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Original Research Report

Childhood Misfortune, Personality, and Heart Attack: Does Personality Mediate Risk of Myocardial Infarction?

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Abstract

Objective: Previous research has revealed a link between childhood experiences and adult health, but the mechanisms underlying this relationship are less clear. To elucidate this relationship, we investigated the pathway from childhood misfortune to nonfatal myocardial infarction (MI) via individual differences in personality.

Method: Longitudinal data were drawn from the National Survey of Midlife Development in the United States, which sampled 3,032 men and women aged 25–74 years at baseline. Big 5 personality traits and multiple measures of childhood misfortune were used to assess whether personality mediated the effect of childhood misfortune on MI risk.

Results: A series of proportional hazards models revealed that neuroticism mediated the effect of additive childhood misfortune on adult MI risk.

Discussion: Childhood misfortune may be formative in the development of personality, which, subsequently, can be consequential to health. These findings highlight the salient roles of early-life experiences and personality to shape health and aging.

Keywords: Childhood misfortune—Life course—Myocardial infarction—Personality

Gerontological theories are giving greater attention to life-course processes, and empirical research on aging increasingly recognizes how early-life events are connected to the leading causes of death (Felitti et al., 1998). Among the leading causes of death in the United States, cardiovascular disease (CVD) demonstrates a particularly salient link to life-course processes of health initiated early in life (e.g., Blackwell, Hayward, & Crimmins, 2001; Hallqvist, Lynch, Bartley, Lang, & Blane, 2004; Morton, Mustillo, & Ferraro, 2014; O'Rand & Hamil-Luker, 2005; Singer & Ryff, 1999) Indeed, the etiologic factors of adult CVD now include influences from conception, birth, and childhood. Many scholars consider these developmental stages as sensitive or critical periods during which individuals are physiologically and psychologically susceptible to environmental exposures (Ben-Shlomo & Kuh, 2002). For instance, misfortune during childhood can negatively impact biological processes, psychological functioning, and health behaviors, all of which affect CVD risk (Dong et al., 2004; Felitti et al., 1998; Taylor, 2010). Whereas prior research has identified biological processes and health behaviors as mechanisms underlying the childhood–adult CVD connection, one potential mechanism linking childhood misfortune to adult CVD has yet to be examined systematically—personality.

Since about half of the variation in personality development is influenced by environmental factors (Clark & Watson, 2008), early-life experiences can be influential on the developing personality (Bowlby, 1969). Exploring

pathways between childhood misfortune and personality development is important because, like childhood misfortune, personality is also a robust predictor of many health behaviors and CVD outcomes throughout the life course (Hampson & Friedman, 2008). For example, higher levels of neuroticism are associated with higher prevalence of CVD (Suls & Bunde, 2005) and mortality risk (Mroczek, Spiro, & Turiano, 2009), whereas higher levels of conscientiousness are associated with lower levels of inflammation (Chapman et al., 2011) and decreased risk of coronary heart disease and stroke mortality (Jokela, Pulkki-Råback, Elovainio, & Kivimäki, 2014).

Given the substantial evidence of both childhood misfortune and personality as key factors in trajectories of health and aging, we ask how childhood events and personality may be connected. Drawing from a life-course conceptual model of accumulation, we posit that a chain of risks beginning in childhood will unfold, resulting in less optimal personality development and adult CVD (Ben-Shlomo & Kuh, 2002). Our objective for the current study is to merge the extant literatures of childhood misfortune and personality as predictors of adult CVD by testing whether personality traits explain (mediate) why misfortune experienced during childhood predicts later-life cardiovascular events. Specifically, childhood misfortune is expected to affect personality development, which, in turn, is expected to influence nonfatal myocardial infarction (MI) risk in adulthood. To test this proposed chain of risks, we use two waves of data from a national U.S. survey of more than 3,000 adults.

The Life Course of Childhood Misfortune

An extensive literature linking childhood events to adult CVD demonstrates that childhood misfortune interrupts the path toward optimal aging (for review, see Braveman & Barclay, 2009). Among the CVDs studied, ischemic heart disease (IHD) warrants special attention for U.S. populations. IHD, which often presents itself as MI or angina, is the most common type of heart disease in the United States (Centers for Disease Control and Prevention [CDC], 2015). Also, there is substantial evidence demonstrating that the antecedents of IHD can occur in the earliest life stages (Barker et al., 1989). During childhood, individuals may experience different types of misfortune—such as low socioeconomic status (SES), maltreatment, and altered family composition—each of which is associated with IHD in adulthood (Hallqvist et al., 2004; Kaplan & Salonen, 1990; Morton et al., 2014; O'Rand & Hamil-Luker, 2005). Moreover, when combined, accumulated childhood misfortune reveals that each additional misfortune increases risk of IHD (Dong et al., 2004; Felitti et al., 1998; Morton et al., 2014). How childhood misfortune structures the risk of adult IHD can be understood through conceptual advances in life-course analysis.

