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## Avoiding the Major Causes of Death: Does Childhood Misfortune Reduce the Likelihood of Being Disease Free in Later Life?

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# Avoiding the Major Causes of Death: Does Childhood Misfortune Reduce the Likelihood of Being Disease Free in Later Life?

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## Abstract

### Objectives

Although previous research reveals the detrimental effects of early misfortune on the development of chronic diseases in later life, few studies have investigated its effects on remaining disease free. This study draws on cumulative inequality theory to investigate whether experiencing childhood misfortune reduces the likelihood of remaining disease free over time.

## Method

This study utilizes five waves of data from the Health and Retirement Study to test whether five domains of childhood misfortune predict being disease free at baseline (2004) and developing disease over time (2004–2012).

## Results

Respondents reporting risky parental behaviors during childhood were less likely to be disease free at baseline and had an increased risk of disease onset over time, the latter driven by having a guardian who smoked in combination with more pack-years smoked in adulthood. Furthermore, we find that adult resources, that is wealth, help to mitigate the noxious effects of other misfortunes, notably poor socioeconomic conditions.

## Discussion

Consistent with cumulative inequality theory, these findings reveal that experiencing multiple types of misfortune during childhood decreases the likelihood of remaining disease free in later life, but engaging in health behaviors, such as physical activity, can help to ameliorate some of the noxious effects of early misfortune.

**Keywords:** Childhood disadvantage, Cumulative inequality theory, Successful aging

Nearly 86% of Americans aged 65 and older live with at least one chronic disease, whereas more than 60% have two or more diseases ([Ward, Schiller, & Goodman, 2014](#)). Many chronic diseases are also the leading causes of death for older Americans: Two thirds of their deaths are due to heart disease, cancer, chronic lower respiratory disease, stroke, Alzheimer’s disease, and diabetes ([Heron, 2016](#); [Xu, Murphy, Kochanek, & Arias, 2016](#)).

Although chronic disease in later life is widespread and studied extensively, relatively few investigators examine how people are able to avoid any of the chronic “killer” diseases during later life. Most gerontologists do not consider being disease free a sine qua non for “successful aging,” but avoiding the leading causes of death greatly improves one’s chances for exceptional longevity ([Evert, Lawler, Bogan, & Perls, 2003](#); [Rowe & Kahn, 1987](#)). Studying the antecedents of remaining free of chronic diseases is a logical way to identify risks that are common to multiple diseases and fundamental factors that promote health and well-being.

Epidemiology’s search for risk factors common across life-threatening diseases also has led to greater consideration of experiences from childhood. Although hundreds of studies have identified *adult* risk factors of various chronic diseases, a now well-established body of research has unveiled the influence of *early-life* risk factors on adult diseases, ranging from lung cancer ([Brown et al., 2010](#)) to sarcopenia ([Sayer, Syddall, Gilbody, Dennison, & Cooper, 2004](#)). [Kuh and Ben-Shlomo \(2016\)](#) describe the shift in epidemiology from an adult *life style* model to one that also accounts for *life course* influences. In this article, we examine life course influences on the likelihood of being disease free in later life. We ask whether childhood misfortune reduces the likelihood that older adults will be able to avoid major, life-threatening diseases.

Although reaching later life free of chronic disease is both rare and remarkable, to our knowledge, there is only one study that systematically examines early-life influences on this outcome ([Schafer & Ferraro, 2012](#)). The present study examines the enduring effects of childhood misfortune on health in later life and is distinct from parallel studies in several ways outlined in the following section.

## **Childhood Misfortune and Avoiding Disease**

Dozens of studies during the past two decades reveal how early-life insults elevate the risk of specific diseases during adulthood. There is compelling evidence that childhood misfortune raises the risk of heart disease ([Morton, Mustillo, & Ferraro, 2014](#); [O’Rand & Hamil-Luker, 2005](#)), diabetes ([Thomas, Hyppönen, & Power, 2008](#)), chronic lung disease ([Svanes et al., 2010](#)), and cancer ([Brown et al., 2010](#); [Kemp, Ferraro, Morton, & Mustillo, 2018](#)) directly, through stress processes, and indirectly, through poor environments and health behaviors. These findings have been valuable for identifying life course influences on conditions often identified as “age-related diseases.” Although these diseases are prevalent among older people, the etiology of these and other conditions begins much earlier.

Investigating individual diseases is fruitful for advancing life course epidemiology, but it is plausible that some risk factors are pluripotent. For example, [Blackwell, Hayward, and Crimmins \(2001\)](#) found that poor childhood health is associated with cancer, lung disease, cardiovascular conditions, and arthritis, even after adjusting for life course socioeconomic status. Furthermore, research examining disease accumulation reveals that early-life misfortune raises the risk of comorbidity in later life ([Ferraro, Schafer, & Wilkinson, 2016](#); [Pavela & Latham, 2016](#)).

Failure to account for related diseases in studies of single diseases, however, may lead to misdirected conclusions. For example, a person may be cancer-free giving the appearance of relative health, yet suffer from other, equally life-threatening conditions. Although studying the early origins of disease-free aging is a promising line of inquiry to identify factors that promote longevity and overall health, it is surprising that so few life course studies have examined being disease free as an outcome.

The study that directly motivated the present investigation used data from the Midlife Development in the United States Study (MIDUS). [Schafer and Ferraro \(2012\)](#) found that a simple sum of childhood misfortune indicators, ranging from physical and mental abuse to financial strain, decreased the likelihood of being disease free in adulthood, both at baseline and 10 years later (1995 and 2015). Although being disease free at the follow-up survey was infrequent (<7%), the influence of early misfortune did not wane. The findings from this study are important but limited in several ways.

