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Alcohol's Collateral Damage: Childhood Exposure to Problem Drinkers and Subsequent Adult Mortality Risk

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Abstract

The importance of childhood circumstances, broadly defined, for shaping adult health and longevity is well-established. But the significance of one of the most prevalent childhood adversities—exposure to problem drinkers—has been understudied from a sociological perspective and remains poorly understood. We address this gap by drawing on cumulative inequality theory, using data from the 1988–2011 National Health Interview Survey-Linked Mortality Files, and estimating Cox proportional hazards models to examine the relationship between exposure to problem drinkers in childhood and adult mortality risk. Childhood exposure to problem drinkers is common (nearly 1 in 5 individuals were exposed) and elevates adult overall and cause-specific mortality risk. Compared to individuals who had not lived with a problem drinker during childhood, those who had done so suffered 17 percent higher risk of death ($p<.001$) over the follow-up period, net of age, sex, and race/ethnicity. We find compelling evidence that the duration, source, and intensity of exposure to problem drinkers in childhood contributes to inequality in adult mortality risk. Favorable socioeconomic status in adulthood does not ameliorate the consequences of childhood exposure to problem drinkers. The primary intervening mechanisms are risky behaviors, including adult drinking and smoking. The findings—which reveal that the influence of problem drinking is far-reaching and long-term—should inform policies to improve childhood circumstances, reduce detrimental effects of problem drinking, and increase life expectancy.

Keywords

alcohol; problem drinking; childhood conditions; mortality; cumulative inequality theory; NHIS

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Introduction

Early life circumstances shape adult well-being, health, and longevity. But which circumstances are most influential and how these conditions shape later outcomes remains poorly understood. In particular, the importance of one of the most prevalent childhood adversities—exposure to problem drinkers—has been understudied from a sociological perspective. We do not yet know the adult consequences of growing up in a home with a problem drinker. Furthermore, it is unclear whether the duration, intensity, and source (e.g., parent, sibling) of childhood exposures matter for adult health and longevity, whether they vary by age and sex, and whether adult circumstances can adequately explain or modify the longevity consequences. We therefore investigate the extent to which various types of exposure to problem drinkers in childhood are associated with adult overall and cause-specific mortality risk and whether adult circumstances can mediate or moderate the association. The novel and important findings from this study contribute to our understanding of the origins of inequalities in adult longevity, and the crucial role that childhood families play in generating these inequities.

Alcohol's Collateral Damage

Alcohol consumption, especially heavy alcohol consumption, is a major risk factor for chronic diseases and external causes of death (Rehm et al. 2009), and it is the third leading preventable cause of death in the United States after smoking and poor diet and physical inactivity (Mokdad et al. 2004). In addition to the many health consequences of alcohol consumption, heavy drinkers often experience social, economic, and legal ramifications, such as family disintegration, severed ties with friends, being passed over for promotions or fired from a job, bankruptcy, spending money on alcohol instead of necessities like food and bills, and having run-ins with the legal and criminal justice systems (McClelland, Michael, and Teplin 2001; Rehm and Russow 2001; Rogers et al. 2015). While the consequences of alcohol consumption on the health of drinkers have been extensively studied, researchers have become increasingly interested in the broader effects on society and on individuals other than drinkers. The societal costs of alcohol consumption (e.g., health care, law enforcement, work productivity) in the United States are estimated to be 2.7 percent of gross domestic product (Rehm et al. 2009); but these estimates may be conservative because they do not account for many of the presumed impacts on individuals connected to drinkers (Casswell, You and Huckle 2011; Giesbrecht, Cukier and Steeves 2010; Livingston, Wilkinson and Laslett 2009).

The myriad consequences of alcohol consumption on individuals other than drinkers—termed "collateral damage" or "second-hand effects of drinking" (Giesbrecht, Cukier and Steeves 2010)—are understudied and poorly understood (Livingston, Wilkinson and Laslett 2009). Moreover, there is little knowledge about the extent to which exposure to others' drinking has *enduring* consequences for the health and well-being of individuals throughout the adult life course. The focus of extant studies has primarily been on the immediate or short-term consequences among children, adolescents, and more recently on young adults. But it is important to understand the *long-term* consequences of exposure to others' drinking. Scholars have underscored the need for this information by drawing parallels with

smoking, noting that information on the dangers of second-hand smoke was crucial in the debate and development of public policy related to smoking (Casswell, You and Huckle 2011; Livingston, Wilkinson and Laslett 2009).