The life-course risk accumulation model provides a conceptual platform for understanding how childhood experiences (a) accumulate and (b) lead to IHD risk. First, the concept of risk clustering suggests that the co-occurrence of disadvantageous or stressful experiences combine to exert an additive effect on health risk (Ben-Shlomo & Kuh, 2002). Accumulation via risk clustering may occur within a single life stage: accumulation can be defined as amassing different types of childhood experiences, such as socioeconomic disadvantage, abuse, or poor health (Ferraro & Morton, 2016). The effect of risk clustering during childhood has been well substantiated by prior research. The cumulative effect of childhood misfortune has been observed for IHD, including angina and MI (Dong et al., 2004; Morton et al., 2014), and associated risk factors, such as hypertension and diabetes (Felitti et al., 1998; Stein et al., 2010). In the present study, we investigate the additive effect of multiple childhood experiences.

Second, to elucidate the path from early-life events to later-life health, we draw upon the chain of risks concept. This concept specifies a sequence of risks following precarious events or experiences (Ben-Shlomo & Kuh, 2002). Whereas risk clustering implies that events and experiences occur during the same time period (e.g., childhood), a chain of risks comprises a series of temporally ordered risks occurring at different life stages (Ben-Shlomo & Kuh, 2002). Related to the present study, childhood misfortune could set off a chain reaction of early risks begetting future risk in later life, culminating in MI risk. Felitti and associates (1998) articulate a pyramid process model in which they posit that childhood adversity increases risk of premature mortality through a temporally outlined chain of risks, which includes social/emotional impairment, risky health behaviors, disability, and disease. Indeed, the additive effect of childhood misfortune on IHD and mortality has been shown to operate through obesity, psychological factors, and smoking (Dong et al., 2004; Morton et al., 2014). However, scholars report that nontraditional mechanisms of misfortune (e.g., psychological risk factors) have produced stronger mediational effects on IHD than traditional risk factors (e.g., health behaviors), urging future research to investigate more nontraditional pathways (Dong et al., 2004). The present study advances the current research by investigating whether personality could also contribute to the chain of risks initiated in childhood.

Several theoretical frameworks, such as cumulative inequality and stress process, proposed that acute or chronic stressors can trigger a domino effect toward poor health (Ferraro & Shippee, 2009; Pearlin, Aneshensel, & LeBlanc, 1997), but these frameworks have yet to explicitly identify personality as a mechanism. The health behavior model's conceptualization of personality, however, suggests that differences in personality characteristics are associated with behavior patterns that either promote or harm health over time (Smith, 2006). Other mechanisms such as stress reactivity may also play a role (see Turiano, Chapman, Gruenewald, & Mroczek, 2015). Therefore, the health consequences of personality are not attributed solely to

behaviors; personality is a distinct pathway of health development. Indeed, recent findings suggest that personality is a theoretically overlooked—but empirically relevant—component of the stress process (Pai & Carr, 2010). Thus, our aim is to contribute to the literature by elucidating personality's role in the cardiovascular health risks of adults who experience early misfortune. As such, our aims are empirical and conceptual.

The Role of Personality

When children are born, there are biological dispositions called temperament that the child displays almost immediately after birth (Thomas, Chess, Birch, Hertzig, & Korn, 1963). As children grow older, more stable and measurable aspects of personality develop, largely influenced by environmental forces (Clark & Watson, 2008). Defined by the five-factor model of personality, individual differences in each of the Big 5 traits—agreeableness, conscientiousness, extraversion, neuroticism, and openness—represent the relatively enduring patterns of thoughts, feelings, and actions that both differentiate and make people alike. Agreeableness refers to the propensity to be prosocial, co-operative, empathetic, generous, and kind versus those who tend to be hostile and aggressive. Conscientiousness refers to the propensity to be goal directed, responsible, disciplined, and in control of impulses. Extraversion implies an inclination to be outgoing, expressive, sociable, and energetic, whereas introversion describes individuals who are quiet, inhibited, and content on following the lead of others. Neuroticism reflects an individual's propensity to experience elevated levels of negative affect and higher emotional reactivity. Finally, imagination, creativity, open mindedness, and intellectuality are all characteristics of a person high in trait openness.