First, given its focus on midlife, the MIDUS sample has a baseline age ceiling of 74 years, whereas the Health and Retirement Study (HRS) does not, enabling us to examine the highly selective situation where septuagenarians and octogenarians are disease free. Second, although [Schafer and Ferraro \(2012\)](#) examined disease prevalence and incidence across two waves of data, separated by 8 years, we use five waves of HRS data collected at 2-year intervals to permit a more fine-grained analysis of transitions involving disease incidence. This longitudinal approach

enables us to examine trends and the risk of getting a major disease over time. Third, the authors incorporated an extensive list of conditions in their conceptualization of disease free, but some of these conditions are symptoms and often temporary (e.g., teeth problems, constipation). Instead, we focus on leading causes of death. Fourth, rather than adding all types of misfortune into a single variable, we examine specific domains of childhood misfortune to identify which types of misfortune are most detrimental in a parsimonious way. Finally, an oversampling of minority respondents in the HRS allows for in-depth investigation into whether the relationship between childhood misfortune and remaining disease free in later life varies by race and ethnicity. Black adults have higher prevalence of several diseases considered to be leading causes of death and higher mortality rates than White adults ([National Center for Health Statistics, 2016](#)). Evidence regarding health disparities between Hispanic and non-Hispanic Americans is less consistent but merits study nonetheless.

## Theoretical Background

Cumulative inequality (CI) theory informs this study of the early origins of adult health ([Schafer, Ferraro, & Mustillo, 2011](#)), integrating elements of the life course perspective ([Elder, 1998](#)), cumulative disadvantage theory ([Dannefer, 2003](#)), and stress process theories ([Pearlin, 1989](#)). Instead of focusing on proximal factors such as adult socioeconomic status and health behaviors, the theory emphasizes how social stratification manifests over the life course, especially how early disadvantage shapes risks and resources over time ([Ferraro & Shippee, 2009](#); [Ferraro et al., 2016](#)). Early disadvantage may alter health trajectories in several ways, and four elements of the theory are germane for the present study.

First, recognizing the role of family lineage, CI theory draws on the life course perspective and “linked lives” to argue that childhood conditions, including inequality across families of origin, are integral to stratification processes ([Ferraro, Shippee, & Schafer, 2009](#)). Second, the theory emphasizes how inequality diffuses across life domains—“disadvantage in one domain (e.g., family life) may spill over to other domains (e.g., mental and physical health)” ([Ferraro & Shippee, 2009](#), p. 339), resulting in stress proliferation ([Pearlin, 1989](#)). Rather than add all indicators of misfortune into one variable, a formerly common practice, the theory calls for examining multiple domains of misfortune, when such measures are available.

Third, a novel contribution of CI theory is that it emphasizes the plasticity of human development and the ways in which disadvantage does *not* accumulate. Although experiencing misfortune in childhood can be a strong predictor of health problems in later life by influencing continued exposure to stressors and behaviors uncondusive to adult health (e.g., smoking), human agency and resource activation can mitigate the unfavorable consequences of early misfortune ([Ferraro et al., 2009, 2016](#)). The theory cautions about “runaway cumulative disadvantage” and calls for identifying factors that can avert the presumed health consequences.

Finally, cumulative inequality often leads to premature mortality among those most exposed to adversity, creating compositional change in a population that can masquerade as decreasing inequality ([Ferraro & Shippee, 2009](#)). It may be more difficult to detect the potential influence of early misfortune on persons belonging to racial and ethnic minorities because of selective

mortality. Thus, we systematically examine racial and ethnic differences and mortality selection in our analyses.

## Research Questions

The present study is innovative in several ways and addresses the following research questions:

1. *Does childhood misfortune decrease the likelihood of older adults being disease free?* We address this question at the initial survey and across 8 years of longitudinal follow-ups, anticipating that childhood misfortune is a threat to disease-free aging.
2. *Do adult resources and lifestyle factors moderate the relationship between childhood misfortune and being disease free in later life?* We anticipate that the relationship between childhood misfortune and being disease free will be moderated by adult resources (e.g., education), and lifestyle factors (e.g., smoking).

## Method

### Sample

Data were obtained from the HRS—a multistage, probability study of adults aged 51 years and older, with an oversampling of Black and Hispanic adults and Florida residents. Waves 7–11 (2004–2012) were used for this research because a detailed battery of childhood misfortune indicators were first measured on the full sample in 2004.

For this study, respondents were excluded if they were age ineligible, deceased as of 2004, or entered the study in 2010. In addition, those who had proxy responses or fell 2 *SD* below the average cognition score were excluded to preserve the validity of the responses, particularly for the retrospective childhood misfortune variables. These restrictions resulted in a final, analytic sample of 13,921 adults.

### Disease Free

There are different ways to define being *disease free*. One could give particular attention to chronic diseases and life-threatening conditions or could include every potential health condition experienced throughout life. However, the majority of adults aged 50 and older experience an array of minor health symptoms and conditions, yet continue to live long and fairly healthy lives until faced with one of the major causes of death. We focus on those diseases that are both chronic and life threatening. As of 2014, the top chronic disease-related causes of death for those aged 50 and older included heart disease, cancer, chronic lower respiratory disease, cerebrovascular disease, diabetes, Alzheimer's disease, kidney disease, and liver disease ([National Center for Health Statistics, 2015](#)); we include the aforementioned diseases to create our indicators of being disease free at 2004 (prevalence) and developing disease (incidence) from 2004 to 2012.

