

# Childhood Adversities and Adult Cardiometabolic Health: Does the Quantity, Timing, and Type of Adversity Matter?

Journal of Aging and Health  
2015, Vol. 27(8) 1311–1338

© The Author(s) 2015

Reprints and permissions:  
sagepub.com/journalsPermissions.nav

DOI: 10.1177/0898264315580122

jah.sagepub.com



Esther M. Friedman, PhD<sup>1\*</sup>,  
Jennifer Karas Montez, PhD<sup>2\*</sup>,  
Connor McDevitt Sheehan, MA<sup>3\*</sup>,  
Tara L. Guenewald, PhD<sup>4</sup>, and  
Teresa E. Seeman, PhD<sup>5</sup>

## Abstract

**Objective:** Adverse events in childhood can indelibly influence adult health. While evidence for this association has mounted, a fundamental set of questions about how to operationalize adverse events has been understudied. **Method:** We used data from the National Survey of Midlife Development in the United States to examine how quantity, timing, and types of adverse events in childhood are associated with adult cardiometabolic health. **Results:** The best-fitting specification of quantity of events was a linear measure reflecting a dose–response relationship. Timing of event mattered less than repeated exposure to events. Regarding the type of event, academic interruptions

---

\*Authors listed alphabetically but contributed equally.

<sup>1</sup>RAND Corporation, Santa Monica, CA, USA

<sup>2</sup>Syracuse University, Syracuse, NY

<sup>3</sup>University of Texas at Austin, USA

<sup>4</sup>University of Southern California, Los Angeles, USA

<sup>5</sup>University of California, Los Angeles, USA

## Corresponding Author:

Esther M. Friedman, RAND Corporation, 1776 Main Street, Santa Monica, CA 90401, USA.

Email: [friedman@rand.org](mailto:friedman@rand.org)

and sexual/physical abuse were most important. Adverse childhood events elevated the risk of diabetes and obesity similarly for men and women but had a greater impact on women's risk of heart disease. **Discussion:** Findings demonstrate the insights that can be gleaned about the early-life origins of adult health by examining operationalization of childhood exposures.

### Keywords

life course, childhood, midlife, gender, obesity, heart disease, diabetes

Adult health is shaped by experiences that occur and accumulate across the life course (Ben-Shlomo & Kuh, 2002; Ferraro, Shippee, & Schafer, 2009). Recently, there has been a proliferation of studies investigating the potential consequences of *early-life* experiences for adult health. In general, these studies find that adverse experiences in childhood, such as parental abuse and sibling death, have enduring consequences for adult health net of many confounders (see reviews in Danese & Tan, 2014; Slopen, Koenen, & Kubzansky, 2012). These findings have important public health implications for at least two reasons. First, adverse experiences in childhood are not uncommon. In a sample of nearly 14,000 adults who completed a standardized medical evaluation at a large Health Maintenance Organization (HMO), Felitti et al. (1998) found that more than one half (52%) of the respondents experienced abuse or serious household dysfunction as a child. Second, the health impact of these adverse exposures can be substantial. For instance, U.S. adults aged 55 to 65 years who experienced a major infectious disease (e.g., pneumonia) in childhood had 2.5 times the odds of cancer, 1.7 times the odds of cardiovascular conditions, 4.9 times the odds of lung conditions, and 1.8 times the odds of arthritis/rheumatism, net of adult socioeconomic circumstances (Blackwell, Hayward, & Crimmins, 2001). Thus, a better understanding of how early-life exposures—individually and collectively—influence adult morbidity and mortality could improve population health and reduce health disparities.

Despite the proliferation of studies examining the association between early exposures and adult health, many fundamental questions remain unanswered. In particular, as noted by several scholars (e.g., Fisher et al., 2010; Green et al., 2010; Morton, Schafer, & Ferraro, 2012; Schilling, Aseltine, & Gore, 2008), insufficient attention has been given to determining how to best operationalize adverse exposures. Operationalization is not merely a technical issue. Rather, it influences substantive interpretation of the developmental mechanisms and etiology of adult morbidity and mortality. In this study, we focus on three dimensions of operationalization—quantity, timing,

and types of childhood adversities—and address several questions. Does the quantity of adverse exposures experienced in childhood exhibit dose–response or threshold effects on adult health? To what extent do the health effects of these exposures depend on the age(s) at which they were experienced? Are certain types of exposures more consequential for adult health than other exposures? Do the quantity, timing, and types of exposures have differential consequences for men’s versus women’s health? The importance of addressing fundamental questions such as these was emphasized by Fisher et al. (2010) who stated that “. . . before the research focus can be shifted to the mechanisms underlying the association between childhood adversity and psychosis, it seems imperative to establish more precisely what aspects of adversity are driving the association” (p. 168). In other words, addressing these fundamental questions is an important first step before testing hypotheses about mechanisms.

In this study, we examine how the quantity, timing, and types of childhood adversities are associated with three measures of adult cardiometabolic health. We address our aims using data from the National Survey of Midlife Development in the United States (MIDUS). The findings provide new insights into the association between early exposures and adult cardiometabolic health, whether certain ages of exposure are sensitive, and which aspects of the early-life environment may be most critical for intervention.

## **Childhood Exposures and Adult Health**

Before discussing the potential importance of quantity, timing, and types of exposures, we briefly review the main frameworks for conceptualizing the relationship between childhood exposures and adult health. Both cumulative inequality theory (Ferraro et al., 2009) and life-course epidemiology (Ben-Shlomo & Kuh, 2002) propose that childhood exposures can affect adult health via indirect and direct mechanisms. They may indirectly affect health through myriad mechanisms such as health-related behaviors, psychosocial well-being, and socioeconomic resources. For instance, individuals raised in 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; Lynch, Kaplan, & Salonen, 1997). In addition, exposure to adverse events in childhood may disrupt the development of psychosocial resources. For example, a study using data from MIDUS found that exposure to physical violence in childhood diminished individuals’ sense of personal control, and strained family relationships in adulthood, which in turn predicted poorer mental and physical health (Shaw & Krause, 2002). Another

mechanism which is especially important is educational attainment (Kestilä et al., 2005; Mirowsky & Ross, 1998; Montez & Hayward, 2014). Its importance in life-course studies stems partly from its relatively early completion in the life span—it generally precedes the attainment of other socioeconomic resources such as occupation and income—and its role in shaping psychosocial well-being and health behaviors (Mirowsky & Ross, 1998). For instance, Krause (1993) found that children who experienced parental death achieved lower levels of education and, in turn, experienced greater financial strain in adulthood and a diminished sense of personal control.

Childhood exposures may also impart an enduring biological imprint on adult health. This “direct” mechanism is often associated with the fetal origins hypothesis, which proposes that prenatal environments have enduring consequences for cardiovascular and metabolic systems (Barker, 1997). In this study, we focus on exposures that occur after birth through age 17. Several studies suggest that exposures during this age range can have a biological imprint on health. For example, Slopen et al. (2010) found that an index of early-life adversity (including events such as dropping out or being expelled from school, parental unemployment, and parental substance abuse) was associated with biomarkers of inflammation among Black adults in MIDUS, and that several of the associations remained significant after adjusting for adult circumstances. A recent meta-analysis found that multiple domains of childhood maltreatment were associated with an elevated odds of developing obesity over the life course and that the association persisted net of childhood and adulthood socioeconomic conditions and health behaviors (Danese & Tan, 2014).

Adverse exposures in childhood, especially when chronic, may overwork stress-response systems and alter biological set points (Repetti, Taylor, & Seeman, 2002). The cardiovascular and metabolic systems appear especially susceptible. Stress-response hormones cause glucose and fatty acids to be released, promote insulin resistance, suppress estrogen levels, and increase glucocorticoid secretion which then elevates food storage and obesity risk (Sapolsky, 2004). Numerous studies have found a relationship between childhood environments and indicators of adult cardiometabolic health, including cardiovascular disease (see Galobardes, Davey Smith, & Lynch, 2006), heart attack risk (Hamil-Luker & O’Rand, 2007), obesity (Heraclides, Witte, & Brunner, 2008), diabetes (Maty, Lynch, Raghunathan, & Kaplan, 2008), and metabolic function (Lehman, Taylor, Kiefe, & Seeman, 2005), even after controlling for adult circumstances.