From a developmental perspective, life experiences, particularly negative experiences, can be very influential on the developing personality during childhood (Bowlby, 1969; Caspi & Shiner, 2009). Emotional, physical, and sexual abuse as well as neglect are some of the main negative childhood experiences (Cicchetti & Barnett, 1991; Felitti et al., 1998). Childhood traumas such as these affect the successful development of a secure attachment with a parent, affect regulation and impulse control and, perhaps most importantly, influence the development of a stable identity (Cicchetti & Toth, 2005). All of these developmental processes occurring during childhood are critical foundations for the development of a stable and healthy personality profile typically reflected by emotional stability (low neuroticism), high conscientiousness, agreeableness, openness, and extraversion (Clark & Watson, 2008). Because personality characteristics are robust predictors of behavior, physiological reactivity, and various health outcomes (Hampson & Friedman, 2008), it is likely that personality may connect the causal chain by which early-life misfortune influences adult health.

There is an established empirical base that early-life misfortune is predictive of later-life psychopathology (Cicchetti & Lynch, 1995; Cicchetti & Toth, 2005), but because misfortune may not lead to a clinically relevant threshold where a diagnosis is given, more attention is needed to examine how such misfortune influences more normative personality trait characteristics. The handful of studies that have linked misfortune to personality characteristics have utilized various types of misfortune measures (e.g., abuse and neglect, low SES), different cultures, and varying life-course ages. From these studies, a consistent pattern has emerged: early-life misfortune is associated with higher levels of neuroticism and lower levels of conscientiousness (Jonassaint, Siegler, Barefoot, Edwards, & Williams, 2011; Kitamura & Fujihara, 2003; Rogosch & Cicchetti, 2004; Rosenman & Rodgers, 2006; Roy, 2002). There also is evidence that misfortune is related to lower levels of agreeableness, openness, and extraversion (Kitamura & Fujihara, 2003; Rogosch & Cicchetti, 2004). Because lower levels of conscientiousness, extraversion, agreeableness, openness, and emotional stability are predictors of CVD and its risk factors (Jokela et al., 2014; Suls & Bunde, 2005), we expect that personality will mediate the relationship between childhood misfortune and adult MI risk.

Method

Data

The present study used two waves of the National Survey for Midlife Development in the United States (MIDUS), a national survey of midlife and older adults residing in the continental United States. These data provide a wealth of information on childhood, disease onset, personality indicators, and potential confounders—such as demographic, psychosocial, and health behavioral factors—making these data ideal for the present study. The first wave of data (W1) collected between 1995 and 1996 employed a national random-digit-dial sample of 3,032 noninstitutionalized, English-speaking men and women aged 25–74 years, with an oversample of men and older adults aged 65–74 years. Respondents were first contacted by phone and participated in a computer-assisted telephone interview (response rate ~ 70%). Then, respondents were mailed a self-administered questionnaire (response rate ~ 87%). The overall response rate for completing both the telephone interview and mailed questionnaire was approximately 61% (.70 * .87 = .61).

From 2004 to 2006, MIDUS conducted a follow-up survey of surviving W1 respondents. This second wave of data (W2) consists of a similar telephone interview and mailed questionnaire. Respondents from W1 participated in the W2 telephone interview (N = 2,101), resulting in a response rate of 69.5% (71% mortality-adjusted response rate). The present study utilized the W1 telephone and mail surveys and the W2 telephone interviews.

Nonfatal MI

The dependent variable for this study is age of first nonfatal MI. Respondents were asked two separate questions that could indicate whether they had experienced a heart attack. The first question asked whether the respondent had "ever had heart trouble suspected or confirmed by a doctor." If so, respondents were asked for the specific diagnosis. Second, respondents were asked whether they ever had a heart attack. Respondents who answered either yes to this question or indicated that their heart trouble diagnosis was a heart attack were then asked in what year they had their first heart attack. Using these items, two variables were created for the event history analysis. First, the censoring variable was created by coding respondents as "1" for heart attack observed by the second wave (2005). Second, the duration variable was created by subtracting respondent's birth year from year of MI to create age at first MI. The duration variable was age at latest survey for respondents who had not reported experiencing an MI. Among respondents who reported experiencing an MI, age of first nonfatal MI ranged from 20 to 76 years.

