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Early Origins of Adult Cancer Risk Among Men and Women: Influence of Childhood Misfortune?

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Abstract

Objective—To examine the effect of five childhood misfortune domains—parental behavior, socioeconomic status, infectious diseases, chronic diseases, and impairments—on all-site and selected site-specific cancer prevalence and all-site cancer incidence.

Method—Panel data from the Health and Retirement Study (2004–2012) were used to investigate cancer risk among adults above the age of 50.

Results—Risky parental behavior and impairment in childhood were associated with higher odds of all-site cancer prevalence, and childhood chronic disease was associated with prostate cancer, even after adjusting for adult health and socioeconomic factors. Moreover, having one infectious disease in childhood lowered the odds of colon cancer. Cancer trends varied by race and ethnicity, most notably, higher prostate cancer prevalence among Black men and lower all-site cancer among Hispanic adults.

Discussion—These findings underscore the importance of examining multiple domains of misfortune because the type and amount of misfortune influence cancer risk in different ways.

Keywords

childhood disadvantage; breast/prostate/colon cancer; cumulative inequality theory; race

Cancer is a major public health concern, responsible for one in four deaths in the United States (Siegel, Miller, & Jemal, 2015). In the near future, cancer is expected to surpass heart disease as the leading cause of death (Siegel et al., 2015). Due to prevention, early detection, treatment, and changing health behaviors, overall cancer incidence and mortality rates began to decline in 1992; however, some site-specific rates, such as thyroid and liver, have

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Declaration of Conflicting Interests

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increased in recent years (Edwards et al., 2014; Siegel et al., 2015; Smith, Smith, Hurria, Hortobagyi, & Buchholz, 2009). Using data from the Census Bureau and the Surveillance Epidemiology and End Result (SEER), Smith et al. (2009) projected that older adults and racial/ethnic minorities will experience increases in cancer diagnoses, warning that the anticipated increases will be driven by some of the most deadly cancers.

The development of cancer involves an accumulation of molecular changes that eventually spark tumor growth. Thus, as a complex process, distal and cumulative insults are important to map in attempting to understand the origins and progression of cancer. Although research has paid close attention to the carcinogenic effects of some cumulative insults such as smoking and dietary habits, research on the early origins of cancer is somewhat underdeveloped.

The purpose of this manuscript is to systematically examine one set of distal influences—misfortune during childhood—as well as adult lifestyle and behavior on all-site and site-specific cancer prevalence and all-site incidence. Attention is also given to sex, race, and ethnic differences in how early insults may be associated with cancer occurrence. Guided by cumulative inequality (CI) theory, this study uses data from the Health and Retirement Study (HRS) to examine the effect of five different domains of childhood misfortune on cancer risk. We begin by reviewing evidence on the early origins of cancer, then draw on CI theory to formulate research questions to advance the literature.

Evidence on the Early Origins of Cancer

Although most cancer epidemiology research is focused on understanding the influence of proximal risk factors such as environmental pollution, smoking, and diet, scholars have investigated aspects of the *social environment*, including life events and experiences, as potential cancer risk factors. Life course epidemiologists emphasize the importance of considering early-life events and experiences that shape health in later life (Ben-Shlomo & Kuh, 2002). Childhood is a time of physiological, psychological, and social development; events and experiences during this sensitive time may lay the foundation of later-life health. In addition, experiencing misfortune in childhood could lead to further misfortune, constructing an early trajectory of accumulated insults with long-reaching effects (Ferraro & Shippee, 2009). Indeed, a burgeoning literature has emerged linking childhood events and experiences to later-life health behaviors, morbidity, and mortality (e.g., Beebe-Dimmer et al., 2004; Felitti et al., 1998).

Studies relating childhood misfortune to adult cancer are limited, and findings are inconsistent. Although some scholars have found that parental abuse (Morton, Schafer, & Ferraro, 2012), illness (Blackwell, Hayward, & Crimmins, 2001), and low socioeconomic status (SES; de Kok et al., 2008) in childhood increase cancer risk in adulthood, others report no association (Korpimäki, Sumanen, Sillanmäki, & Mattila, 2010; McCrory, Dooley, Layte, & Kenny, 2014).

The measurement of childhood misfortune or analytic strategy used may partially explain the mixed findings. Scholars have operationalized childhood misfortune in various ways,

ranging from examining specific types of misfortune (e.g., low parental SES, abuse) to a sum of indicators. Often, only one type of misfortune is investigated (e.g., Fuller-Thomson & Brennenstuhl, 2009). Not adjusting for other types of childhood misfortune could bias estimates as childhood misfortunes may be interrelated and/or related to adult characteristics. For instance, SES disadvantage in childhood has been associated with lower overall ratings of child health, impairments, and limitations in school (Chen, Martin, & Matthews, 2006). However, if other childhood misfortune domains were not included in the model, the effect of childhood SES misfortune would likely be overestimated (Preston & Taubman, 1994). In addition, researchers have found that poor childhood health is associated with lower educational attainment in adulthood (Case, Fertig, & Paxson, 2005; Haas, 2006). Other childhood misfortune domains such as abuse have been associated with higher odds of risky health behaviors such as smoking during adulthood (Felitti et al., 1998). Thus, failing to include childhood misfortune domains may lead to overestimating the effect of adult characteristics on later-life health. Moreover, whereas a count variable of childhood misfortune is telling, a certain type of misfortune may be the driving force influencing later-life health.

