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Childhood Disadvantage and Health Problems in Middle and Later Life: Early Imprints on Physical Health?

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Abstract

Drawing from cumulative inequality theory, we examine the relationship between childhood disadvantage and health problems in adulthood. Using two waves of data from Midlife Development in the United States, we investigate whether childhood disadvantage is associated with adult disadvantage, including fewer social resources, and the effect of lifelong disadvantage on health problems measured at the baseline survey and a 10-year follow-up. Findings reveal that childhood socioeconomic disadvantage and frequent abuse by parents are generally associated with fewer adult social resources and more lifestyle risks. Health problems, in turn, are affected by childhood disadvantage and by lifestyle risks, especially smoking and obesity. Not only was early disadvantage related to health problems at the baseline survey, but childhood socioeconomic disadvantage and frequent abuse also were related to the development of *new* health problems at the follow-up survey. These findings reveal the imprint of early disadvantage on health decades later and suggest greater attention to resources, even during midlife, can interrupt the chain of risks.

Keywords

life course, health, childhood adversity, cumulative inequality

A growing number of sociological studies of life chances and well-being give priority to the life course as the analytic frame of reference. Although studying specific stages of human lives remains a valuable line of inquiry, it is the examination of the interconnectedness of life stages that has led to breakthrough discoveries by sociologists investigating a variety of topics, including educational attainment (Conley and Bennett 2000), occupational status (Elder 1974), criminal offenses (Laub and Sampson 2003), and mental health (Turner and Lloyd 1995). It is understandable that problems early in life, from low birth weight to economic deprivation, may influence status attainment and mental health, but research also shows that

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physical health is influenced by disadvantage experienced early in the life course.

Research on the early origins of adult physical health has proliferated in recent years, as investigators have sought to assess the long-term influence of childhood disadvantage (e.g., financial deprivation and abuse) on life chances in adulthood. Beyond the accumulated empirical evidence, however, there is a shift in *how* scholars study adult health: inquiries increasingly integrate information from early life, recognizing that failure to do so is largely a "downstream" research endeavor. Application of the life course perspective is seen in multiple fields, including medicine (Barker 1997; Felitti 2002), sociology of aging (O'Rand and Hamil-Luker 2005), and epidemiology (Kuh and Ben-Shlomo 2004). Indeed, although the life course concept was pioneered by sociologists (Cain 1964; Elder 1974), the growth of the subfield of *life course epidemiology* reflects this shift toward studying the early origins of adult health (Davey Smith 2012; Kuh 2007).[1]

Considerable research demonstrates important links between early experiences and adult health problems, but questions remain as to how and under what conditions early experiences threaten health in later life. For instance, given the considerable passage of time from childhood to adulthood, do early insults have temporary or enduring effects on health? If enduring, are the effects direct or largely mediated through intervening experiences and exposures? These questions guide the present analysis.

The recent proliferation of empirical research linking childhood experiences and adult health has not been matched with theoretical developments to aid interpretation of the findings and guide future research. Thus, our aims are both theoretical and empirical. First, we draw on recent theoretical developments in sociology and epidemiology to offer a conceptually integrated argument about the early origins of health problems observed decades later. Second, and distinct from most prior studies, we use longitudinal data from a national sample to examine links between multiple forms of early life disadvantage and multiple health problems in adulthood. Our central research question is whether childhood disadvantage has enduring effects on health problems in middle and later life, and we pursue it by examining widely recognized pathways between early disadvantage and adult health. We begin with a consideration of theoretical issues for the sociological analysis of life course health.

THEORETICAL BACKGROUND

The concept of accumulation is central to discussions of how early experiences shape later life outcomes (Ferraro and Morton forthcoming). Whether in sociology or toxicology, a core thesis is that the accumulation of negative exposures raises the risk of subsequent health problems. Sociological thought on the topic often draws from Merton's (1968) analysis of cumulative advantage—referred to as the Matthew effect—for illuminating how early distinction in science leads to additional honor and opportunity. Although Merton (1968:63) drew attention to the "cumulation of prestige for successive accomplishments," he also noted the challenges faced by individuals who do not get off to an auspicious scientific start. Thus, many scholars of life course health use the concept of accumulation to predict that negative exposures lead to additional negative outcomes. Others question the simplicity of this prediction, noting that some forms of disadvantage may not unilaterally lead to negative outcomes. For instance, financial deprivation during the Great Depression actually led to beneficial effects on emotional health for middle-class women (Elder and Liker 1982); and compensatory behaviors may counteract the effects of negative exposures (Ferraro and Kelley-Moore 2003).

The concept of cumulative advantage—growing inequality over time—is appealing for many fields of sociological inquiry, but DiPrete and Eirich (2006) argue for greater precision in the sociological use of the term. Three points in their essay are essential for research on the early origins of adult health.

First, they identify several forms of cumulative advantage, some of which require rather strong assumptions when applied to the study of human lives and social inequality (e.g., future inequality depends solely on current accumulation). Although there may be simple forms of cumulative advantage that operate like compound interest, this is unlikely when discussing life course development involving health (because of the episodic nature of human development and illness onset). Second, DiPrete and Eirich draw a distinction between the direct impact of an event on some outcome over time versus the impact of an event leading to other events or experiences, which combine to affect the outcome (see also Berkman 2009). Third and most germane to studies of health, they call for more attention to mechanisms that "turn off" the influence of accumulated exposures. Although it is quite reasonable to anticipate that initial inequality will persist or grow over time, the influence of accumulation processes may not grow, and this has been demonstrated in studies of life course health (Dupre 2007; House et al. 1994). Moreover, isolating how to turn off the influence of accumulated exposures is especially significant for public policy initiatives.

More recently, scholars have formulated cumulative inequality theory to integrate elements of the cumulative advantage theories (Dannefer 2003; DiPrete and Eirich 2006; O'Rand 1996) with life course (Elder 1998) and stress process (Pearlin 1989) theories. Identifying developmental and demographic processes, cumulative inequality theory emphasizes how negative events and experiences place people at heightened risk, how positive experiences create opportunities, and how the configuration of both can alter life chances for individuals and collectivities (Ferraro, Shippee, and Schafer 2009; Schafer, Ferraro, and Mustillo 2011).

This theory prioritizes childhood as a pivotal life stage leading to social inequality, "especially when differences in experience or status emerge early" (Ferraro et al. 2009:419). The processes leading to social inequality begin early, and we examine two main reasons why childhood is pivotal to adult health. First, childhood conditions reflect intergenerational processes: "influenced by genes and environment, family lineage is critical to status differentiation early in the life course" (Ferraro and Shippee 2009:337). For instance, parental socioeconomic status (SES) during one's childhood may be directly and indirectly related to a child's future health, due to how SES shapes future lifestyles and resources that also influence health.[2] Second, stressors during childhood may alter personal development and social functioning. Indeed, in a review of hundreds of studies on the topic, Miller, Chen, and Parker (2011:960) conclude that "early stress fosters vigilance for threat and mistrust of others, traits that make it difficult to form deep social ties."

Cumulative inequality theory also assigns priority to the configuration of risks and resources over the life course. The theory holds that disadvantage, an unfavorable position in a status hierarchy, increases exposure to risk or the "probability of a hazard or negative event" (Ferraro et al. 2009:422). As such, early disadvantage increases the likelihood of exposure to later risks—and perhaps a life filled with hardship—but resources help actors respond to those exposures. For instance, supportive social relations may help a person cope with early life disadvantage, but strained interpersonal relations make it more difficult to respond effectively to early negative exposures.

For the study of life course health, it is important to examine not only whether early exposures generate chains of risk, but also the midlife resources that may mediate early disadvantage. According to cumulative inequality theory, accounting for accumulated risks and resources may reveal processes that can turn off the presumed influence of early disadvantage. This is not to say that early insults do not have a direct effect on health, only that other life course risks and resources should be considered simultaneously.

Given the large literature showing a link between childhood disadvantage and adult health, we examine two models to elucidate

this relationship. According to Berkman (2009:33–34), the social trajectory model posits that early exposures "shape opportunities or barriers" to later life exposures—and later life exposures are the presumed engine of poor health. Evidence for this model exists when early exposures do not have an independent (i.e., direct) effect on adult health problems; rather, the effect is indirect because of how childhood experiences have "redirected" one's life. By contrast, a cumulative exposure model specifies that early experiences shape adult social conditions and health problems. Parallel to cumulative inequality theory's emphasis on risks and resources, the cumulative exposure model highlights the contingent nature of the development of adult health problems, including how resources may reduce "risks set by trajectories in early childhood" (Berkman 2009:35).

