some college	35.1	41.2	38.0	22.5
Bachelor's degree or higher	44.0	27.3	39.3	71.7
Metabolic syndrome at baseline (%)[a]	24.6	33.1	23.2	18.1
N	1,109	294	552	263

Note: SES = socioeconomic status; SWAN = Study of Women's Health Across the Nation.
[a]Among the 1,077 participants, with non-missing metabolic syndrome scores.

proportional hazards models using elapsed age between visits as the time metric. Model 1 estimates the hazard of MetS from childhood SES. Model 2 adds education to Model 1. Models 3–6 add time-varying adult factors. All models adjust for age, race, and SWAN location and were estimated with SAS 9.4.

Results

At baseline, 265 of 1,077 SWAN participants (24.6%) had MetS. Table 1 shows a graded association between childhood SES and MetS at baseline. The prevalence of MetS was 33.1%, 23.2%, and 18.1% among women raised in adverse, fair, and good childhood SES, respectively. Among the 1,064 respondents in the prevalence analysis, 259 of them (24.3%) had MetS. After baseline, 271 of 826 participants (32.8%) developed MetS.

Prevalence Analysis

We first assess the importance of childhood SES—on its own and net of educational attainment—for women's odds of MetS. Model 1 in Table 2 estimates the basic association between childhood SES and the odds of MetS at baseline, controlling for age, race, and location. Compared with women raised in good childhood SES, the odds of MetS are 77% higher among women raised in adverse SES ($p \leq .01$) and the odds are a nonsignificant 14% higher among women raised in fair SES. In Model 2, the elevated odds of MetS among women raised in adverse SES are attenuated when education is included. Adjusting for education attenuates the log-odds coefficient for adverse SES between Models 1 and 2 by 28%. Nonetheless, adverse childhood SES remains elevated (OR [odds ratio] = 1.51, $p \leq .10$) and education remains significant (OR for high school or less = 1.72, $p \leq .01$; OR for some college = 1.47, $p \leq .05$).

In Models 3–6, we assess the extent to which the associations between childhood SES, women's education, and MetS estimated in Model 2 are mediated by four adult factors. Model 3 adjusts for reproductive factors (measured by number of live births) but does not attenuate the size or significance of the associations. Among the remaining three adult factors, health behaviors (Model 5) appear to be most important. Adjusting for behaviors reduces the log-odds for adverse childhood SES by 25% and to marginal significance. It reduces the log-odds for high school or less by 30% and to marginal significance. Controlling for all adult factors (models not shown) did not further attenuate the associations.

When controlling for all four health behaviors in Model 5, exercise and alcohol are significantly related to MetS but smoking (OR = 1.13, $p > .10$) and sleep (OR = 1.04, $p > .10$) are not (full models available on request). In general, the odds of MetS decline with exercise frequency and alcohol consumption. For instance, compared with women who exercised multiple times per week, women who exercised once per week have more than double the odds of MetS (OR = 2.06, $p \leq .05$) and women who never exercised have more than triple the odds (OR = 3.07 $p \leq .001$). Compared with women who consumed moderate amounts of alcohol, women who abstained have 1.73 times the odds of MetS ($p \leq .01$).

Incidence Analysis

We replicate the analyses above to glean insights into the importance of childhood SES and women's education for incidence of MetS after baseline. This is a period when many SWAN participants began the menopause transition, generally considered an important turning point in women's lives. Model 1 in Table 3 estimates the basic association between childhood SES and the risk of developing MetS after baseline, controlling for age, race, and SWAN location. Compared with women raised in good SES, women raised in adverse SES have an elevated hazard of MetS, but the difference is not significant (HR [hazard ratio] = 1.28, $p > .10$). In contrast to childhood SES, education is a significant predictor. In Model 2, net of childhood SES, women with a high school or less education have 51% higher risk of MetS ($p \leq .05$), and women with some college have a 38% higher risk ($p \leq .05$), compared with college-educated women.