With few exceptions (e.g., D. W. Brown et al., 2010; de Kok et al., 2008), most studies investigate the early antecedents of all-site rather than site-specific cancer risk. This is useful, but the etiology and risk factors associated with cancer vary by site. For instance, smoking, a risky adult health behavior, has been strongly linked to some types of cancer such as lung cancer, but not others such as breast and prostate (Surgeon General of the United States, 2014). Thus, it is possible that childhood misfortune domains behave similarly and influence certain cancers. Also, many studies explore cancer prevalence as an outcome rather than cancer incidence. Although these studies are informative, investigating new diagnoses over time may offer additional insight.

Given that Black children may be more likely to experience some forms of misfortune, such as low SES (Flores, Olson, & Tomany-Korman, 2005; Hussey, Chang, & Kotch, 2006), and that Black adults have greater cancer incidence and mortality rates than White adults (Siegel, DeSantis, & Jemal, 2014; Ward et al., 2004), it is essential to account for racial differences when studying the link between childhood misfortune and cancer. Cancer rates also vary by ethnicity (Siegel et al., 2014; Ward et al., 2004). Hispanic adults generally have better health and lower mortality rates than White adults, a phenomenon referred to as the Hispanic paradox (Markides & Coreil, 1986). Consistent with the paradox, most researchers report lower cancer incidence and death rates of all cancer sites among Hispanic adults compared with non-Hispanic White adults (Lariscy, Hummer, & Hayward, 2015; Siegel et al., 2015; Ward et al., 2004). In addition, Hispanic adults generally have higher incidence of cancers related to infectious agents (Siegel et al., 2015), suggesting that there may be ethnic differences in vulnerability to particular risk factors. Few studies, however, have examined the effect of childhood misfortune on adult cancer risk in a diverse sample of the U.S. population.

Theory

The diversity of human experience means that some people are more likely to face negative events and experiences. The accumulation of negative exposures underlies many theses and theories of aging, health, and survival. Given that single exposures to carcinogens or stress rarely give rise to tumor growth, a life course perspective is particularly important for cancer research. Damage associated with negative exposures generally accumulates over a long period of time before malignancy is manifest; thus, the emphasis of many studies on proximal risk factors may be misplaced.

The life course perspective has been used extensively in many disciplines to help frame life transitions and processes over time in a given context (Elder, 1998). Life course epidemiology, for instance, has focused on the timing of exposures and accumulation of risk, which shape health trajectories (Ben-Shlomo & Kuh, 2002). Indeed, many theoretical frameworks have been proposed to explain how experience “gets under the skin” on an individual level (e.g., Hertzman & Boyce, 2010; O’Rand, 1996; Pearlin, Menaghan, Lieberman, & Mullan, 1981). Among them, CI theory integrates demographic and development processes over the life course, privileging the timing of events situated in historical time (Ferraro & Shippee, 2009).

As a middle-range theory, CI theory offers a framework for understanding the dynamic exchange of forces between social structure and human agency in molding health trajectories. Children often have little control over their situations and rely on their parents for direct or indirect resources and support; therefore, the pressure of social inequality may be more pronounced in childhood, a time when autonomy is limited. This does not assume that human agency and resources should be disregarded. On the contrary, CI theory holds that agency and resources may alter health trajectories (Ferraro & Shippee, 2009). Linking micro- and macro-level processes within a life course framework—biography and history—aids interpretation of the influence of social structure, resources, and agency.

Framed from a life course perspective, CI theory holds that childhood is a sensitive time of development when exposures to insults may have long-term and latent health effects (Ferraro & Shippee, 2009). In addition, misfortune during childhood may set an individual on a health trajectory of amassing damaging residue of exposures over time, increasing the likelihood of morbidity and premature mortality. Insults may accumulate during childhood and initiate other systemic and agentic processes further stacking the odds against healthy aging (Schafer, Ferraro, & Mustillo, 2011). Especially, for persons who are impoverished or members of minority groups, the accumulation of negative exposures may be substantial. It is possible that childhood misfortune alters physiological systems or responses, socialization, behavior, and emotional vulnerability, as well as life course perceptions (Schafer et al., 2011). Scholars have found evidence that smoking and SES mediate the relationship between childhood misfortune and cancer in adulthood (D. W. Brown et al., 2010; de Kok et al., 2008, respectively). Although these mechanisms are important, a better understanding is still needed concerning what domains of misfortune are most influential on types of cancer, by what magnitude, and for whom.

CI theory also specifies that the magnitude of misfortune is related to health and social functioning (Ferraro & Morton, 2016). Evidence for a dose-response effect of misfortune has been noted, where cancer odds increase for each additional childhood misfortune reported (D. W. Brown et al., 2010; Felitti et al., 1998; Kelly-Irving et al., 2013). However, the type of misfortune and response may not be comparable. For instance, children experiencing sexual abuse by a parent and those living with someone with a mental illness likely present different challenges which, in turn, elicit distinct responses. Thus, a logical next step for research on the early origins of adult health is to examine accumulated misfortune within and across domains (e.g., childhood SES and illness).

Whereas many studies have investigated later-life health outcomes of noxious childhood experiences, often referred to as childhood adversity, we use childhood misfortune to represent a more encompassing array of insults that include poor health and low SES in childhood. Different types of misfortune have been described more broadly by others as chronic and acute stressors, which may operate differently across the life course (Pearlin et al., 1981). Indeed, some studies have found that chronic strains increase chances of cancer in adulthood. Blackwell et al. (2010), for instance, found that non-infectious childhood diseases were associated with higher odds of adult cancer, net of childhood SES, and adult health indicators. Therefore, in addition to the more acute childhood adversity that others have examined (e.g., Felitti et al., 1998), we also consider chronic strains experienced during childhood.