The few studies that have examined the lasting consequences of childhood exposure to others' alcohol consumption suggest that the impacts are alarmingly widespread and substantial. "More than half of all adults have a family history of alcoholism or problem drinking" (National Council on Alcoholism and Drug Dependence n.d).[1] One particularly salient body of evidence is based on the Adverse Childhood Experiences Study (Brown et al. 2009; Felitti and Anda 2010; Felitti et al. 1998). Among seven categories of experiences—ranging from sexual, physical, or psychological abuse to having lived with anyone in the household who had abused drugs or alcohol—Felitti and associates (1998) found that the most common was living with someone who abused alcohol. Almost a quarter of respondents reported childhood exposure to a problem drinker or alcoholic. Respondents exposed to a high number of adverse childhood experiences were more likely to engage in risky behaviors, have worse health, and die during the study follow-up (Brown et al. 2009; Felitti et al. 1998).

The Enduring Reach of Childhood Exposure to Problem Drinkers on Adult Health

To examine *how* exposure to problem drinking in childhood affects longevity, we draw on cumulative inequality (CI) theory, which builds and expands on life course and cumulative disadvantage theories (Ferraro and Shippee 2009). Particularly relevant for our study, CI theory contends that early life is a crucial period for physiological development and social stratification, which can contribute to greater social inequality in adulthood. The theory further asserts that childhood conditions influence adult outcomes such as health and longevity, and that family context is particularly important because of the transmission of genes and common environments in which family members live and interact (Ferraro, Schafer, and Wilkinson 2016; Morton, Mustillo, and Ferraro 2014). Indeed, the family is a basic social institution that can contribute to later life health problems and thereby generate health inequality by exposing children to problem drinking.

Our study focuses on the perception of problem drinking, a concept related to but distinct from alcohol consumption. CI theory notes that individuals evaluate their positions, resources, and trajectories in comparison to others and this evaluation influences future trajectories (Schafer, Ferraro, and Mustillo 2011). In evaluating the influence of problem drinking exposure, our measure reflects the individual's perception of problem drinking, rather than an absolute indicator of alcohol consumption. Some individuals may have been sensitive to alcohol consumption, whereas others may have been unaware of household member habits. The powerful impact that the perception of exposure to problem drinking may have on health outcomes is underscored in the classic observation by Thomas and Thomas (1928, 572): "If men [or women] define situations as real, they are real in their

[1]This is also referred to in the literature as heavy drinking, risky drinking, alcohol misuse, or alcohol abuse.

consequences." Respondent assessments of others' problem drinking are often used (e.g., Burke, Schmied and Montrose 2006) and correlate with indicators of respondents' health and well-being (Casswell, You and Huckle 2011).

Additional research establishes the severity and long-term impact of childhood exposure to problem drinking (Adamson and Templeton 2012; Velleman et al. 2008). Velleman and colleagues interviewed children and adolescents who were exposed to parental problem drinking in ten European Union states and concluded that these "children witness significant domestic abuse and interparental violence, and suffer a much greater incidence of physical violence and emotional abuse themselves" (2008, 404). At extreme levels, children have reported that they have been afraid of their parents because of their drinking. Physical assaults of children by their alcoholic parents—including hitting, choking, burning, cutting with a knife, or slamming against a wall—can be severe enough—including lacerations and broken bones—to result in missed school days or to require medical attention (Velleman et al. 2008).