Respondents were asked if a doctor had ever told them that they have diabetes, lung disease, stroke, heart problems, hypertension, or cancer (excluding skin). Respondents also were asked if they had ever been diagnosed with a “memory-related disease.” In 2010 and 2012, this question was dropped in favor of two questions addressing Alzheimer’s disease and dementia separately. Respondents also were asked if they had other diseases or medical conditions not addressed. We used these responses to gather information about chronic lower respiratory disease, liver disease, and kidney disease. We also included decedents from any of the nine aforementioned diseases using the National Death Index linkage. As specified in axiom 5 of CI theory, failure to account for selective mortality may underestimate disease incidence for the most virulent conditions (i.e., diagnosed with a disease and died between interviews).

For the baseline analyses of *prevalence*, a binary variable was created: one or more diseases (=0) and disease free (=1). Respondents who reported a memory disease prior to 2010 or Alzheimer’s disease or dementia during or after 2010 were coded as 0. Respondents reporting one or more diseases were coded as 0, regardless of missingness on other disease variables. However, respondents reporting no diseases were coded as 1 only if they had valid answers for at least half (four of seven), physician-diagnosed diseases. For the longitudinal analyses of *incidence*, we inverted the coding to be consistent with the widely used concept of disease incidence (0 = disease free; 1 = one or more diseases between 2004 and 2012).

## Childhood Misfortune

Childhood misfortune was operationalized differently in the literature. Instead of adding indicators of misfortune into an overall construct as others have done, we draw from CI theory to identify which domains of childhood misfortune are consequential to remaining disease free (proposition 2.b: [Ferraro & Shippee, 2009](#)). In preliminary inquiry, we used both exploratory and confirmatory factor analyses to finalize the indicators used to specify the domains, eliminating two potential indicators. We categorized misfortunes experienced before the age of 18 into five domains: socioeconomic status (SES), risky parental behaviors, infectious diseases, impairments, and risky adolescent behaviors, using 20 retrospective questions collected between 2004 and 2008. *Socioeconomic status (SES)* misfortune consisted of five indicators: reporting poor or fair finances in relation to others, moving due to finances, having a father with an unskilled labor occupation, mother having less than a high school education, and/or father having less than a high school education. *Risky parental behavior* was assessed by having a parent who smoked, abused substances, and/or was physically abusive. *Infectious disease* included measles, mumps, and/or chicken pox. The presence of a childhood *impairment* included head injury, disability for more than 6 months, learning problems, visual impairment, and/or speech impairment. *Risky adolescent behavior* was assessed by having trouble with police, substance abuse, depressive symptoms, and/or other psychological issues.

Each indicator was coded dichotomously and summed to create a count of misfortune within each domain, top coded at 2 (0, 1, and 2+) to examine a threshold effect of accumulating two or more misfortunes as compared to none. Several domain coding schemes were examined (e.g., dichotomous, top coded at 3+); however, differentiating 0, 1, and 2+ indicators in each domain yielded the most useful information. Multiple imputation by Chained Equations (MICE) was used to handle missing data among childhood misfortune domains with missing exceeding 5%

(SES = 30.19%; risky parental behavior = 20.69%; risky adolescent behavior = 20.21%); observations on other variables that initially had missing were excluded prior to analysis ([Von Hippel, 2007](#)). All variables included in the analysis were used to impute 20 datasets using the “mi impute chained” command in Stata with the appropriate link functions for each variable.

## **Adult Resources and Lifestyle Factors**

Although a challenging childhood can predict health problems for older adults, not everyone who experiences childhood misfortune suffers from poor health in later life. Childhood circumstances can have lasting impacts on adult health by influencing continued exposure to stressors and behaviors un conducive to adult health. Yet, resilience through human agency and resource activation can help to protect against some of these health risks ([Ferraro et al., 2009](#)). For example, smoking cessation has been associated with substantial reduction in risk of all-cause mortality among those with coronary heart disease ([Critchley & Capewell, 2003](#)). Thus, we include both adult resources and lifestyle factors that may modify health risks.

We examined four indicators of adult resources: marital status, education, wealth, and health insurance status. Marital status was coded as never married, married (reference), widowed, and divorced/separated. Education was measured in years and top coded at 17+. Wealth is presented in tens of thousands of dollars and cube rooted to correct for skewness. A four-category indicator of health insurance status was created: no insurance (reference), Medicaid only, Medicare only, and private insurance (private only and/or Medigap plans).

Five indicators of adult health characteristics were included in the models: depressive symptoms, smoking status, heavy drinking, body mass index (BMI), and physical activity. Depressive symptoms were measured using an eight-item Center for Epidemiological Studies–Depression (CES-D) scale. Smoking was assessed by a pack-years variable of cigarette smoking (one pack-year is equivalent to smoking 20 cigarettes daily for 1 year) where never smokers were coded as 0. A heavy drinker was dichotomously coded as having an average of five or more drinks per day for men and four or more drinks per day for women ([Dawson, 2011](#)). BMI was based on self-reports and categorized into underweight or normal weight ( $BMI < 25$ ), overweight ( $25 \geq BMI < 30$ ), and obese ( $BMI \geq 30$ ). A self-reported physical activity scale accounts for both frequency and intensity. Respondents were asked the frequency in which they participated in mild, moderate, and vigorous activity; possible answers included never/rarely (=0), one to three times per month (=1), once per week (=2), two or more times per week (=3), and every day (=4). Drawing from other researchers, the physical activity scale was created by weighting the type of physical activity by intensity (mild = 1.2, moderate = 1.4, and vigorous = 1.8) based on metabolic equivalent recommendations ([Latham & Williams, 2015](#); [Umstadd Meyer, Janke, & Beaujean, 2014](#)). Possible scores ranged from 0 (no physical activity) to 17.6 (mild, moderate, and vigorous physical activity daily).