CI theory states that the relationship between misfortune and health risk may not be linear (Ferraro & Shippee, 2009). Similar to the concept of acquired immunity, some misfortune may be protective relative to no misfortune. Seery, Holman, and Silver (2010) found a “U”-shaped relationship between cumulative lifetime adversity and select health indicators. In their study, better mental health was reported by those experiencing some adversity than those reporting no adversity. People who have not experienced any adversity may lack the physiological, psychological, or social resources to cope with or manage the insult.

Although others have investigated one type of insult (e.g., Blackwell et al., 2010) or additive misfortune (e.g., Felitti et al., 1998), relatively few have explored domains of misfortune. Drawing from CI theory and using a nationally representative sample, we consider the influence of five domains of childhood misfortune as well as adult resources and lifestyle on all-site cancer prevalence and incidence as well as colon, breast, and prostate cancer prevalence among older adults from 2004 to 2012. Our primary research questions are twofold.

Research Question 1: Which domains of childhood misfortune increase risk of cancer prevalence and incidence among men and women?

Research Question 2: Owing to racial and ethnic differences in exposure to negative events and experiences, does the effect of childhood misfortune on cancer risk vary by race or ethnicity?

Method

Sample

This study uses data from Waves 7 to 11 (2004–2012) of the HRS, a multistage area probability panel study of adults 51 years of age and older. The HRS began surveying in 1992 and conducts follow-up interviews every 2 years (HRS, 2015). Black adults, Hispanic adults, and residents in the state of Florida are oversampled, and new cohorts are added every 6 years, making the HRS the largest and most representative panel study of older adults in the United States. Response rates have been 85% or greater since 2004 (baseline for this study; Sonnega et al., 2014). The present study analyzes data from 13,921 men and women.[1]

Cancer

Respondents were asked, “Has a doctor ever told you that you have cancer or a malignant tumor, excluding minor skin cancer?” Information from each wave was carried forward to the next wave, at which time respondents were able to dispute their last wave record (Fisher, Faul, Weir, & Wallace, 2005). Because we are interested in prevalence and first diagnosis of cancer, following suggestions from HRS documentation (Fisher et al., 2005), we coded all-site cancer prevalence in 2004 as 1 for respondents who reported *yes* in 2004, disputed their previous wave record and did have cancer, or previously reported being diagnosed with cancer, 0 otherwise.

Respondents also were asked which part of the body the cancer started. The most common types of cancer reported in the sample, which are also among the most common in the U.S. population (Edwards et al., 2014; Siegel et al., 2015), were colon (146 individuals), breast (299 women), and prostate (333 men). Lung cancer (39 individuals) was examined; however, bivariate analysis revealed low cell counts for our independent variables of interest and lung cancer and wide confidence intervals for childhood misfortune in preliminary regressions. Thus, variables indicating colon, breast, and prostate cancer prevalence in 2004 were created and coded 1 if the respondent reported cancer originating from that location (e.g., colon) during or before 2004, and 0 otherwise. (Whereas only one man reported breast cancer, we limited the breast cancer analysis to women.)

All-site cancer incidence from 2004 to 2012 was created in a similar fashion as all-site prevalence for each wave. Cancer decedents were included using the national death index linkage, adding 114 respondents who had cancer at the time of death, but had not reported it to the HRS. The responses in 2004 are included in incidence if the respondent reported cancer diagnosis after their interview date in 2004. Duration in the incidence window was measured in months. Respondents who reported a year, but not month of cancer diagnosis were set to midway (June) of that year (91 cases, 0.75%). If there was no year or month of diagnosis, midway of the wave prior to first affirmative report of cancer (December of the

[1]Inclusion criteria for the sample began with respondents who were age-eligible in 2004. Next, we excluded respondents whose childhood information was collected from proxies, as they may not be able to answer retrospective questions regarding childhood. Finally, we excluded respondents whose mean total cognition score was below 2 standard deviations to increase reliability and validity given our use of retrospective measures for key variables.

year before) was set as the diagnosis month (37 cases, 0.30%). These same rules were used for censored cases if the month (19 cases, 0.16%) or month and year of last interview (120 cases, 0.98%) were unknown. There were too few cases that were diagnosed with cancer between 2004 and 2012 to investigate cancer incidence of any particular site.

Childhood Misfortune

The independent variables of interest include five domains of childhood misfortune—parental behavior, SES, infectious diseases, chronic diseases, and impairments. Each domain is comprised of multiple indicators and each indicator is coded as 1 if the respondent reported the condition or event (0 otherwise). The five domains were created as count variables, informed by CI theory, previous studies (Felitti et al., 1998; Morton et al., 2012), correlation matrices, and polychoric factor analysis. The domains were top coded at 2. Less than 2% of respondents reported three or more childhood chronic diseases, risky parental behaviors, or impairments. Larger percentages were top coded for SES (46%) and infectious diseases (52%). The domains were treated as categorical variables with 0 as the reference.

The parental behavior domain has three indicators. Respondents were asked if they were physically abused by a parent, a parent had an alcohol or substance abuse issue, or a parent or guardian smoked before they were age 18. Five indicators were used for childhood SES (before the age of 16): mother's education, father's education, perception of family finances, moved due to financial situation, and father's occupation. Mother's and father's education were each coded as 1 for less than 12 years of education and 0 for 12 or more years. Perception of family finances was coded 1 for poor, 0 otherwise. Using the U.S. Labor Statistics occupation categories as reference, father's occupation was grouped into non-skilled manual occupations coded as 1, and all other occupations coded as 0 (U.S. Bureau of Labor Statistics, 2010).

