



Pathways linking combinations of early-life adversities to adult mortality: Tales that vary by gender

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ABSTRACT

Rationale: Socioeconomic disadvantage, family instability, and abuse are widely studied early-life adversities (ELAs) that may co-occur in the lives of many. The detrimental effects of these adversities may result in elevated risk of mortality in midlife and old age. **Objective:** We investigate how combinations of these three ELAs affect later-life mortality and the life-course mediators that explain the associations.

Method: Data come from the first two waves of the Midlife in the United States (MIDUS) study and mortality records over a 20-year period (1995/96–2015). We used latent class analysis (LCA) to identify patterns of ELAs across the three domains and incorporated four life-course mediators: material, psychological, social resources, healthy lifestyle and substance abuse.

Results: LCA yielded six distinct combinations of ELAs, with patterns of socioeconomic status (SES) and abuse being most salient. We found that childhood abuse exists across all levels of childhood SES. For both genders, individuals who experienced low SES combined with frequent abuse have the highest risk of death. For women but not men, frequent abuse increases the risk of mortality even if they grew up in middle or high SES families. For both genders, material resource is a significant contributor on the pathway from ELAs to mortality. Life-course mediators partially accounted for the observed associations between ELAs and mortality, but attenuation was stronger for men than women. This is partially attributed to the stronger direct effects of life-course resources on mortality for men than women.

Conclusions: Our findings suggest that ELAs are an important determinant of mortality in midlife and old age. Traumatic experiences during the critical period of early life may compromise later-life health more for women than men.

1. Introduction

1.1. The long-term effects of early-life adversities (ELAs)

Encountering early-life adversities (ELAs) is common across populations. Studies based on both self-reported data and official documents have estimated that about half of individuals in the US are likely to experience one or more adversities as children. These findings are consistent across samples, assessments, and generations. For example, in a sample of nearly 10,000 middle-class adults in California between 1995 and 1996, Felitti et al. (1998) found that around one-half had experienced one or more ELAs, including abuse, neglect, or household dysfunction. Similarly, a study using data from the National Survey of Midlife Development in the US (MIDUS, 2004–2006) found that one out of two middle-aged adults reported negative childhood events,

including abuse, death or illness of a loved one, or parental substance use (Friedman et al., 2015). Using nationally representative data from the 2011/12 National Survey of Children's Health, a recent estimate showed that just under half of the children in the U.S. have experienced at least one adversity, including economic hardship, parental divorce or separation, maltreatment, or parental drug or alcohol abuse (Sacks et al., 2014).

Although ELAs include a host of negative experiences that may occur early in life, socioeconomic disadvantage, family instability (e.g., parental divorce/separation or death), and abuse are among the most common and widely studied ELAs (e.g., Adler and Ostrove, 1999; McLanahan and Sandefur, 1994; Wildeman et al., 2014). Such early-life experiences affect health and well-being throughout the life course (Ferraro and Shippee, 2009). The critical period model suggests that exposure to toxic environments and stress in early life might have

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lifelong consequences by negatively affecting a wide array of health outcomes during adulthood, including mental health, substance abuse, physiological dysregulation, and risk of morbidity (Friedman et al., 2015; Barr et al., 2018; Lee et al., 2017; Montez et al., 2016). Recent studies have documented that the detrimental effects of these adversities may persist for decades after the experiences, ultimately resulting in elevated risk of mortality in midlife and old age (Chen et al., 2016; Galobardes et al., 2008; Pudrovska and Anikputa, 2014). Prior studies, however, have focused on only one domain, mostly childhood socioeconomic status (SES) (e.g., Beebe-Dimmer et al., 2004) or childhood abuse (e.g., Chen et al., 2016). Such an approach neglects the reality that a significant number of individuals are exposed to multiple adversities in early life. Compared to those who experienced a single adversity, individuals who experienced multiple adversities show lower levels of resilience (Rutter, 1981) and suffer more harmful health consequences (Hughes et al., 2017), suggesting the importance of investigating the impact of multiple ELAs.

1.2. Multiple adversities in early life

Conceptualizing and operationalizing multiple adversities as risk factors is a growing area of research (Evans et al., 2013). A common approach for dealing with multiple exposures uses a total count to investigate cumulative exposure effects, for example, adverse childhood experiences (ACEs) scores (Felitti et al., 1998). Unfortunately, this approach obscures the unique effect of each adversity (e.g., financial difficulties vs. abuse). An alternative approach includes multiple domains of early-life disadvantage simultaneously (e.g., Ferraro et al., 2015). Yet, such testing for the additive effects of each ELA domain does not account for the natural clustering of exposure to ELAs. For example, individuals who grow up in difficult family environments—for example, in low SES families (Cancian et al., 2013) or with single parents (Mersky et al., 2009)—are at greater risk of experiencing abuse. Importantly, although socioeconomic disadvantage is a strong predictor of childhood abuse, exposure to such trauma is not just a problem among low SES families. Evidence shows that family violence can exist even in stable middle-class families (Stewart et al., 1987). These findings underscore the complex nature of ELAs.

Recent studies have used person-centered approaches, such as latent class analysis (LCA), to assign individuals to a mutually exclusive class based on their responses to observed variables of childhood adversity. The combinations of ELAs identified in prior studies vary by sample and the type of observed adversities (e.g., economic difficulty, abuse/neglect, witnessing violence, parental incarceration, or parental mental illnesses). For example, using the 2009 to 2012 Behavioral Risk Factor Surveillance Survey, Barboza (2018) used 11 ACE items and identified five subgroups of adults who experienced different combinations of adversities, ranging from normative/low risk to highly abusive/dysfunctional households. Similarly, using a community sample, Shin et al. (2018) identified four groups of young adults, ranging from Low ACEs to High/Multiple ACEs, based on different patterns of 13 ACE items. Despite using different typologies of ELAs, both studies found that individuals who were exposed to more extreme combinations of ELAs were likely to have poor physical and mental health outcomes.

1.3. Intervening pathways from ELAs to adult mortality

Early-life circumstances may influence resources (psychosocial and behavioral), as well as risk factors over the life course which, in turn, may contribute substantially to later-life mortality. According to cumulative inequality theory, early-life conditions shape opportunities and risks that affect later health outcomes (Ferraro and Shippee, 2009). That is, ELAs can generate stressful experiences during adolescence, which can lead to further hardships. For example, individuals who were maltreated as children are likely to experience adverse trajectories, including poor classroom performance in childhood (Zolotor et al.,

1999), lower educational attainment in young adulthood (Jaffee et al., 2018), and unemployment and economic strain in midlife (Currie and Widom, 2010). Cumulatively, these experiences could contribute to low levels of self-control and social support, increasing the risk of affective disorders and poor health decisions or practices (Cohen et al., 2016). Prior studies have found that life-course mediators partially explain why ELAs increase adult mortality, yet these studies have only focused on a single domain of ELAs (childhood SES or abuse) or examined a limited range of mediators, mostly adult SES and health behaviors (Chen et al., 2016; Galobardes et al., 2008; Montez and Hayward, 2014; Pudrovska and Anikputa, 2014).