## **Demographics**

Finally, models adjusted for demographic characteristics reported at baseline including age (in years), gender (female = 1), and race/ethnicity (non-Hispanic white = 0, non-Hispanic black, and

Hispanic). Other races were excluded as the number of cases was insufficient for meaningful comparisons ( $n = 303$ ).

## Analysis

The analysis was conducted in three parts. First, logistic regression was used to estimate the prevalence of being disease free in 2004 in the full sample. Second, Cox proportional hazards models were estimated, using time-on-study with wave (every 2 years) as our time scale, to investigate disease incidence (reporting one or more diseases) between 2004 and 2012 among a more selective, healthy sample—those who were disease free in the prevalence model (2004). The proportional hazards assumption was tested for potential time-varying covariates with no violation encountered, and ties were handled with the [Breslow's \(1974\)](#) method. Finally, a series of theoretically informed interactions were tested to assess the potential moderating effects of adult risks and resources. Models utilized 2004 HRS weights and accounted for the survey design. Analyses were conducted using Stata ST/SE 14.1. Additional information is presented in [Supplementary Material](#).

## Results

### Sample Characteristics

The weighted descriptive statistics for focal variables are presented in [Table 1](#) (see [Supplementary Table S1](#) for descriptive statistics for all variables in the final analyses). At baseline (2004), approximately 35.30% of the sample was disease free. By 2012, only 15.92% of the sample remained disease free. For the childhood misfortune variables, respondents were most likely to report infectious disease (1.74;  $SD = 0.57$ ) and socioeconomic misfortunes (1.45;  $SD = 0.78$ ).

**Table 1.** Select Descriptive Statistics for the Analytical Sample ( $N = 13,921$ )<sup>a</sup>

Variable	Range	Percent Mean (SE)
Disease free (prevalence) <sup>b</sup>		
2004	0, 1	35.30
2006	0, 1	28.02
2008	0, 1	23.02
2010	0, 1	18.81
2012	0, 1	15.92
Childhood misfortune		
Socioeconomic	0–2	1.45 (0.78)
Risky parental behavior	0–2	0.90 (0.67)
Infectious disease	0–2	1.74 (0.57)
Impairments	0–2	0.22 (0.48)
Risky adolescent behavior	0–2	0.29 (0.54)

Note: <sup>a</sup>Weighted values presented. <sup>b</sup>Diseases include cancer, heart disease, hypertension, chronic lower respiratory disease, cerebrovascular disease, diabetes, liver disease, memory disease, and kidney disease.

## Disease-Free Prevalence

[Table 2](#) presents the logistic regression models predicting being disease free in 2004. When adjusting for age, sex, and race in Model 1, respondents who experienced 2+ socioeconomic misfortunes during childhood had 31.2% lower odds of being disease free in 2004, relative to those who experienced no misfortune. Furthermore, respondents who experienced one *or* 2+ risky parental behaviors (odds ratios [ORs] = 0.882, 0.737, respectively), impairments (ORs = 0.863, 0.712), or risky adolescent behaviors (ORs = 0.815, 0.706) also had lower odds of being disease free in 2004 than those who experienced none. As expected, the odds of being disease free decreased with age, and Black adults had about half the odds of being disease free as White adults. We present Model 1 to highlight the relationships between types of childhood misfortune and disease-free status without adjusting for adult resources but urge caution when interpreting the results. Whereas Model 1 omits variables related to being disease free, and omitted variables bias the ORs for the variables that are included in the model ([Mood, 2010](#)), we interpret the ORs recognizing that the estimates are probably biased downward. Estimates from Model 2 will be closer to the true effects.

**Table 2.** Logistic Regression Predicting Prevalence of Being Disease Free at Baseline (2004)<sup>a</sup>

	<b>Model 1</b>		<b>Model 2</b>	
	<b>OR</b>	<b>95% CI</b>	<b>OR</b>	<b>95% CI</b>
<b>Childhood misfortune<sup>b</sup></b>				
Socioeconomic (=1)	0.865	0.717–1.043	0.974	0.784–1.209
Socioeconomic (=2+)	0.688 <sup>†</sup>	0.577–0.820	0.870	0.707–1.069
Risky parent behavior (=1)	0.882*	0.794–0.979	0.908	0.805–1.023
Risky parent behavior (=2+)	0.737 <sup>†</sup>	0.635–0.855	0.791 <sup>‡</sup>	0.667–0.937
Infectious disease (=1)	1.111	0.866–1.427	0.950	0.714–1.264
Infectious disease (=2+)	0.923	0.752–1.132	0.806	0.644–1.009
Impairment (=1)	0.863*	0.772–0.965	0.904	0.787–1.039
Impairment (=2+)	0.712 <sup>‡</sup>	0.564–0.898	0.843	0.637–1.115
Risky adolescent behavior (=1)	0.815 <sup>‡</sup>	0.704–0.943	0.951	0.804–1.127
Risky adolescent behavior (=2+)	0.706*	0.528–0.944	0.935	0.681–1.282
<b>Demographics</b>				
Age (years at baseline)	0.945 <sup>†</sup>	0.940–0.950	0.936 <sup>†</sup>	0.930–0.942
Female <sup>c</sup>	1.053	0.957–1.159	1.085	0.965–1.219
Black <sup>d</sup>	0.504 <sup>†</sup>	0.418–0.608	0.682 <sup>†</sup>	0.558–0.833
Hispanic <sup>d</sup>	0.915	0.780–1.072	1.120	0.901–1.394
<b>Adult resources and lifestyle factors</b>				
Divorced <sup>e</sup>			1.091	0.930–1.280
Widowed <sup>e</sup>			1.229 <sup>‡</sup>	1.077–1.403
Never married <sup>e</sup>			1.165	0.888–1.529
Education (years)			1.001	0.979–1.022
Wealth ( $\sqrt[3]{}$ )			1.004 <sup>†</sup>	1.002–1.005
Medicaid <sup>f</sup>			0.424 <sup>†</sup>	0.309–0.581
Medicare/VA <sup>f</sup>			0.757	0.571–1.003
Private/Medigap <sup>f</sup>			0.739 <sup>‡</sup>	0.578–0.944
Depressive symptoms			0.884 <sup>†</sup>	0.851–0.918
Smoking (pack-years)			0.994 <sup>†</sup>	0.991–0.996
Heavy drinker <sup>g</sup>			1.250	0.914–1.709
25 ≤ BMI < 30 <sup>h</sup>			0.586 <sup>†</sup>	0.523–0.658
BMI ≥ 30 <sup>h</sup>			0.335 <sup>†</sup>	0.289–0.389
Physical activity			1.046 <sup>†</sup>	1.032–1.061
Constant	32.389 <sup>†</sup>	20.877–50.249	74.139 <sup>†</sup>	40.656–135.198
<i>F</i>	51.51 <sup>†</sup>		67.98 <sup>†</sup>	
<i>N</i>	13,824		12,023	