Infectious diseases during childhood include three indicators: chicken pox, measles, and mumps before the age of 16. Chronic diseases before the age of 16 include asthma, diabetes, respiratory disorder, allergies, heart disease, ear problems, seizures, migraines, stomach problems, high blood pressure, and self-rated childhood health; the latter was dichotomized so that poor or fair health was 1, 0 otherwise (Lee et al., 2007). The childhood impairment domain includes five indicators: learning problems, speech impairment, vision impairment even with corrective lenses, disability for 6 months or more, and a head injury or trauma that required medical attention before age 16.

Covariates

Models were adjusted for demographic characteristics, adult resources, and adult lifestyles. Adult SES and health behaviors were included to adjust for social structure influences and expressions of agency that could affect health in later life. Age was measured in years. Following others who have investigated racial and ethnic health variation in the HRS (Heisler et al., 2007), non-Hispanic Black adults and Hispanic adults of any race (26 respondents were Hispanic Black adults and coded as Hispanic adults) were treated as dummy variables with non-Hispanic White as the reference category.

Two indicators of adult SES were used: years of education (0–17) and wealth (total assets minus debts). Because wealth was skewed, the cube root in thousands of dollars was used (Tukey, 1977). Because marriage is beneficial to health, a dummy variable for married was included (Umberson, 1992). Depressive symptoms was measured with an eight-item Center for Epidemiological Studies-Depression (CES-D) scale of experiencing each of the following in the past week: everything was an effort, restless sleep, felt happy, felt lonely, enjoyed life, felt sad, could not get going, had a lot of energy. This variable is a count of the eight items and treated as continuous. Including indicators of mental health may help adjust for recall bias (Vuolo, Ferraro, Morton, & Yang, 2014).

Models also adjust for health behaviors and lifestyle that have been associated with cancer (Bagnardi et al., 2012; Stein & Colditz, 2004). Body mass index (BMI) was measured in kg/m^2 and bottom coded and top coded at 15 and 50, respectively.[2] Heavy alcohol consumption was coded 1 for men who drink an average of five or more drinks per day when they drink or women who drink on average four or more drinks per day when they drink, 0 otherwise (Dawson, 2011). A pack-years variable of cigarette smoking at baseline was created with respondents who never smoked coded as 0.[3]

Analytic Plan

Analyses were conducted in two parts using Stata 14. First, logistic regression was used to estimate all-site and site-specific cancer prevalence in 2004. Next, cancer incidence from 2004 to 2012 was investigated using Cox proportional hazard (PH) models. Using the episode-splitting technique outlined by Allison (2014), the data were reshaped to long format to test the PH assumption of time-varying covariates. Responses were carried forward in each month to the next wave (Allison, 2014). Schoenfeld residuals were used to check the PHs assumption; there were no violations. The Breslow method was used to handle ties (Breslow, 1974). Models predicting cancer incidence were estimated with and without the cancer decedents; the results reported are on the sample that includes cancer decedents. All models were weighted with 2004 HRS weights and adjusted for clustering.

Results

Whereas some cancers are specific to the site and tissue, we present sex-stratified descriptive statistics in Table 1 (Siegel et al., 2015). There are 8,390 women and 5,531 men in the sample; 9.18% of women and 7.21% of men in the sample are Black, 6.58% of women and 6% of men are Hispanic, and 81.73% of women and 84.07% of men are White. Table 1 reveals that 10.62% of men and 11.32% of women had cancer in 2004. Among those with cancer, 75 women and 71 men had colon cancer, 298 women had breast cancer, and 333 men had prostate cancer. Of those who had never been diagnosed by their 2004 interview, approximately 9.32% of women and 12.09% of men reported cancer by 2012.

[2]There were eight individuals who had a BMI of 15 or less and 57 individuals who had a BMI of 50 or greater.

[3]Among smokers, respondents reported when they began smoking and when they quit (unless they were current smokers in 2004) as well as how much they smoked daily, on average. From this information, we calculated the total years each respondent smoked (Year Stopped – Year Started for Former Smokers; 2004 – Year Started for Current Smokers). Next, we multiplied the total years smoked by the average number of cigarettes smoked daily and divided by 20 (amount of cigarettes in a pack). This variable was top coded at 287.

Table 2 shows the frequency and percentage of 0, 1, or 2+ misfortunes in each domain by sex. Within each domain, the majority of respondents reported two or more SES, two or more infectious, one risky parental behavior, no chronic, and no impairment misfortune. Women were somewhat more likely than men to report two or more SES, infectious, and chronic disease misfortune. Men were somewhat more likely than women to report two or more impairments and one or more risky parental behaviors.

All-Site Cancer Prevalence and Incidence

Table 3 shows the logistic regression predicting all-site cancer prevalence in 2004 and the Cox PH model predicting all-site cancer incidence between 2004 and 2012. Respondents who experienced 2+ risky parental behaviors had 27.8% higher odds of cancer in 2004 relative to those who experienced none, even after adjusting for other childhood circumstances, adult resources, and lifestyle factors ($p < .05$). In addition, 2+ impairments were associated with 46.35% higher odds of all-site cancer prevalence ($p < .05$). As for the control variables, the odds of cancer prevalence were higher with age (odds ratio [OR] = 1.05; $p < .001$) and wealth (OR = 1.04; $p < .01$). For each additional pack per year smoked, the odds of all-site cancer increased 0.5% ($p < .001$). Hispanic adults were 34.88% less likely than White adults to have any cancer in 2004 ($p < .05$). Race, ethnicity, and sex were interacted with each of the childhood misfortune domains to test whether these characteristics moderated the effect of childhood misfortune on cancer prevalence; yet, none of the interaction terms were significant ($p > .05$; findings not shown).