Although some studies examine a global measure of CVD (e.g., Blackwell et al., 2001), we focus on MI for substantive reasons. First, MI differs from other types of CVD in risks and diagnoses; an individual is often aware of experiencing an MI whereas certain types of CVD, such as hypertension, may go undetected, particularly for those without access to health care. Moreover, individuals who experience an MI are able to recall the event with relatively high accuracy (Okura, Urban, Mahoney, Jacobsen, & Rodeheffer, 2004). Second, our aim was to investigate when an event occurred to explore the key life-course component of timing. MI is the only CVD for which MIDUS includes year of onset at both waves. Third, MI prevalence estimates from the MIDUS reflect MI prevalence estimates reported in other U.S. population health surveys given the same age group and observation period. For instance, MI prevalence estimates in the MIDUS are similar to those of the National Health and Nutrition Examination Survey (NHANES)—one of the main national health surveys from which the CDC and American Heart Association derive their estimates of MI prevalence (CDC, 2012). Fourth, MI mortality in the United States decreased between 1995 and 2006—the years from which MIDUS respondents were sampled (Krumholz et al., 2009). Therefore, the issue of competing mortality risk is decreased when using MI as an outcome.

Childhood misfortune

Sixteen items of childhood misfortune were drawn from the W1 surveys based on available indicators and prior research (Felitti et al., 1998; Morton et al., 2014; Turner, Wheaton, & Lloyd, 1995). These indicators tap into several early-life domains: childhood SES, family structure, maltreatment, and health. Childhood SES items include (1) family receipt of welfare or Aid to Dependent Children; (2) self-report of being financially worse off than other families; and (3) less than a high school education for father (or mother if father was absent). Family structure items include (4) female head of house; (5) parental divorce; and (6) death of a parent. Child maltreatment items come from Straus's (1979) Conflict Tactics Scale and include (7–10) emotional abuse by father, mother, sibling, or other; and (11–14) physical abuse by father, mother, sibling, or other. For the maltreatment items, respondents were asked how frequently their mother/father/siblings/or other 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 scalded them. Childhood health items include (15–16) self-report of poor physical or mental health at age 16. The other indicators ask respondents to recall a time when they "were growing up."

All childhood misfortune items were first coded as dummy variables, with "1" indicating experiencing the misfortune. Because response categories for physical and emotional abuse ranged from *never* to *often*, respondents who reported experiencing abuse either sometimes or often were coded as "1." Previous research 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 (Dowd, Kinsey, Wheeless, Suresh, & NSCAW Research Group, 2004; Straus, Hamby, Finkelhor, Moore, & Runyan, 1998). To aid internal consistency, we followed Straus and associates' (1998) recommended dichotomization of the scales. Similarly, responses for self-ratings of physical and mental health were ordinal, ranging from *poor* to *excellent*. Respondents who reported their health as poor or fair were coded as "1." Dichotomizing retrospective health measures into more general groups yields more reliable measures of childhood health (Haas, 2007).

To create the main predictor of additive childhood misfortune (ACM), the 16 dummy variables were summed to assess the additive effect of misfortune. This count variable, ACM, follows a negative binomial distribution (Poisson distribution with overdispersion), ranging from 0 to 14.

Personality

The Big 5 personality traits of agreeableness, conscientiousness, extraversion, neuroticism, and openness were drawn from W1 and based on prior research (Mroczek & Kolarz, 1998; Prenda & Lachman, 2001). These scales have good construct validity, and all five traits are significantly correlated with the Neuroticism, Extraversion, and Openness traits, which some consider the gold standard for measuring personality (Mroczek & Kolarz, 1998). Respondents were given 30 adjectives and asked how much each of the words described them, with responses ranging from *a lot* (originally coded as 1) to *not at all* (originally coded as

4). Some items required reverse coding to construct the five personality traits. Agreeableness was created using the adjectives of helpful, warm, caring, softhearted, and sympathetic (alpha = .80). Conscientiousness was created using the adjectives of organized, responsible, hardworking, and careless (alpha = .58). Extraversion was created using the adjectives of outgoing, friendly, lively, active, and talkative (alpha = .78). Neuroticism was created using the adjectives of moody, worrying, nervous, and calm (alpha = .74). Openness was created using the adjectives of creative, imaginative, intelligent, curious, broad minded, sophisticated, and adventurous (alpha = .77). For each of the personality traits, the mean of the associated adjectives was taken. The personality scales range from 1 to 4, with higher scores reflecting higher standings for each trait.

Adult risk factors

To adjust for potential confounders, W1 variables of MI family history, diabetes, hypertension, obesity, and smoking were included. All respondents were asked how many people in their immediate biological family—that is, biological parents, brothers, or sisters—ever had a heart attack. The original variable ranged from 0 to 20 but was top coded at 4 given that few respondents reported more than 4. Diabetes, hypertension, and obesity were coded as dummy variables with "1" indicative of having the condition. Obesity was defined as having a body mass index equal to or greater than 30 kg/m^2. Smoking was measured by pack-years smoked (total years smoked was multiplied by daily average number of cigarettes smoked, divided by 20). Participants who never were smokers were coded as "0." Pack-years smoked ranges from 0 to 203.7.