The above review is not exhaustive; rather, it provides the context for the discussion below about how the quantity, timing, and types of childhood exposures may influence adult health. Given our focus on cardiometabolic

health, we highlight the few studies that examined quantity, timing, and type of exposure on obesity, diabetes, and cardiovascular disease. Such studies are scarce, and thus most of the research below examines other mental and physical health outcomes.

## Quantity of Childhood Adversities

The quantity dimension of operationalization mainly concerns the functional form of the association between adverse childhood exposures and adult health. There are many ways to operationalize childhood adversity. For instance, one option is to define a dichotomous indicator of whether an individual has experienced any adverse childhood event. Another option is to focus on a specific exposure such as physical abuse. However, as Green et al. (2010) noted, assessing a single exposure may overestimate its effect on adult health because exposures tend to cluster. A commonly used approach, which accounts for such clustering, is to create a count of exposures. While this provides a more holistic view of the childhood environment (e.g., H. A. Turner, Finkelhor, & Ormrod, 2006; R. J. Turner & Lloyd, 1995), researchers must then decide whether to specify the count as a linear or nonlinear term. A linear term assumes a dose–response relationship, such that each subsequent exposure damages health by a constant amount. A nonlinear term (e.g., polynomial, categorical) assumes that an individual’s health becomes either sensitized or inured to each additional childhood exposure, or that threshold effects exist.

A handful of studies have examined linear and nonlinear functional forms of childhood exposures on adult health. Many find a dose–response relationship. For instance, a count of socioeconomic adversities experienced in childhood accumulated in a dose–response fashion to shape physical functioning among older U.S. adults (Montez & Hayward, 2014). A study of U.K. adults also found a graded relationship between an index of childhood adversities, such as parental unemployment and being sent away from home, and adult physical functioning (Surtees & Wainwright, 2007). A dose–response relationship between a count of exposures to childhood abuse plus household dysfunction and a wide range of health behaviors and conditions was also reported by Felitti et al. (1998). Another study found a positive curvilinear relationship between adverse childhood exposures and poor mental health among adults (Schilling et al., 2008). However, their sensitivity analyses revealed that for mental health, the nonlinearity reflected a greater severity of exposures among individuals with a greater quantity of exposures. Most relevant to the current study, Non et al. (2014) reported a graded relationship between a count of socioeconomic and family adversities before age 7 and adult cardiometabolic risk scores in a U.S. birth cohort study, although it is

unclear whether the relationship was linear. In sum, multiple childhood exposures appear to shape several health outcomes in a dose–response fashion; however, whether this applies to cardiometabolic health is understudied and poorly understood.

## **Types of Childhood Adversities**

A related set of issues about the “long arm of childhood” that has been paid insufficient attention concerns the type and timing of adverse experiences. As Fisher et al. (2010) noted, studies typically examine one or two adversities (and most of these studies focus on abuse) or a summation index and have paid little attention to timing of exposure or comparing different types of exposures.

One recent study using MIDUS found that childhood abuse had lasting effects on health net of educational attainment, whereas childhood financial strain and family structure showed weaker associations with adult health in similar models (Schafer & Ferraro, 2013). Similarly, Thomas, Hyppönen, and Power (2008) found that while physical abuse in childhood elevated the risk of obesity and diabetes among U.K. adults, family problems in childhood (e.g., parental separation) did not. Several studies have compared the health effects of different types of childhood abuse. A study using MIDUS found that emotional and physical but not sexual abuse elevated the odds of metabolic syndrome among men, whereas physical and sexual but not emotional abuse elevated the odds among women (C. L. Lee, Tsenkova, & Carr, 2014). Another study using MIDUS examined three measures of childhood socioeconomic status, parental divorce, parental death, and parental physical abuse and their relationships with allostatic load, a marker of biological health risk (Friedman, Karlamangla, Gruenewald, Koretz, & Seeman, 2015). Childhood socioeconomic status and parental physical abuse were significantly related to overall biological risk score. Parental death and divorce were not. A study of German adults found that parental unemployment and parental separation, but not death or severe disease in the family, predicted clinical precursors to cardiovascular disease (Herberth et al., 2008). In sum, the types of exposures compared in extant studies are limited but suggest that associations with health may not be uniform across all types of exposures. Abuse, in particular, appears to be very important.

## **Timing of Childhood Adversities**

Certain periods of biopsychosocial development may be “sensitive,” such that exposures that disrupt developmental processes during these periods may be especially difficult to ameliorate in adulthood (Ben-Shlomo & Kuh, 2002; Ferraro & Shippee, 2009). In sociological research, the timing

dimension has been little studied. As Ferraro (2011) noted, while sociologists generally recognize the importance of certain periods in the life course for shaping health, they are skeptical that child development can be disaggregated into discrete stages. In addition, large-scale surveys commonly employed to examine the early-life origins of adult health using retrospective reports of childhood events generally do not inquire about the age of exposures. Nonetheless, evidence from disciplines such as psychology and medicine suggests that timing may matter.

Some studies find that adverse exposures at very young ages have the greatest effect on adult health. Among U.S. adults, parental home ownership (a proxy for socioeconomic conditions and pathogen exposure) was associated with enhanced immune function if the ownership occurred during infancy but not between ages 15 and 18 (Cohen et al., 2004). Similarly, another study of U.S. adults found that low family income during their prenatal and birth year was associated with an elevated obesity risk, but low family income when aged 1 to 5 or 6 to 15 was not (Ziol-Guest, Duncan, & Kalil, 2009). Several studies have assessed the timing of childhood abuse. Slopen, McLaughlin, Dunn, and Koenen (2013) found that physical abuse (but not sexual abuse) which began during the preschool years had greater consequences than abuse that began in adolescence for cell-mediated immunity. Similarly, Fisher et al. (2010) reported that the effect of maternal physical abuse on adult psychosis was stronger when the abuse started during ages 0 to 11 than if it started during ages 12 to 16. Because the latter two studies assessed the timing of initial exposure, it remains unclear whether the greater effect of early ages of exposure reflects a sensitive period or cumulative exposure.

In some cases, exposures during middle or late childhood seem to matter the most. A study of Dutch adolescents found that exposure to childhood adversity (e.g., parental death, being sent away from home) during ages 6 to 11, but not ages 0 to 5 or 12 to 15, was associated with high cortisol levels (Bosch et al., 2012). Abuse and maltreatment that occurred persistently between ages 0 and 17 or only during ages 12 to 17 had stronger consequences on a range of health-related behaviors among U.S. adolescents than did maltreatment occurring only during ages 0 to 11 (Thornberry, Ireland, & Smith, 2001). In sum, while the timing of events seems to matter, it is unclear when events matter the most for adult health in general and for cardiometabolic health in particular.

## Potential Gender Differences

A growing number of studies have examined whether childhood adversities have differential consequences for women's and men's health. Some

evidence suggests a stronger effect on women. Among adults in the U.S. Health and Retirement Study, having low-educated parents was more consequential for women than for men in terms of functional limitations (Montez, 2013) and active life expectancy (Montez & Hayward, 2014). Childhood emotional abuse predicted worse self-rated health among women but not men when controlling for adult circumstances (Irving & Ferraro, 2006). The bulk of evidence for a differential effect comes from studies of cardiometabolic conditions. Obesity, diabetes, and heart attack risk appear more closely tied to early-life socioeconomic conditions among women than men (Hamil-Luker & O'Rand, 2007; Heraclides et al., 2008; Khlata, Jusot, & Ville, 2009; Lehman et al., 2005; Maty et al., 2008). Obesity risk is also more strongly tied to early-life nutritional deprivation among women than men in quasi-experimental studies (Robinson, 2012). However, differential effects have not been found in all studies (e.g., Kuh, Hardy, Langenberg, Richards, & Wadsworth, 2002, examined mortality), and some studies find stronger effects on men (e.g., Taylor, Lerner, Sage, Lehman, & Seeman, 2004, examined cardiovascular reactivity).

The reasons for differential effects are unclear. Some hypotheses include biological sex differences (Karlmanangla et al., 2005), gender differences in opportunities, such as education and employment, for individuals who have historically had to escape early adversity through upward mobility (Hamil-Luker & O'Rand, 2007), gendered physical activity in childhood (H. Lee, Harris, & Gordon-Larsen, 2009), and gendered socialization, which is stronger in poor and working-class families (Lareau, 2011).

