Social Science & Medicine 104 (2014) 133–141

Contents lists available at ScienceDirect

Social Science & Medicine

journal homepage: www.elsevier.com/locate/socscimed

Does childhood misfortune raise the risk of acute myocardial infarction in adulthood?

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ARTICLE INFO

Article history:
Available online 3 December 2013

Keywords:
Heart attack
Cumulative inequality theory
Childhood adversity
Smoking
USA

ABSTRACT

Whereas most research on acute myocardial infarction (AMI) has focused on more proximal influences, such as adult health behaviors, the present study examines the early origins of AMI. Longitudinal data were drawn from the National Survey of Midlife Development in the United States ($N = 3032$), a nationally representative survey of men and women aged 25–74, which spans from 1995 to 2005. A series of event history analyses modeling age of first AMI investigated the direct effects of accumulated and separate domains of childhood misfortune as well as the mediating effects of adult health lifestyle and psychosocial factors. Findings reveal that accumulated childhood misfortune and child maltreatment increased AMI risk, net of several adult covariates, including family history of AMI. Smoking fully mediated the effects of both accumulated childhood misfortune and child maltreatment. These findings reveal the importance of the early origins of AMI and health behaviors as mediating factors.

Introduction

Since the 1950s, cardiovascular disease (CVD) has been the leading cause of death in the United States, accounting for approximately one third of all deaths (Crimmins & Beltran-Sanchez, 2010). Moreover, heart disease is also a major reason why the United States life expectancy has not kept pace with the rising life expectancy in other developed countries – a trend observed since the 1980s (Crimmins, Preston, & Cohen, 2010). Although the prevalence of heart disease among adults aged 75 or older in the United States is similar to most modern societies, heart disease prevalence among Americans under the age of 65 is relatively high compared to other developed countries (Crimmins et al., 2010). Thus, heart disease, while considered a health concern for older adults, has increasingly become a concern in the US among middle-aged adults.

The National Heart, Lung, and Blood Institute (NHLBI) identified several factors that increase the risk of CVD in adulthood, notably smoking, obesity, and high blood cholesterol (NHLBI, 2011). In addition to these proximal influences of CVD, there is substantial evidence from life course research showing that the antecedents of CVD may occur much earlier in life. Indeed, risk factors for adult CVD can occur in the initial stages of development. In utero exposure to famine, poor fetal nutrition, and low birth weight are associated with increased risk of adult CVD (Barker, 1997; McEniry & Palloni, 2010). Subsequent to these pre- and peri-natal influences on adult CVD, studies have also found effects of childhood misfortune. Low socioeconomic status (SES) (Beebe-Dimmer et al., 2004), poor childhood health (Blackwell, Hayward, & Crimmins, 2001), and a harsh family environment (Loucks, Almeida, Taylor, & Matthews, 2011) are associated with increased risk of CVD in adulthood. More recently, scholars have examined types of heart disease individually rather than combining several maladies into one general outcome of CVD. Since CVD encompasses a broad spectrum of diseases that range in severity and etiology, focusing on one type of heart disease can pinpoint which causes and mechanisms are specific to each condition. The most common type of heart disease, coronary heart disease, often appears first as AMI.

Although few studies examine the relationship between childhood misfortune and adult AMI, research by O'Rand and Hamil-Luker revealed that several clusters of childhood misfortune increase AMI risk, independent of adult risk factors. The study is noteworthy because the authors demonstrated that poor health, family instability, and economic disadvantage during childhood place adults at risk for AMI (O'Rand & Hamil-Luker, 2005). To build upon the contributions of those studies, the present research accounts for an additional factor that is strongly associated with AMI—family history of AMI—while examining lifestyle and psychosocial factors as potential mediators. This study draws from cumulative inequality (CI) theory and the National Survey of Midlife

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http://dx.doi.org/10.1016/j.socscimed.2013.11.026

Development in the United States, an age-heterogeneous, longitudinal sample of men and women. We begin by articulating essential elements of CI theory to guide the analysis.

Theoretical background

CI theory is a middle-range theory focused on how stratification processes unfold over the life course. Four elements of CI theory are central to this study. First, CI theory privileges childhood as a critical period for life course development and stratification, stressing how childhood experiences can shape later life outcomes (Ferraro & Shippee, 2009). As such, the family plays an essential role in these processes (for good or for bad). The links between childhood and adulthood entail not only social and psychological processes but also biological embedding because environmental conditions shape early life biological processes (Hertzman & Boyce, 2010). In this sense, experiences such as chronic poverty or child maltreatment may trigger biological processes that might raise the risk of disease in later life independent of the social psychological process at work (e.g., Dong et al., 2004; Felitti et al., 1998).

Second, CI theory also emphasizes the *timing* of life events. By giving attention to *when* health events occur, CI theory enables researchers to better gauge the development of health disparities. Might early misfortune raise not only the ultimate risk of disease and mortality, but when the risk is manifest? Although a study by McEniry and Palloni (2010) concluded that *in utero* stressors did not influence the age of heart disease onset, we focus on AMI as a serious health event to explore the timing of early insults.

Third, unlike related theories of accumulated disadvantage, CI theory places a premium on intergenerational processes. Recognizing both genetic and environmental influences, the theory specifies that family lineage is essential to understanding social and health outcomes in childhood and adulthood (Ferraro & Shippee, 2009), parallel in some respects to the life course component of linked lives (Elder, 1998). According to CI theory, however, these intergenerational links for most families include both social and genetic components. Failure to account for intergenerational influences on health may result in overestimating the effects of childhood misfortune on health risks.