Demographics

Demographic information on cohort, gender, race, education, and marital status was taken from W1. Whereas the risk of CVD and survival may vary by cohorts due to medical and technological advancements, cohorts were used rather than age (Lloyd-Jones et al., 2006). Cohorts are also helpful for identifying health selection processes (Ferraro & Shippee, 2009). Respondents were divided into four birth cohorts: (a) 1920–1929; (b) 1930–1939; (c) 1940–1949; and (d) 1950–1974. Due to the relatively small number of MI occurrences in more recent cohorts, those born after 1950 were collapsed into a single cohort to ensure sufficient statistical power (<1% of those born after 1950 experienced an MI by W2). Cohorts were included as a series of dummy variables, with the 1920s cohort used as the referent. Dummy variables were created for gender (1 = female), race (1 = Black), divorced, and widowed. Education was a continuous variable measured in years, ranging from 4 to 20.

Analytic Strategy

Descriptive statistics and bivariate correlations were estimated in Stata (version 13.0). Cox proportional hazard models, and tests of mediation were estimated in Mplus (version 7.0). Because Cox models with robust variance provide more reliable estimates than logistic regression (Barros & Hirakata, 2003) and the proportional hazards assumption was not violated ($\chi^2 = 25.46$, $df = 19$, $p = .1458$), Cox models were estimated to assess the rate at which nonfatal MI onset occurs. The first Cox model tested whether there was a baseline association between ACM and MI risk without any of the potential mediating influences of personality. We initially included several demographic covariates in this model but retained only the covariates associated with the outcome—MI risk. Preliminary analyses were adjusted for income, early onset of obesity, occupation, and family strain, but these were removed from the final analyses because they were not associated with the outcome in any model specification. Overall, conclusions remained the same with removal of these variables, but model fit indices were improved. Model 2 added each of the Big 5 personality traits as potential mediators of the ACM–MI risk association. Finally, a formal test of mediation was conducted for each personality trait. For these final analyses, we modeled onset of MI and performed tests of mediation using a maximum-likelihood robust estimator and Monte Carlo integration. Paths were created to establish a direct relationship between ACM and neuroticism (a) and neuroticism and MI risk (b); indirect effects were calculated in Mplus by taking the product of the two paths (a * b). Standard errors were calculated using the delta method, and full-information maximum-likelihood was used for missing data on independent variables. Respondents who were missing on the outcome variable were removed from the analyses, decreasing the sample size by 20. All models adjusted for the main MIDUS sample weight.

Results

Descriptive statistics are presented in Table 1. Approximately 5% of the total sample experienced a nonfatal MI by W2, with mean age of first MI of 54.76 (11.046) years. The mean of ACM is 3.139 (2.539), indicating the average number of childhood misfortunes experienced by the sample. Whereas the sample is somewhat evenly distributed by gender, the majority of the sample is White, highly educated, and relatively healthy (e.g., only about 25% is obese and even fewer have diabetes or hypertension). Participants reported relatively high levels of agreeableness, conscientiousness, extraversion, and openness and relatively low levels of neuroticism.

The results of the Cox proportional hazard models are displayed in Table 2. Model 1 estimated the association between ACM and MI risk, adjusting for demographic and adult risk factors, but without any of the potential mediating influences of personality. As shown, ACM was associated with MI onset (hazard ratio [HR] = 1.084, $p < .05$). For each additional type of childhood misfortune, MI risk increased by 8.4% each year, controlling for other covariates. Cohort, gender, MI family history, diabetes, and

Table 1. Descriptive Statistics for Variables: Midlife Development in the U.S. Study (N = 3,012)

Variables	Range	Mean	*SD*
MI occurrence	0, 1	0.045	
Age at which MI occurred (years)	20–76	54.759	11.046
Additive childhood misfortune	0–14	3.139	2.539
Demographics			
Cohort 1 (1920–1929)	0, 1	0.107	
Cohort 2 (1930–1939)	0, 1	0.194	
Cohort 3 (1940–1949)	0, 1	0.241	
Cohort 4 (1950–1974)	0, 1	0.458	
Female	0, 1	0.515	
Black	0, 1	0.069	
Education	4–20	13.800	2.606
Divorced	0, 1	0.186	
Widowed	0, 1	0.058	
Adult risk factors			
MI family history	0–4	0.504	0.786
Diabetes	0, 1	0.054	
Hypertension	0, 1	0.183	
Obese	0, 1	0.247	
Pack-years smoked	0–203.7	16.068	25.812
Personality traits			
Agreeableness	1–4	3.485	0.485
Conscientiousness	1–4	3.406	0.453
Extraversion	1–4	3.204	0.565
Neuroticism	1–4	2.248	0.664
Openness	1–4	3.042	0.524