1.4. Gender differences

Although the number of studies on gender differences is limited, evidence shows that the effects of ELAs on later-life mortality might be more pronounced among women compared to men. For example, using the Wisconsin Longitudinal Study, researchers have shown that childhood SES is inversely associated with the risk of all-cause mortality from midlife to later life, particularly for women (Pudrovska and Anikputa, 2014). Studies based on MIDUS have shown that childhood abuse is significantly associated with an elevated risk of all-cause mortality only for women (Chen et al., 2016). Gender differences in the effects of ELAs might exist because stressful circumstances in early life are more closely tied to subsequent health-promoting resources and lifestyles for women compared to men throughout the life course. For example, compared to those from higher SES families, women raised in disadvantaged families are more likely than men to weigh more in adolescence which, in turn, is linked with reduced academic achievement in high school, as well as low SES in midlife (Pudrovska et al., 2014). Some studies have shown that the inverse association between parental SES and children's physical activity is stronger for girls than boys (Carson and Spence, 2010). Such gender differences have been observed even in midlife (Lee et al., 2018). Similar gendered patterns appear in psychological and social domains. For women only, abuse or negative events in childhood are likely to reduce psychological well-being (Greenfield and Marks, 2009) and to weaken bonds to family members in adulthood (Savla et al., 2013). These findings suggest that exposure to ELAs may increase the risk of mortality to a greater extent for women than men through compromised life-course processes. Prior studies on gender differences, however, have mostly focused on socioeconomic and health behavioral factors (e.g., Pudrovska and Anikputa, 2014).

1.5. Aims of the current study

Based on the above literature, we pursued the following aims. One objective was to identify whether there are distinct subpopulations of individuals based on experiences of the three common domains of ELAs: socioeconomic disadvantage, family instability, and abuse. We expected that some individuals might be exposed to severe adversities in one domain, but not others, while other subgroups might be exposed to adversities in all three domains, thereby representing the most severe category of ELAs. The next objective was to investigate the extent to which profiles of ELAs matter for later life mortality. We expected that individuals exposed to adversities in all three domains would have the greatest risk of death. We then investigated life-course resources that might mediate the association between ELAs and later-life mortality. We anticipated that life-course resources would reduce the risk of mortality, but expected that those who experienced severe combinations of ELAs would possess fewer resources. Throughout these analyses, we paid close attention to gender differences, expecting that women and men may have different likelihoods of experiencing particular combinations of ELAs, which could translate to differences in health-promoting resources/risks, lifestyles, and later-life mortality.

2. Method

2.1. Sample

Data come from the MIDUS study, a national survey designed to assess the role of social, psychological, and behavioral factors on unfolding profiles of mental and physical health ($N = 7,108$; 52% women). MIDUS began in 1995/1996 (Wave [W] 1) with non-institutionalized, English-speaking adults aged 25–74 in the 48 contiguous states (Brim et al., 2004). The study included a two-stage survey: a telephone interview and a self-administered questionnaire (SAQ). Approximately 89% of the sample completed both the telephone interview and SAQ at W1 ($N = 6,325$). A follow-up interview (W2) was completed from 2004 to 2006 and, after adjusting for mortality between W1 and W2, the retention rate was 75%. Our analytic sample comprises data collected in W1 and W2, as well as data for all-cause mortality. We limited the sample to those respondents ($N = 6,320$) who completed both the telephone interview and the SAQ, as well as who had survival time data available from 1995/1996 (W1) to the last mortality data collection in October of 2015.

2.2. Measures

Early-life adversities (W1) includes three ELA domains: childhood SES, family instability, and abuse. Childhood SES includes two continuous indicators that capture objective and subjective social status in early life: parental education (1 = no school/some grade school to 12 = PhD, MD, or other professional degree) and financial level growing up (1 = a lot better off than the average family to 7 = a lot worse off, reverse coded). Family instability is a binary response to the question, “Did you live with both of your biological parents up until you were 16?” Possible reasons for a negative response include, for example, parental death, separation or divorce, parents not living together, and never knowing a biological parent. Childhood abuse was drawn from the Conflict Tactics Inventory (Straus et al., 1996). Respondents were asked how often (i.e., often, sometimes, rarely, or never) they had experienced each of the following: moderate physical abuse (prompts for this domain: pushed, grabbed, or shoved; slapped; object thrown at respondent), severe physical abuse (kicked, bit, or hit with a fist; hit with an object [or attempted]; beat up; choked; burned or scalded), and emotional abuse (made insulting remarks; sulked or refused to talk; stomped away; did something out of spite; made threats; kicked/smashed something in anger). Respondents reported their exposure to abuse from each of five potential perpetrators separately: mother, father, brothers, sisters, or anybody else. We re-coded exposure to each domain of abuse as 1 when respondents reported sometimes or often, otherwise 0.

All-cause mortality. The mortality data currently available to researchers were obtained from multiple sources (e.g., National Death Index reports, mortality closeout interviews, longitudinal sample maintenance), providing information on date-of-death up to October 31, 2015. By the last mortality data collection, there were a total of 1,140 confirmed deaths in the study sample (18% of the 6,320 respondents at W1). Survival times for decedents reflected the number of months between the date when SAQ at W1 was completed and the date of death. Participants who were still alive were right-censored after October of 2015.

For comprehensive life-course mediators (at W1 and W2), we included extensive indicators of health-promoting resources/risks and behaviors in adulthood, categorized into five domains: material resources, psychological resources, social resources, healthy lifestyle, and substance abuse. To facilitate comparison of measures with different distributions, each item was standardized based on the pooled sample and coded so that higher values represent more resources. We then created an index by computing the mean across the items in each domain.

We created an index of material resources (Cronbach's $\alpha = 0.78$ at both waves) that captures both objective and subjective SES. Objective SES includes four items: (a) educational degree (1 = no school/some grade school to 12 = PhD, MD, or other professional degree), (b) household income (\$0–\$300,000 or more), (c) wage/salary income (\$0–\$100,000 or more), and (d) current or previous occupation (1 = never employed or manual labor, 2 = service/sales/administrative, 3 = management/business/financial, 4 = professional). Subjective SES reflects the respondent's perceived financial strain and includes (a) current financial situation (0 = worst possible through 10 = best possible), (b) control over financial situation (0 = worst possible through 10 = best possible), (c) availability of money to meet basic needs (1 = more than enough through 3 = not enough, reverse coded), and (d) level of difficulty paying bills (1 = very difficult through 4 = not at all difficult).

The psychological resources index ($\alpha = 0.85$ at W1 and $\alpha = 0.87$ at W2) reflects three sub-domains: sense of control, hedonic well-being, and eudaimonic well-being. Sense of control is a 12-item index that sums up four measures of personal mastery and eight measures (reverse coded) of perceived constraints. The questionnaire for personal mastery and perceived constraints comes from the Midlife Developmental Inventory (Lachman and Weaver, 1998). Hedonic well-being includes a five-item life satisfaction scale (Prenda and Lachman, 2001) and a six-item positive affect scale (Mroczek and Kolarz, 1998). An 18-item version of Ryff's (1989) psychological well-being scale was used to measure eudaimonic well-being, with three items measuring each of six components: autonomy, environmental mastery, personal growth, positive relationships with others, purpose in life, and self-acceptance.

The social resources index ($\alpha = 0.74$ at W1 and $\alpha = 0.73$ at W2) reflects three sub-domains: social interaction, social support, and social well-being. Based on prior work (Loucks et al., 2006), we created a social interaction index as the sum of the following four binary components: (a) whether the respondent was married, (b) whether the respondent had at least weekly contact with family members or friends, (c) whether the respondent attended religious or spiritual services at least monthly, and (d) whether the respondent participated in some other social activity (e.g., professional groups, sports, or social groups) at least monthly. We created an index of social support with three scales: family, friend, or spousal support (Walen and Lachman, 2000). Each scale was measured by four items reflecting positive relations (the supportive person cares about you, understands how you feel, is reliable when you face a crisis, and is someone you can confide in about worries). Using 14 measures of well-being developed by Keyes (1998), we created a social well-being index that reflects five constructs: social coherence, social integration, social acceptance, social contribution, and social actualization.