Fourth, CI theory gives explicit attention to selection processes. For aging and health research, it is essential to consider the possibility of how selection process may complicate the interpretation of results purportedly addressing inequality. Censoring in research on aging or health may occur due to nonrandom selection in populations or samples. Accumulating disadvantage over the life course can lead to premature mortality and compositional change in a population – and this effect is pronounced among older cohorts (Ferraro & Shippee, 2009). Although many gerontological studies are based on samples of older respondents only, doing so may pose special problems in interpretation: "If the aim is to show how inequality *accumulates*, it should be clear that studying limited age ranges will result in describing changes after considerable population truncation has occurred" (Ferraro & Shippee, 2009, p. 336). Herein, limiting the sample to older people may censor cases that experienced an AMI earlier in life and either died or were so incapacitated that completing a survey was impossible. Of course, all studies face design issues that compromise their validity, but the key is to be alert to selection processes and cautious in interpreting the results based on the data at hand.

Childhood origins of adult health

As stated in CI theory, childhood is a particularly vulnerable period of the life course since it comprises a significant portion of cognitive, neural, and biological maturation (Maggi, Irwin, Siddiqi, & Hertzman, 2010). Research has demonstrated how childhood insults, such as parental loss, can alter biological functioning and neurological response systems (Luecken, 1998). Many physical ailments in later life still bear the imprint of childhood misfortune, illustrating its far-reaching grasp. Childhood misfortune can later manifest as adult obesity (Greenfield & Marks, 2009), cancer (Morton, Schafer, & Ferraro, 2012), and lung disease (Blackwell et al., 2001). Among the diseases studied in relation to childhood misfortune, the most pervasive in the United States remains CVD.

Investigating how childhood misfortune raises the risk of CVD has generally taken two approaches: analyzing unique or cumulative effects of misfortune. Among the commonly examined types of misfortune, such as SES, poor childhood health, and a harsh family environment, many studies treat these as individual CVD risk factors (e.g., Beebe-Dimmer et al., 2004; Blackwell et al., 2001; Luecken, 1998). Although many of these childhood insults have independent effects, the *accumulation* of childhood misfortune also presents a lasting threat to cardiovascular conditions. Additive measures of childhood misfortune that capture child maltreatment, family structure, and the psychosocial environment have displayed a dose–response effect: as the number of adversities a child experiences increases, so does the risk of heart disease and CVD risk factors (Dong et al., 2004; Felitti et al., 1998; Loucks et al., 2011).

Although many studies link childhood misfortune and CVD, we identified only a few that focused on AMI. O'Rand and Hamil-Luker (2005) have found that adults who experienced poor health, family instability, and low SES during childhood were at increased risk for AMI, and they extended their findings to show that childhood misfortune was more consequential for women than for men (Hamil-Luker & O'Rand, 2007). Whereas O'Rand and Hamil-Luker's research identified how different clusters of misfortune raise AMI risk, Hallqvist, Lynch, Bartley, Lang, and Blane (2004) research focused on SES; they found that the life course trajectory of low SES, rooted in childhood, raises AMI risk in adulthood. These three studies paint a compelling picture of the influence of childhood misfortune on the risk of heart attack, but we draw attention to two notable risk factors that have not yet been incorporated in research on the topic.

First, a risk factor that merits attention is family history of heart disease. Previous research shows that having just one first-degree family member with ischemic heart disease *doubles* the risk of AMI, and the risk elevates further with two or more relatives with heart disease (Bertuzzi, Negri, Tavani, & La Vecchia, 2003). We are unaware, however, of any studies on childhood misfortune and AMI that account for family history of AMI or any type of CVD, potentially leading to an overestimation of the effect of childhood misfortune on AMI. In addition to this confounding relationship, childhood misfortune could also moderate or mediate the relationship between family history of AMI and AMI risk. Regardless of the relationship, we build upon prior literature by incorporating family history of AMI into our analyses.

Second, a growing body of literature has revealed that child maltreatment raises the risk of multiple health problems in adulthood, from ulcers (Springer, 2009) to cancer (Morton et al., 2012). Childhood experiences such as household financial strain or living in a fatherless home may have lasting effects on health, but many studies also point to the long-term effects due to traumatic experiences. Child maltreatment is often a traumatic experience that activates a host of physiological, psychological, and social responses—from secretion of glucocorticoids to social withdrawal. Despite substantial literature suggesting that child maltreatment influences cardiac health, we found only one published study examining the link between child maltreatment and AMI (Fuller-Thomson, Bejan, Hunter, Grundland, & Brennenstuhl, 2012). The authors examined one type of maltreatment—sexual abuse—and reported that it raised the risk of AMI for men but not for women.

Most studies of sexual abuse and CVD, however, report that women are at greater risk (e.g., Goodwin & Stein, 2004). The current study does not include measurement of sexual abuse per se, but we are able to capitalize on an extensive battery of questions tapping both physical and emotional abuse. As such, we examine whether AMI risk is related to an overall measure of accumulated misfortune (including maltreatment) or maltreatment as a special type of early misfortune.

Mechanisms of childhood misfortune

Many life course scholars contend that childhood misfortune triggers a *chain of risks*, where one event increases the likelihood of other negative events. Experiencing childhood misfortune may propel an individual toward risky lifestyles and behaviors that lead to poor health. Childhood misfortune has been shown to increase the risk of smoking, alcoholism, drug abuse, and obesity, each of which may mediate the relationship between childhood misfortune and adult health (Brown et al., 2010; Felitti et al., 1998; Greenfield & Marks, 2009).