To investigate new cancer cases, those who had cancer in 2004 or before were excluded from the incidence sample. As seen in Table 3, there was no evidence that childhood misfortune was associated with all-site cancer incidence during 2004–2012. As expected, age (hazard ratio [HR] = 1.03; $p < .001$) was positively associated with cancer incidence. For each additional pack-year smoked, cancer risk ratio increased 0.4% ($p < .001$). Hispanic adults were 30.37% ($p < .05$) less likely than White adults, and females were 27.43% ($p < .01$) less likely than men to get cancer during this time window. Race, ethnicity, and sex were tested as moderators between each misfortune domain and cancer incidence, but none of the interactions were significant ($p > .05$; findings not shown).

Site-Specific Cancer Prevalence

Table 4 presents results of the logistic regression predicting colon, breast, and prostate cancer prevalence. In the model predicting colon cancer in 2004, one infectious disease was associated with 65.43% *lower* odds of colon cancer compared with experiencing no infectious disease in childhood ($p < .05$). Similar to others who report a negative association between SES and colorectal cancer, wealth was inversely associated with colon cancer prevalence (OR = 0.93, $p < .05$; Doubeni et al., 2012). For each additional year of age, the odds of cancer were 5.45% higher ($p < .001$), and each additional depressive symptom was associated with an 11.99% higher likelihood of colon cancer ($p < .05$). Furthermore, the number of pack-years smoked was associated with elevated risk of colon cancer in 2004 by 0.5% ($p < .05$). Product terms between each childhood misfortune domain and race, ethnicity, and sex were tested as potential moderators; however, none were significant ($p > .05$; findings not shown).

There was no evidence that childhood misfortune was related to breast cancer among women in 2004. Among adult resources and demographics, higher education (OR = 1.08; $p < .01$) and older age (OR = 1.02; $p < .05$) were associated with higher odds of breast cancer among women. Race and ethnicity were tested as moderators between each childhood misfortune domain and breast cancer, but none was significant ($p > .05$; findings not shown).

Men who experienced 2+ chronic diseases in childhood were 68.42% more likely to have prostate cancer in 2004 ($p < .05$) than men who did not experience chronic diseases in childhood. Greater wealth (OR = 1.72; $p < .01$) and older age (OR = 1.09; $p < .001$) were associated with higher odds of prostate cancer prevalence as well. Black men were 2.5 times more likely to have prostate cancer than White men ($p < .001$). Product terms between childhood misfortune and race and ethnicity were not significant ($p > .05$; findings not shown).

Given that smoking was not associated with breast or prostate cancer prevalence, sensitivity analyses were conducted to examine the possibility that the coding of smoking may have influenced results. We examined seven alternative coding schemes but reached conclusions similar to those presented herein.[4]

Discussion

Drawing from CI theory, this study used the HRS to examine the early origins of cancer. By disaggregating childhood misfortune into five domains, we were able to test which domains of misfortune are associated with cancer in later life. Findings revealed that childhood misfortune is associated with cancer risk, but the risk is distinct by the type and amount of misfortune (e.g., one or two or more in a domain). In addition, we found that childhood misfortune was associated with either a higher or lower risk of cancer depending on the childhood misfortune and type of cancer. We also investigated the early origins of racial disparities in cancer and found racial and ethnic differences in cancer prevalence and incidence, yet no evidence that the effect of childhood misfortune on adult cancer varied by race or ethnicity ($p > .05$; findings not shown). Product terms investigating sex as a moderator of childhood misfortune and cancer (all-site and colon cancer models) also were not significant.

Our first research question sought to identify which domains of misfortune are associated with risk of cancer. Domains either increased or decreased cancer risk and were dependent on whether the outcome was all-site or site-specific cancer prevalence. In the all-site model, 2+ risky parental behaviors were associated with a higher risk of cancer prevalence. Other researchers have found that physical abuse in childhood by a parent increases cancer risk in adulthood (e.g., Fuller-Thomson & Brennenstuhl, 2009; Morton et al., 2012). Using available information in the HRS, the domain of risky parental behavior includes an

[4]The ways in which we used available information on smoking include (a) a categorical variable of never smoked, former smoker, and current smoker; (b) a duration variable of total years a person smoked along with a dummy variable for heavy smoking (>1.5 packs/day); (c) a pack-years variable; (d) “logcig years” (this is the logarithm of packs smoked per day plus one, then multiplied by number of years smoked). For instance, log(2 packs smoked on average per day + 1)/50 years of smoking (Thurston, Liu, Miller, & Christiani, 2005); (e) the square root transformation of the pack-years variable; (f) the log transformation of the pack-years variable (e.g., log(pack-years)); and (g) pack-years divided into quintiles and treated as categorical.

indicator of abuse as well as two other indicators of parental behavior. Interestingly, 2+ indicators within this domain were associated with higher odds of all-site cancer, suggesting that the amount of misfortune experienced within the domain may be associated with cancer risk.

Indeed, other misfortune domains were predictive of higher odds of cancer only if 2+ misfortunes within the domain were experienced. In the all-site cancer prevalence model, 2+ childhood impairments were also associated with cancer risk. Few researchers have investigated the relationship between childhood impairments and cancer in adulthood. Although the measurement of impairment in the Kelly-Irving and colleagues (2013) article was dichotomous and tapped slightly different elements than how impairments were measured in this study, the authors also reported that childhood impairments are associated with cancer risk in bivariate analyses. Because that study did not adjust for other childhood health indicators, however, it is difficult to compare the results. Moreover, childhood SES may influence the presence or severity of childhood impairments, which could then influence later-life health (Chen et al., 2006). Unfortunately, we were unable to test mediation among childhood misfortune domains because of the coarseness of when the misfortune occurred. In the HRS, one knows only that the misfortune was before age 16 or 18.