Based on guidelines from the American Heart Association (Lloyd-Jones et al., 2010), healthy lifestyle is a summary of three measures (range 3–9): *smoking* (1 = current smoking, 2 = former smoking, 3 = never smoking), *body mass index* (1 = 30 kg/m² or higher, 2 = 25–29.9 kg/m², 3 = less than 25 kg/m²) and *moderate or vigorous physical activity* (1 = about once a month or less, 2 = about once a week or several times a month, 3 = several times a week). The summary score was standardized based on the distribution of the pooled sample with higher values representing a healthier lifestyle.

Substance abuse is a binary indicator that captures whether respondents have abused alcohol or drugs in the past 12 months. Alcohol abuse ($\alpha = 0.67$ at W1 and $\alpha = 0.69$ at W2) was assessed with a modified Michigan Alcoholism Screening Test (Selzer et al., 1975), which includes five items: (1) having psychological problems from using alcohol, (2) having a strong desire to use alcohol, (3) spending a great deal of time using alcohol, (4) using more alcohol to get the same effect, and (5) increased chances of getting hurt due to alcohol. Alcohol abuse was coded as 1 if a respondent reported yes to any of these items. Drug abuse ($\alpha = 0.79$ at W1 and $\alpha = 0.73$ at W2) was measured with five items analogous to the ones used in the alcohol abuse questionnaire.

Respondents who reported using any of ten drugs (e.g., sedatives, stimulants, inhalants, cocaine, LSD, heroin) were asked to respond to the questions about drug abuse. Drug abuse was coded as 1 if a respondent reported yes to any of these items.

We controlled for age and race/ethnicity at W1. Given that MIDUS includes a wide age range of respondents, from 25 to 74, we classified respondents into four cohorts: (1) those born before 1943, (2) those born from 1943 to 1953, (3) those born from 1954 to 1964, and (4) those born after 1964. Race/ethnicity was coded as 1 for Whites and 0 for other racial/ethnic groups. Some children who experience ELAs may develop secure bonds with one or both primary caregivers that can alleviate the adverse impacts of ELAs. Thus, we included indices of maternal warmth ($\alpha = .91$) and paternal warmth ($\alpha = 0.92$) (Rossi, 2001) as confounders.

2.3. Analytic approach

Using a mix of continuous and categorical indicators of ELAs, we applied LCA to identify subgroups of individuals with similar ELA histories. LCA was implemented with three demographic covariates (age, race, gender) which can potentially influence latent group memberships (for up-to-date information related to LCA, see Nylund-Gibson and Choi, 2018). We handled missing data by using full information maximum likelihood (FIML) estimation in Mplus (Muthén and Muthén, 2017). To select the best-fitting model in subsequent comparisons among latent class solutions, we used the Bayesian Information Criterion (BIC), Entropy, *p*-values of the Vuong-Lo-Mendell-Rubin Likelihood Ratio Test (VLMR-LRT) and the Lo-Mendell-Rubin Adjusted LRT (Adj-LRT), as well as interpretability (Collins and Lanza, 2010).

We then investigated whether the risk of death varied by ELA profile. We conducted gender-stratified logistic regression models, which included age, race/ethnicity, and maternal/paternal warmth as controls, to present adjusted odds of death across latent classes of ELAs. Using random effects regression models, we tested the extent to which ELAs compromise life-course resources/lifestyle and whether there are gender differences. Finally, we conducted a series of parametric survival analyses to examine the intervening role of life-course mediators between ELAs and later-life mortality and gender differences therein. We chose the Weibull distribution as the baseline hazard after comparing the Akaike's Information Criterion across competing parametric models (e.g., generalized gamma, Weibull, and log logistic). Since the potential mediators were measured at W1 and then at W2, we have multiple-record survival data with up to two observations per subject. For the first survival span (interview date at W1 through December 2003), we analyzed 6,320 respondents with mediators from W1. Between W1 and W2, 367 respondents died; thus, for the second survival span (interview date at W2 through October 2015), we updated information on mediators for those who were still alive at W2 ($N = 5,953$). Among these respondents, 4,669 completed the W2 survey (4,104 [87.9%] of whom were still alive by October 2015) while 1,284 were lost to follow up (LFU) at W2 (1,076 [83.8%] of whom were still alive by October 2015). Individuals who experienced ELAs were more likely to be LFU, and those who were LFU were more likely to have died by October 2015 than those who attended the W2 survey.

Item-specific non-response varied by variable (0.2% for education and 10% for respondent's wages). Approximately 23% of respondents in MIDUS had missing data for at least one of the variables of interest. We implemented 10 imputations using Stata's *ice* command (Royston and White, 2011). Given missing data on mediators at W2 due to LFU, we conducted supplementary analyses with two alternative analytic approaches: (a) using mediators at W1 only and (b) censoring LFU in January 2004 in survival analysis. Although the latter approach showed a weakened effect of ELAs, both supplementary models yielded similar conclusions to what is presented here (see supplementary materials S1 and S2). Given that the likelihoods of health-promoting resources and death differ by gender (Rogers et al., 2010), the analysis was stratified

by gender. Gender differences were tested by pooling data from both genders and testing gender interaction terms. The contribution of the mediators in the survival analysis was evaluated by the percentage of total effects explained by each mediator. We used Stata's *suest* command to test for significant attenuation of coefficients of ELAs when a potential mediator was added into the model.

3. Results

3.1. Latent classes of ELAs

We started the LCA model process by estimating a one-class model and stopped at a nine-class model, which is when the smallest latent group consisted of less than 1% of the sample ($N = 56$). Fit indices show significant model improvement for the six-class solution compared to other class solutions (for details, see supplementary materials S3). Each group showed distinct combinations of ELAs, with patterns of SES and abuse being most salient. The contribution of family instability toward identifying multi-domain ELA groups was relatively small, although individuals who were disadvantaged in the SES and abuse domains were more likely to experience family instability. Thus, we labeled the six latent classes of ELAs in terms of SES and abuse only. Specifically, the first two groups of respondents, which consisted of 14% and 12% of the sample, respectively, had highly educated parents (BA degree or higher) and reported that their family was better off financially than the average family, but their experiences of childhood abuse substantially differed: Rare Abuse vs. Frequent Abuse. Similarly, another two groups of respondents had parents who were high school graduates and reported that their family was the same as the average family financially, but their experiences of childhood abuse were substantially different: Rare Abuse vs. Frequent Abuse (24% and 22% of the sample, respectively). The last two groups of respondents had poorly educated parents (8th grade level of education) but their experiences of childhood abuse were also different: Rare Abuse vs. Frequent Abuse (16% and 12% of the sample, respectively). In the last two groups, respondents in the frequently abused group reported that their family was worse off than the average family, while those in the rarely abused group reported the financial level of their family as close to average. We labeled the six groups as (1) High SES/Rare Abuse, (2) High SES/Frequent Abuse, (3) Middle SES/Rare Abuse, (4) Middle SES/Frequent Abuse, (5) Low SES/Rare Abuse, and (6) Low SES/Frequent Abuse.

3.2. Gender differences in ELAs and midlife resources

Table 2 shows gender differences in exposure to ELAs. Men from middle SES or high SES families were more likely than women to experience frequent abuse (*p*-value trends $< .001$), but there was no gender difference in prevalence of frequent abuse in low SES families. In terms of midlife resources, women, compared to men, had significantly fewer material resources (objective and subjective SES, *p*-value trends $< .001$) and fewer psychological resources (eudaimonic well-being and sense of control, *p*-value trends $< .05$), yet women were less likely to become substance abusers and more likely to maintain a healthy lifestyle (*p*-value trends $< .001$).