Felitti et al. (1998) developed a conceptual model that includes both health behaviors and psychosocial factors as potential mediators of the childhood–adult health relationship. Applying this model to childhood misfortune and heart disease, Dong et al. (2004) found that both health behaviors and psychological risk factors mediated the relationship, with the latter, psychological risk factors, producing a stronger mediation effect. Therefore, the mechanisms linking childhood experiences to adult AMI may extend beyond the adult health behaviors, social support, and SES trajectories already identified by Hallqvist et al. (2004) and O'Rand and Hamil-Luker (2005). Prior research reveals that anxiety, locus of control, and family strain are potential mechanisms. Previous studies have reported associations between childhood misfortune and risk of anxiety, low locus of control, and familial relationship strain (Irving & Ferraro, 2006; Kessler, Davis, & Kendler, 1997; Loucks et al., 2011). Anxiety, low locus of control, and family strain have also been associated with increased AMI risk (Kubzansky, Cole, Kawachi, Vokonas, & Sparrow, 2006; Rosengren et al., 2004). Bridging together this research, we investigate psychosocial factors of anxiety, locus of control, and family strain as potential mediators that may link childhood misfortune to adult AMI risk.[1]

Selection issues

Selection processes are also important for the study of AMI. Although AMI is often considered a disease of old age, many AMI studies also identify cases of early onset – as early as 25 years old (e.g., Fang, Alderman, Keenan, & Ayala, 2010). Moreover, a Swedish study of AMI survivors found that approximately one-fourth of adults who had an AMI between the ages of 25 and 55 died within 5 years (Isaksson et al., 2011). In the US, women under the age of 55 have the highest AMI mortality rates (Vaccarino et al., 2009). This type of mortality leads to a selection problem when studying the effects of childhood misfortune on AMI risk in adulthood (people die from an AMI or other causes before they are eligible to be surveyed). This is an important consideration because previous studies of childhood misfortune and adult AMI rely on samples of adults aged 51 and older (Hallqvist et al., 2004; Hamil-Luker & O'Rand, 2007; O'Rand & Hamil-Luker, 2005). By restricting the sample to persons 51 years and older, existing AMI studies may be underestimating the effects of childhood misfortune, especially if those who were faced with early disadvantage and heart trouble did not survive until late middle age. As Vaccarino et al. note (1998), limiting samples to older populations can lead to underestimating the prevalence of AMI at younger ages as well as the potential effect of childhood misfortune on AMI risk. To account for those earlier heart attacks, we use an age-heterogeneous sample of adults.

To extend the literature, we propose three hypotheses:

H1: Childhood misfortune is associated with greater risk of AMI.
H2: Health lifestyle factors will mediate the relationship between childhood misfortune and AMI.
H3: Psychosocial factors will mediate the relationship between childhood misfortune and AMI.

Methods

Data

To test these hypotheses, this study used two waves of the National Survey of Midlife Development in the United States (MIDUS). The first wave (W1) of MIDUS participants were selected from working telephone banks in the contiguous United States using a national random digit-dialing sample. During 1995, MIDUS surveyed 3032 English-speaking, non-institutionalized men and women aged 25–74 with an over-sample of adults aged 65–74 and men. After completing the initial computer assisted telephone interview, respondents were mailed self-administered questionnaires. The response rate for completing both the telephone interview and mail questionnaires was 61%, an expected response rate based on response rate trends of telephone sampling (Curtin, Presser, & Singer, 2005). In addition, a response rate of at least 60% is considered good and is comparable to other surveys with similar methods (Babbie, 2008).

Approximately ten years later, between 2004 and 2006, W1 participants were re-interviewed with similar telephone interview and self-administered mail questionnaires (W2). Of the 3032 W1 respondents, 2101 participated in the W2 telephone interview, yielding a response rate of 69.5% (71% mortality-adjusted response rate). This study drew from both the telephone and mail surveys from W1, and from the W2 telephone interviews.

Acute myocardial infarction

MIDUS included a battery of questions probing "heart trouble suspected or confirmed by a doctor." AMI was measured in the telephone interviews by asking respondents at each wave if they had ever been diagnosed as having a heart attack. Two variables were created for the event history analysis. The censoring variable was coded one for AMI observed. The duration variable was created from a question asking respondents who reported having an AMI in what year they experienced their first heart attack. Subtracting respondent's birth year from year of AMI yields age at first AMI. For those who did not experience an AMI, the duration variable was age at the latest survey.

Childhood misfortune

Item measures for childhood misfortune were drawn from both the MIDUS telephone interview and mailed questionnaire at W1 per previous research and theory (see Felitti et al., 1998; Ferraro & Shippee, 2009; Hertzman & Boyce, 2010; Morton et al., 2012; O'Rand & Hamil-Luker, 2005; Turner, Wheaton, & Lloyd, 1995).

[1] We investigated anxiety instead of depression because Kubzansky et al. (2006) found that anxiety, but not depression, predicted AMI. Moreover, Davidson, Rieckmann, and Rapp (2005) contend that the effects of depression on CVD are more reliable when using clinical depression measures rather than self-reported depressive symptoms. The MIDUS only measures self-reported depressive symptoms.