LIFE COURSE ANALYSES OF ADULT HEALTH

The body of research documenting the early origins of adult health is impressive, but important questions remain regarding how and under what conditions childhood disadvantage influences health in later life. In reviewing the empirical literature to address our main research question, three characteristics of prior research are noteworthy.

Domains of Disadvantage

Researchers have used two basic approaches to study a wide range of childhood statuses, events, and experiences as antecedents of adult health. One approach focuses on a single domain of life; in some cases, this is a singular event such as death of a parent. The domains of childhood experience that have received the most systematic attention include household income (Case, Lubotsky, and Paxson 2002), household SES (O'Rand and Hamil-Luker 2005), child abuse (Greenfield and Marks 2009a; Shaw and Krause 2002), changes in family composition such as parental death or divorce (Cherlin, Chase-Lansdale, and McRae 1998; McLanahan, Tach, and Schneider 2013), and health during childhood or adolescence (Blackwell, Hayward, and Crimmins 2001). These types of detailed investigations of a single domain have greatly advanced our understanding of the long-term effects of specific childhood insults.

At the same time, a second approach calls for greater attention to multiple domains of childhood disadvantage, emphasizing the fact that one domain may be related to others (e.g., parental divorce and household financial strain). From this perspective, failure to consider related negative exposures may overestimate the effect of a single domain studied. Pearlin and colleagues (2005:209–210) poignantly describe the problems associated with studying one exposure in isolation: "It cannot be assumed that a continuous or repeated strain has a presence separate and apart from other strains that individuals might experience. Such an assumption may result in erroneously attributing health effects to exposure to but a single serious stressor when these effects might also be the consequence of unobserved stressors having a simultaneous, overlapping, or sequential presence in time." Thus, rather than study each early exposure in isolation, the present study simultaneously considers multiple domains of early disadvantage.[3]

Health Outcomes Studied

Second, previous research on childhood disadvantage has studied a striking array of health outcomes, but investigators have used different analytic strategies when studying these diseases. Most studies examine a single disease, such as cancer (Morton, Schafer, and Ferraro 2012), heart attack (O'Rand and Hamil-Luker 2005), or hypertension (Stein et al. 2010), as well as various measures of psychological disorder (e.g., depressive symptoms [Booth, Rustenbach, and McHale 2008]).[4] Other studies examine multiple diseases but analyze each separately (Blackwell et al. 2001; Schafer, Wilkinson, and Ferraro 2013). One advantage of analyzing each disease separately is to identify specific etiologic mechanisms, but some stressors and behaviors are associated with multiple diseases

(i.e., shared risk factors). From a different angle, Schafer and Ferraro (2012) examine being disease free in later life, finding that childhood disadvantage reduces the likelihood of avoiding disease. In short, ample evidence shows that early disadvantage is associated with the development of one or more diseases, but few studies examine whether childhood disadvantage leads to disease accumulation in later life. Studying multiple diseases is distinct from studying one disease at a time, because the former may elucidate risk factors that lead to comorbidity, which, in turn, raises the risk of poor functional status and premature mortality (Gijsen et al. 2001).

Closely related to the issue of disease accumulation is the fact that most studies rely on a cross-sectional report of health problems (e.g., Springer 2009; Stein et al. 2010). This is useful for estimating prevalence, but it limits studies of the early origins of adult health. Given the length of time between exposure and the presumed outcome, the effect could be exhausted before, or not appear until after, the cross-sectional assessment. Longitudinal studies do not eliminate this concern but provide the opportunity to observe the onset of new health problems. Relatively few population-based studies examine the co-occurrence of physical health problems resulting from early disadvantage; Greenfield and Marks (2009a) is the only one of which we are aware that uses population-based longitudinal data. Its focus, however, is one domain of disadvantage—child abuse—and it does not account for the influence of important mediating variables (e.g., smoking or obesity). We are unaware of any prior study that uses longitudinal data to examine the link between multiple domains of childhood disadvantage and accumulated health problems.

Processes Linking Childhood Disadvantage and Adult Health Problems

The literature raises the concern that some studies of the link between childhood disadvantage and adult disease omit important events and experiences in the intervening period—reflective of different life course pathways. According to cumulative inequality theory, this omission is usually manifest in two ways: (1) inadequate attention to mediating factors in adulthood and (2) left-censoring due to studying older people only (e.g., the most noxious experiences during childhood may lead to premature mortality). Whether due to sample composition or inadequate mediating or control variables, failure to account for midlife experiences and selection processes may yield misleading conclusions regarding the effect of negative childhood experiences on adult health. As such, it is important to identify whether the effect of early risk factors on adult health is direct or more or less fully accounted for by intervening experiences (i.e., cumulative exposure versus social trajectory models, respectively).

Three main factors have garnered the most attention in previous studies of the period between childhood disadvantage and adult health. First, recognizing that childhood disadvantage may reduce the likelihood of SES attainment, scores of studies reveal independent effects of early disadvantage on adult health after accounting for SES attainment (Blackwell et al. 2001; Schafer et al. 2013).

Second, studies explicate how early disadvantage leads to behavioral responses and lifestyle choices in adolescence and young adulthood that compromise health; early disadvantage may raise the risk of smoking (Lloyd and Taylor 2006), alcohol dependence (Lloyd and Turner 2008), and obesity (Greenfield and Marks 2009b). Although one may regard these choices as "irrational" coping mechanisms because of their consequences to physical health, people sometimes adopt these behaviors to help manage emotional distress following major or traumatic events (Felitti et al. 1998; Lloyd and Taylor 2006).

Third, many studies examine psychosocial resources, but much more attention has been given to psychological factors, such as personal control (Irving and Ferraro 2006), than to social resources per se. Ample evidence shows that risky families lead to vulnerabilities in offspring's social functioning (Repetti,

Taylor, and Seeman 2002), but few studies adjust for social support and strain during adulthood or examine the possibility that these factors mediate the relationship between childhood disadvantage and adult health. A logical next step is to examine interpersonal relationships during adulthood more closely, given that victimization and other types of traumatic childhood experiences may lead to a mistrust of others or a sense of social detachment (Macmillan 2001; Miller et al. 2011).

Although adult SES, lifestyle choices, and psychosocial resources have garnered the most attention, it is possible that early disadvantage manifests in poor health long before adulthood. For instance, Hussey, Chang, and Kotch (2006) report that the health consequences of child abuse are observed as early as adolescence, but we are unaware of any study that treats adolescent health as a potential mediator of the relationship between childhood disadvantage and adult health. It is certainly reasonable to consider it as exogenous—another form of *early life* disadvantage—but we examine whether poor health in adolescence is a mediator between childhood disadvantage and adult health problems.

Research Questions

The present investigation uses longitudinal data to systematically examine the influence of multiple domains of early disadvantage on multiple health problems during adulthood, while testing for potential mediation due to adolescent health and adult SES, lifestyle factors, and social and psychological functioning. Using longitudinal data enables us to examine not only initial health problems but also new health problems during the 10-year follow-up period. Building on the contributions of prior studies, we specify three research questions.

1. *Is childhood disadvantage associated with more risks and fewer resources in adulthood?* Given the accumulated research on the influence of childhood disadvantage on human development, we anticipate that childhood disadvantage will lead to more adult risks and fewer resources.
2. *Is childhood disadvantage associated with more health problems in adulthood?* Using data from the baseline survey and a 10-year follow-up, we examine whether childhood disadvantage has an independent effect on initial and new health problems, respectively. We anticipate a direct effect on each outcome, reflecting the cumulative exposure model.
3. *Do adolescent health problems and adult lifestyle factors and resources mediate the relationship between childhood disadvantage and adult health problems?* Building on our answers to questions 1 and 2, we anticipate partial, but not complete, mediation.

METHODS

Sample

Data for this study come from the National Survey of Midlife Development in the United States (MIDUS), a sample of adults age 25 to 74 years. This sample is useful because of its (1) battery of retrospective questions about childhood disadvantage, (2) extensive measurement of adult risks and resources, and (3) 10-year longitudinal follow-up.

Data collection commenced in 1995 to 1996 with random-digit dialing to obtain a sampling frame of English-speaking noninstitutionalized adults age 25 to 74 in the contiguous 48 states (Brim, Ryff, and Kessler 2004). The response rate from the telephone interviews was 70 percent. The second stage included a two-part follow-up questionnaire mailed to respondents who participated in the telephone interview (86.6 percent response rate). The overall response rate was 61 percent (.70 x .87 = .61). The sample consists of 3,032 participants who completed both the telephone interview and mail questionnaire at Wave 1 (W1). The average age of the sample was about 47 years, and 52 percent of respondents were women. We used poststratification weights in all analyses to adjust

for differences in probability of selection and nonresponse.