3.3. Gender differences in effects of ELAs on all-cause mortality

Fig. 1 shows that exposure to ELAs increased the odds of death even after adjusting for age, race/ethnicity, and maternal/paternal warmth. Compared to women who experienced High SES/Rare Abuse during childhood, there was a higher probability of death among those who experienced High SES/Frequent Abuse (Odds ratio [OR] = 1.62, $p = .07$), Middle SES/Frequent Abuse (OR = 1.73, $p < .05$), Low SES/Rare Abuse (OR = 1.71, $p < .01$), and Low SES/Frequent Abuse (OR = 1.91, $p < .01$). Men who experienced Middle SES/Frequent

Table 1
Latent class memberships of early-life adversity ($N = 6,325$).

	Range	Full sample	Class 1 (14%)	Class 2 (12%)	Class 3 (24%)	Class 4 (22%)	Class 5 (16%)	Class 6 (12%)
			High SES/Rare Abuse	High SES/Frequent Abuse	Middle SES/Rare Abuse	Middle SES/Frequent Abuse	Low SES/Rare Abuse	Low SES/Frequent Abuse
Parental education (mean)	1–12	5.4	9.6	9.5	5.1	5.1	2.1	2.1
Financial level growing up (mean)	1–7	4.0	3.2	3.5	3.9	4.2	4.2	4.7
Family instability (%)	0,1	21%	15%	21%	19%	26%	19%	29%
Emotional abuse (%)	0,1	61%	32%	93%	30%	96%	30%	94%
Moderate physical abuse (%)	0,1	50%	6%	93%	11%	95%	11%	97%
Severe physical abuse (%)	0,1	24%	1%	44%	1%	47%	3%	56%

Note. Item response probabilities of childhood abuse indicate the probability of responding sometimes/often vs. never/rarely to each type of childhood abuse. For continuous indicators of ELAs, parental education ranges from 1 = no school to 12 = PhD, MD, or other professional degree (e.g., 2 = 8th grade, 5 = high school graduate, 9 = BA degree). Financial level growing up ranges from 1 = a lot better off than other families to 7 = a lot worse off (4 = same as average family).

Abuse ($OR = 1.41, p = .08$), Low SES/Rare Abuse ($OR = 1.43, p = .09$) and Low SES/Frequent Abuse ($OR = 1.75, p < .01$) had higher mortality than those who experienced High SES/Rare Abuse. Although gender differences in the effect of ELAs on mortality were insignificant, except for High SES/Frequent Abuse ($p = .06$), we note that in almost every case, the OR for women is larger than the OR for men. The results also suggest that frequent abuse (regardless of childhood SES) is more strongly associated with mortality for women than men. Moreover, age and ELA interaction terms are negative and statistically significant, indicating that ELAs have a stronger effect on mortality at younger ages (see Table S4).

3.4. ELAs and life-course intervening factors

Table 3 shows the inverse associations between ELAs and health-promoting resources/behaviors, most of which are statistically significant for both men and women. Individuals who experienced ELAs had fewer material, psychological, and social resources, and they were more likely to have unhealthy lifestyles and to become substance abusers. In general, more severe combinations of ELAs were associated with fewer resources, except that women who experienced High SES/Frequent Abuse had a greater risk of substance abuse than those who experienced Low SES/Frequent Abuse ($p < .05$). To a large extent, the associations between ELAs and life-course factors were generally stronger for women than men.

3.5. Gender differences in intervening processes

Tables 4 and 5 present Hazard Ratios (HR s) from Weibull Proportional Hazard Models for women and men, respectively. Estimated HR s in Model 1 resemble the pattern of estimated OR s in Fig. 1. Compared to women who experienced High SES/Rare Abuse, there was increased mortality among women who experienced Middle SES/Frequent Abuse ($HR = 1.60, p < .05$), Low SES/Rare Abuse ($HR = 1.47, p < .05$), and Low SES/Frequent Abuse ($HR = 1.62, p < .01$). Although the mortality risk for High SES/Frequent Abuse was not statistically significant, the effect was in the expected direction ($HR = 1.41$). For men, we found a greater mortality risk for Low SES/Frequent Abuse than High SES/Rare Abuse ($HR = 1.45, p < .05$).

Comparing the nested models, we found that the intervening process between ELAs and mortality varied by gender (for percentage changes in coefficients, see supplementary materials S5). Lower material resources was a significant contributor on the pathway from ELAs to mortality for both genders (p -values for the attenuation test between Models 1 and 2 < 0.001), but more so for men than for women. For example, among those exposed to Low SES/Frequent Abuse, material resources accounted for 60% of mortality risk (relative to those with High SES/Rare Abuse) among men, but only 45% for women. In a comparison of Models 1 and 3, we found that psychological resources

was a significant mediator for women but not for men, but observed no substantial gender differences in the contribution of healthy lifestyle or social resources. Although substance abuse independently increased mortality risk for women ($HR = 1.54, p < .05$), the mediating role of substance abuse was negligible for both genders ($< 5\%$). By including all mediators in Model 7, we found that life-course mediators partially accounted for the observed associations between ELAs and mortality, with attenuation being stronger for men (67%) than women (42–52%). This is partially attributed to the stronger direct effects of mediators on mortality for men than women.

4. Discussion

4.1. Summary

Several methodological contributions and key findings emerged from our study. First, using LCA, we revealed ELA profiles defined by distinct combinations of childhood SES, family instability, and history of abuse. Although prior studies have used LCA to categorize individuals into distinct histories of ELAs, such work has been limited to isolated domains, such as childhood SES (Montez et al., 2016; O'Rand and Hamil-Luker, 2005), or childhood abuse (Lee et al., 2017; Noonan et al., 2010). Our findings on the distribution of social class in early life—Low (26% of the sample), Middle (46%), and High (28%)—mirror those from prior studies that classified middle-aged women into three childhood SES groups: Adverse (26%), Fair (50%), and Good (24%) (Matthews et al., 2016; Montez et al., 2016). Nonetheless, we advanced prior work by incorporating other domains of ELAs. For example, we consider the fact that, within each SES group, there are groups of individuals who have different histories of childhood abuse (e.g., frequent vs. rare).

Living in families with economic hardship and poorly educated parents is known to be associated with elevated risk of childhood abuse (Cancian et al., 2010; Cancian et al., 2013), but our findings indicate that some individuals from affluent families also experience frequent abuse (12% of the sample in Table 1). In addition, we found that the contribution of family instability toward identifying multi-domain ELA groups was relatively small. As expected, gender differences in exposure to multi-domain ELAs were evident (Table 2). Prior studies have indicated greater risk of physical violence in childhood for men than women (Thompson et al., 2004), but our findings show that this pattern appears only in middle and high SES families. Alternatively, the likelihood of experiencing frequent abuse combined with low SES is similar for both genders. One caveat of our findings is that the battery of questions about childhood experiences in MIDUS 1 does not explicitly address sexual abuse, which is more common among women (Gilbert et al., 2009). Yet, individuals who experience sexual abuse often experience other types of abuse (Lee et al., 2017). Thus, sexual violence may be embedded in the assessments we have, but we cannot ascertain

Table 2
Descriptive Statistics at Baseline (% or standardized mean) by Gender.