Note: BMI = body mass index; CI = confidence interval; OR = odds ratio.

<sup>a</sup>Analyses weighted to adjust for clustering. <sup>b</sup>Reference group for each of the five domains is no misfortune. <sup>c</sup>Reference group is male. <sup>d</sup>Reference group is non-Hispanic White. <sup>e</sup>Reference group is married. <sup>f</sup>Reference group is no insurance. <sup>g</sup>Reference group is non-heavy drinker. <sup>h</sup>Reference group is BMI < 25.

\* $p < .05$ ; <sup>‡</sup> $p < .01$ , <sup>†</sup> $p < .001$ .

When further adjusting for adult resources and lifestyle factors in Model 2, the magnitude of several relationships between childhood misfortune and being disease free is reduced; the ORs for socioeconomic, impairments, and risky adolescent behavior misfortunes are no longer significant. However, experiencing 2+ risky parental behaviors in childhood was still associated with being disease free in 2004 (OR = 0.791). Age (OR = 0.936) and race (Black; OR = 0.682) were associated with lower odds of being disease free. We used linear probability models to examine potential interactions between race/ethnicity and each of the childhood misfortune domains (not shown) but did not find any evidence that childhood misfortune differentially affects the odds of being disease free by race.

Among adult resources, wealth (OR = 1.004) and being widowed as opposed to married (OR = 1.229) increased one's odds of being disease free. Conversely, older adults with Medicaid exclusively (OR = 0.424) and private and/or Medigap insurance plans (OR = 0.739) were less likely to report being disease free compared with those with no insurance. Depressive symptoms (OR = 0.884), smoking (OR = 0.994), and being overweight (OR = 0.586) or obese (OR = 0.335) decrease the odds, whereas physical activity increases those odds of being disease free (OR = 1.046).

## **Disease Incidence**

[Table 3](#) presents the longitudinal results for disease incidence among those who were disease free in 2004. Cox proportional hazard models are used to predict disease onset by 2012. When adjusting for demographic factors only (Model 1), respondents who experienced 2+ SES or risky adolescent misfortunes in childhood, had 17.1% and 34.1%, respectively, higher hazard of developing disease between 2004 and 2012 compared with those who experienced no misfortune within those domains. In addition, age (hazard ratio [HR] = 1.014) increased the hazard of getting one or more diseases, whereas being female lowered the hazard by 15.6%. Finally, Black and Hispanic adults had 26.3% and 25.4% greater hazard of developing disease than White adults.