W1 respondents were re-contacted about 10 years later to participate in Wave 2 (W2). Of the 3,032 respondents who completed both the W1 telephone and self-administered questionnaire, 1,748 completed both modes of W2 data collection (58 percent). A MIDUS technical report regarding W2 measurement revealed that 10 respondents were actually younger than 25 years at W1; these cases were removed from all analyses. About 16 percent of the sample died during the follow-up period. Nonresponse attrition was higher for persons who were nonwhite, male, not married, and in poorer health (Radler and Ryff 2010). To adjust for nonrandom attrition, we used selection bias models in all longitudinal analyses (Heckman 1979).

MEASUREMENT

Health Problems

Adult health problems were measured by self-report of 31 diseases or conditions.[5] Two diseases, heart trouble and cancer, were queried in the telephone interview. For heart disease, respondents were asked: "Have you ever had heart trouble suspected or confirmed by a doctor?" For cancer, respondents were asked: "Have you ever had cancer?"

The remaining 29 items were asked in the mail questionnaire with a checklist format: "In the past 12 months, have you experienced or been treated for any of the following?" (Each condition was scored 1 if present, zero otherwise.) Although the battery of health problems probed was extensive, age of onset is not available for these conditions.[6]

We coded *W1 health problems* as the sum of the 31 conditions, consistent with other investigators (e.g., Shaw and Krause 2002). For W2, *new health problems* was coded as the sum of all diseases reported at W2 but not at W1. The mean for W1 health problems is 2.742 (SD = 2.820), and new health problems is 1.628 (SD = 1.707) (see Table 1). Given the skewed distributions for these variables, we used a negative binomial regression model for analyses (Long 1997).

Early Life Disadvantage

We initially examined the prevalence of and correlations among 12 indicators of early life disadvantage as well as the prognostic validity of alternative coding schemes on the outcomes.[7] Those analyses and conclusions from previous studies led us to formulate three domains of childhood disadvantage—*SES, family composition,* and *abuse. Adolescent health problems* is also a type of early life disadvantage, but it may be endogenous to childhood disadvantage; we thus analyze it throughout as a potential mediator.

Childhood SES was measured with three items: education of household head (father, or mother if the father was not present, ranging from 1 = less than 8 years of schooling to 12 = professional degree); financial strain (from a lot better off to a lot worse off, compared to other families); and receipt of welfare (ever during childhood or adolescence). Whereas the early life indicators were measured on different scales, we standardized the 12 variables on a 0 to 1 scale before summing into domains of disadvantage.[8]

Family composition was also measured with three items: no male in the household (based on questions probing residence with biological parents and head of household); parental divorce (before age 16); and death of a parent (before age 16).

Child abuse by parents (physical and emotional) was drawn from the Conflict Tactics Scale applied to children (Straus et al. 1998).[9] The physical abuse measures tap behaviors during childhood by mother and by father (or figure who raised the respondent) presented in two lists, the latter assessing severe violence: (1) pushed, grabbed, or shoved; slapped; threw something at and (2) kicked, bit, or hit with a fist; hit or tried to hit with something; beat up; choked; burned or scalded. Measures of emotional abuse came from a list of six items: insulted; sulked; stomped away; did something to spite; threatened; and kicked/smashed something. Response categories for each behavior were coded as never (0), rarely (1), and sometimes or often (2). MIDUS never used the word "abuse" but focused

Table 1. Descriptive Statistics of Study Variables from the National Survey of Midlife Development in the United States (MIDUS), $N = 3,022$

	Range	Mean	SD
Health Problems, W1	0 to 28	2.742	2.820
New Health Problems, W2	0 to 24	1.628	1.707
Early Life Disadvantage			
Childhood SES	0 to 3	1.186	.507
Childhood Family Composition	0 to 2	.263	.558
No Abuse by Parents (reference group)	0,1	.273	
Rare Abuse	0,1	.315	
Frequent Abuse, 1 Type	0,1	.184	
Frequent Abuse, 2 Types	0,1	.228	
Adolescent Health Problems	0 to 2	.392	.424
Adult Characteristics			
Education	4 to 20	13.783	2.615
Income	1 to 5	2.887	1.412
Financial Strain	1 to 3	2.144	.652
Lifetime Smoking (10,000 cigarettes)	0 to 148.701	11.908	19.092
Heavy Drinker	0,1	.244	
Obese	0,1	.246	
Family Support	1 to 4	3.420	.617
Friend Support	1 to 4	3.221	.677
Family Strain	1 to 4	2.125	.616
Friend Strain	1 to 4	1.947	.518
Social Integration	1 to 7	4.674	1.456
Ever Divorced	0,1	.342	
Personal Control	1 to 7	5.455	1.044
Demographics			
Age	25 to 74	47.140	13.073
Nonwhite	0,1	.121	
Female	0,1	.516	
Cognition, W2	–2.940 to 3.011	–.057	1.005

Note: Descriptive statistics are from W1 unless otherwise noted. To create domains of childhood disadvantage, we inverted the coding of selected items.

questioning on specific acts that draw on episodic rather than semantic memory (Kessler, Mroczek, and Belli 1999). All abuse questions (and financial strain) were collected via the mail questionnaire, which is a preferred survey mode for measuring sensitive topics (Tourangeau and Yan 2007).

After identifying both types of abuse by mother and father, we created profiles that incorporate the frequency and type of abuse: (1) never experienced physical or emotional abuse, (2) rarely experienced one or both types of abuse, (3) frequently (sometimes or often) experienced one type of abuse, and (4) frequently experienced both types of abuse. (We adapted the classification scheme from Greenfield and Marks [2009a]; category 1 is the reference group.)

Adolescent health problems was measured with two items, self-rated physical and mental health, each referencing age 16. Response categories ranged from poor to excellent but were standardized to range from zero to one ($\alpha = .71$ when combined).

Because all measures of childhood and adolescent disadvantage were derived from

retrospective questions, the conclusions should be interpreted with caution. Most studies comparing retrospective reports to administrative records (e.g., court-substantiated cases of abuse) reveal that false positives from the former are rare, but false negatives are more common (Hardt and Rutter 2004; Widom and Shepard 1996). Thus, the likely bias is that childhood disadvantage is *underestimated* in these analyses, implying that significance tests are "much more conservative than would be the case under perfect measurement" (Alwin 2007:32).[10]

Potential Mediators and Covariates

As noted earlier, it is important to consider multiple potential mediators when studying the link between childhood disadvantage and adult health (Marmot 2004). Based on the literature, we examined four domains of potential mediators in detail: adolescent health (described earlier), SES, behavioral (lifestyle) responses, and social psychological resources and functioning.

To account for potential SES mediation, we included covariates for respondent's *education* (number of years of completed education) and *household income* adjusted by household size and recoded into five percentile categories (bottom 20 percent, 21st to 40th percentile, and so on). We also incorporated a measure of *financial strain* during adulthood; responses range from 1 (no difficulty paying monthly bills) to 3 (very difficult to pay monthly bills).

The substantial literature on the behavioral responses that ensue from childhood disadvantage led us to examine three lifestyle risk variables (Felitti et al. 1998; Lloyd and Turner 2008). Two are health-related behaviors measured at W1 (*lifetime smoking, heavy drinking*).[11] The third, *obesity,* is not a health behavior but results from diet, lifestyle, genes, and environment; obesity is defined by a body mass index [kg/m^2] $\geq$ 30 (self-reported weight and height).

Given that social psychological resources and functioning have received relatively little attention in prior studies on the topic, we included seven variables. Some types of childhood disadvantage may lead to difficulty in forming close social ties, so we included two measures of social support: *family support* and *friend support*. Each domain was measured with four items reflecting positive relations (i.e., care about you, understand how you feel, reliable when facing crisis, and confide about worries). The indices manifest good reliability (α = .82 and α = .88, respectively, for family and friend support). We also included two measures of relationship strain: *family strain* and *friend strain*. Each domain was measured with four items reflecting difficult relations (i.e., too many demands, criticize you, let you down, and get on your nerves) (Schuster, Kessler, and Aseltine 1990). The indices manifest good reliability (each with α = .79).

We also adjusted for the degree of *social integration* with three Likert items (e.g., I don't feel I belong to anything I'd call a community), each with seven response categories (strongly agree to strongly disagree) (α = .73). A binary variable indicates if the person was *ever divorced*.

Given that negative childhood experiences may make life seem out of control, we incorporated a 12-item index to tap *personal control* (e.g., "what happens to me in the future depends on me"). Responses for the items range from 1 (strongly disagree) to 7 (strongly agree), and we calculated the average score across all responses. The index was coded so that higher values indicate greater personal control, and it manifests high reliability (α = .84).