Variables	Men (N = 3,002)	Women (N = 3,318)
Early life adversities		
High SES/Rare Abuse	13%	14%
High SES/Frequent Abuse	14%	11%***
Middle SES/Rare Abuse	23%	24%
Middle SES/Frequent Abuse	25%	20%***
Low SES/Rare Abuse	13%	18%***
Low SES/Frequent Abuse	12%	13%
Death between 1995/1996 (W1) and 2015	20%	16%***
Life-course mediators		
Material resources		
Objective SES	.16	-.15***
Subjective SES	.04	-.04***
Personal resources		
Sense of control	.07	-.06***
Hedonic well-being	.01	-.01
Eudaimonic well-being	.02	-.02*
Social resources		
Social well-being	.02	-.02*
Social support	-.05	.04***
Social interaction	.00	.00
Healthy lifestyle		
Smoking status		
Current smoking	22%	22%
Former smoking	34%	25%***
Never smoking	43%	53%***
Body mass index		
≥ 30 kg/m ²	20%	22%
25–29 kg/m ²	47%	28%****
< 25 kg/m ²	32%	49%****
Moderate or vigorous physical activity		
Less than once a month	6%	5%
Less than once a week	27%	33%***
Several times a week	67%	62%***
Substance abuse		
Alcohol abuse	20%	8%***
Drug abuse	6%	3%***
Index of life-course mediators		
Material resources	.10	-.09***
Psychological resources	.03	-.02**
Social resources	.00	.00
Healthy lifestyle	-.08	.07***
Substance abuse	22%	10%***
Controls		
Maternal warmth	.14	-.13***
Paternal warmth	.03	-.03*
Cohort 1 (1920–1929)	12%	11%
Cohort 2 (1930–1939)	17%	19%
Cohort 3 (1940–1949)	25%	24%
Cohort 4 (1950–1974)	46%	45%
White	92%	90%*

Note. The final analytic sample includes 6,320 respondents (out of 6,325) because five respondents did not have information on death date.

Baseline refers to W1. Continuous variables were standardized (to mean = 0, SD = 1) based on the pooled distribution (both genders combined).

For gender differences, **p* < .05, ***p* < .01, ****p* < .001.

to what extent.

Moving to mortality analyses, we found that individuals exposed to multiple ELAs—for example, growing up in low SES families and experiencing frequent abuse—had increased risk of all-cause mortality (Fig. 1). Among the three domains of ELAs, childhood abuse had the most pernicious consequences, particularly for women. For example, even among individuals from middle or high SES families, exposure to frequent abuse was significantly associated with mortality for women. The modest effects of ELAs for men were somewhat unexpected. We consider two possible interpretations. Prior research has indicated positive associations between childhood abuse, incarceration, and premature death, particularly for boys (Wolff and Shi, 2012; Lee and White, 2012). Thus, some men who experienced extreme ELAs may not

have been included in the MIDUS sample because they died during young adulthood or were institutionalized at the time of data collection. Second, research has indicated that men are less likely than women to disclose experiences of abuse in early life, particularly sexual abuse, due to the stigma against submissiveness and same-sex contact (Holmes and Slap, 1998). Such under-reports may result in reduced effects of ELAs due to misclassification of ELA group memberships.

We also found that individuals exposed to ELAs have fewer health-promoting resources, a less healthy lifestyle, and more substance abuse, which echoes prior studies (e.g., Ferraro et al., 2015). Nonetheless, the strength of the association varied by ELA profile, the type of resource/risk, and gender (Table 3). In general, there were SES gradients in health-related resources and risks, indicating that lower SES in childhood is associated with fewer resources in midlife, yet exposure to frequent abuse in childhood had a strong impact regardless of early-life SES. For example, individuals in the Middle SES/Frequent Abuse group showed similar or worse health outcomes than those in the Low SES/Rare Abuse group. Overall, the adverse effects of ELAs on life-course factors appeared stronger for women than men. For instance, within high SES families, frequent abuse had a more negative impact on healthy behaviors for women than men. This finding reiterates prior findings showing that exposure to childhood abuse weakens the health benefits of having high SES in early life (Andersson, 2016).

Finally, our life-course models probing explanations for links between ELAs and adult mortality differed by gender. For men (Table 4), material resources and healthy lifestyle in adulthood explained most of the observed association, with personal and social resources having relatively little additional impact. These findings converge with prior work, indicating that early-life SES influences adult mortality by shaping subsequent socioeconomic and behavioral trajectories (Beebe-Dimmer et al., 2004; Pudrovska and Anikputa, 2014). In contrast, for women (Table 5), the role of life-course mediators in the association between ELAs and later-life mortality was complex. Although exposure to ELAs substantially reduced all health-promoting resources and lifestyles, the direct effects of these mediators on mortality was weaker for women relative to men. Thus, the intervening role of these mediators was smaller for women than men. After accounting for all potential mediators, the effect of ELAs was no longer statistically significant for both genders, but the direct effect of ELAs was larger for women than men.

These findings suggest that intervention strategies seeking to improve adult SES and health lifestyles may be effective at reducing later-life mortality for men who were exposed to multiple adversities. For women, however, early life might be a sensitive period for altering the structure and function of physiological systems (Barker, 1990; Ben-Shlomo and Kuh, 2002), for example, compromised health in cardio-metabolic systems (Lee and Ryff, 2016; Senese et al., 2009). Such perspectives underscore the importance of reducing exposure to multiple ELAs as well as interventions that occur during early life.

4.2. Limitations

Several methodological limitations should be acknowledged. There may be recall bias in reporting some measures of childhood experiences, yet memories of specific adversities in early life are highly stable (Yancura and Aldwin, 2009) and recall accuracy of childhood SES has been supported in twin studies (Krieger et al., 1998). Although LCA has several strengths over the traditional cluster analysis to capture population heterogeneity, LCA is primarily an exploratory and data reduction technique; correct class assignments rely on factors in the model, such as sample size and covariate effects (Lubke and Muthén, 2007). Thus, we could not rule out the possibility of model misspecification. The missingness in the outcomes in our analysis may not be ignorable since individuals who experienced ELAs tend to have fewer resources, and those who have fewer resources are more likely to drop out between waves. We also cannot provide a foolproof guarantee that the

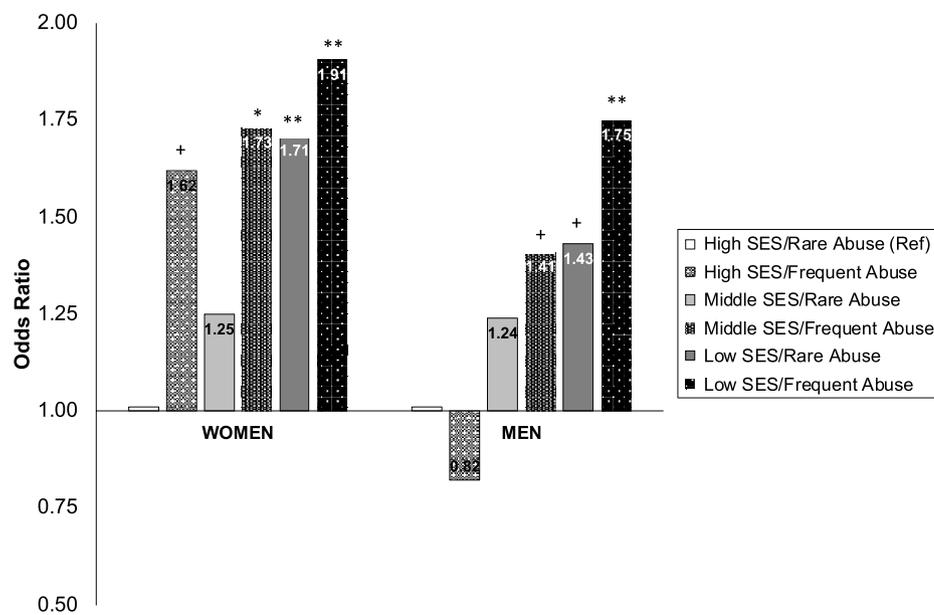


Fig. 1. Odds Ratio of death between 1995/1996 and 2015 by latent class membership of early-life adversity and gender. *Note.* Based on gender-stratified logistic regression models that include age, race/ethnicity and maternal/paternal warmth as controls. Statistical differences in odds between High SES/Rare Abuse (reference) and the other groups are presented as + $p < .10$, * $p < .05$, ** $p < .01$.