Sixteen items of childhood misfortune were used as main predictors of AMI risk: (1) family receipt of welfare or Aid to Dependent Children assistance; (2) self-report of being financially worse off than other families; (3) less than a high school education for father (or mother if father was absent); (4) female household head; (5) parental divorce; (6) death of a parent; (7–14) physical and/or emotional abuse by father, mother, sibling or other; and (15–16) self-report of poor mental or physical health at age sixteen. The maltreatment variables were created using Straus's (1979) Conflict Tactics Scale and are comprised of fifteen different physical and emotional abuse measures. Respondents were asked how frequently their mother/father/siblings/or anybody else insulted or swore at them; sulked or refused to talk to them; did or said something spiteful; threatened to hit them; smashed or kicked something in anger; pushed grabbed, or shoved them; slapped them; threw something at them; kicked, bit, or hit them with a fist; hit or tried to hit them with something; beat them; choked them; burned or scaled them.

Different specifications of childhood misfortune were created to systematically investigate how childhood misfortune affects adult AMI. All sixteen variables were initially coded as dummy variables. Since physical and emotional abuse response categories ranged in frequency from never to often, respondents who reported experiencing abuse "sometimes" or "often" were coded as "1" and those who reported "never" or "rare" were coded as "0". Previous research has demonstrated that the Conflict Tactics Scale measures have high validity, but low internal consistency reliability due to the rare occurrence of certain events, underreporting due to social desirability, and the lack of association among some indicators (Straus, Hamby, Finkelhor, Moore, & Runyan, 1998). However, dichotomizing the scales increases internal consistency (Straus et al., 1998).

Dummy variables for poor mental and physical health at age 16 were created by coding those who reported "poor" or "fair" health as "1" and those who reported "good," "very good," or "excellent" as "0". Previous research suggests that retrospective measures of childhood health may be more reliable if health is categorized into more general terms (e.g., good v. bad) than more specific terms (Haas, 2007).

Drawing from Felitti et al. (1998) and Morton et al. (2012), the first set of models tested the cumulative effect of misfortune using additive childhood misfortune (ACM). ACM was derived from a count of the number of misfortunes, ranging from 0 to 14.

In the second set of models, four domains of childhood misfortune were created to test the unique effect of each misfortune. The first domain of childhood SES was created using measures of welfare, financially worse, and low parental education. Household structure, the second domain, was comprised of female household head, parental divorce, and parental death measures. The third domain—child maltreatment—consisted of physical and/or emotional abuse by respondent's mother, father, sibling or other. The fourth domain—poor health—was based on reports of poor mental or physical health at age sixteen. For consistency, a dummy variable was created for each domain; respondents who reported experiencing any of the domain's item measures were coded as one for that domain. Prior research guided our creation of each domain of childhood misfortune (Draper et al., 2008; Haas, 2007; Hamil-Luker & O'Rand, 2007; Kessler et al., 1997). A confirmatory factor analysis confirmed these four domains.[2]

In the third set of models, child maltreatment measures were transformed into profiles to test the effect of the frequency of emotional and physical abuse by either parent. Drawing from Greenfield and Marks (2009), child maltreatment response categories were recoded into four different profiles: (1) never experienced physical or emotional abuse, (2) experienced one or both types of abuse rarely, (3) experienced one type of abuse frequently, and (4) experienced both types of abuse frequently. Dummy variables of the remaining domains of childhood misfortune were included.

Family history of acute myocardial infarction

Family history of AMI was drawn from W1. Regardless of AMI status, all respondents were asked how many people in their immediate biological family ever had a heart attack. Immediate biological family (first degree) was defined as biological parents, brothers, and sisters. Although the original variable ranged from 0 to 20, few respondents reported more than 4 biological family members experiencing an AMI, so the variable was top coded at 4.

Health lifestyle factors

To examine the mediating effect of unhealthy lifestyles, smoking and obesity variables were created using W1 measures. We created a variable that measured smoking in pack-years to capture the cumulative effect of smoking. To calculate pack-years, we multiplied total years smoked by daily average number of cigarettes smoked during that time. Then, we divided the product by 20. Participants who were never smokers were coded as "0". Total pack-years smoked ranges from 0 to 203.7. Obesity was coded as a dummy variable, with a BMI $\geq$ 30 coded as one.

Psychosocial factors

Anxiety, locus of control, and family strain variables measured at W1 were used as psychosocial mediators. The scale for anxiety follows criteria set in the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders (DSM-III-R, 1987), and is based upon the World Health Organization (WHO) Composite International Diagnostic Interview (CIDI), for which classification accuracy of anxiety is over 99% (Kessler, Andrews, Mroczek, Ustun, & Wittchen, 1998; WHO, 1990). Assessments of the WHO CIDI scales have also reported high validity and reliability (Wittchen, 1994). Anxiety used 10 items to measure how often worrying affected respondents (e.g., how often worries affected sleep, memory, energy, etc). Answers ranged from never to most days. Anxiety was constructed by summing the number of "most days" responses for each of the 10 items. Anxiety ranges from 0 to 10, with higher levels indicating higher anxiety.

Locus of control was a scale constructed from the mean of twelve items that tap self-reports of personal mastery and perceived constraints (alpha = 0.85). Measures of personal mastery and perceived constraints were selected based on Pearlin and Schooler's (1978) Mastery Scale and prior research (Lachman & Weaver, 1998). By combining both personal mastery and perceived constraints, we operationalized locus of control per Skinner's (1996) conceptualization, which includes components of both competence (i.e., mastery) and contingency (i.e., constraints). Questions for both personal mastery and perceived constraints were drawn from the self-administered questionnaire. For personal mastery, respondents indicated to what degree they agreed or disagreed with four statements, such as "I can do just about anything I really set my mind to." Similarly, for perceived constraints, respondents indicated to what degree they agreed or disagreed with eight statements, such as "I often feel helpless in dealing with

[2] Results show that the indicator variables specified in each domain load on the latent domain variable in the expected direction ($p < 0.001$). *R*-squared values for each indicator are above .45 with the exception of financially worse off ($R^2 = 0.41$), parental divorce ($R^2 = 0.23$), and parental education ($R^2 = 0.016$). Overall model fit was slightly lower than what is generally considered good (RMSEA = 0.09 and CFI = 0.84). Output available upon request.

the problems of life." Items for personal mastery were first reverse-coded so that higher scores represent higher levels of control, reflecting the coding scale for perceived constraints. Locus of control ranges from 1 to 7.