Similar to the prevalence results, adjusting for reproductive factors (births, hormone use, and menopausal status) in Model 3 does not attenuate the association. Because of the theoretical importance of those measures, we retain them in all subsequent models. Similar to the prevalence analysis, health behaviors are the most important mediating factors we examined. Adjusting for behaviors reduced the log-hazard between Models 3 and 5 by 22%. The magnitude of the education coefficients in Model 5 is similar to the prevalence models, although the coefficients in the incidence models remain significant at $p \leq .05$.

Table 2. Odds of Having Metabolic Syndrome at Baseline by Childhood SES and Women's Educational Attainment (n = 1,064)

	Model[a]											
	1		2		3		4		5		6	
	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI
Childhood SES[b]												
Good	1.00	Referent	1.00	Referent	1.00	Referent	1.00	Referent	1.00	Referent	1.00	Referent
Fair	1.14	0.77, 1.69	1.01	0.67, 1.51	1.01	0.68, 1.52	0.98	0.65, 1.47	0.94	0.62, 1.42	1.00	0.66, 1.49
Adverse	1.77**	1.14, 2.73	1.51†	0.96, 2.36	1.52†	0.97, 2.39	1.44	0.92, 2.27	1.36	0.85, 2.16	1.46	0.93, 2.29
Educational attainment												
Bachelors or more			1.00	Referent	1.00	Referent	1.00	Referent	1.00	Referent	1.00	Referent
Some college			1.47*	1.03, 2.10	1.53*	1.07, 2.19	1.38†	0.96, 1.99	1.42†	0.98, 2.04	1.46*	1.02, 2.09
High school or less			1.72**	1.15, 2.58	1.81**	1.20, 2.73	1.65*	1.09, 2.51	1.46†	0.96, 2.23	1.67*	1.11, 2.51
AIC	1,159		1,155		1,161		1,155		1,127		1,163	

Note: AIC = Akaike information criterion; CI = confidence interval; OR = odds ratio; SES = socioeconomic status; SWAN = Study of Women's Health Across the Nation.

[a]Model 1 adjusts for race, SWAN location, and age at baseline. Model 2 adds educational attainment to Model 1. Model 3 adds number of live births to Model 2. Model 4 adds economic well-being (employment status, family income, and economic hardship) to Model 2. Model 5 adds health behaviors (smoking, alcohol, sleep, and exercise) to Model 2. Model 6 adds psychosocial resources (marital status, social support, and depression) to Model 2. [b]All participants are classified into one of three latent classes of childhood SES. See Table 1 for a description of each class.

†$p < .10$. *$p < .05$. **$p < .01$.

Table 3. Hazard of Developing Metabolic Syndrome After Baseline by Childhood SES and Women's Educational Attainment (n = 826)

	Model[a]											
	1		2		3		4		5		6	
	HR	95% CI	HR	95% CI	HR	95% CI	HR	95% CI	HR	95% CI	HR	95% CI
Childhood SES[b]												
Good	1.00	Referent	1.00	Referent	1.00	Referent	1.00	Referent	1.00	Referent	1.00	Referent
Fair	1.08	0.78, 1.49	0.98	0.70, 1.36	0.99	0.71, 1.38	0.97	0.70, 1.36	0.98	0.71, 1.37	0.99	0.71, 1.38
Adverse	1.28	0.88, 1.85	1.12	0.76, 1.64	1.16	0.79, 1.71	1.10	0.74, 1.62	1.10	0.75, 1.63	1.13	0.76, 1.66
Educational attainment												
Bachelors or more			1.00	Referent	1.00	Referent	1.00	Referent	1.00	Referent	1.00	Referent
Some college			1.38*	1.04, 1.85	1.49**	1.11, 2.00	1.46*	1.09,1.96	1.44*	1.07, 1.93	1.49**	1.11, 2.00
High school or less			1.51*	1.07, 2.14	1.63**	1.15, 2.32	1.63**	1.14, 2.32	1.47*	1.04, 2.08	1.62**	1.14, 2.29
AIC	3,388		3,385		3,364		3,366		3,360		3,368	

Note: AIC = Akaike information criterion; CI = confidence interval; HR = hazard ratio; SES = socioeconomic status; SWAN = Study of Women's Health Across the Nation.
[a]Model 1 adjusts for race and SWAN location. Model 2 adds educational attainment to Model 1. Model 3 adds reproductive function (live births, menopausal status, and hormone use) to Model 2. Model 4 adds economic well-being (employment, family income, and economic hardship) to Model 3. Model 5 adds health behaviors (smoking, alcohol, sleep, exercise) to Model 3. Model 6 adds psychosocial resources (marital status, social support, and depression) to Model 3. [b]All participants are classified into one of three latent classes of childhood SES. See Table 1 for a description of each class.
$*p \le .05$. $**p \le .01$.