The main aim of this study is to glean insights into the early-life origins of cardiometabolic health by examining how the quantity, timing, and types of adverse events in childhood are associated with adult obesity, diabetes, and heart trouble. The findings provide important insights into the developmental mechanisms that underlie the association. We extend the informative yet small number of studies on operationalization reviewed above by examining: (a) three dimensions of operationalization—quantity, timing, and type; (b) three measures of cardiometabolic health that have strong etiological ties to early-life stressors; and (c) potential gender differences.

## **Method**

### *Data and Sample*

The data for this study come from MIDUS, a nationally representative panel study of adults aimed at examining the behavioral, psychological, and social causes of adult health (MIDUS, 2007). The first wave of MIDUS contained

7,108 respondents surveyed during 1995 to 1996. Respondents in the main MIDUS sample are representative of 25- to 74-year-old, noninstitutionalized, English-speaking residents of the contiguous United States. The second wave occurred during 2004 to 2006, with responses from 4,963 of the original respondents. Questions about adverse childhood events were asked as part of the self-administered questionnaire (SAQ) in Wave 2. Thus, our analytic sample consists of the 4,028 respondents in Wave 2 who completed the SAQ. After excluding the few respondents with missing values on key dependent and independent variables, our final sample contains 3,996 respondents (or 99.2% of those who completed the SAQ).

### *Cardiometabolic Health*

We analyze three binary indicators of cardiometabolic health. *Diabetes* is coded as 1 if the respondent reported in either wave that they had experienced or been treated for diabetes or high blood sugar in the past 12 months. *Heart disease* is coded as 1 if the respondent reported in either wave ever having heart trouble suspected or confirmed by a doctor. *Obesity* is coded as 1 if the respondent had a body mass index (BMI) of 30 or greater in either wave. The few respondents not reporting their height or weight ( $n = 22$ , or 0.54%) in both waves were excluded.

### *Adverse Childhood Events*

Childhood environments can be conceptualized and operationalized in many different ways, and the choice is often a function of the types of information available within a survey. Childhood environments can be captured with measures of chronic, day-to-day exposures such as socioeconomic conditions. They can also be captured with measures of acute events, such as sibling death or parental divorce. It is the latter operationalization that is the focus of this study.

Information on adverse childhood events was gathered from two successive sets of survey questions asked only in Wave 2. In one set of questions, respondents were asked if they experienced the following eight events as a child or a teenager: repeating a school year, being sent away from home because they did something wrong, having a parent out of a job when they wanted to be working, one or both parents drank so often it caused problems, one or both parents used drugs so often it caused problems, ever dropping out of school, ever flunking out of school, and ever being expelled or suspended from school. Respondents were also asked if they ever experienced the following 19 events and, if so, at what age: fired from a job; did

not have a job for a long time when they wanted to be working; parental death; parental divorce; spouse/partner engaged in marital infidelity; significant difficulties with in-laws; sibling death; child death; child experienced life-threatening accident or injury; lost home to fire, flood, or natural disaster; physically assaulted or attacked; sexually assaulted; serious legal difficulties/prison; detention in jail or comparable institution; declared bankruptcy; suffered a financial or property loss unrelated to work; went on welfare; entered the armed forces; experienced combat. Some of these events are highly unlikely to occur before age 18 (e.g., having a spouse who engaged in infidelity); thus, we only consider events in our analysis if the respondent reported the event occurred before age 18. The inventory of events in MIDUS is similar to inventories used in prior studies (e.g., R. J. Turner & Lloyd, 1995) and the National Comorbidity Study (Kessler, David, & Kendler, 1997).

To assess how the *quantity* of different childhood events shapes adult health, we test three specifications. These include a binary measure indicating the respondent experienced at least one type of adverse event, a (cumulative) linear measure capturing the total number of adverse events experienced, and a categorical measure of the total number of events, ranging from 0 to 5 or more (for a similar approach, see Fisher et al., 2010; Non et al., 2014). We set the limit at 5 or more due to small numbers of respondents at this high end. The categorical measure captures nonlinearities; in ancillary analyses, we also examined a quadratic measure but it fit the data poorly.

To assess how the *type* of adverse event shapes adult health, we collapsed the events into seven meaningful categories: academic, interpersonal, physical/sexual abuse, work or financial problems, death or illness of a loved one, legal problems, and parental substance abuse.<sup>1</sup> We collapsed the events to ensure that we had sufficient sample sizes for the analysis. Respondents who experienced multiple types of events are represented in multiple categories.

We assess the extent to which the *timing* of childhood events matters for adult health using a six-category measure. Although age cutoffs are somewhat arbitrary, certain categories have been proposed in the field of child development as representing meaningful periods of physical, cognitive, and socioemotional development (McDevitt & Omrod, 2015). We draw on these categories to define the following groups: (a) experienced events only between ages 0 and 5 ("infancy and early childhood"), (b) experienced events only between ages 6 and 10 ("middle childhood"), (c) experienced events only between ages 11 and 14 ("early adolescence"), (d) experienced events only between ages 15 and 17 ("late adolescence"), (e) experienced events across multiple age categories, and (f) experienced no events between ages 0 and 17 (McDevitt & Omrod, 2015).

Most childhood events that we examine are likely to occur across all age categories. One exception includes academic events. They are less likely to occur during ages 0 to 5, but they do occur. For example, 5% to 6% of children repeat kindergarten (Zill, Loomis, & West, 1997), which is a type of academic event we capture. Nonetheless, to illustrate that the type and timing of the events are not confounded, Table 1 shows the timing of events within each type of event. The events are nicely dispersed across multiple age categories. For instance, interpersonal events were experienced by 39 respondents only when aged 0 to 5, 56 respondents only when aged 6 to 10, 72 respondents only when aged 11 to 14, 45 respondents only when aged 15 to 17, and across multiple age groups for 260 respondents. As expected, academic events were least like to occur “only when aged 0 to 5.” However, this pattern applies to all events, and thus, it is not problematic in terms of confounding timing and type of event.

We also note that legal events were very rare. Only 18 respondents experienced legal events, which occurred only during adolescence or during multiple age ranges. We considered dropping legal events from the analysis or combining them with another category, but because the event is so rare it does not skew our results, and thus, we retain it as a distinct event. We also note that we do not have the statistical power necessary to examine the interaction between the type and timing of event—that is, whether experiencing a certain event in one age category has a greater effect on health than experiencing the event in another age category.

### *Covariates*

All analyses adjust for age in years (30-84) and race (White, non-White), as reported in MIDUS Wave 2. Less than 8% of the respondents in MIDUS are non-White, which precluded a more detailed measure of race. Analyses that combine men and women also adjust for gender.

### *Analytic Approach*

We estimate a series of logistic regression models to assess how the quantity, timing, and types of adverse childhood events predict the odds of diabetes, heart trouble, and obesity. For each health measure, we estimate a model for each of the three functional forms for quantity of events. To select the form that best fits the data for each health measure, we calculate the Bayesian Information Criterion (BIC) for all models. The BIC penalizes overspecified models and is preferable over a likelihood ratio  $\chi^2$  test when the models to be compared are nonnested (Raftery, 1995). In addition to the three models that

**Table 1.** Number of Respondents Reporting Each Type of Childhood Event by the Age(s) the Event Was Experienced.

	Type of childhood event						
	Academic	Interpersonal	Physical or sexual abuse	Work or financial problems	Death or illness of a loved one	Legal	Parental substance abuse
Timing of childhood event							
Only when aged 0 to 5	14	39	15	11	35	0	26
Only when aged 6 to 10	162	56	51	73	74	0	96
Only when aged 11 to 14	79	72	47	80	69	1	78
Only when aged 15 to 17	179	45	31	93	69	4	36
Multiple age groups	339	260	150	186	167	13	235

Note. See Note 1 for a description of each type of event. Respondents who experienced an academic event only when aged 0 to 5 reported that this event referred to "repeating a grade." Respondents who experienced work or financial problems only when aged 0 to 5 reported that this event referred to "parents out of work" or their childhood family "experienced a great financial loss."

assess quantity of events, we estimate one model to assess timing and one model to assess type. We then estimate gender-stratified models. In all models, we estimate robust standard errors to adjust for shared variance between siblings.