Family strain was constructed by calculating the mean of four items; it ranges from 1 to 4, with higher values indicating higher levels of strain (alpha = 0.80). Measures of family strain were guided by the work of Schuster, Kessler, and Aseltine (1990) and prior use of the scale (e.g., Walen & Lachman, 2000). Respondents were asked how often family members made demands on and criticized them, let them down, and got on their nerves.

Covariates

Covariates for all models included cohort, race, sex, education, occupation, marital status, diabetes, and hypertension, all of which were taken from W1. Birth year was used to divide respondents into 4 birth cohorts: (1) 1920–1929; (2) 1930–1939; (3) 1940–1949; (4) 1950–1974. Our initial aim was to use 10-year period cohorts, but preliminary analyses lacked sufficient statistical power due to the few number of AMI occurrences among those born during the 1950s, 1960s, and 1970s. Less than 1% of those born between 1950 and 1974 experienced an AMI by W2. Cohorts were included as a series of dummy variables, with the earliest cohort—those born during the 1920s—as the reference group. Cohorts were used instead of age because preliminary findings revealed an unanticipated age pattern: age was inversely associated with AMI onset. The retrospective nature of the timing of AMI onset (i.e., a respondent may have had an AMI prior to joining the MIDUS in 1995) means that respondents who reported having an AMI at W1 actually consisted of those who had an AMI and survived. To account for these selection processes, we separated respondents into cohorts. Dummy variables were also created for race (1 = Black) and sex (1 = female). Education is a continuous variable measured in years, ranging from 4 to 20.

Occupation, marital status, diabetes, and hypertension were included to adjust for adult risk factors and stressors. Following Carr and Friedman (2005), occupation was coded into three categories: upper white-collar (professional, executive, and managerial occupations), lower white-collar (sales and clerical occupations), and blue-collar (crafts, operatives, labor, and farm occupations). If respondent's current occupation was missing, then previous occupation was used. Dummy variables were created for those who were divorced (1 = divorced) or widowed (1 = widowed). Diabetes and hypertension were coded as dummy variables, with one indicative of the condition. Additional variables were examined in preliminary analyses, but were excluded from the final models because they were not significantly related to the outcome in any specification; these included early onset of obesity and income. The range, mean, and standard deviation for each variable used in the final analyses are presented in Table 1.

Analytic plan

Preliminary analyses were performed using logistic regression in Stata (i.e., separate equations for experienced AMI at W1 and at W2) to examine alternative specifications of childhood misfortune, perform sensitivity tests for alternative coding of independent variables, and obtain initial estimates. Whereas accumulated childhood misfortune and two domains of childhood misfortune—maltreatment and low SES—predicted AMI, profiles of maltreatment were not significant predictors and, therefore, were not included in subsequent analyses. We also estimated sex-stratified analyses, but did not observe any sex differences. These non-significant findings may be attributed to limited statistical power given the small proportion of respondents who experienced an AMI (~5%). Therefore, the models presented are not stratified by sex. Supplementary analyses investigated AMI family history as a potential moderator and depression as a potential mediator. These yielded non-significant findings, and, therefore, are not included in the final analyses.

Guided by these preliminary analyses, the final analyses were completed with event history analysis in Mplus to account for the time dependence of first AMI and to perform tests of mediations. Using a maximum-likelihood robust estimator and Monte Carlo integration allowed us to model the age of first AMI and examine pathways between childhood misfortune and AMI onset—via health lifestyle and psychosocial factors—while assessing both direct and indirect effects. Paths were created for each mediating variable to establish a direct relationship between childhood misfortune and the mediator (a), and the mediator and AMI risk (b); indirect effects were calculated by taking the product of the two paths (a*b). Standard errors were computed using the delta method. Although presented results include all mediators in one model, we also examined each mediator separately, which yielded the same results. In all models, full information maximum likelihood estimation was used for missing cases.

Table 1
Descriptive statistics from the midlife development in the U.S. study at baseline (1995).

Variables	Range	Mean	SD
AMI occurrence[a]	0–1	0.051	
AMI age[a,b]	20–76	54.659	11.068
Covariates			
Cohort 1 (1920–1929)	0–1	0.110	
Cohort 2 (1930–1939)	0–1	0.195	
Cohort 3 (1940–1949)	0–1	0.240	
Cohort 4 (1950–1974)	0–1	0.455	
Female	0–1	0.515	
Black	0–1	0.068	
Education	4–20	13.780	2.618
Upper white-collar	0–1	0.347	
Lower white-collar	0–1	0.414	
Blue-collar	0–1	0.239	
Divorced	0–1	0.185	
Widowed	0–1	0.059	
Diabetes	0–1	0.055	
Hypertension	0–1	0.184	
AMI family history	0–4	0.507	0.79
Additive childhood misfortune (ACM)	0–14	3.144	2.542
Childhood misfortune categories			
Child maltreatment	0–1	0.656	
Childhood SES	0–1	0.526	
Household structure	0–1	0.205	
Poor health at age 16	0–1	0.098	
Health lifestyle factors			
Total pack-years smoked	0–203.7	16.273	26.125
Obese	0–1	0.246	
Psychosocial factors			
Anxiety	0–10	0.183	1.022
Locus of control	1–7	5.454	1.043
Family strain	1–4	2.126	0.616
N		3023	

[a] Drawn from W1 (1995) and W2 (2005).
[b] Age calculated for only those respondents who reported having an AMI by W2 ($N = 138$).