Table 3
Random effects estimates for the effects of early-life adversities on life-course resources/lifestyle for women and men.

	Material resources (OLS)	Psychological resources (OLS)	Social resources (OLS)	Healthy lifestyle (OLS)	Substance abuse (logit)
	Beta	Beta	Beta	Beta	OR
Panel 1: Women (N = 3,318)					
Early-life adversities					
High SES/Frequent Abuse	-0.09*	-0.06	-0.06 +	-0.33*** ^b	2.14**
Middle SES/Rare Abuse	-0.19***	-0.08*	-0.09**	-0.33*** ^a	0.91
Middle SES/Frequent Abuse	-0.28***	-0.15***	-0.19***	-0.38***	1.60*
Low SES/Rare Abuse	-0.34***	-0.15***	-0.19*** ^a	-0.42***	0.61
Low SES/Frequent Abuse	-0.41***	-0.15**	-0.20***	-0.51***	1.07
Panel 2: Men (N = 3,002)					
Early-life adversities					
High SES/Frequent Abuse	-0.09*	-0.06	-0.05	-0.07 ^b	1.57*
Middle SES/Rare Abuse	-0.25***	-0.04	-0.04	-0.18*** ^a	0.75
Middle SES/Frequent Abuse	-0.34***	-0.12**	-0.11**	-0.31***	1.53*
Low SES/Rare Abuse	-0.38***	-0.09*	-0.14*** ^a	-0.41***	0.57
Low SES/Frequent Abuse	-0.42***	-0.11*	-0.19***	-0.48***	1.27

Note. High SES/Rare Abuse is a reference group.

OLS = ordinary least square regression model; Logit = logistic regression model; Beta = standardized coefficient; OR = odds ratio.

All models include age, race/ethnicity, maternal/paternal warmth as controls.

a refers to significant gender differences ($p < .10$). *b* refers to significant gender differences ($p < .05$).

* $p < .05$, ** $p < .01$, *** $p < .001$.

Table 4
Hazard ratios (HR) with 95% confidence interval (CI) from Weibull proportional hazard models for women (N = 3,318).

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
	HR [95% CI]	HR [95% CI]	HR [95% CI]	HR [95% CI]	HR [95% CI]	HR [95% CI]	HR [95% CI]
Early-life adversities							
High SES/Frequent Abuse	1.41 [.90, 2.20]	1.32 [.84, 2.06]	1.44 [.92, 2.26]	1.40 [.90, 2.19]	1.28 [.82, 2.00]	1.40 [.90-2.19]	1.24 [.80, 1.95]
Middle SES/Rare Abuse	1.19 [.86, 1.67]	1.07 [.77, 1.50]	1.17 [.84, 1.63]	1.16 [.83, 1.62]	1.06 [.76, 1.48]	1.20 [.86-1.68]	1.03 [.74, 1.45]
Middle SES/Frequent Abuse	1.60* [1.11, 2.29]	1.38+ [.95, 1.99]	1.53* [1.06, 2.20]	1.48* [1.03, 2.14]	1.42+ [.98, 2.04]	1.60* [1.11-2.30]	1.25 [.87, 1.81]
Low SES/Rare Abuse	1.47* [1.07, 2.03]	1.26 [.91, 1.74]	1.40* [1.02, 1.92]	1.36+ [.99, 1.88]	1.32+ [.96, 1.82]	1.50* [1.09-2.06]	1.20 [.86, 1.66]
Low SES/Frequent Abuse	1.62** [1.14, 2.31]	1.31 [.91, 1.88]	1.53* [1.07, 2.18]	1.48* [1.04, 2.12]	1.42+ [.99, 2.03]	1.64** [1.15-2.34]	1.17 [.82, 1.68]
Life-course mediators							
Material resources		.58*** [.49, .69]					.72** [.60, .87]
Psychological resources			.65*** [.56, .74]				.77** [.64, .95]
Social resources				.69*** [.58, .82]			1.06 [.83, 1.36]
Healthy lifestyle					.70*** [.63, .77]		.74*** [.67, .82]
Substance abuse						1.54* [1.09, 2.16]	1.29 [.87, 1.91]

Note. All models include age, race/ethnicity, maternal/paternal warmth as controls. High SES/Rare Abuse is a reference group.

+ $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$.

Table 5
Hazard ratios (HR) with 95% confidence interval (CI) from Weibull proportional hazard models for men (N = 3002).

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
	HR [95% CI]	HR [95% CI]	HR [95% CI]	HR [95% CI]	HR [95% CI]	HR [95% CI]	HR [95% CI]
Early-life adversities							
High SES/Frequent Abuse	.81 [.53, 1.23]	.77 [.50, 1.17]	.79 [.52, 1.20]	.76 [.50, 1.17]	.80 [.52, 1.22]	.80 [.53, 1.22]	.73 [.48, 1.11]
Middle SES/Rare Abuse	1.22 [.89, 1.67]	1.06 [.77, 1.46]	1.21 [.88, 1.65]	1.18 [.86, 1.62]	1.21 [.88, 1.65]	1.22 [.89, 1.67]	1.11 [.81, 1.53]
Middle SES/Frequent Abuse	1.23 [.90, 1.70]	1.05 [.76, 1.45]	1.19 [.86, 1.64]	1.15 [.83, 1.58]	1.15 [.83, 1.58]	1.23 [.89, 1.69]	.97 [.70, 1.34]
Low SES/Rare Abuse	1.26 [.91, 1.73]	1.04 [.75, 1.43]	1.20 [.87, 1.66]	1.16 [.84, 1.61]	1.10 [.79, 1.52]	1.26 [.91, 1.74]	.99 [.72, 1.38]
Low SES/Frequent Abuse	1.45* [1.05, 2.01]	1.16 [.83, 1.61]	1.40* [1.01, 1.93]	1.33+ [.96, 1.84]	1.30 [.94, 1.80]	1.45* [1.05, 2.00]	1.08 [.78, 1.50]
Life-course mediators							
Material resources		.52*** [.44, .61]					.66*** [.56, .79]
Psychological resources			.57*** [.50, .65]				.76** [.64, .90]
Social resources				.61*** [.52, .72]			.95 [.78, 1.16]
Healthy lifestyle					.69*** [.62, .77]		.76*** [.69, .85]
Substance abuse						1.28 [.94, 1.73]	1.07 [.74, 1.54]

Note. All models include age, race/ethnicity, maternal/paternal warmth as controls. High SES/Rare Abuse is a reference group.
+p < .10, *p < .05, **p < .01, ***p < .001.

missing at random assumption is met since there could be unobserved covariates that are omitted. Although we included extensive sets of covariates, we could not exclude potential bias due to omitted variables that might be strongly correlated with both predictors and outcomes. Finally, given that only around 10% of the sample was non-White, our findings may not be generalizable to diverse racial/ethnic groups.