### *Additional Analyses*

In some models, we also adjust for educational attainment to glean insights into the extent to which the associations between quantity, timing, and types of childhood events and adult health persist, net of adult circumstances. Like many prior studies examining the association between childhood circumstances and adult health, we use education as a singularly strong and informative proxy for adult circumstances (e.g., Kestilä et al., 2005; Montez & Hayward, 2014). While other adult circumstances, such as health behaviors and income, can affect health, these circumstances cluster tightly with educational attainment and are generally thought to be mediators linking education to adult health (Ross & Wu, 1995). A comprehensive, life-course mediation model is outside the scope of this article. We measure education as a three-category variable indicating 0 to 11 years, a high school credential or some college, and a bachelor's degree or higher. Prior research indicates that this

measure is a good functional form when information on completed years of school is unavailable (Montez, Hayward, Brown, & Hummer, 2009).

Finally, to ensure that the health effects of adverse events in childhood do not simply reflect the effects of unfavorable socioeconomic conditions in childhood more generally, we estimate additional models which control for the latter. Numerous studies find that the long-term health effects of adverse childhood events are independent of childhood socioeconomic conditions (e.g., Danese & Tan, 2014; Fisher et al., 2010; Thomas et al., 2008).

## Results

Table 2 contains descriptive statistics of the analytic sample. On average, respondents were 56 years old, White, and well educated, with only 6% of the sample having less than a high school credential and 38% completing a bachelor's degree or higher. Many respondents reported a cardiometabolic health risk or condition. Almost 11% reported a doctor's diagnosis of diabetes, 22% reported a heart disease diagnosis, and 31% were obese. Men were slightly more likely than women to report diabetes (13% of men, 9% of women) and heart disease (25% of men, 20% of women). A similar proportion (31%) of men and women were obese.

Experiencing an adverse event in childhood was common. Almost one half (48%) of the sample experienced one or more types of events. Among the total sample, 28% experienced one type of event, 12% experienced two types, 5% experienced three types, 2% experienced four types, and 1% experienced five or more types. With respect to timing of events, 3% of respondents reported events only when aged 0 to 5; 11% only when aged 6 to 10; 9% only when aged 11 to 14; 10% only when aged 15 to 17; and 14% reported events across multiple age groups. Unlike the quantity and timing of adverse childhood events, the types of events differed somewhat between men and women. The largest difference is for academic events, where 24% of men and 16% of women reported an event. Differences in physical/sexual abuse were also large, with 5% of men and 10% of women reporting an event.

### *Quantity, Timing, and Types of Adverse Childhood Events*

Table 3 reports odds ratios (ORs) estimated from logistic regression models predicting the odds of diabetes, heart disease, and obesity from various specifications of childhood events. All models control for age, race, and gender. The table also contains the BIC for each model; the model with the lowest BIC value fits the data the best.

**Table 2.** Descriptive Characteristics of the Analytic Sample Overall and by Gender.

	Overall	Men	Women
<b>Demographics</b>			
Average age in years ( <i>SD</i> )	56.2 (12.4)	56.5 (12.2)	56.0 (12.5)
White (%)	91.9	92.5	91.4
Educational attainment (%)			
Less than high school	6.0	5.8	6.2
High school or some college	55.7	49.5	60.7
Bachelor's or higher	38.3	44.8	33.1
<b>Adult cardiometabolic health (%)</b>			
Diabetes	10.6	12.8	8.8
Heart disease	22.0	25.0	19.6
Obesity	31.1	31.0	31.2
<b>Adverse childhood events (%)</b>			
Any event	47.7	50.0	45.7
Total number of events ( <i>SD</i> )	0.8 (1.1)	0.8 (1.1)	0.8 (1.1)
<b>Number of events (%)</b>			
None	52.4	50.0	54.3
One	28.3	30.1	26.8
Two	11.8	12.7	11.1
Three	4.5	4.2	4.8
Four	2.0	1.9	2.0
Five or more	1.2	1.2	1.1
<b>Timing of events (%)</b>			
None	52.4	50.0	54.3
Only events when aged 0 to 5	3.2	2.7	3.6
Only events when aged 6 to 10	10.9	11.8	10.3
Only events when aged 11 to 14	9.3	10.3	8.4
Only events when aged 15 to 17	10.0	10.4	9.7
Events at multiple age groups	14.2	14.9	13.7
<b>Types of events<sup>a</sup> (%)</b>			
None	52.4	50.0	54.3
Academic	19.3	23.8	15.8
Interpersonal	11.8	11.0	12.5
Physical/sexual abuse	7.4	4.7	9.5
Work/financial problems	11.1	12.6	9.9
Death/illness of a loved one	10.4	9.9	10.7
Legal	0.5	0.8	0.2
Parental substance abuse	11.8	11.7	11.9
<i>n</i>	3,996	1,787	2,209

Note. Descriptive statistics are not weighted.

<sup>a</sup>Columns will not add to 100 because respondents could report more than one type of event.

**Table 3.** Odds Ratios From Logistic Regression Models Estimating the Odds of Cardiometabolic Health Conditions From Adverse Childhood Events.

	Diabetes				Heart disease				Obesity			
	OR	SE	p	BIC	OR	SE	p	BIC	OR	SE	p	BIC
Model 1: Any event	1.22	0.13	†	2,622	1.31	0.10	**	3,998	1.22	0.09	**	4,972
Model 2: Total number of events	1.10	0.05	*	2,621	1.18	0.04	***	3,989	1.14	0.04	***	4,961
Model 3: Number of events (none)				2,650				4,019				4,987
One	1.15	0.14			1.21	0.11	*		1.04	0.08		
Two	1.23	0.20			1.31	0.17	*		1.47	0.16	***	
Three	1.38	0.34			1.40	0.26	†		1.38	0.23	†	
Four	2.44	0.80	**		2.20	0.60	**		1.42	0.35		
Five or more	0.77	0.46			3.23	1.03	***		2.89	0.87	***	
Model 4: Timing of events (none)				2,652				4,024				4,994
Only events when aged 0 to 5	1.13	0.34			1.26	0.28			1.00	0.20		
Only events when aged 6 to 10	1.06	0.19			1.34	0.17	*		1.06	0.12		
Only events when aged 11 to 14	1.18	0.22			0.98	0.15			1.18	0.14		
Only events when aged 15 to 17	1.13	0.22			1.33	0.18	*		1.10	0.13		
Events at multiple age groups	1.49	0.22	**		1.53	0.18	***		1.54	0.16	***	
Model 5: Type of event (none)				2,664				4,034				4,998
Academic	1.42	0.17	**		1.37	0.13	**		1.54	0.13	***	
Interpersonal	1.10	0.20			1.05	0.14			0.98	0.11		
Physical/sexual abuse	1.09	0.25			1.45	0.22	*		1.05	0.14		
Work/financial problems	1.09	0.18			1.22	0.15			1.12	0.12		
Death/illness of a loved one	1.09	0.18			0.91	0.12			1.19	0.14		
Legal	0.98	0.76			0.91	0.52			1.49	0.72		
Parental substance abuse	0.77	0.15			1.19	0.15			1.03	0.11		

Note. Reference groups in parentheses. All models control for age, race, and gender. N = 3,996 for all models. OR = odds ratio; BIC = Bayesian Information Criterion.

†p < .10. \*p < .05. \*\*p < .01. \*\*\*p < .001.

Regarding quantity of events, the model with the lowest BIC for all three cardiometabolic outcomes includes early-life events as a linear term (Model 2). This implies that adverse events elevate the risk of cardiometabolic conditions in a dose–response fashion. However, for diabetes the dichotomous “any event” measure fits the data about as well as the linear (cumulative) number of events measure. The worst fitting model for all health outcomes is the categorical measure.

Comporting with previous research, the models show that adverse childhood events are statistically significant and substantively important predictors of adult health. In the best-fitting linear models, each additional event increased the odds of diabetes by 10% ( $p < .05$ ), the odds of heart disease by 18% ( $p < .001$ ), and the odds of obesity by 14% ( $p < .001$ ). Adults who experienced multiple adversities had significantly worse cardiometabolic health. For instance, adults reporting five or more events in childhood had

a 223% greater odds of heart disease ( $100[3.23-1]$ ,  $p < .001$ ) than adults who experienced none of these events.