**Table 3.** Cox Proportional Hazards Predicting Disease Incidence (2004–2012)<sup>a</sup>

	Model 1		Model 2		Model 3	
	HR	95% CI	HR	95% CI	HR	95% CI
Childhood misfortune <sup>b</sup>						
Socioeconomic (=1)	1.105	0.946–1.292	1.118	0.946–1.323	1.114	0.940–1.322
Socioeconomic (=2+)	1.171*	1.017–1.349	1.110	0.932–1.322	1.107	0.928–1.322
Risky parent b. (=1)	1.046	0.935–1.171	1.057	0.939–1.191	0.977	0.852–1.121
Risky parent b. (=2+)	1.063	0.927–1.217	1.072	0.930–1.235	1.022	0.849–1.230
Infectious disease (=1)	1.081	0.894–1.306	1.164	0.936–1.447	1.162	0.936–1.443
Infectious disease (=2+)	1.048	0.881–1.247	1.075	0.885–1.306	1.077	0.888–1.307
Impairment (=1)	1.015	0.896–1.151	0.962	0.840–1.101	0.968	0.846–1.107
Impairment (=2+)	1.084	0.865–1.358	1.134	0.880–1.462	1.147	0.888–1.480
Risky adolescent b. (=1)	1.058	0.931–1.204	1.049	0.910–1.209	1.049	0.912–1.206
Risky adolescent b. (=2+)	1.341*	1.033–1.740	1.265	0.948–1.687	1.239	0.924–1.663
Demographics						
Age (years at baseline)	1.014 <sup>†</sup>	1.009–1.019	1.017 <sup>†</sup>	1.011–1.022	1.017 <sup>†</sup>	1.011–1.022
Female	0.844 <sup>‡</sup>	0.761–0.935	0.873*	0.785–0.969	0.873*	0.786–0.971
Race/ethnicity (White = rf)						
Black	1.263 <sup>‡</sup>	1.107–1.440	1.137	0.981–1.318	1.132	0.975–1.314
Hispanic	1.254 <sup>‡</sup>	1.089–1.443	1.166	0.990–1.375	1.179*	1.005–1.384
Adult resources and lifestyle						
Marital status (married = rf)						
Divorced			0.925	0.812–1.054	0.929	0.815–1.060
Widowed			1.011	0.873–1.170	1.001	0.866–1.158
Never married			1.097	0.906–1.328	1.102	0.909–1.337
Education (years)			1.012	0.992–1.033	1.014	0.994–1.034
Wealth ( $\sqrt[3]{}$ )			0.998*	0.997–1.000	0.998*	0.997–1.000
Insurance (none = rf)						
Medicaid			1.152	0.839–1.581	1.203	0.891–1.623
Medicare/VA			1.096	0.876–1.371	1.093	0.874–1.366
Private/Medigap			1.022	0.858–1.218	1.023	0.857–1.222
Depressive symptoms			1.059 <sup>†</sup>	1.034–1.084	1.059	1.034–1.084
Smoking (pack-years)			1.000	0.998–1.002	0.996*	0.992–1.000
Heavy drinker			1.365*	1.078–1.729	1.371 <sup>‡</sup>	1.089–1.727
BMI (<25 = rf)						
25 ≤ BMI < 30			1.205 <sup>†</sup>	1.096–1.324	1.206	1.097–1.325
BMI ≥ 30			1.611 <sup>†</sup>	1.441–1.801	1.619	1.448–1.810
Physical activity			0.982*	0.966–0.998	0.982*	0.966–0.998
1 Risky parent × smoking					1.007 <sup>‡</sup>	1.002–1.012
2+ Risky parent × smoking					1.005	0.998–1.012
<i>F</i>	6.96 <sup>†</sup>		16.36 <sup>†</sup>		18.84 <sup>†</sup>	
<i>N</i>	4,101		3,594		3,594	

Note: b. = behavior; BMI = body mass index; CI = confidence interval; HR = hazard ratio; rf = reference group.

<sup>a</sup>Analyses weighted to adjust for clustering. <sup>b</sup>Reference group is no misfortune.

\**p* < .05; <sup>‡</sup>*p* < .01, <sup>†</sup>*p* < .001.

When adjusting for adult resources and lifestyle factors in Model 2, the relationship between domains of childhood misfortune and disease incidence attenuated, such that the HRs for 2+ SES and risky adolescent misfortunes are no longer significant. As in Model 1, both age (HR = 1.017) and being female (HR = 0.873) were associated with disease incidence, but Black and Hispanic adults no longer differed from White adults. We tested interaction terms for race and childhood misfortune domains, but none were significant (not shown). Depressive symptoms (HR = 1.059), heavy drinking (HR = 1.365), and being overweight (HR = 1.205) or obese (HR = 1.611) as compared to normal weight increased the risk of incident disease; however, more wealth (HR = 0.998) and physical activity (HR = 0.982) decreased the risk of disease.

## Tests of Moderation

To address our second research question, we estimated a series of models with product terms to assess the moderating effects of adult resources and lifestyle factors on the relationship between childhood misfortune and disease incidence. None of the product terms were significant in the fully adjusted prevalence model. The process was repeated for the incidence analyses, where we identified one significant interaction. As shown in Model 3 of [Table 3](#), among those who were disease free in 2004, disease incidence by 2012 was higher for persons with one risky parental behavior and a high level of pack-years smoking (HR = 1.007). Most HRs remained stable from Model 2 except for the effect for Hispanic adults (HR = 1.179,  $p < .05$ ).

Finding that one risky parental behavior was significant, we subsequently tested product terms between each of the three indicators of risky parental behavior and pack-years. Analyses showed that parental smoking was the source of the interaction. Specifically, having a parent who smoked, in combination with the number of pack-years smoked in adulthood, increased the risk of disease onset by 2012 (HR = 1.006,  $p < .01$ ; not shown).

## Discussion

This research contributes to the literature on the early origins of adult health by identifying types of childhood misfortune associated with being free of any disease during middle and later life. Our first research question asked whether experiencing childhood misfortune decreased the odds of being disease free in later life. Results from logistic regression of baseline data revealed that experiencing childhood misfortune from several domains—socioeconomic, risky parental behavior, impairments, and/or risky adolescent behavior—reduces the odds of being disease free in later life. Even when adjusting for adult behaviors and resources, experiencing 2+ risky parental behaviors reduced the odds of being disease free.

These findings add to the literature demonstrating the long-term influence of early misfortune. Growing up in a risky household, experiencing poor socioeconomic conditions, and/or exhibiting risky behavior in adolescence are the types of childhood misfortune most consequential to being disease free. This is consistent with conclusions of prior studies examining selected diseases among HRS respondents ([O’Rand & Hamil-Luker, 2005](#)) as well as those using the MIDUS ([Schafer & Ferraro, 2012](#)), Wisconsin Longitudinal Study ([Springer, 2009](#)), and Adverse

Childhood Experiences (ACE) Study ([Brown et al., 2010](#); [Felitti et al., 1998](#)). This study extends the early origins of health literature by identifying types of childhood misfortune that reduce the likelihood of being disease free.