Note: MI = myocardial infarction.

pack-years smoked were also significant predictors of MI risk. The 1940s cohort had a higher risk of MI than the 1920s cohort (HR = 2.542, $p < .01$). Women had a lower risk of MI compared with men (HR = 0.582, $p < .05$). The number of immediate family members who experienced an MI was directly associated with MI risk (HR = 1.456, $p < .001$). Diabetics had an increased risk of MI (HR = 2.117, $p < .01$). The more pack-years smoked, the higher the risk of MI (HR = 1.011, $p < .001$).

Model 2 examined whether personality mediated the effect of ACM on MI risk. When the Big 5 personality traits were introduced as mediators, the effect of ACM was attenuated, becoming nonsignificant ($p = .14$). The variables of cohort, gender, MI family history, diabetes, and pack-years smoked that were significant in Model 1 remained significant in Model 2. Among the five personality traits that were entered into the model, only neuroticism was significantly associated with MI risk (HR = 1.441, $p < .05$).

A formal test of mediation to elucidate the path from ACM to MI was also conducted. This revealed a significant direct effect of ACM on neuroticism (HR = 1.057, $p < .001$) as well as a significant indirect effect of ACM on MI via neuroticism (HR = 1.020, $p < .05$). The total effect was also significant (HR = 1.079, $p < .05$). These findings reveal that neuroticism mediated the relationship between ACM and MI, net of adult risk factors and other personality traits. These results are illustrated in Figure 1.

Formal mediation tests for the other four personality traits revealed that the direct effects of ACM on conscientiousness and extraversion were both significant ($p < .001$; $p < .05$, respectively), but neither indirect effect was significant. Neither the direct nor indirect effects of agreeableness and openness were significant.

Supplementary analyses were conducted to further explore the childhood misfortune–MI risk association. First, we examined three alternative specifications of childhood misfortune—individual types, domains, and an alternative ACM measure—because prior research has revealed that the unique and additive effects of misfortune can operate differently (e.g., Morton, Schafer, & Ferraro, 2012). The effect of individual types of childhood misfortune was investigated by using each dummy variable of misfortune as a separate predictor. (Supplementary analyses investigating which types of misfortune had the strongest effect on personality revealed that emotional abuse (β = .09; SE = .02; p = .001), physical abuse (β = .07; SE = .02; p = .001), and health at 16 years of age (β = .21; SE = .03; p = .00) had the strongest associations with neuroticism.) Domains that classified misfortune by childhood SES, family structure, child maltreatment, and childhood health were used as four separate predictors. An alternative ACM measure that excluded mental and physical health was examined to ensure that these variables were not solely driving the relationship between ACM and personality. Second, gender-stratified analyses were also performed because there are gender differences in MI risk. However, none of these supplementary analyses revealed any findings to alter the conclusions presented herein. Although these analyses are not shown, they are available upon request.

Discussion

The present study identifies neuroticism as a life-course mechanism for how childhood misfortune influences adult health, potentially threatening optimal aging. Although we expected that all components of personality would play a significant role in mediating the effect of childhood misfortune on adult nonfatal MI risk, only neuroticism was a significant mediator. Neuroticism is related to risk factors for vascular damage and the pathogenesis of MI risk; it is associated with increased risk of hypertension (Phillips et al., 2010) and higher prevalence of smoking (Turiano, Whiteman, Hampson, Roberts, & Mroczek, 2012). Recent research also suggests that neurotic individuals often self-medicate feelings of depressed affect and anxiety by using alcohol, tobacco, and illicit drugs, which also raise MI risk (for review, see Turiano et al., 2012). In addition, neuroticism is related to higher levels of adiposity that can induce inflammatory responses leading to CVD (Dandona, Aljada, & Bandyopadhyay, 2004; Guzik, Mangalat, & Korbut, 2006; Yudkin, Kumari,

Table 2. Cox Proportional Hazards Models for MI, Midlife Development in the U.S. Study (1995–2005)