Timing of events mattered less than we anticipated. We found little difference in the odds of diabetes or obesity between individuals who experienced no adverse child events and those who experienced events within a single age category. Only for heart disease did experiencing an event during a single age category elevate the odds of the condition. The odds were significantly elevated among adults who only experienced an event when aged 6 to 10 ( $OR = 1.34$ ) or only when aged 15 to 17 ( $OR = 1.33$ ); the odds were also elevated if events occurred only when aged 0 to 5 ( $OR = 1.26$ ) but this was not statistically significant likely due to a small number of events reported for this age group. One consistent finding in Table 3 is that experiencing events across *multiple* age categories was associated with the highest odds of each health condition. Compared with individuals who did not experience any of these childhood events, individuals who experienced events during multiple age categories had 49% greater odds of diabetes, 53% greater odds of heart disease, and 54% greater odds of obesity. Interestingly, this finding is consistent with our finding above that the (cumulative) linear measure of events provides the best-fitting model of quantity of events.

Certain types of childhood events were more consequential than others. Academic events (e.g., repeating school, dropping out, being expelled) were particularly important for all three cardiometabolic health measures. An academic event was associated with a 42% higher odds of diabetes ( $p < .01$ ), a 37% higher odds of heart disease ( $p < .01$ ), and a 54% higher odds of obesity ( $p < .001$ ) compared with individuals who did not experience any adverse childhood events. Physical/sexual abuse was also associated with an elevated odds of heart disease ( $OR = 1.45$ ). No other types of events we examined significantly elevated the risk of cardiometabolic conditions.

In Table 4, we examine whether the association between childhood events and adult cardiometabolic health persists, net of educational attainment. Even after adjusting for education, the (cumulative) linear measure of childhood events provides the best-fitting model for predicting heart disease and obesity, and it provided a similarly good fit as the dichotomous "any event" measure for predicting diabetes. The attenuation of ORs for childhood events from Table 3 to Table 4 implies that adults who experience adverse events in childhood have an elevated risk of diabetes and obesity in part because they experience adverse adult circumstances (proxied by low levels of education). This appears to be most true for diabetes, as most childhood coefficients became nonsignificant after controlling for education. In contrast, childhood events remained highly significant predictors of heart disease and obesity after accounting for education.

**Table 4.** Odds Ratios from Logistic Regression Models Estimating the Odds of Cardiometabolic Health Conditions From Adverse Childhood Events and Educational Attainment.

	Diabetes				Heart disease				Obesity			
	OR	SE	p	BIC	OR	SE	p	BIC	OR	SE	p	BIC
Model 1: Any event	1.13	0.12		2,617	1.26	0.10	**	4,005	1.13	0.08	†	4,932
Model 2: Total number of events	1.05	0.05		2,618	1.15	0.04	***	3,998	1.10	0.04	**	4,926
Model 3: Number of events (none)				2,646				4,029				4,953
One	1.11	0.14			1.19	0.11	†		1.01	0.08		
Two	1.09	0.19			1.24	0.16	†		1.33	0.15	*	
Three	1.21	0.29			1.32	0.25			1.21	0.20		
Four	1.99	0.65	*		2.00	0.56	*		1.19	0.29		
Five or more	0.56	0.34			2.83	0.91	**		2.32	0.70	**	
Model 4: Timing of events (none)				2,649				4,032				4,958
Only events when aged 0 to 5	1.12	0.34			1.25	0.28			0.96	0.19		
Only events when aged 6 to 10	1.05	0.19			1.33	0.17	*		1.02	0.12		
Only events when aged 11 to 14	1.14	0.22			0.96	0.14			1.15	0.14		
Only events when aged 15 to 17	1.00	0.20			1.25	0.17	†		1.01	0.12		
Events at multiple age groups	1.29	0.19	†		1.43	0.17	**		1.34	0.14	**	
Model 5: Type of event (none)				2,664				4,043				4,969
Academic	1.19	0.16			1.26	0.13	*		1.32	0.12	**	
Interpersonal	1.09	0.19			1.05	0.14			0.95	0.11		
Physical/sexual abuse	1.11	0.24			1.46	0.22	*		1.06	0.14		
Work/financial problems	1.11	0.18			1.23	0.15	†		1.14	0.12		
Death/illness of a loved one	1.07	0.17			0.89	0.12			1.15	0.13		
Legal	0.93	0.72			0.89	0.52			1.46	0.70		
Parental substance abuse	0.76	0.14			1.18	0.15			1.01	0.12		

Note. Reference groups in parentheses. All models control for age, race, gender, and educational attainment. N = 3,996 for all models. OR = odds ratio; BIC = Bayesian Information Criterion. †p < .10. \*p < .05. \*\*p < .01. \*\*\*p < .001.

### Potential Gender Differences

Table 5 shows the models from Table 4 (adjusted for age, race, and adult education) but now stratified by gender. Gender differences in the association between childhood events and cardiometabolic health are tested in ancillary models that include an interaction term between gender and events. Significant gender differences are identified in Table 5 by superscript “b” ( $p < .05$ ) or “c” ( $p < .10$ ). Given the large number of interactions tested, we focus on significance at  $p < .05$  to minimize Type I error.

The best-fitting specification of childhood events is generally the (cumulative) linear measure for both men and women, although it only fits the data slightly better than the dichotomous outcome. We find few gender differences at  $p < .05$  in the extent to which adverse childhood events elevate the

**Table 5.** Odds Ratios From Gender-Stratified Logistic Regression Models Estimating the Odds of Cardiometabolic Health Conditions From Adverse Childhood Events and Educational Attainment.

	Diabetes						Heart disease						Obesity													
	Men			Women			Men			Women			Men			Women										
	OR	SE	p	BIC	OR	SE	p	BIC	OR	SE	p	BIC	OR	SE	p	BIC	OR	SE	p	BIC						
Model 1: Any event	1.09	0.16		1,323	1.12	0.19		1,314	0.97	0.12	<sup>a</sup>	1,873	1.54	0.06	<sup>***b</sup>	2,138	1.20	0.13	<sup>†</sup>	2,219	1.06	0.10	2,742			
Model 2: Total number of events	1.10	0.07		1,322	0.99	0.07		1,315	1.04	0.06	<sup>a</sup>	1,873	1.23	0.06	<sup>***b</sup>	2,135	1.18	0.06	<sup>***b</sup>	2,210	1.04	0.04	<sup>b</sup>	2,741		
Model 3: Number of events (none)				1,344				1,344				1,900				2,159				2,233				2,770		
One	0.97	0.17			1.23	0.23			0.93	0.13	<sup>a</sup>		1.44	0.18	<sup>***a</sup>		1.04	0.13						0.98	0.11	
Two	1.14	0.26			1.00	0.26			0.92	0.18	<sup>a</sup>		1.57	0.28	<sup>**</sup>		1.43	0.22	<sup>*</sup>					1.25	0.19	
Three	1.51	0.48			0.91	0.34			1.30	0.35			1.30	0.34			1.28	0.33						1.14	0.25	
Four	3.31	1.43	<sup>**</sup>		1.08	0.61			0.97	0.44	<sup>a</sup>		3.27	1.12	<sup>***a</sup>		1.36	0.49						1.09	0.36	
Five or more	0.38	0.38			0.73	0.54			1.83	0.87	<sup>b</sup>		4.06	1.72	<sup>***b</sup>		5.70	2.76	<sup>***ba</sup>					1.08	0.49	<sup>a</sup>
Model 4: Timing of events (none)				1,346				1,344				1,898				2,165				2,237				2,761		
Only events when aged 0 to 5	1.32	0.58			0.97	0.43			1.48	0.51			1.12	0.35			1.17	0.38						0.83	0.22	
Only events when aged 6 to 10	0.88	0.21			1.22	0.32			0.96	0.18	<sup>a</sup>		1.74	0.30	<sup>***a</sup>		0.79	0.14	<sup>b</sup>					1.25	0.20	<sup>b</sup>
Only events when aged 11 to 14	0.98	0.25			1.33	0.36			0.74	0.16	<sup>b</sup>		1.21	0.26	<sup>b</sup>		1.18	0.21						1.11	0.19	
Only events when aged 15 to 17	0.86	0.23			1.11	0.32			0.91	0.18	<sup>a</sup>		1.61	0.30	<sup>**</sup>		1.26	0.22						0.82	0.15	
Events at multiple age groups	1.57	0.31	<sup>*</sup>		0.98	0.24			1.14	0.20	<sup>a</sup>		1.70	0.27	<sup>***a</sup>		1.61	0.24	<sup>**</sup>					1.14	0.16	
Model 5: Type of event (none)				1,359				1,350				1,914				2,176				2,254				2,780		
Academic	1.50	0.24	<sup>***b</sup>		0.75	0.17	<sup>b</sup>		1.08	0.15			1.41	0.22	<sup>*</sup>		1.34	0.17	<sup>*</sup>					1.29	0.17	<sup>†</sup>
Interpersonal	1.28	0.31			0.92	0.25			1.16	0.24			0.93	0.17			1.18	0.20						0.81	0.13	
Physical/sexual abuse	0.75	0.31			1.32	0.37			0.92	0.26	<sup>a</sup>		1.63	0.30	<sup>***a</sup>		1.11	0.26	<sup>a</sup>					1.06	0.17	<sup>a</sup>
Work/financial problems	1.07	0.24			1.18	0.30			1.07	0.18			1.41	0.25	<sup>†</sup>		1.06	0.17						1.24	0.18	
Death/illness of a loved one	1.15	0.98			1.04	0.26			0.71	0.15	<sup>†</sup>		1.10	0.20			1.22	0.21						1.10	0.17	
Legal	1.13	0.26			— <sup>c</sup>	— <sup>c</sup>			0.92	0.66			1.23	1.37			1.61	0.89						0.88	0.95	
Parental substance abuse	0.70	0.19			0.70	0.19			1.05	0.19			1.32	0.23			1.18	0.20						0.90	0.14	