All multivariate estimates were adjusted for *age, sex,* and *race* (white versus nonwhite). Finally, although most previous studies fail to show meaningful age differences in the accuracy of self-reports of factual information (Alwin 2007; Rodgers and Herzog 1987), we incorporated a measure of cognition to examine the possibility of retrieval errors for the retrospective questions. Unfortunately, MIDUS did not assess cognition until the second wave, but it provides an opportunity to address the potential influence of cognitive status on parameter estimates in our longitudinal analyses. Cognition was measured with the Brief Test of Adult Cognition by Telephone

(BTACT), a comprehensive battery encompassing six cognitive domains, including episodic verbal memory, working memory span, verbal fluency, inductive reasoning, speed of processing, and task-switching. We used the composite BTACT score, standardized in *z*-units, as a single score measuring various cognitive domains; the composite score demonstrates strong external validity and high reliability ($\alpha = .82$; Tun and Lachman 2006).

ANALYSIS

The analysis was completed in two main stages. First, anticipating that childhood disadvantage would lead to more adult risks and fewer resources, we examined the relationship between early disadvantage and 14 potential mediators. Given differences in the measurement of the 14 variables, we used general linear, binary logistic, and ordered logistic models.

Second, we estimated the relationships between childhood disadvantage and adult health problems while accounting for the potential mediators. We analyzed health problems at the baseline survey as well as new health problems at W2. Given that health problems is a count variable, we estimated relationships with negative binomial regression and present incidence rate ratios and 95 percent confidence intervals for ease of interpretation (Long 1997). We performed these analyses sequentially by adding potential mediating variables, separately and in blocks, to examine changes in the incidence rate ratios.

Finally, to identify different pathways leading from childhood disadvantage to adult health, we estimated indirect and total effects using Mplus 7.3 for all 14 potential mediators (Muthén and Muthén 2014). To formally test for mediation, we examined the product of the relationship between domains of childhood disadvantage and each potential mediator, and the relationship between each potential mediator and adult health problems (MacKinnon, Fairchild, and Fritz 2007). We estimated standard errors for the indirect effects in Mplus using the delta method.

To address missing data, we first examined the prevalence of missing observations and patterns of nonresponse. Over 93 percent of respondents had valid scores on all indicators of childhood characteristics (and 4.8 percent were missing on one item only). Among the indicators of childhood characteristics, missing data were most frequent for education of household head (4.1 percent). For the adult characteristics, over 90 percent of respondents had valid scores on all of the indicators of adult characteristics, and about 8 percent were missing one item only. The adult characteristics with the most missing data were obesity (6.1 percent), computed from self-reported weight and height, and household income (5.4 percent). Income was imputed by the MIDUS team based on each respondent's sex, education, and age. To handle all other item-missing data, we used Stata's ICE routine for multiple imputation (Royston 2005). We implemented 10 imputations, using values sampled from the posterior predictive distribution and combined using Rubin's rules. Estimates varied slightly when the models used listwise deletion, but the major conclusions were unchanged.[12]

RESULTS

Risks and Resources

To examine our first research question, we tested whether childhood disadvantage is related to each of the variables assessing risks and resources identified as potential mediators. For ease of presentation, we present the 14 equations in two tables, with Table 2 displaying the nine continuous outcomes (estimated with the general linear model) and Table 3 displaying the five categorical outcomes (estimated with binary or ordered logistic regression). Table 2 displays results from regressing the continuous outcomes on early life disadvantage and demographic characteristics.

As Table 2 shows, three variables reflecting early life disadvantage are associated with six or more outcomes: childhood SES, one type of frequent abuse, and frequent physical and emotional abuse. Being raised in an SES-disadvantaged household is negatively associated with educational attainment, friend support, social integration, and personal

Table 2. Early Antecedents of Risks and Resources: Continuous Outcomes, $N = 3{,}016$

	Adolescent Health Problems	Education	Lifetime Smoking	Family Support	Friend Support	Family Strain	Friend Strain	Social Integration	Personal Control
Early Life Disadvantage									
Childhood SES	.003	−1.293***	2.492***	−.049	−.113**	.067*	−.011	−.226***	−.128**
	(.020)	(.110)	(.679)	(.030)	(.033)	(.029)	(.026)	(.062)	(.045)
Childhood Family Composition	.004	.074	1.215*	−.112***	.035	.052*	.004	−.041	.027
	(.016)	(.084)	(.607)	(.029)	(.026)	(.026)	(.021)	(.058)	(.037)
Rare Abuse[a]	−.014	.378*	.205	−.055	−.015	.180***	.071*	−.055	.000
	(.022)	(.146)	(.837)	(.033)	(.038)	(.036)	(.032)	(.080)	(.056)
Frequent Abuse, 1 Type[a]	.074**	.170	2.692*	−.175***	−.066	.293***	.082*	−.340***	−.183**
	(.027)	(.145)	(1.043)	(.039)	(.044)	(.038)	(.035)	(.092)	(.067)
Frequent Abuse, 2 Types[a]	.158***	−.013	3.974***	−.393***	−.202***	.458***	.189***	−.342***	−.418***
	(.028)	(.138)	(.952)	(.040)	(.045)	(.040)	(.037)	(.091)	(.069)
Demographics									
Age	−.002**	−.026***	.373***	.004***	.001	−.007***	−.004***	.016***	−.010***
	(.001)	(.004)	(.030)	(.001)	(.001)	(.001)	(.001)	(.002)	(.002)
Nonwhite	−.020	−.462*	−4.673***	−.019	−.107*	.076	.087*	.005	−.035
	(.028)	(.181)	(.772)	(.044)	(.051)	(.045)	(.040)	(.097)	(.068)
Female	.021	−.297**	−6.516***	.065*	.210***	.174***	−.040	.023	−.254***
	(.018)	(.103)	(.697)	(.026)	(.029)	(.026)	(.023)	(.060)	(.043)
Constant	.434***	16.047***	−6.342***	3.412***	3.257***	2.027***	2.072***	4.288***	6.316***
	(.042)	(.245)	(1.568)	(.059)	(.071)	(.060)	(.057)	(.147)	(.103)
R-squared	.035	.114	.144	.093	.050	.125	.035	.044	.067

Note: Unstandardized coefficients; robust standard errors are in parentheses.
[a]Reference group is no abuse by parents.
*$p < .05$; **$p < .01$; ***$p < .001$ (two-tailed tests).

control as well as higher lifetime smoking and family strain. Childhood family composition is associated with three outcomes: higher lifetime smoking, less family support, and more family strain. In comparison to respondents reporting no abuse, the frequent abuse variables show a clear pattern of greater risk and fewer resources. In most equations, the slope for frequent physical and emotional abuse is greater than for one type of frequent abuse (e.g., .158 versus .074, respectively, predicting adolescent health problems). Generally speaking, childhood disadvantage is associated with fewer resources and more lifestyle risks, and the influence of the demographic variables is as anticipated (e.g., lower lifetime smoking among women and nonwhite adults).

Results from Table 3 also reveal the gravity of growing up in an SES-disadvantaged household on three outcomes, raising the risk of low-income and financial strain during adulthood as well as becoming a heavy drinker. Childhood family composition is associated with one outcome: respondents raised in single-parent households were more likely to report financial strain during adulthood. Frequent abuse—both one type and two types—raised the likelihood of the respondent becoming a heavy drinker and divorced. People who experienced frequent physical and emotional abuse (two types) also reported greater financial strain during adulthood.

Findings from Tables 2 and 3 reveal that some forms of childhood disadvantage have a long-term influence on adult resources and risks. Most notably, when young people experienced frequent physical and emotional abuse by parents, we find consequences for 11 of the 14 outcomes, ranging from adolescent health to eventual divorce. Childhood SES disadvantage influenced nine of the 14 outcomes.

Health Problems in Adulthood

To examine the effects of childhood disadvantage on health problems, as well as the potential mediating influence of later life resources and risks, Table 4 displays the incidence rate ratios from six negative binomial regression models. Model 1 of Table 4 regresses health problems at W1 on childhood disadvantage and demographic variables, Models 2 through 5 add blocks of potential mediators (adolescent health problems, adult SES, lifestyle risks, and social psychological resources, respectively), and Model 6 includes all variables.

The analyses reveal that childhood family composition is related to health problems in all models; respondents who grew up in nontraditional households experienced more health problems. Frequent physical and emotional abuse is associated with more health problems in Models 1 through 4 but not after the social and psychological resources are added in Model 5. Model 2 reveals that adolescent health problems are associated with more adult health problems, suggestive of life course continuity, but not after adjustment for adult characteristics. Model 6 clarifies that five of the potential mediators are related to accumulated health problems: smoking, obesity, family support, family strain, ever divorced, and low personal control are associated with more health problems.