Table 4. Probability of Surviving from Pre/Early Menopausal Baseline Until at Least Age 50, 55, and 60 Without Developing Metabolic Syndrome by Childhood SES and Women's Educational Attainment (n = 802)

	A	B	C	D
	Adverse childhood SES and high school or less	Fair childhood SES and some college	Good childhood SES and college or more	Columns (C) − (A)
Age (years)	% Surviving[a]	% Surviving	% Surviving	
50	82.3	85.7	89.1	6.8
55	68.6	74.2	79.9	11.3
60	57.3	64.3	71.8	14.5

Note: SES = socioeconomic status. [a]Survival probabilities were generated from Cox proportional hazards models in SAS PROC PHREG that included childhood SES, educational attainment, race, and SWAN location (i.e., Model 2 in Table 3). The probabilities are estimated at the reference values for race (black) and location (Pittsburgh).

As CIT underscores the importance of assessing inter-individual differences, we next examine how disparities in MetS between life-course SES groups change as women age. Table 4 shows the probability of surviving from baseline to at least age 50, 55, and 60 without developing MetS (probabilities derived from Model 2 in Table 3 using the reference values for race [black] and location [Pittsburgh]). The probability of surviving without MetS to age 50 ranged from 89% among women with good childhood SES and a college degree to 82% among women with adverse childhood SES and low education. The differences grew with age: the probability of surviving to age 65 without MetS ranged from 72% to 57% among these two groups.

Supplementary Analyses

We conducted two ancillary analyses. First we examined whether the association between childhood SES and MetS was moderated by women's education. We added four SES-by-education interaction terms to the prevalence and incidence models; none were significant at $p \le .05$. Second, given the central role of obesity in MetS, we assessed whether our results primarily reflect obesity, following Hall and colleagues (2008, 2012). Results are available in Supplementary Table S1. Using Model 3 in Table 2, we added a control for obesity. This attenuated the OR for adverse childhood SES (1.52, $p \le .10$ to 1.13, $p > .10$) but not for education. We also estimated Model 3 to predict each MetS component. Childhood SES was significantly associated with obesity and glucose; education was significantly associated with hypertension and triglycerides. Next, given the high correlation between obesity and glucose intolerance (Grundy et al. 2005), we estimated odds of obesity among women not glucose intolerant, and odds of intolerance among women not obese. Childhood SES was not significant in either model suggesting its association with MetS reflects an obesity-glucose synergy rather than obesity alone.

Discussion

Drawing on CIT, this study examined whether childhood SES and adult SES (measured by education level) have enduring consequences for MetS among midlife women. To our knowledge, it is the first study to examine a prospective data set with repeated measures of MetS and assess how SES-related disparities in MetS change with age. The findings provide new evidence on the life-course SES origins of MetS.

Consistent with the CIT proposition that childhood conditions are important for adult health, childhood SES was inversely related to MetS at baseline. Compared with women who grew up in "good" circumstances (high-educated, nonpoor parents), women who grew up in "fair" circumstances (low-educated, nonpoor parents) had marginally higher odds of MetS, while women who grew up in "adverse" circumstances (low-educated, poor parents) had significantly greater odds. As the primary distinction between fair and adverse childhood SES was economic hardship, it appears that economic hardship in childhood, rather than low levels of parental education alone, has pernicious and enduring consequences for women's metabolic health. It is important to note that our analysis did not include women raised by high-educated, poor parents. Thus, we cannot assess whether economic hardship in childhood would have similarly deleterious consequences for women with high-educated parents or if high-educated parents could buffer against such consequences.