	Model 1	Model 2
Independent variables	Hazard ratio (confidence interval)	Hazard ratio (confidence interval)
Additive childhood misfortune	1.084* (1.009, 1.161)	1.058 (0.982, 1.138)
Demographics		
Cohort 2[a] (1930–1939)	1.347 (0.755, 2.404)	1.280 (0.731, 2.243)
Cohort 3[a] (1940–1949)	2.542** (1.338, 4.826)	2.370** (1.269, 4.428)
Cohort 4[a] (1950–1974)	2.119 (0.770, 5.824)	1.833 (0.682, 4.928)
Female	0.582* (0.363, 0.932)	0.531* (0.324, 0.872)
Black	1.081 (0.481, 2.428)	1.161 (0.513, 2.630)
Education	0.953 (0.866, 1.049)	0.943 (0.845, 1.052)
Divorced	1.311 (0.809, 2.123)	1.331 (0.825, 2.147)
Widowed	0.571 (0.266, 1.224)	0.611 (0.284, 1.314)
Adult risk factors		
MI family history	1.456*** (1.208, 1.756)	1.445*** (1.214, 1.719)
Diabetes	2.117** (1.232, 3.846)	2.038* (1.115, 3.725)
Hypertension	1.250 (0.795, 1.966)	1.223 (0.783, 1.908)
Obese	1.338 (0.882, 2.028)	1.383 (0.907, 2.109)
Pack-years smoked	1.011*** (1.005, 1.016)	1.010*** (1.005, 1.015)
Personality traits		
Agreeableness		1.116 (0.574, 2.173)
Conscientiousness		0.864 (0.461, 1.618)
Extraversion		0.772 (0.446, 1.334)
Openness		1.297 (0.757, 2.223)
Neuroticism		1.441* (1.017, 2.040)
Log likelihood	−41,044.544	−52,680.178
AIC	82,355.088	105,666.356
N	3,012	3,012

Notes: AIC = Akaike information criterion; MI = myocardial infarction.
[a]Referent is cohort 1 (1920–1929).
*$p < .05$. **$p < .01$. ***$p < .001$ (two-tailed tests).

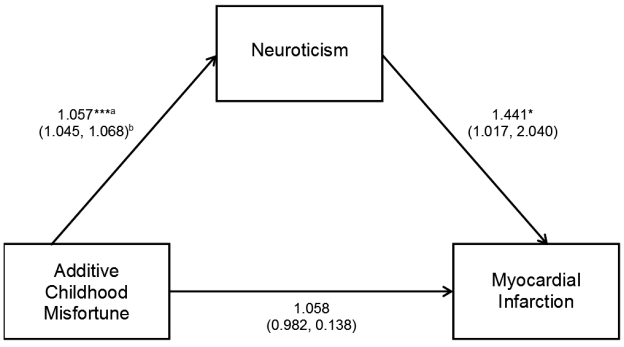

Figure 1. Relationship among additive childhood misfortune, neuroticism, and myocardial infraction. Adjusted for all covariates in Table 2, Model 2. [a]Hazard ratio (HR). [b]Confidence interval (CI). Indirect effect: HR = 1.020* (95% CI: 1.000, 1.041).Total effect: HR = 1.076* (95% CI: 1.003, 1.160). *$p < .05$. ***$p < .001$ (two-tailed tests).

Humphries, & Mohamed-Ali, 2000). Thus, neuroticism can exert influences on cardiovascular outcomes such as MI directly through increased cardiovascular processes as well as indirectly through several detrimental behaviors. However, even when adjusting for adult risk factors, including obesity, hypertension, and smoking, and the other four personality traits, neuroticism's mediating effect, albeit modest, remained significant.

These findings are also consistent with those of prior life-course studies showing that childhood misfortune, including abuse and adverse economic conditions, raises the risk of MI in adulthood (Hallqvist et al., 2004; Morton et al., 2014; O'Rand & Hamil-Luker, 2005). At the same time, the present findings reveal two notable substantive contributions to the literature on childhood misfortune and adult health. First, this study demonstrated a relationship between childhood misfortune and neuroticism decades later, revealing the salience of childhood experiences in the development of personality traits. With mounting evidence that personality may be a root cause of detrimental health behaviors and poorer health outcomes, information on the antecedents of personality development is useful for gerontological research on health over the life course (Shiner & Caspi, 2003; Shiner, Masten & Roberts, 2003).

Second, and most importantly, this study identified neuroticism as an important mediator between early-life events and adult health. This complements research showing that personality is an important mechanism when assessing the aftermath of misfortune (Pai & Carr, 2010). We are unaware

of any previous studies that considered the influence of both childhood exposures and personality on MI risk. Rather than separately assessing the impact of childhood misfortune and personality on MI, we revealed the intertwining nature of these life domains. This finding bridges two previously distinct bodies of research, uncovering an important link among early misfortune, personality development, and adult health: the effect of childhood misfortune on adult MI risk operates through higher levels of neuroticism. This finding remained even when controlling for common risk factors, such as smoking, that often explain associations of childhood disadvantage and personality with adult health.