To examine pathways leading from childhood disadvantage to adult health problems, we estimated indirect effects using Mplus. We found evidence that childhood disadvantage has an indirect effect on health problems through lifetime smoking and personal control. Although childhood SES disadvantage does not directly affect adult health, it is indirectly related through two separate pathways: smoking ($z = 3.825$, $p < .001$) and personal control ($z = 3.925$, $p < .001$) (z-scores associated with indirect effects [product of coefficients]). In addition, frequent parental abuse, involving one type of maltreatment, shows an indirect effect on health problems through smoking ($z = 2.473$, $p < .05$), family support ($z = –1.966$, $p < .05$), and greater family strain ($z = 2.197$, $p < .05$). The effect of frequent physical and emotional abuse on health problems is mediated via four pathways: smoking ($z = 2.892$, $p < .01$), family support ($z = –2.023$, $p < .05$), family strain ($z = 2.240$, $p < .05$), and personal control ($z = 4.240$, $p < .001$).

Table 3. Early Antecedents of Risks and Resources: Categorical Outcomes, $N = 3{,}016$

	Income	Financial Strain	Heavy Drinker	Obese	Ever Divorced
Early Life Disadvantage					
Childhood SES	–.608***	.198*	.317**	.079	.106
	(.082)	(.092)	(.107)	(.107)	(.094)
Childhood Family Composition	.059	.192*	.075	–.086	.033
	(.072)	(.078)	(.095)	(.091)	(.081)
Rare Abuse[a]	.184	–.112	.074	.100	.169
	(.101)	(.111)	(.152)	(.143)	(.130)
Frequent Abuse, 1 Type[a]	.276*	.116	.364*	.204	.382**
	(.121)	(.132)	(.158)	(.161)	(.141)
Frequent Abuse, 2 Types[a]	.032	.365**	.355*	.248	.528***
	(.108)	(.125)	(.153)	(.150)	(.136)
Demographics					
Age	.025***	–.018***	–.039***	.020***	.008*
	(.003)	(.003)	(.005)	(.004)	(.003)
Nonwhite	–.659***	.718***	–.386*	.473**	–.022
	(.128)	(.142)	(.194)	(.158)	(.152)
Female	–.381***	.374***	–.754***	.055	.140
	(.076)	(.086)	(.107)	(.105)	(.093)
Constant	Cut 1: –.744	Cut 1: –2.199	.474*	–2.330***	–1.614***
	(.177)	(.199)	(.241)	(.241)	(.214)
	Cut 2: .251	Cut 2: .654			
	(.175)	(.193)			
	Cut 3: 1.171				
	(.177)				
	Cut 4: 2.221				
	(.184)				
AIC	9447.980	5672.154	3238.436	3412.027	3848.079
BIC	9520.121	5732.271	3292.542	3466.132	3902.184

Note: Logistic regression estimates for ever divorced, heavy drinker, and obese; ordered logistic regression estimates for income and financial strain. Robust standard errors are in parentheses.
[a]Reference group is no abuse by parents.
*$p < .05$; **$p < .01$; ***$p < .001$ (two-tailed tests).

New Health Problems in Adulthood

Table 5 replicates the W1 modeling sequence on new health problems at W2, but adds two modifications to account for the longitudinal design. First, all models include the count of W1 health problems to acknowledge that a higher baseline disease burden may play a role in the accumulation of additional health problems. Indeed, such is the case in Models

Table 4. Negative Binomial Regression of W1 Health Problems on Early Life and Adult Characteristics, $N = 2{,}995$

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Early Life Disadvantage						
Childhood SES	1.035[a]	1.036	.988	1.016	.992	.970
	(.945 – 1.134)[b]	(.946 – 1.135)	(.894 – 1.092)	(.928 – 1.113)	(.901 – 1.093)	(.873 – 1.078)
Childhood Family Composition	1.121*	1.121*	1.113*	1.120*	1.143**	1.135**
	(1.026 – 1.225)	(1.028 – 1.224)	(1.018 – 1.216)	(1.025 – 1.224)	(1.046 – 1.249)	(1.040 – 1.240)
Rare Abuse[c]	.918	.925	.940	.910	.893	.903
	(.794 – 1.062)	(.800 – 1.069)	(.814 – 1.086)	(.787 – 1.053)	(.768 – 1.038)	(.780 – 1.045)
Frequent Abuse, 1 Type[c]	1.127	1.117	1.126	1.096	1.028	1.015
	(.969 – 1.311)	(.960 – 1.299)	(.971 – 1.305)	(.942 – 1.274)	(.881 – 1.200)	(.872 – 1.182)
Frequent Abuse, 2 Types[c]	1.333***	1.299**	1.304**	1.258**	1.114	1.080
	(1.135 – 1.565)	(1.107 – 1.524)	(1.118 – 1.521)	(1.075 – 1.472)	(.957 – 1.298)	(.931 – 1.255)
Adolescent Health Problems		1.189**				1.090
		(1.078 – 1.311)				(.991 – 1.199)
Adult Characteristics						
Age	1.016***	1.017***	1.016***	1.013***	1.015***	1.012***
	(1.012 – 1.020)	(1.013 – 1.021)	(1.012 – 1.021)	(1.008 – 1.017)	(1.011 – 1.019)	(1.008 – 1.017)
Nonwhite	.960	.962	.925	.975	.949	.950
	(.786 – 1.173)	(.789 – 1.174)	(.749 – 1.141)	(.796 – 1.194)	(.770 – 1.170)	(.768 – 1.173)
Female	1.326***	1.320***	1.292***	1.397***	1.227***	1.272***
	(1.206 – 1.458)	(1.202 – 1.451)	(1.176 – 1.419)	(1.270 – 1.538)	(1.120 – 1.344)	(1.160 – 1.394)
Education			.968**			.985
			(.948 – .988)			(.965 – 1.006)
Income			1.003			1.012
			(.971 – 1.036)			(.981 – 1.044)
Financial Strain			1.189***			1.066
			(1.096 – 1.290)			(.991 – 1.146)
Lifetime Smoking				1.007***		1.006***
				(1.005 – 1.009)		(1.003 – 1.008)

(continued)

Table 4. *(continued)*

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Heavy Drinker				1.054		.999
				(.944 – 1.176)		(.901 – 1.108)
Obese				1.299***		1.227***
				(1.175 – 1.435)		(1.114 – 1.352)
Family Support					1.086*	1.085*
					(1.003 – 1.176)	(1.004 – 1.172)
Friend Support					.973	.974
					(.914 – 1.035)	(.916 – 1.035)
Family Strain					1.153*	1.136*
					(1.032 – 1.290)	(1.017 – 1.268)
Friend Strain					1.009	.995
					(.886 – 1.150)	(.878 – 1.128)
Social Integration					.963	.972
					(.927 – 1.001)	(.936 – 1.009)
Ever Divorced					1.154**	1.110*
					(1.047 – 1.271)	(1.006 – 1.225)
Personal Control					.825***	.842***
					(.780 – .873)	(.796 – .890)
–2LL	6484.994	6476.881	6453.770	6437.062	6375.981	6336.985

[a]Incidence rate ratio.
[b]Confidence interval (95%).
[c]Reference group is no abuse by parents.
*$p < .05$; **$p < .01$; ***$p < .001$ (two-tailed tests).

Table 5. Negative Binomial Regression of New W2 Health Problems on Early Life and Adult Characteristics, $N = 1{,}733$