One particularly interesting finding is that the associations between childhood and adult SES and MetS appear to change with age. At the pre/early menopausal baseline, both childhood SES and women's education predicted MetS. However, only education predicted incident MetS after baseline. One interpretation is that childhood SES may leave its stamp on MetS in early to mid- life; it appears to be less relevant than own education for later onset MetS, particularly as women traverse the menopause transition. Midlife in general—and the years surrounding the menopause transition in particular—may be a time when the importance of distal SES exposures for MetS onset give way to more proximal ones. This concurs with the CIT assertion that the health effects of early exposures are not static; rather, they unfold as a life-course process. Our findings also imply that the CIT concept of turning points should not be confined to the conventional set of discrete events such as military service and retirement. Rather, it should incorporate more fluid and subtle processes, some of which may be biological in nature.

The association between childhood SES and the odds of MetS at baseline appears to largely reflect a pathway of low education and lifestyle behaviors. Accounting for education and behaviors attenuated the adverse childhood SES coefficient by roughly one-half; and while it was no longer statistically significant, the odds remained elevated (OR = 1.36). Similarly, the association between women's education and MetS at baseline was largely accounted for by behaviors rather than other factors we examined. Frequent exercise and light-to-heavy alcohol consumption predicted lower odds of MetS; and SES-disadvantaged women were less likely to exercise and more likely to abstain. Our finding is consistent with studies documenting a positive correlation between exercise and alcohol thought to reflect an active lifestyle that mixes team sports (e.g., running clubs, tennis) with socializing centered on alcohol consumption (Poortinga, 2007). Indeed, the correspondence between alcohol consumption and exercise frequency among SWAN participants is significant ($\rho = .11$, $p \leq .001$).

Although health behaviors partly accounted for the inverse relationship between participants' education and incident MetS, other adult factors did not. Prior research has similarly found that even when controlling for health behaviors and number of live births, education remains a significant predictor of women's MetS (Loucks et al., 2007). These results concur with many U.S. studies finding education is the strongest and most consistent adult SES predictor of health behaviors and health (e.g., Mirowsky & Ross, 2003). Education may be a robust predictor because it is a stable measure of lifelong SES. Moreover, in our study, it reflects participant's own SES across several decades before baseline. Exposures measured from baseline onward may be unable to account for much of the association between education and MetS.

Also noteworthy is that disparities in MetS across life-course SES groups widened with age. The difference in surviving without MetS between the most and least advantaged women nearly doubled between ages 50 and 60. Corroborating several CIT components, this finding illustrates that disparities in MetS reflect the accumulation of SES dis/advantages across life-course stages, and these disparities expand with age. It also underscores the need to consider respondent ages when examining the life-course origins of MetS.

Limitations

One potential limitation of our study is that childhood indicators were retrospectively reported and could be affected by measurement errors, such as recall bias. This could produce conservative estimates of the effects of childhood SES because false negatives (not recalling adversities) are more common than false positives (Hardt & Rutter, 2004). Nonetheless, reviews (Hardt & Rutter, 2004) and recent studies (Havari & Mazzonna, 2015) find retrospective measures of "objective" childhood conditions, such as parental education, are sufficiently reliable and valid to be used judiciously.

Although we focused on psychosocial and behavioral mediators, future studies may want to examine physiologic mediators. Other early-life indicators, such as childhood obesity, might also be important but were not measured. As our study is based on four U.S. cities, it is not representative of the U.S. population. Our findings should not be extrapolated to other ages or men. Lastly, although the prevalence analysis could be affected by reverse causation (i.e., MetS may cause inactivity), the graded association between activity and MetS suggests this is not the primary explanation.

Conclusions

Childhood and adult SES predict the odds of MetS among women as they approach the menopause transition; however, adult SES (measured by education) is primarily important afterwards. Health care providers should consider women's SES in childhood, and especially adulthood, when assessing MetS risks. Strategies to decrease the prevalence of MetS should target the first few decades of life—specifically by reducing childhood poverty and raising educational attainment. Strategies that facilitate an active lifestyle may also be beneficial.

Supplementary Material

Please visit the article online at http://gerontologist.oxfordjournals.org/ to view supplementary material.