Results

Table 2 displays the results of the Cox proportional hazard models estimated in Mplus. Models 1 and 2 examined the effect of

Table 2
Cox proportional hazards model for acute myocardial infarction, midlife development in the United States (1995).

Independent variables	Model 1	Model 2	Model 3	Model 4
Covariates				
Cohort 2[a] (1930–1939)	1.284[b]	1.319	1.338	1.354
	(0.715, 2.309)[c]	(0.710, 2.450)	(0.738, 2.425)	(0.725, 2.524)
Cohort 3[a] (1940–1949)	2.499**	2.586**	2.527**	2.604**
	(1.278, 4.894)	(1.262, 5.291)	(1.289, 4.958)	(1.265, 5.360)
Cohort 4[a] (1950–1974)	1.791	2.092	1.846	2.145
	(0.640,5.013)	(0.748, 5.853)	(0.668, 5.099)	(0.767, 5.989)
Female	0.445**	0.496*	0.447**	0.495*
	(0.272, 0.729)	(0.285, 0.863)	(0.274, 0.729)	(0.288, 0.850)
Black	0.926	1.148	0.922	1.125
	(0.408, 2.104)	(0.498, 2.649)	(0.398, 2.134)	(0.474, 2.670)
Education	0.931	0.954	0.929	0.951
	(0.829, 1.046)	(0.844, 1.080)	(0.820, 1.052)	(0.837, 1.082)
Upper white-collar[d]	0.936	1.017	0.912	1.007
	(0.511, 1.716)	(0.538, 1.923)	(0.497, 1.675)	(0.536, 1.893)
Lower white-collar[d]	0.894	0.952	0.888	0.947
	(0.457, 1.492)	(0.569, 1.592)	(0.534, 1.474)	(0.568, 1.581)
Divorced	1.52	1.201	1.502	1.188
	(0.906, 2.550)	(0.720, 2.002)	(0.895, 2.519)	(0.710, 1.984)
Widowed	0.643	0.644	0.647	0.652
	(0.290, 1.426)	(0.289, 1.438)	(0.296, 1.413)	(0.298, 1.429)
Diabetes	2.246**	1.980*	2.164*	1.893*
	(1.221, 4.129)	(1.090, 3.597)	(1.168, 4.011)	(1.035, 3.466)
Hypertension	1.358	1.223	1.379	1.254
	(0.847, 2.175)	(0.766, 1.952)	(0.858, 2.212)	(0.784, 2.004)
AMI family history	1.473***	1.448***	1.480***	1.452***
	(1.217, 1.782)	(1.198, 1.751)	(1.213, 1.806)	(1.197, 1.763)
Additive childhood misfortune (ACM)	1.095*	1.063		
	(1.019, 1.178)	(0.981, 1.151)		
Childhood misfortune categories				
Child maltreatment			1.598*	1.484
			(1.004, 2.545)	(0.911, 2.418)
Childhood SES			1.330	1.269
			(0.752, 2.349)	(0.724, 2.223)
Household structure			1.258	1.212
			(0.761, 2.171)	(0.720, 2.036)
Poor health at age 16			1.022	0.886
			(0.455, 2.300)	(0.358, 2.190)
Health lifestyle factors				
Total pack-years smoked		1.011***		1.011***
		(1.005, 1.017)		(1.005, 1.016)
Obese		1.184		1.178
		(0.753, 1.865)		(0.748, 1.859)
Psychosocial factors				
Anxiety		0.098		1.106
		(0.972, 1.252)		(0.972, 1.260)
Locus of control		0.908		0.902
		(0.737, 1.121)		(0.734, 1.108)
Family strain		1.241		1.271
		(0.826, 1.865)		(0.848, 1.904)
Log likelihood	−701.726	−25846.201	−700.701	−25997.434
AIC	1431.452	51758.402	1435.401	52066.868
N	2832	2832	2835	2835

*$p < 0.05$; **$p < 0.01$; ***$p < 0.001$ (two-tailed tests).
[a] Reference group is cohort 1 (1920–1929).
[b] Hazard ratio.
[c] Confidence interval.
[d] Reference group is blue-collar occupations.

additive childhood misfortune (ACM) on AMI risk; Models 3 and 4 examined the four domains of childhood misfortune. To establish an association between ACM and AMI risk, Model 1 estimated AMI risk without any of the potential mediators. As shown, ACM increased AMI risk (HR = 1.095, $p < 0.05$). Each additional misfortune increases AMI risk by 9.5%. A positive relationship was also observed between AMI family history and AMI risk (HR = 1.473, $p < 0.001$). Other significant predictors included cohort, female, and diabetes: the 1940s cohort had a higher risk of AMI than the 1920s cohort; women had a lower risk of AMI than men; and being diabetic increased AMI risk.