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
W1 Health Problems	1.027*[a]	1.027**	1.024*	1.015	1.019	1.009
	(1.006 – 1.047)[b]	(1.007 – 1.048)	(1.004 – 1.044)	(.994 – 1.037)	(1.000 – 1.039)	(.988 – 1.030)
Early Life Disadvantage						
Childhood SES	1.124*	1.126*	1.130*	1.110	1.120*	1.120*
	(1.010 – 1.252)	(1.012 – 1.254)	(1.016 – 1.257)	(.996 – 1.236)	(1.006 – 1.247)	(1.005 – 1.248)
Childhood Family Composition	.979	.979	.978	.980	.991	.990
	(.893 – 1.072)	(.894 – 1.073)	(.891 – 1.072)	(.894 – 1.075)	(.902 – 1.088)	(.901 – 1.088)
Rare Abuse[c]	1.086	1.085	1.086	1.077	1.075	1.066
	(.937 – 1.259)	(.936 – 1.256)	(.938 – 1.257)	(.935 – 1.241)	(.933 – 1.238)	(.930 – 1.222)
Frequent Abuse, 1 Type[c]	1.218*	1.219*	1.199*	1.213*	1.184*	1.172
	(1.025 – 1.449)	(1.025 – 1.450)	(1.007 – 1.427)	(1.020 – 1.443)	(1.005 – 1.395)	(.994 – 1.383)
Frequent Abuse, 2 Types[c]	1.276**	1.281**	1.253*	1.267**	1.216*	1.209*
	(1.071 – 1.520)	(1.072 – 1.530)	(1.052 – 1.494)	(1.067 – 1.505)	(1.025 – 1.443)	(1.019 – 1.435)
Adolescent Health Problems		.972				.955
		(.863 – 1.095)				(.850 – 1.072)
Adult Characteristics						
Age	1.014***	1.014***	1.014***	1.013***	1.015***	1.013***
	(1.008 – 1.020)	(1.008 – 1.020)	(1.008 – 1.020)	(1.006 – 1.019)	(1.009 – 1.021)	(1.006 – 1.020)
Nonwhite	.929	.928	.910	.946	.915	.907
	(.734 – 1.176)	(.734 – 1.174)	(.709 – 1.167)	(.742 – 1.206)	(.727 – 1.150)	(.704 – 1.169)
Female	1.238**	1.241***	1.232**	1.284***	1.209**	1.281***
	(1.098 – 1.397)	(1.099 – 1.401)	(1.081 – 1.405)	(1.134 – 1.455)	(1.071 – 1.364)	(1.122 – 1.462)
Cognition, W2	.950	.949	.951	.948	.957	.951
	(.877 – 1.028)	(.877 – 1.028)	(.877 – 1.032)	(.874 – 1.028)	(.886 – 1.033)	(.876 – 1.032)
Education			.997			1.004
			(.966 – 1.030)			(.972 – 1.037)
Income			1.038			1.036
			(.995 – 1.082)			(.995 – 1.080)
Financial Strain			1.113*			1.071
			(1.013 – 1.223)			(.978 – 1.173)
Lifetime Smoking				1.004*		1.004*
				(1.000 – 1.007)		(1.000 – 1.007)

(continued)

Table 5. *(continued)*

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Heavy Drinker				1.041		1.057
				(.910 – 1.192)		(.923 – 1.209)
Obese				1.377***		1.344***
				(1.209 – 1.567)		(1.187 – 1.521)
Family Support					1.021	1.009
					(.915 – 1.140)	(.904 – 1.126)
Friend Support					.952	.963
					(.858 – 1.057)	(.869 – 1.067)
Family Strain					1.084	1.055
					(.961 – 1.222)	(.937 – 1.188)
Friend Strain					1.051	1.058
					(.934 – 1.182)	(.939 – 1.193)
Social Integration					1.021	1.026
					(.977 – 1.067)	(.983 – 1.072)
Ever Divorced					.979	.973
					(.874 – 1.097)	(.868 – 1.090)
Personal Control					.939*	.942
					(.882 – .998)	(.885 – 1.002)
Nonresponse Hazard	2.201***	2.215***	2.220**	1.894**	2.015***	1.948*
	(1.522 – 3.183)	(1.529 – 3.207)	(1.325 – 3.721)	(1.285 – 2.792)	(1.372 – 2.958)	(1.100 – 3.449)
–2LL	2826.438	2826.294	2822.644	2807.602	2818.654	2797.795

[a]Incidence rate ratio.
[b]Confidence interval (95%).
[c]Reference group is no abuse by parents.
*$p < .05$; **$p < .01$; ***$p < .001$ (two-tailed tests).

1, 2, and 3, but not after accounting for early life disadvantage and adult risks and resources (Models 4, 5, and 6). Second, each model includes an adjustment for attrition to account for nonrandom changes in the sample over the 10-year follow-up period (Heckman 1979).[13] Given that more than 200 respondents died during the follow-up period, the nonresponse hazard shows that those who dropped out would have displayed more new health problems at the follow-up. In addition, each model includes the W2 measure of cognition to adjust for recall difficulty.

As Table 5 shows, two domains of childhood disadvantage are related to new health problems at W2: low SES during childhood and frequent child abuse. Although childhood SES is not independently related to W1 health problems, it is associated with new health problems at W2, even after adjusting for a long vector of risks and resources, including adult SES, financial strain, and the nonresponse hazard. Frequent abuse of one type is significant only in Models 1 through 5, but the combination of frequent physical and emotional abuse is associated with more health problems across all models. Neither family composition during childhood nor adolescent health problems are associated with the development of new health problems at W2.

Table 5 reveals that two of the potential mediators are significant predictors of new health problems at W2 in reduced models only (financial strain and personal control), and two are significant in Model 6 (smoking and obesity), each of which raises the risk of new health problems. The only mediator found to be significant, however, is obesity. Low childhood SES raises the risk of obesity, which, in turn, leads to new health problems ($z = 2.042$, $p < .05$).

Profiles of Early Disadvantage

To illustrate these results, Figure 1 presents the predicted number of health problems at W1 and W2 by three contrasts of childhood disadvantage (after adjusting for all covariates): (1) no child abuse coupled with no indications of family or SES disadvantage; (2) two types of frequent child abuse coupled with no indications of family or SES disadvantage; and (3) two types of frequent child abuse coupled with the highest observed indications of family or SES disadvantage (90th percentile)—the archetypical risky family. The difference between categories 1 and 3 at W1 is about one health problem. In other words, persons raised in a home with frequent physical and emotional abuse, coupled with low SES and some type of family disruption, had at least one more disease at W1 than those raised in a home free of such disadvantage. For new diseases at W2, the pattern is similar but the expected difference is about .5 health problems. Respondents whose only type of early disadvantage was child abuse have a higher likelihood of health problems at both waves.

Supplementary Analyses

Extending the analyses from Tables 4 and 5, we examined potential moderation of the relationships reported above by testing for interactions between childhood disadvantage and gender, age, and race. We found no evidence that childhood disadvantage, even when tested with alternative coding algorithms, had distinct effects for men and women on health problems at W1 or W2 (i.e., models with gender interactions do not produce better fit when log likelihood tests are compared). Nor could we find any evidence of statistical interaction by age or race.

We also examined cohort differences in exposure to childhood disadvantage and whether the effect of childhood disadvantage on health problems varied by cohort. Using four birth cohorts, defined as Great Depression, War Years, Baby Boomers, and Post-Boomers, we uncovered differences in exposure to disadvantage. As expected, the Great Depression cohort was most likely to report SES disadvantage, and each successive cohort reported less SES disadvantage. By contrast, Baby Boomers and Post-Boomers were more likely than the Great Depression and War Years cohorts to report frequent acts by parents indicative of child abuse. When testing interactions of cohort-by-disadvantage

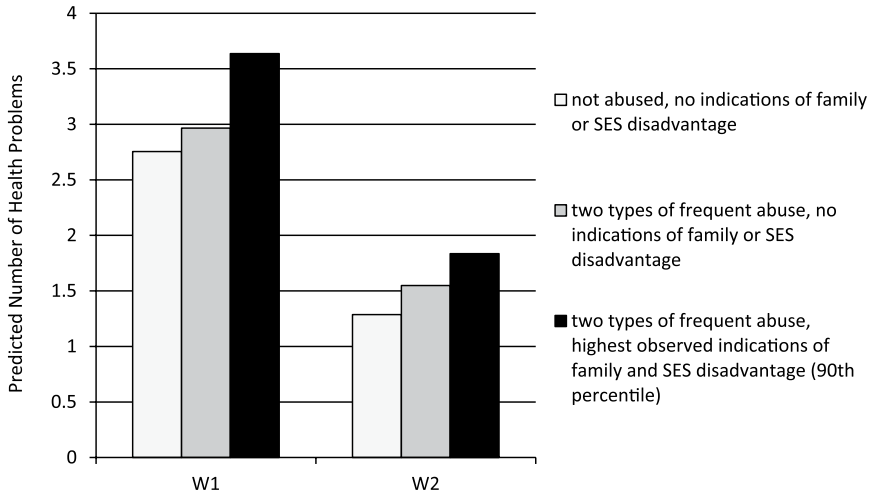

Figure 1. Predicted Number of Adult Health Problems at W1 and W2 by Categories of Childhood Disadvantage

(with the Great Depression cohort as the reference group), however, none are significant. Baby Boomers and Post-Boomers experienced fewer W1 health problems, but the relationships between childhood disadvantage and health problems are similar. We subsequently repeated the analyses for new health problems at W2, but we again found no significant interaction terms. (Replicating the analyses using 10-year cohorts did not alter the conclusions.)

Finally, although the focus of the analysis is whether childhood disadvantage has long-term effects on multiple health conditions (disease accumulation), we conducted supplementary analyses to examine each health problem and groups of conditions based on the International Classification of Diseases (ICD). Those analyses reveal that more than a dozen separate health problems and six ICD categories are influenced by childhood disadvantage. These outcomes include life-threatening conditions such as heart disease, lung disease, and cancer as well as less virulent conditions such as gum and mouth problems, lumbago, hemorrhoids, and migraine headaches.