Funding

This work was supported in part by the Robert Wood Johnson Foundation Health & Society Scholars program at Harvard University (to J. K. Montez). The Study of Women's Health Across the Nation (SWAN) has grant support from the National Institutes of Health (NIH), Department of Health and Human Services, through the National Institute on Aging (NIA), the National Institute of Nursing Research (NINR), and the NIH Office of Research on Women's Health (ORWH) (Grants U01NR004061; U01AG012505, U01AG012535, U01AG012531, U01AG012539, U01AG012546, U01AG012553, U01AG012554, and U01AG012495). The content of this article is solely the responsibility of the authors and does not necessarily represent the official views of the NIA, NINR, ORWH, or NIH.

Clinical Centers: University of Michigan, Ann Arbor – Siobán Harlow, PI 2011–present, MaryFran Sowers, PI 1994–2011; Massachusetts General Hospital, Boston, MA – Joel Finkelstein, PI 1999–present; Robert Neer, PI 1994–1999; Rush University, Rush University Medical Center, Chicago, IL – Howard Kravitz, PI 2009–present; Lynda Powell, PI 1994–2009; University of California, Davis/Kaiser – Ellen Gold, PI; University of California, Los Angeles – Gail Greendale, PI; Albert Einstein College of Medicine, Bronx, NY – Carol Derby, PI 2011–present, Rachel Wildman, PI 2010–2011; Nanette Santoro, PI 2004–2010; University of Medicine and Dentistry – New Jersey Medical School, Newark – Gerson Weiss, PI 1994–2004; and the University of Pittsburgh, Pittsburgh, PA–Karen Matthews, PI.

NIH Program Office: National Institute on Aging, Bethesda, MD – Winifred Rossi 2012 - present; Sherry Sherman 1994–2012; Marcia Ory 1994–2001; National Institute of Nursing Research, Bethesda, MD – Program Officers. Central Laboratory: University of Michigan, Ann Arbor – Daniel McConnell (Central Ligand Assay Satellite Services). Coordinating Center: University of Pittsburgh, Pittsburgh, PA – Maria Mori Brooks, PI 2012 - present; Kim Sutton-Tyrrell, PI 2001–2012; New England Research Institutes, Watertown, MA - Sonja McKinlay, PI 1995–2001. Steering Committee: Susan Johnson, Current Chair; Chris Gallagher, Former Chair.

We thank the study staff at each site and all the women who participated in SWAN.

Acknowledgments

J. K. Montez initiated and designed the study, conducted the analysis, and led the writing of the manuscript. J. T. Bromberger, S. D. Harlow, H. M. Kravitz, and K. A. Matthews coordinated data collection at their respective SWAN locations, advised on all aspects of study design and interpretation of data, and edited the article.

Conflict of Interest

The authors declare no conflicts of interest.

References

Beltrán-Sánchez, H., Harhay, M. O., Harhay, M. M., & McElligott, S. (2013). Prevalence and trends of metabolic syndrome in the adult U.S. population, 1999–2010. *Journal of the American College of Cardiology*, **62**, 697–703. doi:10.1016/j.jacc.2013.05.064

Ben-Shlomo, Y., & Kuh, D. (2002). A life course approach to chronic disease epidemiology: Conceptual models, empirical challenges and interdisciplinary perspectives. *International Epidemiological Association*, **31**, 285–293. doi:10.1093/ije/31.2.285

Blackwell, D. L., Hayward, M. D., & Crimmins, E. M. (2001). Does childhood health affect chronic morbidity in later life? *Social Science and Medicine*, **52**, 1269–1284. doi:10.1016/S0277-9536(00)00230-6

Carr, M. C. (2003). The emergence of the metabolic syndrome with menopause. *The Journal of Clinical Endocrinology and Metabolism*, **88**, 2404–2411. doi:10.1210/jc.2003-030242

Felitti, V. J., Anda, R. F., Nordenberg, D., Williamson, D. F., Spitz, A. M., Edwards, V., … Marks, J. S. (1998). Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults: The Adverse Childhood Experiences (ACE) Study. *American Journal of Preventive Medicine*, **14**, 245–258. doi:10.1016/S0749-3797(98)00017-8

Ferraro, K. F., Shippee, T. P., & Schafer, M. H. (2009). Cumulative inequality theory for research on aging and the life course. In V. L. Bengtson, M. Silverstein, N. M. Putney, & D. Gans (Eds.), *Handbook of theories of aging* (pp. 413–433). New York, NY: Springer.