Both CI theory and life course epidemiology theorize that childhood exposures can have enduring consequences for adult health and longevity through multiple indirect and direct mechanisms (Ben-Shlomo and Kuh 2002). Children who live with an alcoholic parent may be at higher mortality risk as adults because they were raised in a risky family environment. Alcoholic adults may provide less parental supervision, be more likely to neglect or abuse children, and be less foresighted about and active with disease prevention and health promotion (Burke, Schmied and Montrose 2006). Long-term and repeated exposure to problem drinking in childhood may create chronic stress, hormonal dysregulation, and unhealthy behaviors and become biologically embedded to produce proinflammatory propensities that can result in negative physiological changes, including subsequent chronic disease (Danese and McEwen 2012; Hertzman 1999; Miller, Chen and Parker 2011; Odgers and Jefee 2013; Taylor, Repitti, and Seeman 1997). Still, resourceful and resilient individuals can overcome or tolerate and adjust to childhood adversity (Schafer, Ferraro and Mustillo 2011).

In addition to direct mechanisms, childhood exposure can indirectly affect health and longevity through adult socioeconomic resources, health-related behaviors, and psychosocial well-being (Montez and Hayward 2011). For instance, being raised by a problem drinker may disrupt socioeconomic achievement processes. A review of studies that interviewed children of problem drinkers found that the most prevalent theme was educational failure (Tunnard 2002). Children often arrived late to school or were kept home to care for parents, had difficulty concentrating because they were too tired from events at home, had parents who showed little interest in their schoolwork, and often changed schools due to family separation. Felitti and Anda (2010) found that adult workers who experience adverse events in childhood were more likely to exhibit impaired performance, including greater levels of absenteeism and serious problems on the job, all of which can adversely impact economic well-being and limit financial resources necessary to create a healthy lifestyle and avoid premature death.

Individuals exposed to problem drinkers in childhood are more likely to discount their future, possess less self-regulation and more impulsive behavior, and prefer immediate gratification, all of which increases their propensity to engage in health compromising behaviors (Miller, Chen, and Parker 2011). As advanced by social learning theory, they may imitate their parents' drinking behavior (Bandura 1977). Adolescents may also develop unhealthy coping behaviors from family and friends—such as problem drinking, smoking, and overeating—to alleviate stress, trauma, hardship, neglect, and dysfunction experienced in the childhood home (Pearlin 1989). Adults who experienced abuse and family dysfunction in childhood are more likely to become heavy smokers (Lloyd and Taylor 2006) and to consume alcohol; among those who drink, they are more likely to initiate drinking at earlier ages and to suffer from alcohol use disorders (Dube et al. 2006). And compared to a control group, obese adults who had experienced adversity in childhood, including exposure to alcoholic parents, were more likely to overeat as a coping mechanism (Felitti 1993).

Another indirect pathway may occur through family structure. Families headed by problem drinkers often experience dysfunction, perceive their home environment to lack cohesion, have few routines and rituals, exhibit high levels of arguing and unresolved conflict, and express colder, less caring, and negative feelings (Burke, Schmied and Montrose 2006). Alcoholic parents are associated with almost one-third of child abuse cases (Dube et al. 2001). Dube and colleagues (2001) found that compared to adults who did not grow up with an alcoholic parent, individuals who did generally experienced at least twice the risk of emotional, physical, and sexual abuse; emotional and physical neglect; witnessing violence against his or her mother; and parental separation or divorce. These stressful environments can compromise the development of psychosocial resources (Hussong et al. 2005) and interpersonal skills, which provide the bedrock for important social relationships, such as marriage, in adulthood.

Marriage generally contributes to superior mental and physical health and longer lives because of stronger social support, higher socioeconomic status (SES), and healthier behaviors (Ross, Mirowsky, and Goldsteen 1990; Umberson 1992; Waite and Gallagher 2000). Nevertheless, marriage can also be a staging area for conflict, strain, physical and emotional abuse, and bad behavior. Some spouses can act as co-conspirators, encouraging the other spouse to indulge in poor diet, sedentary lifestyle, risky driving, violent behavior, criminal activities, drug use, tobacco consumption, and excessive drinking (Knight 2011). In addition, alcohol abuse by one spouse may lead to alcohol abuse by the other spouse (Leonard and Mudar 2004). Thus, we expect that compared to adults who were exposed, those who were not exposed to problem drinkers in childhood may have more emotional intelligence and greater social skills and attachments, which could increase their likelihood of getting and staying married to an individual who engages in healthy behaviors.