Note. Reference groups in parentheses. All models control for age, race, and education. N = 3,996 for all models. OR = odds ratio; BIC = Bayesian Information Criterion.

<sup>a</sup>Association significantly different for men and women, as measured by an interaction term in models not shown ( $p < .05$ ).

<sup>b</sup>Association significantly different for men and women, as measured by an interaction term in models not shown ( $p < .10$ ).

<sup>c</sup>This cell had  $n = 4$  observations and caused the diabetes model to be unstable; thus, we removed these respondents from the diabetes model.

<sup>†</sup> $p < .10$ . <sup>\*</sup> $p < .05$ . <sup>\*\*</sup> $p < .01$ . <sup>\*\*\*</sup> $p < .001$ .

**Table 6.** Odds Ratios Estimated From Logistic Regression Models Predicting Cardiometabolic Health Conditions From Childhood Socioeconomic Conditions and Adverse Events.

	Diabetes			Heart disease			Obesity		
	OR	SE	p	OR	SE	p	OR	SE	p
<b>Model 1</b>									
Average parental education	0.93	0.02	***	0.97	0.01	*	0.91	0.01	***
<b>Model 2</b>									
Father occupational status	0.74	0.06	***	0.93	0.04		0.79	0.04	***
<b>Model 3</b>									
Average parental education	0.96	0.02	†	0.97	0.02		0.93	0.01	***
Father occupational status	0.79	0.07	**	1.00	0.05		0.90	0.05	*
Total number of events	1.13	0.06	*	1.18	0.05	***	1.15	0.04	***

Note. Reference groups in parentheses. All models control for age, race, and gender. N = 3,273 for all models. OR = odds ratio.

†p < .10. \*p < .05. \*\*p < .01. \*\*\*p < .001.

odds of diabetes or obesity. However, there is a strong gender difference ( $p < .05$ ) for heart disease that exists across all specifications of childhood events.<sup>2</sup> For instance, each additional childhood event elevated the odds of heart disease by 4% ( $p > .10$ ) for men and by 23% ( $p < .001$ ) for women. We found a significant gender difference associated with physical/sexual abuse and heart disease. Experiencing abuse in childhood elevated the odds of heart disease by 63% among women ( $p < .01$ ) but had little effect on men ( $p > .10$ ).

### Childhood Events and Socioeconomic Conditions

Because exposure to adverse childhood events and socioeconomic status (SES) tends to be correlated, we assess the extent to which the association between childhood events and adult health persisted after controlling for childhood SES. We use average parental education and father’s occupational status as measures of SES. The models in Table 6 show that, controlling for age, race, and gender, parental education was significantly associated with all three health measures. In addition, father’s occupational status was associated with diabetes and obesity but not heart disease. Model 3 mutually adjusts for parental education, father’s occupational status, and the (cumulative) linear measure of events. The association between the linear measure of adverse events and health is robust to controlling for socioeconomic conditions.

## Discussion

Adult health is shaped by experiences that occur and accumulate across the life course. Experiences such as parental divorce and sibling death which occur in early life can impart a strong and enduring effect on adult health, often irrespective of adult circumstances. The empirical evidence for this association has been quickly growing. However, as several scholars have noted (e.g., Fisher et al., 2010; Green et al., 2010; Morton et al., 2012; Schilling et al., 2008), there has been considerably less attention given to determining how to best operationalize childhood experiences. In this study, we address this gap by systematically examining several specifications of childhood events. We identified the operationalization that best reflects the association between childhood events and adult cardiometabolic health, and the extent to which the association persists after adjusting for educational attainment and stratifying by gender.

Five findings are particularly noteworthy. First, the best-fitting functional form for quantity of events is a (cumulative) linear term for all three measures of cardiometabolic health. This implies that adverse childhood events elevate the risk of cardiometabolic conditions in a dose–response fashion. Each additional event increased the odds of diabetes by 10%, the odds of heart disease by 18%, and the odds of obesity by 14%. This finding is fairly robust, as a dose–response model was generally the best-fitting functional form even after controlling for adult education and stratifying by gender. Our findings largely corroborate the handful of studies commenting on functional form (e.g., Montez & Hayward, 2014; Morton et al., 2012; R. J. Turner & Lloyd, 1995).

Second, the timing of childhood events mattered less than repeated exposure to events. We found little difference in the odds of diabetes or obesity between individuals who experienced no adverse child events and individuals who experienced events within a single age category. Rather, experiencing events across *multiple* age categories was important. In contrast, experiencing an event in just one age category (except ages 0–5 and 11–14) elevated the risk of heart disease. Nonetheless, timing mattered much less than we anticipated. This may be because, as Ben-Shlomo and Kuh (2002) asserted, it is difficult to empirically find sensitive periods of exposure for chronic disease outcomes given that these physiological systems are malleable throughout life. Our results suggest that systems related to obesity and diabetes are more malleable than systems related to heart disease. Compared with chronic disease outcomes, sensitive periods may be demonstrated more easily for infectious disease outcomes given certain developmental periods of immune function development (e.g., Cohen et al., 2004; Hall, Yee, & Thomas, 2002).

Third, different childhood events have different consequences for adult health. Academic events (e.g., being expelled, dropping out) were not only the most common type of event, but they were also most strongly associated with adult cardiometabolic health. For heart disease and obesity, the effect remained even after controlling for educational attainment. For women, physical/sexual abuse in childhood was also a particularly strong predictor of heart disease, consistent with other studies (Midei, Matthews, Chang, & Bromberger, 2013; Rich-Edwards et al., 2012).

Fourth, among this sample of adults, adverse early-life events had similar consequences for men's and women's risk of diabetes and obesity but a greater consequence for women's risk of heart disease. This finding was robust across all specifications of early-life events and even after controlling for educational attainment, indicating that these experiences may lead to physiological scarring among women. A study of adults in the Health and Retirement Study similarly found that poor SES in childhood was more consequential for women's than men's heart attack risk (Hamil-Luker & O'Rand, 2007). Given that heart disease is currently the leading killer of U.S. women, improving childhood contexts may be particularly valuable for improving women's health.

Fifth, our results corroborate extant studies that find the effect of childhood events on adult health persists even when controlling for childhood SES. This implies that acute events or shocks (e.g., sibling death) exert an enduring imprint on health over and above the health impact of chronic day-to-day circumstances associated with socioeconomic conditions.

Taken together, our findings indicate that the developmental origins of cardiometabolic health among U.S. men and women are strongly tied to early-life circumstances and that the health consequences of these circumstances develop in a cumulative manner. Moreover, the developmental origins are protracted, spanning childhood and adulthood. Our findings also suggest that improving educational outcomes and reducing exposure to sexual/physical abuse in childhood (especially for women) are two potentially powerful levers for improving population health.