Ford, E. S. (2005). Risks for all-cause mortality, cardiovascular disease, and diabetes associated with the metabolic syndrome: A summary of the evidence. *Diabetes Care*, **28**, 1769–1778. doi:10.2337/diacare.28.7.1769

Galobardes, B., Smith, G. D., & Lynch, J. W. (2006). Systematic review of the influence of childhood socioeconomic circumstances on risk for cardiovascular disease in adulthood. *Annals of Epidemiology*, **16**, 91–104. doi:10.1016/j.annepidem.2005.06.053

Gold, E. B., Bromberger, J., Crawford, S., Samuels, S., Greendale, G. A., Harlow, S. D., & Skurnick, J. (2001). Factors associated with age at natural menopause in a multiethnic sample of midlife women. *American Journal of Epidemiology*, **153**, 865–874. doi:10.1093/aje/153.9.865

Goldin, C. (1998). America's graduation from high school: The evolution and spread of secondary schooling in the twentieth century.

The Journal of Economic History, **58**, 345–374. doi:10.1017/S0022050700020544

Grundy, S. M., Cleeman, J. I., Daniels, S. R., Donato, K. A., Eckel, R. H., Franklin, B. A., … Costa, F. (2005). Diagnosis and management of the metabolic syndrome: An American Heart Association/National Heart, Lung, and Blood Institute scientific statement. *Circulation*, **112**, 2735–2752. doi:10.1161/CIRCULATIONAHA.105.169405

Gustafsson, P. E., & Hammarström, A. (2012). Socioeconomic disadvantage in adolescent women and metabolic syndrome in mid-adulthood: An examination of pathways of embodiment in the Northern Swedish Cohort. *Social Science and Medicine*, **74**, 1630–1638. doi:10.1016/j.socscimed.2012.01.044

Hall, M. H., Muldoon, M. F., Jennings, J. R., Buysse, D. J., Flory, J. D., & Manuck, S. B. (2008). Self-reported sleep duration is associated with the metabolic syndrome in midlife adults. *Sleep*, **31**, 635–643.

Hall, M. H., Okun, M. L., Sowers, M., Matthews, K. A., Kravitz, H. M., Hardin, K., … Sanders, M. H. (2012). Sleep is associated with the metabolic syndrome in a multi-ethnic cohort of midlife women: The SWAN Sleep Study. *Sleep*, **35**, 783–790. doi:10.5665/sleep.1874

Hardt, J., & Rutter, M. (2004). Validity of adult retrospective reports of adverse childhood experiences: Review of the evidence. *Journal of Child Psychology and Psychiatry*, **45**, 260–273. doi:10.1111/j.1469-7610.2004.00218.x

Havari, E., & Mazzonna, F. (2015). Can we trust older people's statements on their childhood circumstances? Evidence from SHARELIFE. *European Journal of Population*, **31**, 233–257. doi:10.1007/s10680-014-9332-y

Heckman, J. J., & Lafontaine, P. A. (2010). The American high school graduation rate: Trends and levels. *The Review of Economics and Statistics*, **92**, 244–262. doi:10.1162/rest.2010.12366

Heraclides, A., Witte, D., & Brunner, E. J. (2008). The association between father's social class and adult obesity is not explained by educational attainment and an unhealthy lifestyle in adulthood. *European Journal of Epidemiology*, **23**, 573–579. doi:10.1007/s10654-008-9245-3

Krause, N. (1993). Early parental loss and personal control in later life. *Journal of Gerontology: Psychological Sciences*, **48**, P117–P126. doi:10.1093/geronj/48.3.p117

Langenberg, C., Kuh, D., Wadsworth, M. E., Brunner, E., & Hardy, R. (2006). Social circumstances and education: life course origins of social inequalities in metabolic risk in a prospective national birth cohort. *American Journal of Public Health*, **96**, 2216–2221. doi:10.2105/AJPH.2004.049429