One strategy for identifying the mechanisms linking childhood exposures to adult mortality is to examine how the exposures are associated with specific causes of death. Our review of studies above suggests that exposure will more strongly increase the risk of death from causes that are more commonly associated with risky behaviors and stress. Specifically, if as some studies demonstrate, exposure to problem drinking leads to inactivity, poor diets, smoking, alcohol use, and externalizing behaviors such as aggression and reckless driving,

then the mortality consequences may be most pronounced for deaths from external (e.g., car accidents) and behavioral (e.g., diseases of the liver and heart disease) causes. Additionally, the biological embedding model claims that exposure to problem drinking contributes to hypertension, atherosclerosis, insulin resistance, and tumor development and spread, which can increase risks of such chronic diseases as cancer, circulatory diseases, type 2 diabetes, and autoimmune disorders (Miller, Chen and Parker 2011).

Potential Heterogeneity in the Association

Determining whether the mortality risk associated with exposure to problem drinking varies according to the duration, quantity, and source(s) of exposure further contributes to the CI theory and addresses the call for "greater specification of the *content* and *process* of accumulation" (Ferraro, Schafer, and Wilkinson 2016, 128). A study of children aged 6–15 found that deficits in social competence were greater for children with two rather than one alcoholic parent(s) (Hussong et al. 2005), which may reflect the fact that children with two alcoholic parents lack a potentially compensating effect of a nonalcoholic parent. In addition, the deleterious effects of childhood exposure to problem drinkers may increase with duration of exposure. A review by Burke and associates (2006) concluded that the influence may be cumulative, such that the longer children were exposed, the greater the impact on their health and well-being. Studies that have combined quantity and duration of exposure to problem drinkers into a single index have also found a strong, inverse relationship between exposure and personal well-being and self-reported health in adulthood (Casswell, You and Huckle 2011).

Related to the life course principle of "linked lives" (Elder 1998), CI theory postulates that childhood conditions are influential because of shared genetic background and living environments (Ferraro, Schafer, and Wilkinson 2016; Ferraro and Shippee 2009). It is conceivable that the closer the family relationship, the stronger the mortality effect of exposure to problem drinking in childhood. In other words, stronger links produce stronger effects. Therefore, problem drinking among parents may have greater weight than problem drinking among more distant relatives, including grandparents, aunts and uncles, and cousins.

Although early-life exposures may have a long reach in shaping later life outcomes, CI theory rejects a deterministic view, noting that human agency and resources can alter trajectories over the life course (Schafer, Ferraro and Mustillo 2011). Can adult circumstances exacerbate or alleviate the consequences of childhood exposure to problem drinkers? On one hand, the health of adults who were exposed to a problem drinker in childhood may be more vulnerable to stressors such as poverty and marital disruption in adulthood than adults who were not exposed. On the other hand, their health may disproportionately benefit from protective buffers in adulthood such as high incomes and stable marriages. Educational attainment may be a particularly central buffering resource for adults exposed to problem drinkers in early life because adults with higher levels of SES tend to use effective coping strategies and have higher levels of self-esteem, sense of mastery, self-efficacy, and problem-solving skills (Aneshensel 1992). Empirical support for moderating effects of adult circumstances also comes from studies finding that positive

social, familial, and external supports increased one's resilience to the consequences of others' problem drinking (see review in Burke, Schmied and Montrose 2006).

The consequences of exposure to problem drinkers in childhood on adult health and longevity may vary by age and sex, although the literature is mixed. On one hand, because older adults have been exposed to disadvantages (or advantages) for a longer time, inequalities in mortality between individuals exposed and not exposed to problem drinkers may be greatest among older adults. On the other hand, inequality may stabilize or decline at older ages due in part to such government programs as Medicare and Social Security (Willson, Shuey, and Elder 2007). In addition, the inequalities may shrink with age if the mechanisms linking early exposure and later mortality become less relevant with age. For instance, the inequalities may contract if risky behaviors—including thrill-seeking, risk-acceptance, and aggressive and reckless driving—are an important mechanism because the prevalence of risky behaviors declines with age (Grant et al. 2004; Lyvers, Duff and Hasking 2011; Turner and McClure 2003).