5. Conclusions

Despite the limitations noted, our study used an integrative life-course approach to elaborate gender-specific pathways through which ELAs affect adult mortality. Policy programs initiated in early life that minimize exposure to multiple adversities could improve survival rates for disadvantaged children. Intervention strategies seeking to promote protective factors and thereby to reduce the adverse effects of ELAs may need to be designed differently for women compared to men. More research is needed to illuminate why women are more vulnerable than men when they experience ELAs in multiple domains as well as how protective resources that promote well-being in midlife and old age may vary by gender.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.socscimed.2019.112566>.

References

Adler, N.E., Ostrove, J.M., 1999. Socioeconomic status and health: what we know and what we don't. *Ann. N. Y. Acad. Sci.* 896 (1), 3–15.
 Andersson, M.A., 2016. Chronic disease at midlife: do parent-child bonds modify the effect of childhood SES? *J. Health Soc. Behav.* 57 (3), 373–389.
 Barker, D.J., 1990. The fetal and infant origins of adult disease. *Br. Med. J.* 301 (6761), 1111.
 Barboza, G.E., 2018. Latent classes and cumulative impacts of adverse childhood experiences. *Child. Maltreat.* 23 (2), 111–125.
 Barr, P.B., Silberg, J., Dick, D.M., Maes, H.H., 2018. Childhood socioeconomic status and longitudinal patterns of alcohol problems: variation across etiological pathways in genetic risk. *Soc. Sci. Med.* 209, 51–58.

Beebe-Dimmer, J., Lynch, J.W., Turrell, G., Lustgarten, S., Raghunathan, T., Kaplan, G.A., 2004. Childhood and adult socioeconomic conditions and 31-year mortality risk in women. *Am. J. Epidemiol.* 159 (5), 481–490.
 Ben-Shlomo, Y., Kuh, D., 2002. A life course approach to chronic disease epidemiology: conceptual models, empirical challenges and interdisciplinary perspectives. *Int. J. Epidemiol.* 31 (2), 285–293.
 Brim, O.G., Ryff, C.D., Kessler, R.C., 2004. *How Healthy Are We?: A National Study of Well-Being at Midlife*. University of Chicago Press, Chicago, IL.
 Cancian, M., Slack, K.S., Yang, M.Y., 2010. The Effect of Family Income on Risk of Child Maltreatment. Institute for Research on Poverty, Madison, WI.
 Cancian, M., Yang, M.Y., Slack, K.S., 2013. The effect of additional child support income on the risk of child maltreatment. *Soc. Serv. Rev.* 87 (3), 417–437.
 Carson, V., Spence, J.C., 2010. Seasonal variation in physical activity among children and adolescents: a review. *Pediatr. Exerc. Sci.* 22, 81–92.
 Chen, E., Turiano, N.A., Mroczek, D.K., Miller, G.E., 2016. Association of reports of childhood abuse and all-cause mortality rates in women. *JAMA Psychiatry* 73 (9), 920–927.
 Cohen, S., Gianaros, P.J., Manuck, S.B., 2016. A stage model of stress and disease. *Perspect. Psychol. Sci.* 11 (4), 456–463.
 Collins, L.M., Lanza, S.T., 2010. *Latent Class and Latent Transition Analysis: with Applications in the Social, Behavioral, and Health Sciences*. John Wiley & Sons, Inc, Hoboken: NJ.
 Currie, J., Widom, C.S., 2010. Long-term consequences of child abuse and neglect on adult economic well-being. *Child. Maltreat.* 15 (2), 111–120.
 Evans, G.W., Li, D., Sepanski Whipple, S., 2013. Cumulative risk and child development. *Psychol. Bull.* 139, 1342–1396.
 Felitti, V.J., Anda, R.F., Nordenberg, D., Williamson, D.F., Spitz, A.M., Edwards, V., et al., 1998. Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults. The Adverse Childhood Experiences (ACE) Study. *Am. J. Prev. Med.* 14 (4), 245–258.
 Ferraro, K.F., Schafer, M.H., Wilkinson, L.R., 2015. Childhood disadvantage and health problems in middle and later life: early imprints on physical health? *Am. Sociol. Rev.* 81 (1), 107–1333.
 Ferraro, K.F., Shippee, T.P., 2009. Aging and cumulative inequality: how does inequality get under the skin? *Gerontol.* 49 (3), 333–343.
 Friedman, E.M., Montez, J.K., Sheehan, C.M., Guenewald, T.L., Seeman, T.E., 2015. Childhood adversities and adult cardiometabolic health: does the quantity, timing, and type of adversity matter? *J. Aging Health* 27 (8), 1311–1338.
 Galobardes, B., Lynch, J.W., Smith, G.D., 2008. Is the association between childhood socioeconomic circumstances and cause-specific mortality established? Update of a systematic review. *J. Epidemiol. Community Health* 62 (5), 387–390.
 Gilbert, R., Widom, C.S., Browne, K., Fergusson, D., Webb, E., Janson, S., 2009. Burden and consequences of child maltreatment in high-income countries. *The Lancet* 373, 68–81.
 Greenfield, E.A., Marks, N.F., 2009. Profiles of physical and psychological violence in childhood as a risk factor for poorer adult health: evidence from the 1995-2005 National Survey of Midlife in the United States. *J. Aging Health* 21 (7), 943–966.
 Holmes, W.C., Slap, G.B., 1998. Sexual abuse of boys: definition, prevalence, correlates, sequelae, and management. *J. Am. Med. Assoc.* 280, 1855–1862.
 Hughes, K., Bellis, M.A., Hardcastle, K.A., Sethi, D., Butchart, A., Mikton, C., et al., 2017. The effect of multiple adverse childhood experiences on health: a systematic review and meta-analysis. *Lancet Publ. Health* 2 (8), e356–e366.
 Jaffee, S.R., Ambler, A., Merrick, M., Goldman-Mellor, S., Odgers, C.L., Fisher, H.L., ... Arseneault, L., 2018. Childhood maltreatment predicts poor economic and educational outcomes in the transition to adulthood. *Am. J. Publ. Health* 108 (9), 1142–1147.
 Keyes, C.L.M., 1998. Social well-being. *Soc. Psychol. Q.* 61 (2), 121–140.
 Krieger, N., Okamoto, A., Selby, J.V., 1998. Adult female twins' recall of childhood social class and father's education: a validation study for public health research. *Am. J. Epidemiol.* 147 (7), 704–708.
 Lachman, M.E., Weaver, S.L., 1998. The sense of control as a moderator of social class