Lawlor, D. A., Ebrahim, S., & Smith, G. D. (2003). The association of socio-economic position across the life course and age at menopause: The British Women's Heart and Health Study. *BJOG: An International Journal of Obstetrics and Gynaecology*, **110**, 1078–1087. doi:10.1111/j.1471-0528.2003.02519.x

Lee, C. L., Tsenkova, V., & Carr, D. (2014). Childhood trauma and metabolic syndrome in men and women. *Social Science and Medicine*, **105**, 122–130. doi:10.1016/j.socscimed.2014.01.017

Lehman, B. J., Taylor, S. E., Kiefe, C. I., & Seeman, T. E. (2005). Relation of childhood socioeconomic status and family environment to adult metabolic functioning in the CARDIA study. *Psychosomatic Medicine*, **67**, 846–854. doi:10.1097/01.psy.0000188443.48405.eb

Lin, J. W., Caffrey, J. L., Chang, M. H., & Lin, Y. S. (2010). Sex, menopause, metabolic syndrome, and all-cause and cause-specific mortality—Cohort analysis from the Third National Health and Nutrition Examination Survey. *The Journal of Clinical Endocrinology and Metabolism*, **95**, 4258–4267. doi:10.1210/jc.2010-0332

Link, B. G., & Phelan, J. C. (1995). Social conditions as fundamental causes of disease. *Journal of Health and Social Behavior*, (extra issue), 80–94. doi:10.2307/2626958

Loucks, E. B., Rehkopf, D. H., Thurston, R. C., & Kawachi, I. (2007). Socioeconomic disparities in metabolic syndrome differ by gender: Evidence from NHANES III. *Annals of Epidemiology*, **17**, 19–26. doi:10.1016/j.annepidem.2006.07.002

Lucove, J. C., Kaufman, J. S., & James, S. A. (2007). Association between adult and childhood socioeconomic status and prevalence of the metabolic syndrome in African Americans: The Pitt County Study. *American Journal of Public Health*, **97**, 234–236. doi:10.2105/AJPH.2006.087429

Luo, Y., & Waite, L. J. (2005). The impact of childhood and adult SES on physical, mental, and cognitive well-being in later life. *The Journals of Gerontology, Series B: Psychological Sciences and Social Sciences*, **60**, S93–S101. doi:10.1093/geronb/60.2.S93

Matthews, K. A., Chang, Y., Bromberger, J. T., Karvonen-Gutierrez, C., Kravitz, H. M., Thurston, R. C., & Montez, J. K. (in press). Childhood socioeconomic circumstances, inflammation, and hemostasis among midlife women: Study of Women's Health across the Nation. *Psychosomatic Medicine*.

Matthews, K. A., Räikkönen, K., Gallo, L., & Kuller, L. H. (2008). Association between socioeconomic status and metabolic syndrome in women: Testing the reserve capacity model. *Health Psychology*, **27**, 576–583. doi:10.1037/0278-6133.27.5.576

Maty, S. C., Lynch, J. W., Raghunathan, T. E., & Kaplan, G. A. (2008). Childhood socioeconomic position, gender, adult body mass index, and incidence of type 2 diabetes mellitus over 34 years in the Alameda County Study. *American Journal of Public Health*, **98**, 1486–1494. doi:10.2105/AJPH.2007.123653

McEniry, M., Palloni, A., Dávila, A. L., & Gurucharri, A. G. (2008). Early life exposure to poor nutrition and infectious diseases and its effects on the health of older Puerto Rican adults. *The Journals of Gerontology, Series B: Psychological Sciences and Social Sciences*, **63**, S337–S348. doi:10.1093/geronb/63.6.S337

Miech, R. A., & Shanahan, M. J. (2000). Socioeconomic status and depression over the life course. *Journal of Health and Social Behavior*, **41**, 162–176. doi:10.2307/2676303

Miller, G. E., Lachman, M. E., Chen, E., Gruenewald, T. L., Karlamangla, A. S., & Seeman, T. E. (2011). Pathways to resilience: Maternal nurturance as a buffer against the effects of childhood poverty on metabolic syndrome at midlife. *Psychological Science*, **22**, 1591–1599. doi:10.1177/0956797611419170

Mirowsky, J., & Ross, C. E. (2003). *Education, social status, and health*. New York, NY: Aldine de Gruyter.