Research Aims

The study addresses three central aims. First, we establish the magnitude of the association between childhood exposure to problem drinking and adult mortality risk. We posit that childhood exposure to problem drinking within the family elevates adult mortality in large part through an adult life characterized by social, economic, and behavioral risks. Thus, we expect that adults who were exposed to a problem drinker during childhood have higher mortality risk than individuals who were never exposed during childhood, and that the mortality gap declines with additional controls for adult socioeconomic, family structure, and behavioral factors.

Second, we examine the effects of the source, duration, and magnitude of exposure to problem drinkers in childhood on adult mortality risk. We expect that the greater number, the closer the relationship, and the longer duration of exposure will each elevate adult mortality risk. Finally, as explained above, we expect that childhood exposure to problem drinking increases the risks of death from all major causes, and in particular, external causes.

Data and Methods

Data

We use the 1988–2011 National Health Interview Survey Alcohol Supplement Linked Mortality File (NHIS-LMF; NCHS 2015). The NHIS Alcohol Supplement, cosponsored by the National Institute on Alcoholism and Alcohol Abuse, includes an extensive set of questions on past and current alcohol use by the respondent and other key individuals, including parents, siblings, spouses, and partners (Schoenborn 1991). Although other years of the NHIS ask about drinking habits, the 1988 NHIS Alcohol Supplement is the only survey year that asks about problem drinking in childhood. Further, this year provides a substantial follow-up period of survival status.

The NHIS is a nationally-representative survey of the non-institutionalized U.S. population. It is the major source of information on the health of the population and the social, economic, and behavioral correlates of health (it does not collect biomarkers). Although the 1988 NHIS Alcohol Supplement interviewed 43,809 individuals aged 18 and over, we focus on the 41,308 adults aged 21 and above who met the legal drinking age. The dataset of individuals aged 21 and older includes 47.4 percent males and 52.6 percent females; 74.4 percent non-Hispanic whites, 10.0 percent non-Hispanic blacks, 6.6 percent Hispanics, 2.0 percent non-Hispanic Asians, and 7.0 percent others; with an average age of 45.2.

In 2013 the National Center for Health Statistics created the 1988–2011 NHIS-LMF by linking the 1988 NHIS respondents to death certificate information in the National Death Index through December 31, 2011 using a probabilistic matching algorithm (NCHS 2013). Thus, respondents' vital status was monitored from their NHIS survey until death or the end of 2011 for survivors. Over the 1988–2011 period, 13,293 individuals (32 percent) in our analytic sample died. The exceptionally rich and detailed information on alcohol use, including retrospective questions, the large nationally representative sample, the full age range of adults of legal drinking age, and the extended mortality follow-up are major strengths of this dataset.

Mortality

The main outcome of interest is death from all causes. Compared to other potential outcomes, mortality studies have the clear advantage of temporal ordering—death is the terminal event in a person's life—and relatedly, avoiding reverse causation. To illuminate the mechanisms that link childhood exposure to problem drinkers to subsequent mortality risk in adulthood, we also examine the risk of death from nine leading causes. We use the classification of causes of death from the current International Classification of Diseases (ICD)-10 codes (WHO 2007) and separately examine the risk of death from heart diseases (I00–I09, I11, I13, I20–I51); malignant neoplasms [cancer] (C00–C97); chronic lower respiratory diseases (J40–J47); cerebrovascular diseases (I60–I69); accidents (V01–X59, Y85–Y86); Alzheimer's disease (G30); diabetes mellitus (E10–E14); nephritis, nephrotic syndrome, nephrosis [kidney diseases] (N00–N07, N17–N19, N25–N27); influenza and pneumonia (J09–J18); and all other causes. Although it would be informative to identify a major cause of death that would be unaffected by exposure to problem drinking, such exposure could affect a wide range of causes of death. Instead, we speculate that causes of death associated with risky behaviors and stress, such as heart disease, cirrhosis of the liver, and external causes, especially unintentional injuries, will have stronger associations with exposure to problem drinking than many other causes of death. Our codes are based on the selected leading causes of death in the United States (Kochanek et al. 2016).