- differences in health and well-being. *J. Personal. Soc. Psychol.* 74 (3), 763–773.
- Lee, C., Coe, C., Ryff, C., 2017. Social disadvantage, severe child abuse and biological profiles in adulthood. *J. Health Soc. Behav.* 58 (3), 371–386.
- Lee, C., Ryff, C.D., 2016. Early parenthood as a link between childhood disadvantage and adult heart problems: a gender-based approach. *Soc. Sci. Med.* 171, 58–66.
- Lee, C., Tsenkova, V.K., Boylan, J.M., Ryff, C.D., 2018. Gender differences in the pathways from childhood disadvantage to metabolic syndrome in adulthood: an examination of health lifestyles. *SSM Popul. Health* 4, 216–224. <https://doi.org/10.1016/j.ssmph.2018.01.003>.
- Lee, C., White, H.R., 2012. Effects of childhood maltreatment on violent injuries and premature death during young adulthood among urban high-risk men. *Arch. Pediatr. Adolesc. Med.* 166, 814–820.
- Lloyd-Jones, D.M., Hong, Y., Labarthe, D., Mozaffarian, D., Appel, L.J., Van Horn, L., et al., 2010. Defining and setting national goals for cardiovascular health promotion and disease reduction the American Heart Association's Strategic Impact Goal through 2020 and beyond. *Circulation* 121 (4), 586–613.
- Loucks, E.B., Sullivan, L.M., D'Agostino Sr., R.B., Larson, M.G., Berkman, L.F., Benjamin, E.J., 2006. Social networks and inflammatory markers in the framingham heart study. *J. Biosoc. Sci.* 38 (6), 835–842.
- Lubke, G.H., Muthén, B.O., 2007. Performance of factor mixture models as a function of model size, covariate effects and class-specific parameters. *Struct. Equ. Model.* 14, 26–47.
- Matthews, K.A., Chang, Y., Bromberger, J.T., Karvonen-Gutierrez, C.A., Kravitz, H.M., Thurston, R.C., et al., 2016. Childhood socioeconomic circumstances, inflammation, and hemostasis among midlife women: study of Women's Health across the Nation (SWAN). *Psychosom. Med.* 78 (3), 311–318.
- McLanahan, S., Sandefur, G., 1994. *Growing up with a Single Parent: what Hurts, what Helps*. Harvard University Press, Cambridge, MA.
- Mersky, J.P., Berger, L.M., Reynolds, A.J., Gromoske, A.N., 2009. Risk factors for child and adolescent maltreatment: a longitudinal investigation of a cohort of inner-city youth. *Child. Maltreat.* 14 (1), 73–88.
- Montez, J.K., Bromberger, J.T., Harlow, S.D., Kravitz, H.M., Matthews, K.A., 2016. Life-course socioeconomic status and metabolic syndrome among midlife women. *J. Gerontol.: Series B* 71 (6), 1097–1107.
- Montez, J.K., Hayward, M.D., 2014. Cumulative childhood adversity, educational attainment, and active life expectancy among US adults. *Demography* 51 (2), 413–435.
- Mroczek, D.K., Kolarz, C.M., 1998. The effect of age on positive and negative affect: a developmental perspective on happiness. *J. Personal. Soc. Psychol.* 75 (5), 1333–1349.
- Muthén, L.K., Muthén, B.O., 2017. *Mplus User's Guide*, eighth ed. Muthén and Muthén, Los Angeles, CA.
- Nooner, K.B., Litrownik, A.J., Thompson, R., Margolis, B., English, D.J., Knight, E.D., et al., 2010. Youth self-report of physical and sexual abuse: a latent class analysis. *Child Abuse Negl.* 34 (3), 146–154.
- Nylund-Gibson, K., Choi, A.Y., 2018. Ten frequently asked questions about latent class analysis. *Transl. Issues Psychol. Sci.* 4 (4), 440–461.
- O'Rand, A.M., Hamil-Luker, J., 2005. Processes of cumulative adversity: childhood disadvantage and increased risk of heart attack across the life course. *J. Gerontol. Ser. B Psychol. Sci. Soc. Sci.* 60, 117–124.
- Prenda, K.M., Lachman, M.E., 2001. Planning for the future: a life management strategy for increasing control and life satisfaction in adulthood. *Psychol. Aging* 16 (2), 206–216.
- Pudrovska, T., Anikputa, B., 2014. Early-life socioeconomic status and mortality in later life: an integration of four life-course mechanisms. *J. Gerontol. Ser. B Psychol. Sci. Soc. Sci.* 69 (3), 451–460.
- Pudrovska, T., Reither, E.N., Logan, E.S., Sherman-Wilkins, K.J., 2014. Gender and reinforcing associations between socioeconomic disadvantage and body mass over the life course. *J. Health Soc. Behav.* 55 (3), 283–301.
- Rogers, R.G., Everett, B.G., Saint Onge, J.M., Krueger, P.M., 2010. Social, behavioral, and biological factors, and sex differences in mortality. *Demography* 47 (3), 555–578.
- Royston, P., White, I.R., 2011. Multiple imputation by chained equations (MICE): implementation in Stata. *J. Stat. Softw.* 45 (4), 1–20.
- Rossi, A., 2001. Developmental roots of adult social responsibility. In: Rossi, A.S. (Ed.), *Caring and Doing for Others: Social Responsibility in the Domains of Family, Work, and Community*. University of Chicago Press, Chicago, IL, pp. 227–320.
- Rutter, M., 1981. Stress, coping and development: some issues and some questions. *J. Child Psychol. Psychiatry* 22 (4), 323–356.
- Ryff, C.D., 1989. Happiness is everything, or is it? Explorations on the meaning of psychological well-being. *J. Personal. Soc. Psychol.* 57 (6), 1069.
- Sacks, V., Murphey, D., Moore, K., 2014. *Adverse Childhood Experiences: National and State-Level Prevalence*. Research Brief Child Trends. Publication #2014-28. Retrieved from <http://hdl.handle.net/11212/1663>.
- Savla, J.T., Roberto, K.A., Jaramillo-Sierra, A.L., Gambrel, L.E., Karimi, H., Butner, L.M., 2013. Childhood abuse affects emotional closeness with family in mid- and later life. *Child Abuse Negl.* 37 (6), 388–399.
- Senese, L.C., Almeida, N.D., Fath, A.K., Smith, B.T., Loucks, E.B., 2009. Associations between childhood socioeconomic position and adulthood obesity. *Epidemiol. Rev.* 31 (1), 21–51.
- Selzer, M.L., Vinokur, A., van Rooijen, L., 1975. A self-administered short Michigan alcoholism screening test (SMAST). *J. Stud. Alcohol* 36, 117–126.
- Shin, S.H., McDonald, S.E., Conley, D., 2018. Patterns of adverse childhood experiences and substance use among young adults: a latent class analysis. *Addict. Behav.* 78, 187–192.
- Stewart, C., Senger, M.M., Kallen, D., Scheurer, S., 1987. Family violence in stable middle-class homes. *Soc. Work* 32 (6), 529–531.
- Straus, M.A., Hamby, S.L., Boney-McCoy, S., Sugarman, D.B., 1996. The revised conflict tactics scales (CTS2) development and preliminary psychometric data. *J. Fam. Issues* 17 (3), 283–316.
- Thompson, M.P., Kingree, J., Desai, S., 2004. Gender differences in long-term health consequences of physical abuse of children: data from a nationally representative survey. *Am. J. Publ. Health* 94 (4), 599–604.
- Walen, H.R., Lachman, M.E., 2000. Social support and strain from partner, family, and friends: costs and benefits for men and women in adulthood. *J. Soc. Pers. Relatsh.* 17 (1), 5–30.
- Wildeman, C., Emanuel, N., Leventhal, J.M., Putnam-Hornstein, E., Waldfogel, J., Lee, H., 2014. The prevalence of confirmed maltreatment among US children, 2004 to 2011. *JAMA Pediatr.* 168 (8), 706–713.
- Wolff, N., Shi, J., 2012. Childhood and adult trauma experiences of incarcerated persons and their relationship to adult behavioral health problems and treatment. *Int. J. Environ. Res. Public Health* 9 (5), 1908–1926.
- Yancura, L.A., Aldwin, C.M., 2009. Stability and change in retrospective reports of childhood experiences over a 5-year period: findings from the Davis Longitudinal Study. *Psychol. Aging* 24 (3), 715–721.
- Zolotor, A., Kotch, J., Dufort, V., Winsor, J., Catellier, D., Bou-Saada, I., 1999. School performance in a longitudinal cohort of children at risk of maltreatment. *Matern. Child Health J.* 3 (1), 19–27.