Among models investigating site-specific cancer prevalence, 2+ chronic diseases in childhood were associated with higher prostate cancer risk. Blackwell, Hayward, and Crimmins (2001) reported that non-infectious childhood diseases are associated with higher odds of all-site cancer among adults aged 55 to 65. By investigating cancer by site, our findings indicate that this association is specific to some types of cancer such as prostate cancer among men.

Somewhat unanticipated was the finding that one infectious disease during childhood was associated with lower colon cancer prevalence. Seery and colleagues (2010) argued that some childhood misfortune may be protective, and we suggest two plausible mechanisms. First, some research points to acquired immunity related to infectious disease (Preston, Hill, & Drevenstedt, 1998) and suggests that one infectious disease may aid immunological responses related to cancer as well as to the original infection. Second, an infectious disease during childhood may have led to the development of coping strategies or behavior constraint to prevent further insult.

By contrast, there is clear evidence that two or more types of childhood misfortune are related to higher risk of cancer. In the HRS, we observed that 2+ risky parental behaviors and 2+ impairments misfortune was associated with higher odds of all-site cancer prevalence, and 2+ chronic diseases were associated with higher odds of prostate cancer prevalence. These findings reveal the importance of *accumulated misfortune*—and a possible threshold effect: 2+ childhood types of misfortune within a domain may be the tipping point leading to poor health.

Our results also revealed racial and ethnic variation in cancer risk. Reflected in our results, Black men have higher rates of prostate cancer than White men (Bostwick et al., 2004;

Siegel et al., 2015; Ward et al., 2004). Racial and ethnic disparities in cancer are often attributed to access to screening, timely diagnosis and treatment, lifestyle and behavioral risk factors, dietary patterns, and occupational and living exposures to toxins (Bostwick et al., 2004; Siegel et al., 2015; Ward et al., 2004). Consistent with prior research, we also observed that Hispanic adults had lower all-site cancer prevalence and incidence than White adults, suggestive of a Hispanic paradox (Lariscy et al., 2015; Siegel et al., 2015; Ward et al., 2004). Some theses have been offered to explain why this paradox exists. For instance, the salmon bias hypothesis posits that immigrants in poor health return to their country of origin, thus leaving behind a select group of healthy immigrants (Abraido-Lanza, Dohrenwend, Ng-Mak, & Turner, 1999). However, this bias is less likely in a sample of older adults, and other explanations should be explored (Wong & Gonzalez-Gonzalez, 2010). Examining why the Hispanic paradox exists is important, yet it is beyond the scope of this manuscript and should be a goal for future studies.

Adult resources and lifestyles were also associated with later-life cancer. Smoking was positively associated with all-site prevalence, all-site incidence, and colon cancer prevalence. One pack-year is the equivalent of smoking a pack a day for a year. Although the effect sizes appear small (less than 1%), there is a compounding effect. Given that nicotine is addictive and this sample is comprised of older adults who most likely began smoking before the Surgeon General's warning in 1964, these adults smoked for many years (Garfinkel, 1997).

Although a relationship with smoking was not revealed for breast or prostate cancer prevalence, literature suggests that the smoking–cancer association varies considerably by type of cancer (Kuper, Boffetta, & Adami, 2002; Siemiatycki, Krewski, Franco, & Kaiserman, 1995; Surgeon General of the United States, 2014; Yun et al., 2005). The review by Kuper et al. (2002) showed a strong risk of tobacco use on many types of cancer, including lung, larynx, pharynx, bladder, kidney, and stomach. For other sites, such as colon, liver, and cervical cancer, the relationship is described as modest or "limited" in one or more ways. The authors also state that "other forms of cancer, including breast, ovarian and prostate cancer, are unlikely to be linked to tobacco use" (Kuper et al., 2002, p. 206). Therefore, null findings for certain cancer sites are not unusual, but it should be emphasized that smoking is a risk factor for many other cancer types as well as a host of other diseases. Bayesian analysis may be suitable for future research examining the variable effect of smoking on types of cancer as a competing risk.

SES is related to cancer risk through other risk factors associated with SES such as smoking and diet; however, SES may also influence diagnosis and treatment (Siegel et al., 2015; Ward et al., 2004). Given that the respondents were asked if a doctor ever told them that they had cancer, it is likely that the SES indicators positively associated with cancer reflect greater access and earlier stage of diagnosis among wealthier and/or highly educated individuals (Ward et al., 2004). We also reveal that wealth is negatively associated with colon cancer prevalence. This finding is consistent with others; a closely linked risk factor to colon cancer is diet that is also related to SES (e.g., Doubeni et al., 2012; Siegel et al., 2014; Surgeon General of the United States, 2014). Despite adjusting for these well-established risk factors of cancer (Siegel et al., 2015; Ward et al., 2004), the salient effects of early misfortune persisted on cancer prevalence.

Interpretations should be made in light of some limitations. First, retrospective data were used; thus, recall bias may be an issue. To minimize the impact of recall bias on our statistical estimates, we restricted our sample to those within two standard deviations from the mean total cognition score as well as adjusted our models for socioeconomic and mental health status confounders as recommended by others to minimize recall bias (Vuolo et al., 2014). Second, although the HRS has multiple measures for childhood diseases and SES, there are relatively few measures tapping adverse experiences or trauma. Other researchers have included different types of child abuse and risky events and experiences such as living with someone who had been in jail (e.g., Felitti et al., 1998). Third, we investigated direct associations only between childhood misfortune and cancer risk in adulthood. A helpful goal for future research would be to examine the succession of events from childhood that may lead to an increased likelihood of cancer in later life, thereby explicating the potential pathways and mechanisms involved.