### *Limitations*

As with any research, there are some limitations. One concern in any national survey is whether nonrespondents differed in important ways from survey respondents. The MIDUS study does somewhat underrepresent African Americans and less educated individuals, but this is consistent with the characteristics of survey nonrespondents in general and is not unique to MIDUS (e.g., Fitzgerald, Gottschalk, & Moffitt, 1998; Kapteyn, Michaud, Smith, &

van Soest, 2006; Lindenberger et al., 1999; Radler & Ryff, 2010). If anything, this bias should make it more difficult to detect significant effects as the underrepresented groups are most likely to have experienced childhood adversities. Another potential limitation concerns the accuracy of retrospective reporting of childhood experiences, and whether recall differs for women and men. However, recent studies find that memories of specific childhood experiences are stable and fairly accurate (Hardt & Rutter, 2004; Yancura & Aldwin, 2009). In addition, a recent study comparing recall of childhood abuse reported across the two MIDUS waves found a strong correlation (C. L. Lee et al., 2014) and other recent MIDUS work found consistent reports of physical abuse within twin pairs (Friedman et al., 2015), both of which provide further evidence that recall of trauma is quite reliable.

While we focused on educational attainment as the main adult pathway, other pathways linking childhood events and adult health also exist. For example, childhood events may directly affect adult health by increasing the odds of smoking over and above the effects of low education, and other adult circumstances such as income may affect health over and above educational attainment. Modeling the full array of life-course circumstances is outside the scope of this study. Future research may want to incorporate these circumstances to glean additional insights.

Another potential limitation is that we did not have a sufficient sample size to analyze specific events (e.g., flunked out of school) and thus had to collapse them into meaningful groups (e.g., an academic event). In addition, similar to many studies, we did not have information on how often each type of event occurred, which could potentially be important. Despite these limitations, the MIDUS survey provides an exceptionally rich and underutilized source of information on type and timing of childhood exposures. This study is the first that we are aware of that has taken advantage of this information to assess how childhood events are associated with adult health; and as a result it provides novel and important findings about how the quantity, timing, and type of adverse events in early life can have enduring implications for adult cardiometabolic health.

## **Conclusion**

As with previous research on the early-life origins of later life health, our study indicates that strategies and policies aimed at reducing exposure to adverse experiences in childhood could have substantial population health benefits. Our results echo the conclusions of previous studies, which assert that enhancing the childhood environment should be treated as a public health priority. The 20th century underwent dramatic increases in health and

longevity. Acknowledging the importance of childhood circumstances in adult health and longevity, and developing strategies and policies aimed at improving childhood conditions, could facilitate continued improvement.

### Declaration of Conflicting Interests

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

### Funding

The authors disclosed receipt of the following financial support for the research, authorship, and/or publication of this article: This research was supported by a grant from the National Institute on Aging (P01-AG020166) to conduct a longitudinal follow-up of the MIDUS (Midlife in the U.S.) investigation. The original study was supported by the John D. and Catherine T. MacArthur Foundation Research Network on Successful Midlife Development. Friedman and Montez were funded in part by the Robert Wood Johnson Foundation Health & Society Scholars Program at Harvard University. Sheehan was funded in part by grant 5 R24 HD042849 (PI: Mark D. Hayward) awarded to the Population Research Center at the University of Texas at Austin by the Eunice Kennedy Shriver National Institute of Child Health and Human Development. This work was also funded by grants M01-RR000865 (UCLA CTRC grant, supported MIDUS data collection), and P30-AG028748 (Older Americans Independence Center; supported MIDUS data collection) from the National Institute of Health.

### Notes

1. *Academic* includes repeating school, dropping out of school, expelled from school, flunking out of school. *Interpersonal* includes being sent away from home, parental divorce, spousal infidelity, in-law trouble, losing home due to flood/fire (<1% of sample). *Abuse* includes ever being physically or sexually abused. *Work/financial* includes parents being unemployed for a while, ever fired, great financial loss, ever on welfare, ever unemployed for a while. *Death/illness* includes having a parent die, having a sibling die, having a child die, having a child die or suffer a life-threatening condition. *Legal* includes serious legal problems, ever being detained in jail or prison. *Parental substance abuse* includes parents abusing alcohol or drugs.
2. Men with no adverse childhood events had slightly higher prevalence of heart disease than their female counterparts (25% vs. 17%, respectively). To ensure that the stronger association between childhood adversities and heart problems among women was not an artifact of women having lower prevalence of heart problems compared with men, we ran a linear probability model with robust standard errors. The gender-by-adversity interaction was still statistically significant ( $p < .05$ ), and thus, our conclusions were robust to gender differences in prevalence of heart trouble.

## References

- Barker, D. J. P. (1997). Maternal nutrition, fetal nutrition, and disease in later life. *Nutrition, 13*, 807-813.
- 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.
- Blackwell, D. L., Hayward, M. D., & Crimmins, E. M. (2001). Does childhood health affect chronic morbidity in later life? *Social Science & Medicine, 52*, 1269-1284.
- Bosch, N. M., Riese, H., Reijneveld, S. A., Bakker, M. P., Verhulst, F. C., Ormel, J., & Oldehinkel, A. J. (2012). Timing matters: Long term effects of adversities from prenatal period up to adolescence on adolescents' cortisol stress response. The TRAILS study. *Psychoneuroendocrinology, 37*, 1439-1447.
- Cohen, S., Doyle, W. J., Turner, R. B., Alper, C. M., & Skoner, D. P. (2004). Childhood socioeconomic status and host resistance to infectious illness in adulthood. *Psychosomatic Medicine, 66*, 553-558.
- Danese, A., & Tan, M. (2014). Childhood maltreatment and obesity: Systematic review and meta-analysis. *Molecular Psychiatry, 19*, 544-554.
- 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.
- Ferraro, K. F. (2011). Health and aging: Early origins, persistent inequalities? In R. A. Settersten & J. A. Angel (Eds.), *Handbook of sociology of aging* (pp. 465-475). Springer Science+Business Media. New York, NY.
- Ferraro, K. F., & Shippee, T. P. (2009). Aging and cumulative inequality: How does inequality get under the skin? *The Gerontologist, 49*, 333-343.
- 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.
- Fisher, H. L., Jones, P. B., Fearon, P., Craig, T. K., Dazzan, P., Morgan, K., . . . Morgan, C. (2010). The varying impact of type, timing and frequency of exposure to childhood adversity on its association with adult psychotic disorder. *Psychological Medicine, 40*, 1967-1978.
- Fitzgerald, J., Gottschalk, P., & Moffitt, R. (1998). An analysis of the impact of sample attrition on the second generation of respondents in the Michigan Panel Study of Income Dynamics. *Journal of Human Resources, 33*, 300-344.
- Friedman, E. M., Karlamangla, A., Gruenewald, T., Koretz, B., & Seeman, T. E. (2015). Early life adversity and adult biological risk profiles. *Psychosomatic Medicine, 77*, 176-185.
- Galobardes, B., Davey Smith, G., & 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.

- Green, J. G., McLaughlin, K. A., Berglund, P. A., Gruber, M. J., Sampson, N. A., Zaslavsky, A. M., . . . Kessler, R. C. (2010). Childhood adversities and adult psychiatric disorders in the National Comorbidity Survey Replication I: Associations with first onset of DSM-IV disorders. *Archives of General Psychiatry, 67*, 113-123.
- Hall, A. J., Yee, L. J., & Thomas, S. L. (2002). Life course epidemiology and infectious diseases. *International Journal of Epidemiology, 31*, 300-301. doi:10.1093/ije/31.2.300
- Hamil-Luker, J., & O'Rand, A. M. (2007). Gender differences in the link between childhood socioeconomic conditions and heart attack risk in adulthood. *Demography, 44*, 137-158.
- 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.
- 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.
- Herberth, G., Weber, A., Röder, S., Elvers, H.-D., Krämer, U., Schins, R., . . . Lehmann, I. (2008). Relation between stressful life events, neuropeptides and cytokines: Results from the LISA birth cohort study. *Pediatric Allergy and Immunology, 19*, 722-729.
- Irving, S. M., & Ferraro, K. F. (2006). Reports of abusive experiences during childhood and adult health ratings. *Journal of Aging and Health, 18*, 458-485.
- Kapteyn, A., Michaud, P.-C., Smith, J. P., & van Soest, A (2006, August). *Effects of attrition and non-response in the health and retirement study*. IZA Discussion Paper No. 2246; RAND Working Paper No. WR-407. Retrieved from <http://ssrn.com/abstract=928369>
- Karlamangla, A. S., Singer, B. H., Williams, D. R., Schwartz, J. E., Matthews, K. A., Kiefe, C. I., . . . Seeman, T. E. (2005). Impact of socioeconomic status on longitudinal accumulation of cardiovascular risk in young adults: The CARDIA Study (USA). *Social Science & Medicine, 60*, 999-1015.
- Kessler, R. C., David, C. G., & Kendler, K. S. (1997). Childhood adversity and adult psychiatric disorder in the US National Comorbidity Study. *Psychological Medicine, 27*, 1101-1119.
- Kestilä, L., Koskinen, S., Martelin, T., Rahkonen, O., Pensola, T., Aro, H., . . . Aromaa, A. (2005). Determinants of health in early adulthood: What is the role of parental education, childhood adversities, and own education? *European Journal of Public Health, 16*, 305-314.
- Khlat, M., Jusot, F., & Ville, I. (2009). Social origins, early hardship and obesity: A strong association in women, but not in men? *Social Science & Medicine, 68*, 1692-1699.
- Krause, N. (1993). Early parental loss and personal control in later life. *Journals of Gerontology: Psychological Sciences, 48*, P117-P126.