CI theory specifies pathways by which disadvantage may or may not accumulate, highlighting how the consequences of early insults are partly contingent on resource activation. Prior to including adult resources and lifestyles in our models, many domains of misfortune were negatively associated with being disease free. However, several of these findings were attenuated after including these adult factors, highlighting the importance of resources and human agency in shaping health trajectories. For instance, remaining physically active increases the likelihood of being disease free at baseline and remaining so over time, consistent with work by others ([Barengo, Antikainen, Borodulin, Harald, & Jousilahti, 2017](#)), despite experiencing considerable childhood misfortune. In addition, SES resources are central to overcoming the anticipated deleterious sequelae of early misfortune. In both the prevalence and incidence models, we observed that childhood SES misfortune was related to disease-free status but that these associations were no longer significant after adjusting for adult wealth. This suggests that some of the negative health consequences associated with financial strain or poverty during childhood can be offset by sufficient socioeconomic assets during adulthood.

Our second research question asked if and to what extent adult lifestyle and resources moderate the relationship between childhood misfortune and developing disease over time. Our analyses revealed an interaction associated within the risky parental behavior domain: having a parent who smoked during one's childhood—in combination with the number of pack-years smoked in adulthood—significantly increased the risk of developing disease over time. To our knowledge, no previous study of the early origins of adult health reports such an interaction. To illustrate the dose–response relationship when a parent smoked during one's childhood, the risk of getting more or more disease rises 12% between a 40 pack-year smoker and a 60 pack-year smoker (i.e., 10 additional cigarettes daily for 40 years).

This finding shines a light on the intergenerational and life course implications of tobacco consumption. Parental smoking may influence children's health through environmental exposure to tobacco smoke (i.e., second-hand smoke), by modeling a lifestyle of tobacco use, and perhaps biologically by inducing symptoms of early nicotine dependence in the womb ([Alberg & Korte, 2014](#)). Any sustained exposure to tobacco smoke is harmful, but our analyses show the potency of *lifelong exposure* to smoking. Some mothers and fathers stop smoking during pregnancy, which is certainly beneficial, yet smoking resumption among parents after birth remains a serious threat to their progeny's likelihood of disease-free aging.

In addition to the general influence of one's upbringing, the imprint of family lineage is clear (proposition 1.c, [Ferraro & Shippee, 2009](#)): risky parental behaviors have long-term consequences on the health of the next generation. Although future research may be able to probe more carefully how much of the family lineage effect is due to genetic and environmental influence, CI theory and the results of this study call for more attention to family lineage as a core element of life course epidemiology.

In addition to our main findings, we briefly discuss those pertaining to gender and race. Gender did not predict being disease free at baseline, but predicted disease incidence with women less likely to develop disease. This finding stands in contrast to much of the literature that reports women are more likely to develop chronic diseases ([Rieker & Bird, 2005](#)). Although men and women suffer from similar diseases in later life, women live longer lives and more likely to suffer from more chronic, *nonlife-threatening* diseases (i.e., arthritis) that are not accounted for in our measure.

Black adults had significantly lower odds of being disease free in 2004, even in the fully adjusted model. These prevalence results are consistent with much of the disparities literature showing that stark Black–White racial inequalities develop early in the life course, which may be understated in studies of older adults because of early-life mortality selection ([Ferraro, Kemp, & Williams, 2017](#); [Woolf & Braveman, 2011](#)). By contrast, although both Black and Hispanic adults had higher risk of developing disease by 2012, these effects became nonsignificant after accounting for adult resources and lifestyle factors—confirming that these factors can ameliorate or exacerbate racial disparities, even during later life.

These findings should be interpreted with limitations in mind. First, childhood misfortune was measured using retrospective data, which may involve recall bias. We attempted to reduce the effect of this bias by adjusting for depressive symptoms and socioeconomic resources as recommended by [Vuolo, Ferraro, Morton, and Yang \(2014\)](#). In addition, we restricted the sample to adults who fell within 2 *SD* of the mean cognition score. Nevertheless, results should be interpreted with care.

Second, interpretation of the moderation findings regarding risky parental behaviors, particularly smoking, is limited because of the crudeness of the measure. Although it taps exposure to family tobacco use during childhood, there is no information on duration or quantity of smoking. Future studies that capture more detailed information on parental smoking may help identify the processes at work, including potential mediation.

Third, self-reports of a number of variables used here can be limiting. For example, in a review of physical activity measures, [Prince and colleagues \(2008\)](#) found that correlations between self-report and direct measures of physical activity were only low to moderate, creating potential issues of reliability. Furthermore, self-reported, physician-diagnosed disease may underestimate prevalence and incidence due to limited access to medical care, particularly for uninsured and minority adults. Although we adjusted for insurance status and wealth, these three preceding limitations probably lead to a conservative bias, underestimating the influence of early origins on adult health.

Finally, studies of disease-free aging have to grapple with the issue of which diseases should be included in one's definition of this highly favorable health status. Although some include an exhaustive list of diseases and symptoms (see [Schafer & Ferraro, 2012](#)), we chose to focus on the leading causes of death in the United States. Empirical generalizations across studies need to consider the types of conditions examined.

## Conclusion

[Kuh and Ben-Shlomo \(2016\)](#) call for more “study of common risk and protective factors” (p. 117). Knowledge about risk factors for specific diseases is vitally important; yet, the public is often confused by conflicting reports about health practices derived from “single-disease epidemiology.” Consequently, identifying risk factors associated with *any* of the leading causes of death is important for fundamental scientific and public understanding of health. Our findings show that risky parental behaviors, especially smoking, have *long-term health consequences on their children*. Perhaps more attention on the intergenerational transmission of health risks would aid smoking cessation programs.