Beyond empirical contributions, these findings also contribute to the conceptual life-course framework which guided this study. Foremost, the confirmation of our statistical mediation tests of neuroticism supported our theoretically proposed chain of risks—that childhood misfortune would influence personality, which, in turn, would influence MI risk. Our findings do not support a "runaway" model of a critical period during childhood; experiencing multiple forms of misfortune does not inevitably raise MI risk in later life. Rather, intermediate human developmental processes shape MI risk. Our conclusions draw attention to the potent influence of early negative experiences but also that the associated health consequences are contingent on intervening factors (Ferraro & Shippee, 2009).

Our results also are consistent with the concept of risk clustering. The pattern of different types of childhood misfortune co-occurring was evidenced by ACM. On average, most people experienced three types of misfortune during childhood. Moreover, when multiple experiences were combined, they raised the risk of both neuroticism and MI. In line with the concept of risk clustering, these findings reveal that the summation of events and experiences co-occurring during childhood can later influence trajectories of health and aging (Ben-Shlomo & Kuh, 2002). These findings empirically demonstrate the lasting consequences of childhood misfortune, both to shape personality and risk of a leading cause of death in the United States.

These contributions are, however, tempered by this study's limitations. First, the childhood measures are retrospective and, therefore, subject to recollection bias. As such, the results presented herein probably underestimate the prevalence of childhood misfortune (Hardt & Rutter, 2004). Second, there is no actual temporal spacing between the data collection of the exogenous predictor and the mediator due to the survey design. Although the exogenous predictor of childhood misfortune is retrospective and, therefore occurred prior to the mediator, these data were nonetheless collected at the same time; personality could influence retrospective reporting of misfortune. Third, early-life personality traits could influence the likelihood of experiencing certain types of childhood misfortune. Although these data do not include information on early-life personality, many of these misfortunes would have likely occurred independent of the respondent's childhood personality, such as childhood SES (e.g., parent's education and finances) and family structure (e.g., death of parent and female-headed household). Fourth, attrition bias may be present due to the longitudinal survey design. Fifth, given the age-heterogeneous sample, there are selection issues along the lines of health and survival to be eligible for participation in the MIDUS survey, particularly for the earliest cohort. Sixth, those who experienced the most misfortune in early-life are likely underrepresented in the sample given their increased risk of incarceration and premature mortality (Brown et al., 2009). Seventh, although the models were weighted, these findings are most likely generalizable to college-educated, White Americans.

Despite these limitations, we found evidence—both direct and indirect effects—for a mediating effect of neuroticism on the relationship between childhood misfortune and adult nonfatal MI. Moreover, with the low prevalence of MI (~5%), the findings are a testament to how important the mediating effect of personality may be and the salience of childhood misfortune. Future studies with higher prevalence of MI should be employed to replicate the findings presented and assess effect size.

Beyond replication studies with greater power, there are additional implications of these findings for research on health and aging, most notably the need to better understand the personality development reflected in these data. The negative exposures occurred during childhood and adolescence, and we were able to uncover how they led to higher levels of neuroticism in adulthood. In terms of needed research, however, it would be invaluable to determine whether there are early signs of the development of neuroticism in late adolescence and early adulthood; whether there are other stressors during these times that led to the development of neurotic traits; or whether early-life personality traits contribute to the likelihood of experiencing childhood misfortune. Given this study's finding that childhood experiences could influence components of personality coupled with the fact that both childhood misfortune and personality are related to health behaviors and psychosocial resources, then perhaps childhood misfortune sets off a cascade of events toward poor adult health—a multipronged chain of risks that threatens optimal aging. Although the MIDUS does not enable one to identify when neurotic tendencies emerged or have sufficient waves to examine multiple mediators, doing so would be very useful for public health policy and interventions. Given the relationship between personality and health behaviors, creating interventions that target traits such as neuroticism is important. By targeting the personality characteristic rather than a sole behavior, there can be downstream improvements in multiple health behaviors because the root cause of the behavioral problems is addressed. Cognitive behavioral therapy targeting neuroticism, for example, could reduce levels of anxiety or provide an individual a more optimal behavioral outlet to relieve anxiety (e.g., exercise). Nonetheless, this study is a first step toward elucidating

the role of personality when examining health over the life course and paves the way for additional avenues of research.

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