- Kuh, D., Hardy, R., Langenberg, C., Richards, M., & Wadsworth, M. E. J. (2002). Mortality in adults aged 26-54 years related to socioeconomic conditions in childhood and adulthood: Post war birth cohort study. *British Medical Journal*, *325*, 1076-1080.
- Lareau, A. (2011). *Unequal childhoods: Class, race, and family life*. Berkeley: University of California Press.
- Lee, C. L., Tsenkova, V., & Carr, D. (2014). Childhood trauma and metabolic syndrome among in men and women. *Social Science & Medicine*, *105*, 122-130.
- Lee, H., Harris, K. M., & Gordon-Larsen, P. (2009). Life course perspectives on the links between poverty and obesity during the transition to young adulthood. *Population Research and Policy Review*, *28*, 505-532.
- 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.
- Lindenberger, U., Gilberg, R., Little, T. D., Nuthmann, R., Pötter, U., & Baltes, P. B. (1999). Sample selectivity and generalizability of the results of the Berlin Aging Study. In P. B. Baltes & K. U. Mayer (Eds.), *The Berlin Aging Study: Aging from 70 to 100* (pp. 56-82). New York, NY: Cambridge University Press.
- Lynch, J. W., Kaplan, G. A., & Salonen, J. T. (1997). Why do poor people behave poorly? Variation in adult health behaviours and psychosocial characteristics by stages of the socioeconomic lifecourse. *Social Science & Medicine*, *44*, 809-819.
- 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.
- McDevitt, T. M., & Omrod, J. E. (2015). *Child development and education* (6th ed.). Upper Saddle River, NJ: Pearson.
- Midei, A. J., Matthews, K. A., Chang, Y.-F., & Bromberger, J. T. (2013). Childhood physical abuse is associated with incident metabolic syndrome in mid-life women. *Health Psychology*, *32*, 121-127.
- Midlife Development in the United States. (2007). *Mid-life in the United States: A national study of health and well-being*. Retrieved from [http://midus.wisc.edu/newsletter/MIDUS\\_Final.pdf](http://midus.wisc.edu/newsletter/MIDUS_Final.pdf)
- Mirowsky, J., & Ross, C. E. (1998). Education, personal control, lifestyle and health: A human capital hypothesis. *Research on Aging*, *20*, 415-449.
- Montez, J. K. (2013). The socioeconomic origins of physical functioning among older U.S. adults. *Advances in Life Course Research*, *18*, 244-256.
- Montez, J. K., & Hayward, M. D. (2014). Cumulative childhood adversity, educational attainment, and active life expectancy among U.S. adults. *Demography*, *51*, 413-435.
- Montez, J. K., Hayward, M. D., Brown, D. C., & Hummer, R. A. (2009). Why is the educational gradient in mortality steeper for men? *Journals of Gerontology: Social Sciences*, *64*, 625-634.
- Morton, P. M., Schafer, M. H., & Ferraro, K. F. (2012). Does childhood misfortune increase cancer risk in adulthood? *Journal of Aging and Health*, *24*, 948-984.

- 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.
- Radler, B. T., & Ryff, C. D. (2010). Accounting for longitudinal retention in the MIDUS national study of health and well-being. *Journal of Aging and Health, 22*, 307-331.
- Raftery, A. E. (1995). Bayesian model selection in social research. *Sociological Methodology, 25*, 111-163.
- Repetti, R. L., Taylor, S. E., & Seeman, T. E. (2002). Risky families: Family social environments and the mental and physical health of offspring. *Psychological Bulletin, 128*, 330-366.
- Rich-Edwards, J. W., Mason, S., Rexrode, K., Spiegelman, D., Hibert, E., Kawachi, I., . . . Wright, R. J. (2012). Physical and sexual abuse in childhood as predictors of early-onset cardiovascular events in women. *Circulation, 126*, 920-927.
- Robinson, W. R. (2012). Gender-specific effects of nutritional restriction on adult obesity risk: Evidence from quasi-experimental studies. *Obesity, 20*, 2464-2466.
- Ross, C. E., & Wu, C.-L. (1995). The links between education and health. *American Sociological Review, 60*, 719-745.
- Sapolsky, R. M. (2004). *Why zebras don't get ulcers* (3rd ed.). New York, NY: Henry Holt.
- Schafer, M. H., & Ferraro, K. F. (2013). Childhood misfortune and adult health: Enduring and cascading effects on somatic and psychological symptoms? *Journal of Aging and Health, 25*, 3-28.
- Schilling, E. A., Aseltine, R. H., & Gore, S. (2008). The impact of cumulative childhood adversity on young adult mental health: Measures, models, and interpretations. *Social Science & Medicine, 66*, 1140-1151.
- Shaw, B. A., & Krause, N. (2002). Exposure to physical violence during childhood, aging, and health. *Journal of Aging and Health, 14*, 467-494.
- Slopen, N., Koenen, K. C., & Kubzansky, L. D. (2012). Childhood adversity and immune and inflammatory biomarkers associated with cardiovascular risk in youth: A systematic review. *Brain, Behavior, and Immunity, 26*, 239-250.
- Slopen, N., Lewis, T. T., Gruenewald, T. L., Mujahid, M. S., Ryff, C. D., Albert, M. A., & Williams, D. R. (2010). Early life adversity and inflammation in African Americans and Whites in the Midlife in the United States survey. *Psychosomatic Medicine, 72*, 694-701.
- Slopen, N., McLaughlin, K. A., Dunn, E. C., & Koenen, K. C. (2013). Childhood adversity and cell-mediated immunity in young adulthood: Does type and timing matter? *Brain, Behavior, and Immunity, 28*, 63-71.
- Surtees, P. G., & Wainwright, N. W. J. (2007). The shackles of misfortune: Social adversity assessment and representation in a chronic-disease epidemiological setting. *Social Science & Medicine, 64*, 95-111.
- Taylor, S. E., Lerner, J. S., Sage, R. M., Lehman, B. J., & Seeman, T. E. (2004). Early environment, emotions, responses to stress, and health. *Journal of Personality, 72*, 1365-1393.

- Thomas, C., Hyppönen, E., & Power, C. (2008). Obesity and type 2 diabetes risk in midadult life: The role of childhood adversity. *Pediatrics, 121*(5), e1240-e1249.
- Thornberry, T. P., Ireland, T. O., & Smith, C. A. (2001). The importance of timing: The varying impact of childhood and adolescent maltreatment on multiple problem outcomes. *Development and Psychopathology, 13*, 957-979.
- Turner, H. A., Finkelhor, D., & Ormrod, R. (2006). The effect of lifetime victimization on the mental health of children and adolescents. *Social Science & Medicine, 62*, 13-27.
- Turner, R. J., & Lloyd, D. A. (1995). Lifetime traumas and mental health: The significance of cumulative adversity. *Journal of Health and Social Behavior, 36*, 360-376.
- Yancura, L. A., & Aldwin, C. M. (2009). Stability and change in retrospective reports of childhood events over a five-year period: Findings from the Davis Longitudinal Study. *Psychology and Aging, 24*, 715-721.
- Zill, N., Loomis, L. S., & West, J. (1997). *The elementary school performance and adjustment of children who enter kindergarten late or repeat kindergarten: Findings from national surveys* (NCES 98-097). Washington, DC: U.S. Department of Education, National Center for Educational Statistics.
- Ziol-Guest, K. M., Duncan, G. J., & Kalil, A. (2009). Early childhood poverty and adult body mass index. *American Journal of Public Health, 99*, 527-532.