Model 2 examined whether health lifestyle or psychosocial factors mediated the effect of ACM on AMI risk. The 1940s cohort, female, diabetes, and family history of AMI remained significant, but the effect of ACM was attenuated when the potential mediators were added to the model ($p > 0.05$). Among the five mediators, only total pack-years of smoking was significant, increasing the risk of AMI (HR = 1.011, $p < 0.001$). Each additional pack-year of smoking

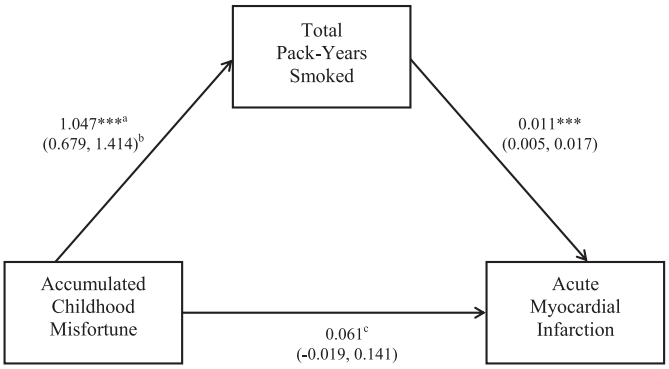

Fig. 1. Relationship between accumulated childhood misfortune, smoking, and acute myocardial infarction. [a]Unstandardized coefficient. [b]Confidence interval. [c]Indirect effect: $b = 0.011$**. *$p < 0.05$; **$p < 0.01$; ***$p < 0.001$ (two-tailed tests).

increased the hazard by 1.1%. A formal test of mediation revealed the pathway of ACM to AMI, demonstrating the direct effect of ACM on smoking ($b = 1.047$, $p < 0.01$) and the indirect effect of ACM on AMI via smoking ($b = 0.011$, $p < 0.01$). The results are displayed graphically in Fig. 1.

Models 3 and 4 parallel Models 1 and 2, but replaced ACM with four domains of childhood misfortune. Among the four domains, Model 3 reveals that only child maltreatment predicted AMI risk; those who experienced maltreatment have a 59.8% higher risk of AMI than those who did not experience maltreatment (HR = 1.598, $p < 0.05$). Similar to the ACM Models, being born in the 1940s, diabetes, and family history of AMI increased AMI risk, whereas women had lower risk of AMI compared to men.

Model 4 examined whether health lifestyle or psychosocial factors mediated the effect of child maltreatment on AMI risk. Results yielded findings parallel to Model 2: the addition of potential mediators fully attenuated the effect of child maltreatment; the 1940s cohort, female, diabetes, and family history of AMI remained significant; and smoking was the only significant predictor of AMI risk among the 5 potential mediators (HR = 1.011, $p < 0.001$). Each additional pack-year of smoking increased the hazard by 1.1%. A formal test of mediation demonstrated that smoking mediated the relationship between child maltreatment and AMI. A direct association between child maltreatment and smoking ($b = 2.738$, $p < 0.01$) was established in addition to an indirect effect of child maltreatment on AMI via smoking ($b = 0.029$, $p < 0.05$). Results are displayed in Fig. 2.

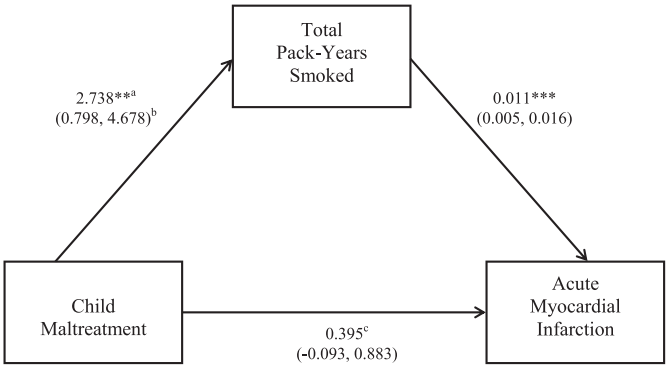

Fig. 2. Relationship between child maltreatment, smoking, and acute myocardial infarction. [a]Unstandardized coefficient, [b]Confidence interval. [c]Indirect effect: $b = 0.029$*. *$p < 0.05$; **$p < 0.01$; ***$p < 0.001$ (two-tailed tests).

Discussion

Our aim was to systematically examine the effect of childhood misfortune on adult AMI risk. In doing so, we tested three main hypotheses. First, childhood misfortune was hypothesized to increase adult AMI risk. Results for the analyses of both additive childhood misfortune and domains of misfortune supported H1, revealing that childhood is a sensitive period for adult health. Parallel to findings by Felitti et al. (1998) for ischemic heart disease, higher levels of childhood misfortune among MIDUS respondents were associated with heightened risk of adult AMI. When domains of childhood misfortune were examined separately, only child maltreatment predicted AMI risk. Although some scholars position SES as a salient social determinant of health status, childhood SES did not predict AMI risk when adjusting for adult covariates. Given the growing evidence on the short- and long-term health consequences of child maltreatment, this study's observed effect of child maltreatment suggests that child maltreatment could potentially be another fundamental cause of disease (e.g., Greenfield & Marks, 2009; Morton et al., 2012).

The second and third hypotheses predicted that health lifestyle factors (H2) and psychosocial factors (H3) would mediate the effect of childhood misfortune on AMI risk. The present study found partial support for H2 whereas H3 was not supported. Among the five potential mediators—two of which were health lifestyle factors—only smoking mediated the effect of childhood misfortune on AMI risk. Although findings revealed that neither obesity nor psychosocial factors mediated the effects of ACM or child maltreatment on AMI risk, the mediating effect of smoking for both ACM and maltreatment implies that smoking may act as a detrimental coping mechanism for children of misfortune. Previous research has connected childhood misfortune to adult health via smoking (Brown et al., 2010), indicating that children of misfortune are at higher risk for smoking—and that interventions to reduce smoking risk should target this vulnerable population with resources for healthy coping. Despite this finding for smoking as a mediator, our list of mediators is in no way exhaustive; future research should continue to investigate potential mechanisms that include both typical and novel risk factors.