DISCUSSION

The aims of this research are both substantive and theoretical, related to a basic question: does childhood disadvantage—encompassing low SES, family composition, and abuse—have long-term effects on health? For the substantive inquiry, we found compelling evidence that *childhood disadvantage is associated with adult health problems,* both directly and indirectly. Even after adjusting for a host of adult resources and health risks, the results reveal that childhood disadvantage has independent effects on the number of health problems afflicting persons in middle and later life. Moreover, the analysis reveals that such early disadvantages also shape the very resources that could help assuage their effects.

Substantive Conclusions

Theories and previous research emphasize that childhood disadvantage, especially traumatic experience, may be directly related to adult health and also indirectly due to how early stressors shape future lifestyles and resources. Thus, our first research question focused on whether childhood disadvantage is associated with more risks and fewer resources in adulthood. Does childhood disadvantage increase the likelihood of an adult lifestyle with more health risks? Does it also lead to problems in social functioning? The general answer to these questions is affirmative. The analysis reveals that all three

domains of childhood disadvantage are consequential to adult risks and resources. The influence of family composition is confined to four of the 14 outcomes, clearly shaping adult family functioning (less support, more strain). It also increases the likelihood of smoking and financial strain during adulthood. The influence of childhood SES is more striking: it is associated with nine of the 14 adult risks and resources.

Beyond its association with educational attainment and adult income, being raised in an SES-disadvantaged household is associated with behaviors that harm health—smoking and heavy alcohol consumption—as well as less social integration. The consequences of frequent child abuse are even more evident, affecting 11 out of the 14 outcomes, including adolescent health problems, smoking, heavy drinking, and interpersonal relationships with high levels of strain and low levels of support. Consistent with results of other studies, some early insults are sufficient to alter the development of social psychological resources and predispose people to engage in risky behaviors (see Greenfield and Marks 2009b; Lloyd and Turner 2008). But childhood SES disadvantage and abuse also take their toll on social integration and personal control. When viewed in context, childhood disadvantage sets the stage for an adult life filled with disadvantage. Scholars often refer to stress proliferation when discussing accumulated negative exposures during relatively short periods of time, but the current research provides empirical support for *stress proliferation as a life course phenomenon* (Pearlin et al. 2005; Turner, Wheaton, and Lloyd 1995). Some, but not all, childhood stressors are related to adult stressors, but accumulated childhood stressors increase adult health risks and decrease social resources to manage subsequent stressors.

Our other research questions address whether these accumulated exposures influence health. One may anticipate that childhood disadvantage will influence status attainment and social psychological resources, but does it also influence health problems decades later? For health problems at W1, the analysis reveals that respondents who grew up in nontraditional households had more health problems during adulthood, and this was not mediated by any of the adult characteristics considered. Frequent child abuse was related to W1 health problems, but this relationship was mediated by lifestyle risks and social psychological resources, namely lifetime smoking, family support, family strain, and personal control; the effect of frequent child abuse on W1 health problems was indirect. Adolescent health problems were related to more W1 morbidity, reflective of life course continuity in health—consistent with findings by others using different surveys (e.g., Blackwell et al. 2001; Springer et al. 2007)—but there is no evidence that adolescent health problems mediate the relationship between childhood disadvantage and adult health.

We also found evidence that new health problems at W2 were more likely among persons who suffered more childhood disadvantage. Distinct from W1 health problems, however, the effect of child abuse on W2 health problems was not mediated by lifestyle factors or adult resources. Frequent abuse and low SES during childhood were associated with the development of new health problems during the 10-year follow-up. Although one might be tempted to attribute these new health problems solely to adult behavior and lifestyle, such an attribution would be misdirected. Lifetime smoking and obesity are, indeed, related to new health problems, but these lifestyle factors do not fully mediate the influence of childhood SES and frequent abuse. In many respects, it is unsurprising that lifetime smoking and obesity are related to new health problems in middle and later life, because lifetime smoking reflects accumulated tobacco consumption and obesity rises in middle and older ages (Ferraro and Kelley-Moore 2003).

What is novel from these analyses, however, is that childhood SES and abuse are associated with *new* health problems, despite being distal stressors. Moreover, the link between these two forms of childhood disadvantage and new health problems remained after adjusting for more than a dozen adult

characteristics related to health. These findings add to the growing literature that distal or "upstream" risk factors are important when studying social factors and health (Hayward and Gorman 2004; Kuh and Ben-Shlomo 2004; Lloyd and Taylor 2006). The paradigm for studying adult health has long emphasized proximal risk factors, but both science and practice would benefit from a paradigm shift to more fully address the early origins of adult disease. As Felitti (2002:44) argues, "Our findings are of direct importance to the everyday practice of medicine and psychiatry because they indicate that much of what is recognized as common in adult medicine is the result of what is not recognized in childhood."

The health consequences of a life filled with disadvantage are not limited to the development of just a few diseases. Rather, accumulated disadvantage is related to disease accumulation, consistent with Hertzman and Boyce's (2010:331) claim that "adverse social conditions yield broad, pluripotential pathogenicity rather than focal, specific morbidities." Many previous studies examine the relationship between a single negative childhood exposure and a single health outcome, the present study, however, examines multiple domains of childhood disadvantage—to account for stress proliferation—and multiple health problems. Even when considering three broad domains of childhood exposures, the imprint of early disadvantage is clear.

The third research question guiding the analyses focuses on mediating effects. Once one has experienced childhood disadvantage, perhaps the most important question is whether any resources can protect against the early insults (Steptoe and Marmot 2003). One of the difficulties for life course epidemiologic work is that there are so many potential mediators—what could be called the million mediator problem. Although any study is limited to a set of potential mediators, we sought to tap domains of mediation along four primary pathways: adolescent health, adult SES, lifestyle risk factors, and social psychological resources.

Findings from the MIDUS reveal that the effects of childhood family composition on W1 health problems are not mediated by any of the 14 adult indicators of risks and resources. By contrast, the effect of child abuse on W1 health is fully mediated by smoking and the social psychological resources, especially low family support, high family strain, and personal control (Irving and Ferraro 2006). Although low childhood SES does not result directly in more health problems, it is indirectly related to W1 morbidity through its influence on lifetime smoking and personal control. This is consistent with evidence indicating that intervening effects may be detected even in the absence of a direct relationship between the predictor and outcome variables (Hayes 2009). For new health problems at W2, however, the effects of childhood SES and abuse remain despite adjustment for adult risks and resources, reflecting support for a cumulative exposure model (Berkman 2009).

When considering the findings presented here, several limitations are notable. First, caution is warranted because data about childhood conditions rely on recollections of childhood disadvantage. Potential bias is always possible when using retrospective questions, but the MIDUS questionnaire was devised to minimize bias by asking for specific information on experiences and never mentioning words such as adversity or abuse. Although the details of distal events may begin to fade over time, previous research shows that the ability to recall whether a significant event occurred appears to be relatively stable (e.g., Hardt and Rutter 2004). The greater concern is likely an underreporting of negative childhood exposures, especially abuse, which would likely yield conservative tests of significance (Alwin 2007).

Second, MIDUS unfortunately does not include a measure of parental income when respondents were children. Although very difficult to measure retrospectively, prior studies show that parental income is important to children's health, and the effect does not diminish quickly as children age (Case et al. 2002). To compensate for this limitation, we included parental education, welfare receipt, and financial strain in our measure of childhood SES disadvantage. Whereas parental

education is generally antecedent to and correlated with parental income (and more stable than income [Elo 2009]), we consider its inclusion to be useful for the analysis. Nevertheless, we welcome future studies that include parental income as part of SES disadvantage.

Third, the sampling frame for this study of non-institutionalized adults is relevant for both the independent and dependent variables considered. Although we selected the MIDUS because it is an age-heterogeneous sample, the possibility of left-censoring remains, because adults with the *most adverse* early life experiences may be excluded from the study in the first place (perhaps due to higher risk of incarceration, institutionalization, or early death). To partially address selection, we adjusted the parameter estimates to account for attrition between survey waves and tested for whether cohort differences in exposure to childhood disadvantage influenced adult health problems. Nevertheless, selection processes may be operant prior to W1.

Fourth, detailed information on the timing and actor's view of disadvantage is not available in the MIDUS. The survey is exemplary for the breadth of childhood disadvantage studied and its longitudinal design, but studies with more detailed information on the sequencing and pacing of accumulated exposures, perhaps through life history calendar methods, will greatly advance our understanding of these life course phenomena. Integrating specific information on the timing of exposures is a propitious avenue of inquiry.