Limitations notwithstanding, this study contributes to the mounting research on early origins of adult health. With use of a large, nationally representative data set, we addressed some of the issues in childhood misfortune and cancer research that other investigators were unable to, gaining a clearer understanding of what types of childhood misfortunes are most detrimental, in what amount, and for which type of cancer. We uncovered three domains of childhood misfortune—parental behaviors, chronic diseases, and impairments—to be early risk factors of cancer, and one domain—infectious disease—to be protective at a low dose. In addition, and similar to other cancer risk factors, domains of misfortune influenced types of cancer differentially. For instance, risky parental behaviors and impairments were associated with higher odds of all-site cancer prevalence, whereas chronic diseases were associated with prostate cancer prevalence.

We found that variation in cancer rates by race in the HRS reflect trends in other data sets (e.g., SEER). Although the effect of childhood misfortune on cancer did not seem to vary by race or ethnicity, it is possible that indirect effects of childhood misfortune on cancer contribute to cancer disparities by race. Therefore, further investigation is needed to explore these various pathways. It should be noted that childhood misfortune was not associated with cancer incidence, after adjustment for an array of adult resources and lifestyle factors.

Given population aging and the dynamic link between cancer and aging, identifying the early origins of cancer is paramount for prevention. By identifying what type of misfortune affects cancer in later life, which type of cancer, and at what dose, we offer insight into the process of cancer development as well as progression. This information may help target cancer prevention strategies.

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Table 1

Descriptive Statistics From the Health and Retirement Study (2004–2012).[a]

	Women (*n* = 8,390)			Men (*n* = 5,531)		
Variable	**Range**	***M* (*SD*)**	**Cancer cases[b]**	**Range**	***M* (*SD*)**	**Cancer cases**
Cancer incidence 2004–2012	0, 1	0.093	711	0, 1	0.121	667
Cancer prevalence 2004						
All-site	0, 1	0.113	1,003	0, 1	0.106	712
Colon	0, 1	0.008	75	0, 1	0.011	71
Breast	0, 1	0.034	298			
Prostate				0, 1	0.046	333
Childhood misfortune domain						
SES	0–2	1.349 (0.841)		0–2	1.232 (0.817)	
Risky parental behaviors	0–2	0.846 (0.681)		0–2	0.864 (0.604)	
Chronic diseases	0–2	0.503 (0.752)		0–2	0.402 (0.629)	
Infectious diseases	0–2	1.748 (0.568)		0–2	1.684 (0.604)	
Impairments	0–2	0.188 (0.455)		0–2	0.268 (0.502)	
Adult resources						
Education	0–17	12.860 (2.849)		0–17	13.414 (2.863)	
Wealth	−8.20–31.58	5.858 (3.681)		−13.09–33.37	6.234 (3.577)	
Married	0, 1	0.602		0, 1	0.782	
Adult lifestyle						
Smoking (Pack-Years)	0–75	11.411 (21.192)		0–79	21.589 (28.816)	
Heavy drinking	0, 1	0.015 (0.123)		0, 1	0.052 (0.212)	
BMI	15–50	27.745 (6.273)		16.4–50	28.063 (4.608)	
Depressive symptoms	0–8	1.498 (2.054)		0–8	1.133 (1.690)	
Demographics						
Age	50–97	63.651 (10.233)		50–95	62.346 (8.849)	
Black	0, 1	0.092		0, 1	0.072	
Hispanic	0, 1	0.066		0, 1	0.060	
White	0, 1	0.817		0, 1	0.841	

Note. SES = socioeconomic status; BMI = body mass index.

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[a] Statistics are weighted and adjusted for clustering.

[b] Absolute number of cancer cases.

Table 2

Frequency of Childhood Misfortune by Sex.

	Women (n = 8,390)		Men (n = 5,531)	
	Frequency	**%**	**Frequency**	**%**
SES ***				
0	1,527	18.200	1,189	21.497
1	1,677	19.999	1,085	19.617
2+	5,186	61.812	3,257	58.886
Risky parental behavior ***				
0	2,776	33.087	1,646	29.760
1	4,476	53.349	3,183	57.548
2+	1,138	13.564	702	12.692
Infectious diseases ***				
0	566	6.746	581	10.504
1	1,178	14.041	792	14.319
2+	6,646	79.213	4,158	75.176
Chronic diseases ***				
0	5,525	65.852	3,900	70.512
1	1,749	20.846	1,121	20.268
2+	1,116	13.302	510	9.221
Impairments ***				
0	7,082	84.410	4,375	79.100
1	1,135	13.528	971	17.556
2+	173	2.062	185	3.345

Note. χ^2 tests were performed for each domain of misfortune by sex. SES = socioeconomic status.

*** $p < .001$.

Table 3

Logistic Regression Predicting Cancer Prevalence 2004 and Cox PH Predicting Cancer Incidence 2004–2012.