This study extends the findings of [Schafer and Ferraro \(2012\)](#), demonstrating the early origins of remaining disease-free in middle and later life. Family upbringing characterized by SES misfortune and risky parental behaviors place notable long-term health risks on the children. One might expect that the effect of childhood misfortune on disease onset would be limited to early and middle adulthood, but these results show elevated risk decades after the insults. We do not claim that avoiding a noxious home environment guarantees that children will be able to escape disease altogether, but avoiding or even delaying these killer diseases begins long before symptoms are identified. Our findings may seem disheartening; yet, it is valuable to remember that the trajectories set into motion by experiencing early adversity are not determined for life. Engaging in regular physical activity, abstaining from—or even reducing—tobacco consumption, and accumulating socioeconomic resources may help to interrupt or counteract the likely sequelae of childhood misfortune.

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**Conflict of Interest** None reported.

**Supplementary Material follows the References**

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**Table S1.** Descriptive Statistics for the Analytical Sample (N=13,921)<sup>a</sup>

Variable	Range	Percent	Mean (SE)
Disease Free (Prevalence) <sup>b</sup>			
2004	0,1	35.30	
2006	0,1	28.02	
2008	0,1	23.02	
2010	0,1	18.81	
2012	0,1	15.92	
Childhood Misfortune			
Socioeconomic	0-2		1.45 (0.78)
Risky parental behavior	0-2		0.90 (0.67)
Infectious disease	0-2		1.74 (0.57)
Impairments	0-2		0.22 (0.48)
Risky adolescent behavior	0-2		0.29 (0.54)
Demographics			
Age (years at baseline)	50-97		63.10 (9.64)
Female	0,1	56.90	
Race/ethnicity			
White (reference)	0,1	84.34	
Black	0,1	8.84	
Hispanic	0,1	6.83	
Marital Status			
Married (reference)	0,1	67.97	

Never married	0,1	3.91	
Divorced/separated	0,1	14.25	
Widowed	0,1	13.87	
Education (years)	0-17		13.04 (2.92)
Wealth ( $\sqrt[3]{}$ in \$1,000s)	-13.10-33.33		5.98 (3.65)
Health Insurance			
None (reference)	0,1	7.41	
Medicaid only	0,1	4.79	
Medicare only	0,1	13.86	
Private/supplemental	0,1	73.93	
Adult Lifestyle Factors			
Depressive symptoms	0-8		1.36 (1.92)
Smoking (pack-years)	0-287		15.74 (25.59)
Heavy drinker	0,1	0.03	
BMI			
BMI<25 (reference)	0,1	31.56	
25≤BMI<30	0,1	39.16	
BMI≥30	0,1	29.29	
Physical Activity	0-17.6		8.15 (3.87)

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<sup>a</sup>Weighted values presented. <sup>b</sup>Diseases include cancer, heart disease, hypertension, chronic lower respiratory disease, cerebrovascular disease, diabetes, liver disease, memory disease, and kidney disease.

## SUPPLEMENTARY MATERIAL

### Analytic Strategy

Cox proportional hazards models (Cox, 1972) were estimated to investigate disease incidence (reporting one or more diseases) between 2004 and 2012 among those who were disease free in 2004. This cox regression model expresses the logarithm of the risk of getting one or more diseases over this eight-year period as a linear function of the predictors. The effect of a predictor is expressed as a “hazard ratio,” which can be viewed as a relative risk. Hazard ratios below 1 reflect a decreased risk, hazard ratios above one reflect an increased risk. We use time-on-study as our time scale (with waves occurring every 2 years), which Pencina, Larson, & D'Agostino (2007), cited as being both the most commonly used method in survival analyses as well being more reliable than unadjusted age-scale models.

Among the advantages of using a Cox model is that it does not make any assumptions about the shape of the survival distribution. Given that this study examines older adults, an appropriate underlying distribution is not known. Another advantage is allowing independent variables to vary over time. Although satisfying the proportionality assumption may be a concern, it can be tested and the model can be modified to allow for non-proportionality (Allison, 2014). For this study, the proportional hazards assumption was tested for potential time-varying covariates with no violation encountered and ties were handled with the Breslow method (1974).

### Sensitivity Analyses

To check the robustness of our results, we conducted multiple sensitivity analyses. First, we used different coding schemes of physical activity from the approach specified herein but

reached similar conclusions. These include: (1) sedentary (3x/month or less) versus active (weekly or more), (2) Frequency ONLY as presented, and (3) Intensity ONLY as a scale.

Second, we conducted sensitivity analyses setting the thresholds to 0, 1, 2, 3+ for each of the childhood misfortune domains. Conclusions from baseline analyses were very similar: 2 risky parental behaviors decreased the odds of being disease free in 2004 (OR=0.807, 95% CI=0.670-0.971,  $p<.01$ ). In addition, 3+ risky parental behaviors were marginally significant in predicting being disease free (OR=0.708, SE=0.121,  $p=0.05$ ). For longitudinal analysis, we set thresholds to 0, 1, 2, 3+ for each of the CM domains, except risky adolescent behaviors because there were too few cases with 3 or 4 misfortunes to successfully estimate the models (11 total cases). Again, the conclusions did not change substantively; 2 SES misfortunes increased the likelihood of getting one or more diseases (HR=1.213, SE=0.103,  $p<0.05$ ) and we observed a significant interaction (HR=1.007, SE=0.002,  $p<0.01$ ) between risky parental behaviors and respondent pack-years (parental smoking\*respondent smoking).

Third, we tested for additional interactions (product terms). We first examined whether the influence of childhood misfortune on disease free status differed for men and women; and subsequently examined parallel interactions by race and ethnicity. There was no evidence that the effect of childhood misfortune on disease free status varied across these status characteristics.