Finally, although the analysis does not reveal any racial differences in how childhood disadvantage influences adult health, the size and racial composition of the MIDUS sample may constrain such tests, especially for the longitudinal analyses, because African Americans had a lower retention rate (Radler and Ryff 2010).

Theoretical Implications

The analyses and substantive findings also lay the foundation for theoretical and methodological contributions for research on the life course. To begin, many models and theories rely on the concept of accumulation, but usage of the term varies widely. Although cumulative inequality theory prioritizes the accumulation of risk and resources, the theory needs greater specification of the *content* and *process* of accumulation. Pertaining to the content of accumulation, we offer two suggestions.

First, it may be useful to distinguish the accumulation of events or experiences that are irreversible, such as death of a family member, attaining a degree, or publication of a book, from events or experiences that are reversible, such as debt, wealth, fame, or social support. This distinction may aid understanding of responses to accumulation. Second, content hinges on an actor's view of the situation, which is also linked to responses such as compensation, resignation, or discounting. Except for a few exemplars (e.g., Surtees and Wainwright 2007), however, most studies offer little on how actors view the events, statuses, and experiences often presumed to be a disadvantage.

The process of accumulation has received considerable attention, with life course analyses privileging the timing of exposures and events along historical and biographical axes (Elder 1998; Ferraro and Morton forthcoming). Identifying the onset and duration of exposures has become commonplace in studies of health (Kuh and Ben-Shlomo 2004), leading to discussion of triggering events and sequential processes. The rate of accumulation after onset has received much less attention. Hill (1949) advanced the concept of stress *pile up* to describe the accumulation of related stressors (e.g., divorce often triggers financial and residential changes), but many authors appear to use the term as a synonym for accumulation (Boss 2002). Still others use *pile up* to refer to short-term accumulation—over multiple days (Diehl, Hay, and Chui 2012)—which may be a useful distinction. Although we know little about the rate of accumulation (or loading) in most fields of life course inquiry, studying bursts of accumulated exposures, both positive and negative, may be pivotal for identifying triggering events, resilience, and sensitive periods of the life course (Berkman 2009; Elder 1998). Perhaps

medical or crime records with precise dates could be linked to survey data to illuminate such processes.

Finally, more theoretical and empirical attention is needed to identify how phenomena can interrupt or "shut down" the noxious consequences of accumulation processes (DiPrete and Eirich 2006). Some of these consequences may appear immutable, such as the link between child abuse and new health problems observed herein, but this may simply mean that the proper mediator has not yet been uncovered, that it operates only with another mediator, or that the measurement of the presumed mediator is too coarse. For other relationships, where mediators have been identified—for instance, smoking as a mediator between childhood disadvantage and adult health—both the theoretical and policy implications are noteworthy.

Conclusion

These results clearly show that domains of childhood disadvantage are associated with adult disadvantage, ranging from fewer social psychological resources to more health problems. Early disadvantage was related to health problems at the baseline survey, but this study also reveals that childhood SES and abuse are associated with the onset of *new* health problems, even after adjusting for a wide array of potential mediators. The fact that new health problems are more likely to develop among adults who experience childhood disadvantage sheds new light on the imprint of distal or "upstream" risk factors when studying social factors and health.

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Notes

1. Although scholars debate whether the life course is a perspective or a theory, it is quite clear that scientists from a variety of fields use key concepts for life course analysis (Alwin 2012). For the present research, we view it largely as a perspective that can be effectively integrated with any number of theories.
2. Failure to consider SES during both childhood and adulthood may lead to overestimating the importance of the one studied on adult health.
3. Many early studies examining multiple forms of childhood disadvantage probe the possibility of a dose-response effect by adding childhood exposures (Felitti et al. 1998; Turner and Lloyd 1995). These studies reveal the import of multiple negative exposures, but more recent studies have used latent-class models to identify clusters of disadvantage (O'Rand and Hamil-Luker 2005) or compared clusters to additive formulations (Morton, Mustillo, and Ferraro 2014).
4. Beyond social factors that shape physical and mental health, related areas of research on the early origins of adult health include (1) discoveries that low birth weight is a predictor of multiple adult diseases—including heart disease, diabetes, and cancer (Barker 1997; Eriksson et al. 2010) and (2) breakthroughs linking poor childhood health with a higher prevalence of multiple chronic diseases during adulthood, including cardiovascular disease, cancer, and lung disease (Blackwell et al. 2001).
5. The 31 health problems queried were AIDS/HIV; alcohol or drug problems; anxiety, depression, or some other emotional problem; arthritis, rheumatism, or other bone or joint diseases; asthma, bronchitis, or emphysema; cancer; constipation; diabetes; foot trouble, persistent (e.g., bunions, ingrown toenails); gallbladder trouble; gum or mouth problems, persistent; hay fever; heart trouble (e.g., attack, failure); hernia or rupture; high blood pressure or hypertension; lung problem (other); lupus or other autoimmune disease; multiple sclerosis, epilepsy, or other neurological problem; migraine headaches; piles or hemorrhoids; sciatica, lumbago, or recurring backache; skin trouble, persistent (e.g., eczema); sleeping problems, chronic; stomach trouble (recurring), indigestion, or diarrhea; stroke; teeth problems, persistent; thyroid disease; tuberculosis; ulcer; urinary or bladder troubles; and varicose veins (requiring treatment).
6. A limitation of the MIDUS for this analysis is the inconsistency in question wording to measure health problems. For heart trouble and cancer, respondents were asked if they *ever* had the condition. By contrast,

the remaining 29 conditions were queried for the *past 12 months*. The likely consequence of the different measurement time frame is an underestimation of morbidity for the 29 conditions. Although most of the 29 are chronic conditions (e.g., arthritis, asthma, diabetes, and recurring stomach trouble), some conditions are remediable through surgery or other treatment (e.g., gum or tooth problems, hernia, and varicose veins). Thus, measurement of this latter set of health problems is more likely to be underestimated in the analysis.

7. We examined additional specifications of disadvantage (separate indicators, sum of disadvantage, and latent classes), but these results did not alter the main conclusions.
8. Specifically, indicators for no male in household, parents divorced, parent(s) died, and welfare receipt were measured as binary variables; self-rated physical and mental health at age 16 were measured on a 1 to 5 scale, from "excellent" to "poor"; financial strain was measured on a 1 to 7 scale, from "a lot better off" to "a lot worse off"; and education of household head was measured on a 1 to 12 scale, from professional degree to "no school/some grade school." Items about child abuse were measured on a 1 to 4 scale and used to create abuse profiles capturing frequency and type of abuse. Where appropriate, variables were recoded so that higher scores uniformly indicate greater disadvantage.
9. MIDUS also included indicators for abuse by siblings or "anybody else," but we excluded these from the analyses after preliminary examination. Similar to other studies, we focus on parental abuse.
10. Given that older people had a longer time for the retrospective window, we also examined age differences in missingness. The analyses reveal that older adults were significantly more likely to have missing data on some measures of early disadvantage (education of household head, receipt of welfare, and abuse) but not others (childhood family composition, financial strain, and adolescent health problems). The fact that older adults were more likely to have incomplete data on select retrospective measures—but not on other measures tapping the same period of life—suggests that respondents were engaged with the survey and declined response rather than fabricating an uncertain response (Alwin 2007).
11. Lifetime smoking was calculated from information reported by respondents: age when started smoking, year stopped (for former smokers), and average number of cigarettes smoked daily. Using a yearly metric, lifetime smoking is the product of years smoked and annual number of cigarettes, divided by 10,000. The measurement of heavy drinking was sex differentiated and tapped respondents' period of greatest lifetime consumption: five or more drinks per day for men and four or more drinks for women (Wechsler et al. 2000).
12. Although missing data were not frequent for the variables used in these analyses, we nonetheless examined whether missingness was associated with other study variables and compared the results using alternative procedures for handling missing data. Those analyses revealed that respondents with missing data on the variables of interest were more likely to report several types of childhood disadvantage (i.e., received welfare, financial strain, and no male in household). To avoid excluding people who experienced multiple forms of childhood disadvantage, we used multiple imputation. Estimates derived from multiple imputation and listwise deletion of missing cases were very similar for W1 health problems, but the effect sizes for health problems at W2 were reduced with listwise deletion.
13. A probit model estimated the likelihood of W2 participation using a series of predictors from W1 (age, race, sex, education, self-rated health, report of discrimination, missing income, missing household assets, count of years lived in neighborhood, and an index of civic obligation). We then calculated a nonresponse hazard instrument, based on the inverse Mills ratio of the function derived from the probit model.

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