	Cancer prevalence		Cancer incidence	
	OR[a]	95% CI	HR	95% CI
Childhood misfortune				
1 SES	1.001	[0.791, 1.267]	1.172	[0.960, 1.431]
2+ SES	1.085	[0.862, 1.364]	1.164	[0.936, 1.447]
1 risky parental behavior	1.035	[0.890, 1.204]	1.056	[0.896, 1.244]
2+ risky parental behaviors	1.278*	[1.040, 1.570]	1.122	[0.866, 1.454]
1 chronic disease	0.949	[0.775, 1.162]	1.274	[0.902, 1.800]
2+ chronic diseases	1.253	[0.996, 1.577]	1.323	[0.998, 1.752]
1 infectious disease	1.014	[0.782, 1.313]	0.943	[0.773, 1.149]
2+ infectious diseases	1.032	[0.832, 1.281]	1.143	[0.949, 1.377]
1 impairment	0.969	[0.794, 1.184]	0.961	[0.812, 1.139]
2+ impairments	1.463*	[1.039, 2.061]	0.755	[0.495, 1.152]
Adult resources				
Education	1.020	[0.992, 1.050]	1.003	[0.972, 1.035]
Wealth	1.035**	[1.010, 1.061]	0.991	[0.967, 1.015]
Married	0.917	[0.785, 1.071]	0.926	[0.772, 1.110]
Adult lifestyles				
Pack-years	1.005***	[1.002, 1.007]	1.004***	[1.002, 1.006]
Heavy drinking	0.652	[0.362, 1.177]	1.235	[0.868, 1.757]
BMI	1.011	[0.997, 1.025]	1.010	[0.997, 1.024]
Depressive symptoms	1.045*	[1.004, 1.087]	0.994	[0.956, 1.033]
Demographics				
Age	1.053***	[1.046, 1.060]	1.029***	[1.022, 1.035]
Black	0.875	[0.704, 1.088]	1.142	[0.943, 1.384]
Hispanic	0.651*	[0.425, 0.997]	0.696*	[0.512, 0.947]
Female	1.037	[0.917, 1.174]	0.726**	[0.608, 0.866]
Constant	0.002***	[0.001, 0.004]		

	Cancer prevalence		Cancer incidence	
	OR[a]	**95% CI**	**HR**	**95% CI**
n	12,631		1,1053	
F	18.21***		12.59***	

Note. PH = proportional hazard; OR = odds ratio; CI = confidence interval; HR = hazard ratio; SES = socioeconomic status; BMI = body mass index.

* $p < .05$.

** $p < .01$.

*** $p < .001$.

Table 4

Logistic Regression Predicting Colon, Breast, and Prostate Cancer in 2004.

	Colon cancer		Breast cancer (Women)		Prostate cancer (Men)	
	OR	**95% CI**	**OR**	**95% CI**	**OR**	**95% CI**
Childhood misfortune						
1 SES	0.516	[0.238, 1.121]	1.269	[0.737, 2.185]	0.775	[0.478, 1.255]
2+ SES	1.134	[0.640, 2.012]	1.164	[0.737, 1.838]	0.789	[0.532, 1.170]
1 risky parental behavior	0.991	[0.655, 1.498]	1.004	[0.718, 1.404]	1.054	[0.785, 1.414]
2+ risky parental behaviors	0.752	[0.345, 1.642]	1.115	[0.670, 1.856]	1.180	[0.691, 2.014]
1 chronic disease	1.275	[0.803, 2.025]	0.839	[0.596, 1.180]	0.913	[0.607, 1.373]
2+ chronic diseases	0.684	[0.294, 1.591]	0.774	[0.493, 1.215]	1.684*	[1.093, 2.594]
1 infectious disease	0.346*	[0.145, 0.822]	0.788	[0.424, 1.465]	1.228	[0.695, 2.172]
2+ infectious diseases	0.849	[0.423, 1.703]	0.785	[0.456, 1.350]	1.186	[0.751, 1.873]
1 impairment	0.772	[0.431, 1.383]	0.828	[0.532, 1.291]	1.033	[0.707, 1.508]
2+ impairments[a]			1.814	[0.748, 4.395]	1.148	[0.577, 2.284]
Adult resources						
Education	0.999	[0.931, 1.073]	1.080**	[1.021, 1.143]	0.968	[0.914, 1.024]
Wealth	0.930*	[0.871, 0.992]	1.034	[0.993, 1.077]	1.072**	[1.029, 1.117]
Married	0.979	[0.602, 1.590]	0.827	[0.604, 1.133]	0.940	[0.671, 1.316]
Adult lifestyles						
Pack-years	1.005*	[1.000, 1.010]	1.002	[0.994, 1.011]	1.002	[0.997, 1.007]
Heavy drinking	0.486	[0.080, 2.948]	1.160	[0.334, 4.028]	0.787	[0.216, 2.862]
BMI	1.012	[0.974, 1.051]	1.010	[0.985, 1.034]	1.027	[0.996, 1.058]
Depressive symptoms	1.120*	[1.020, 1.230]	0.960	[0.892, 1.034]	0.920	[0.827, 1.023]
Demographics						
Age	1.054***	[1.028, 1.082]	1.020*	[1.004, 1.036]	1.093***	[1.080, 1.106]
Black	0.987	[0.606, 1.609]	0.778	[0.471, 1.284]	2.499***	[1.540, 4.057]
Hispanic	0.385	[0.121, 1.220]	0.768	[0.462, 1.277]	0.602	[0.221, 1.642]
Female	0.634	[0.402, 0.999]	—	—	—	—
Constant	0.000***	[0.000, 0.013]	0.003***	[0.001, 0.020]	0.000***	[0.000, 0.000]

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	Colon cancer		Breast cancer (Women)		Prostate cancer (Men)	
	OR	**95% CI**	**OR**	**95% CI**	**OR**	**95% CI**
n	12,312		7,631		5,000	
F	10.77***		2.99**		26.60***	

Note. OR = odds ratio; CI = confidence interval; HR = hazard ratio; SES = socioeconomic status; BMI = body mass index.

[a]No respondent with colon cancer in 2004 reported 2+ childhood impairments.

*$p < .05$.

**$p < .01$.

***$p < .001$.