Monte, L. M., & Ellis, R. R. (2014). *Fertility of women in the United States: 2012. Current Population Reports, P20-575*. Washington DC: U.S. Census Bureau.

Montez, J. K., & Hayward, M. D. (2011). Early life conditions and later life mortality. In R. G. Rogers & E. M. Crimmins (Eds.), *International handbook of adult mortality* (pp. 187–206). The Netherlands: Springer.

Non, A. L., Rewak, M., Kawachi, I., Gilman, S. E., Loucks, E. B., Appleton, A. A., … Kubzansky, L. D. (2014). Childhood social disadvantage, cardiometabolic risk, and chronic disease in adulthood. *American Journal of Epidemiology*, **180**, 263–271. doi:10.1093/aje/kwu127

Poon, C. Y., & Knight, B. G. (2011). Impact of childhood parental abuse and neglect on sleep problems in old age. *The Journals of Gerontology, Series B: Psychological Sciences and Social Sciences*, **66**, 307–310. doi:10.1093/geronb/gbr003

Poortinga, W. (2007). Associations of physical activity with smoking and alcohol consumption: A sport or occupation effect? *Preventive Medicine*, **45**, 66–70. doi:10.1016/j.ypmed.2007.04.013

Radloff, L. S. (1977). The CES-D scale. A self-report depression scale for research in the general population. *Applied Psychological Measurement*, **1**, 385–401. doi:10.1177/014662167700100306

Ramsay, S. E., Whincup, P. H., Morris, R., Lennon, L., & Wannamethee, S. G. (2008). Is socioeconomic position related to the prevalence of metabolic syndrome? *Diabetes Care*, **31**, 2380–2382. doi:10.2337/dc08-1158

Rosenberg-Lee, M., Barth, M., & Menon, V. (2011). What difference does a year of schooling make? Maturation of brain response and connectivity between 2nd and 3rd grades during arithmetic problem solving. *Neuroimage*, **57**, 796–808. doi:10.1016/j.neuroimage.2011.05.013

Scuteri, A., Vuga, M., Najjart, S. S., Mehta, V., Everson-Rose, S. A., Sutton-Tyrrell, K., … Lakatta, E. G. (2008). Education eclipses ethnicity in predicting the development of the metabolic syndrome in different ethnic groups in midlife: The Study of Women's Health Across the Nation (SWAN). *Diabetic Medicine*, **25**, 1390–1399. doi:10.1111/j.1464-5491.2008.02596.x

Sowers, M., Crawford, S. L., Sternfeld, B., Morganstein, D., Gold, E. B., Greendale, G. A., … Kelsey, J. (2000). SWAN: A multicenter, multi-ethnic, community-based cohort study of women and the menopause. In R. A. Lobo, J. Kelsey, & R. Marcus (Eds.), *Menopause: Biology and pathobiology* (pp. 175–188). San Diego, CA: Academic Press.

Umberson, D., & Montez, J. K. (2010). Social relationships and health: a flashpoint for health policy. *Journal of Health and Social Behavior*, **51**(Suppl.), S54–S66. doi:10.1177/0022146510383501

Wamala, S., Lynch, J., Horsten, M., Mittleman, M. A., Schenck-Gustafsson, K., & Orth-Gomer, K. (1999). Education and the metabolic syndrome in women. *Diabetes Care*, **22**, 1999–2003. doi:10.2337/diacare.22.12.1999

Wang, J., & Wang, X. (2012). *Structural equation modeling*. West Sussex: John Wiley.

Wickrama, K. A. S., Conger, R. D., & Abraham, W. T. (2005). Early adversity and later health: The intergenerational transmission of adversity through mental disorder and physical illness. *The Journals of Gerontology, Series B: Psychological Sciences and Social Sciences*, **60**, 125–129. doi:10.1093/geronb/60.Special_Issue_2.S125