Although not hypothesized, one interesting finding is the effect of cohort on AMI risk, which reveals two noteworthy observations. First, the relationship between cohort and AMI risk is not linear. Note that cohorts 2 (1930–1939) and 4 (1950–1974) did not differ from cohort 1, but cohort 3 (1940–1949) had notably higher AMI risk. Second, whereas one would expect earlier cohorts to have a higher AMI risk, it appears that one of the more recent cohorts (cohort 3) is at higher risk of AMI than earlier cohorts. Due to the retrospective nature of how age at AMI was measured (i.e., a respondent may have had an AMI prior to joining MIDUS in 1995), respondents who reported having an AMI at W1 actually consisted of those who had an AMI and survived. Medical and technological advancements increasing survival rates may explain why earlier cohorts who had an AMI prior to W1 are less likely to survive, and, therefore, underrepresented at W1. Although recent cohorts may not be more likely to experience an AMI, they may be more likely to *survive* an AMI, and, therefore, that is what these data capture.

Based on these survival issues, one may intuitively expect the most recent cohort to also have a higher AMI risk, but respondents in this cohort (1950–1974) were much younger at W1 than the rest of the sample and, therefore, have not reached the average age when most AMIs occur (at W1, they were aged 25–45). Therefore, cohort 3 (1940s–1949) is the most likely cohort to have a substantial amount of individuals who experienced an AMI and survived—and these are AMIs experienced *before* age 65. These cohort and survival effects speak to CI theory's axiom 5, which addresses

selection processes and how they alter the composition of samples. In addition to selection issues, these data also present several other limitations.

Due to the longitudinal nature of the data, sampling bias arises from attrition, specifically attrition owing to mortality. Although mortality records are maintained for MIDUS participants, data on cause of death are not available. Therefore, the number of AMI cases may be underreported as respondents could have had an AMI between W1 and W2, but did not survive. This may also be related to the small proportion of AMI occurrence (~5%). Although AMI occurrence is relatively rare in these data, the fact that childhood misfortune is related to AMI attests to the importance of childhood misfortune in the etiology of AMI and related cardiovascular diseases. A final limitation is that the childhood data are retrospective and, therefore, may be subject to recall bias.

Despite these limitations, the present study addresses a growing health concern among Americans by identifying early life course antecedents for one of the major types of cardiovascular disease afflicting modern societies: acute myocardial infarction. Building on life course research examining AMI risk, this study contributes to the current literature by using an age-heterogeneous sample to systematically investigate a vector of childhood misfortune and its subsequent life course pathways. It adds credence to previous studies that observed a link between childhood misfortune and AMI (e.g., Hallqvist et al., 2004; Hamil-Luker & O'Rand, 2007), but also shows the importance of incorporating family lineage—including family risk of AMI—in such epidemiologic studies. The effect of family history of AMI on AMI risk was observed in all four Models, and suggests at least two implications.

First, findings highlight the importance of including information on family history when examining health. Scholars investigating the role of the environment in health outcomes should not neglect the intergenerational transmission of health risks. Second, the findings speak to the salient nature of childhood misfortune on a major disease. Although it may be easy to conceptualize childhood misfortune having long-term effects on mental health, the results from this analysis reveal that such misfortune can exert a notable impact on cardiovascular health. Additive childhood misfortune and child maltreatment remained significant predictors of AMI even when accounting for AMI family history. This finding corroborates others that have uncovered a link between childhood misfortune and AMI risk (e.g., Hallqvist et al., 2004; O'Rand & Hamil-Luker, 2005), but also highlights the importance of simultaneously considering the intergenerational transmission of health risks.

Although the empirical tests are consistent with several of the expectations of CI theory—notable influences of childhood experiences and family lineage as well as selection processes—few of the resources postulated by CI theory actually held sway in affecting AMI risk. Psychosocial factors such as locus of control have been found to mediate the relationships between childhood misfortune and self-rated health (e.g., Irving & Ferraro, 2006), but psychosocial factors did not mediate the effect of childhood misfortune on AMI risk. Psychosocial factors can influence the relationships between childhood misfortune and some health outcomes, but such expectations from CI theory may need to be tempered in the presence of putative family lineage effects.

An association between childhood misfortune and AMI risk was observed in various statistical specifications, net of adult risk factors and family history of AMI. Still, the present study draws attention to the role of smoking as a mediator. Using a pack-years formulation of smoking, we found that tobacco consumption was predictive of heightened AMI risk. Childhood misfortune, especially maltreatment, increases the likelihood of smoking, including early smoking, and people with extensive smoking histories are at much higher risk of AMI. Given the findings of the present research, we should prioritize efforts to prevent or stop smoking by teenagers who have faced childhood maltreatment or multiple types of misfortune. Even greater attention should be given to interventions for those who have suffered early misfortune and have a family history of AMI. Interrupting the pathway from childhood misfortune to smoking during the teen and early adult years would pay dividends in lowering heart disease risk. This would benefit not only older people at risk of heart attack but also middle-aged men and women.

Acknowledgments

Support for this research was provided by a grant from the National Institute on Aging (R01 AG033541). Data were made available by the Inter-university Consortium for Political and Social Research, Ann Arbor, MI. Neither the collector of the original data nor the Consortium bears any responsibility for the analyses or interpretations presented here. The authors would like to thank Margaret Favorite, Glen Ray Hood, and Kadari Taylor-Watson for their comments on an earlier version of the manuscript.

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