Childhood Exposure to Problem Drinkers

Our key exposure of interest is living with a problem drinker or alcoholic during the first 18 years of life. After being told, "People have different opinions about heavy, moderate, and light drinking," respondents were asked "When you were growing up, that is, during your first 18 years, did you live with anyone who was a problem drinker or alcoholic?" This variable carries the benefit of temporal ordering—exposure occurs before all of the

mediating variables, which are measured in adulthood. Because problem drinker and alcoholic were defined by the respondents, the designations may not fit strict clinical definitions (Schoenborn 1991) but they nonetheless reflect the importance of perceptions of childhood stressors and disadvantages (Ferraro and Shippee 2009; Ferraro, Shippee and Schafer 2009; Schafer, Ferraro and Mustillo 2011).[2] Although adults provide accurate responses to retrospective questions about childhood health and SES (Haas 2008), responses about childhood exposure to problem drinking may be slightly underreported (Felitti et al. 1998; Schafer and Ferraro 2012), which could result in conservative estimates of the actual mortality risk.[3] We code all respondents who lived with a problem drinker or alcoholic in childhood as 1, and all other respondents as 0.

Respondents stating that they lived with a problem drinker or alcoholic during childhood were then asked to identify their relationship to the individual (e.g., parent, sibling, other relative). This survey allows the respondent to identify up to five individuals. Furthermore, it asked respondents to state how long they lived with each individual while they were a problem drinker or alcoholic. For simplicity, we hereafter refer to these individuals as a "problem drinker." Using this information we create three additional measures of childhood exposure. The measures include the number of problem drinkers the respondent ever lived with when aged 0–18 (0, 1, 2 or more), the relationship to each problem drinker (parent, sibling, other relative), and the number of months the respondent lived with a problem drinker. Because respondents may have lived with more than one problem drinker relative, we prioritized reports of parental problem drinkers over siblings and other relatives, and siblings over other relatives. We were able to separately examine mother, father, and both mother and father problem drinkers. Because respondents may have lived with more than one problem drinker in their childhood, we coded the length of time for the problem drinker who lived with the respondent the longest. For descriptive statistics, we report months lived with a problem drinker. For multivariate analyses, we code years lived with a problem drinker into four categories: 0, never lived with a problem drinker, the most common response; less than 6 years; 6 to less than 12 years; and 12 or more years.[4]

Hypothesized Mediators

We hypothesize that three types of adult circumstances—SES, adult health behaviors, and family structure—partly mediate the association between childhood exposure to problem

[2]Alcohol consumption varies by time and place. U.S. alcohol consumption per capita was low during Prohibition (1920–1933), increased from the mid-1930s to peak in early 1980s, and has declined to lower levels today. For example, the per capita consumption in gallons of ethanol per year for individuals aged 14 and older was 2.34 in 2013, 2.76 in 1980, and 2.52 in 1970 (Haughwout, LaVallee, and Castle 2015). Today, almost all adults grew up when alcohol was legal and drinking was socially acceptable (Keyes, Hatzenbuehler, and Hasin 2011). There are also regional, state, and county variations in drinking patterns. For example, in 2013, per capital alcohol consumption in gallons was 1.89 or below for five states, including Arkansas, Kansas, and Utah, and 2.31 or above for 31 states, including California, Colorado, Florida, and Illinois (Haughwout, LaVallee, and Castle 2015). Thus, problem drinking might be more prevalent among adults who were raised during periods and in states with higher levels of alcohol consumption. An examination of time and space variations in alcohol consumption would be an interesting topic for future research, but is beyond the scope of this study. Importantly, problem drinking is not just a measure of average consumption, but drinking behaviors, for example, heavy regular or episodic versus moderate average drinking. Compared to more mild problems, severe problems with drinking might be less time and space dependent.

[3]False positive reports of child maltreatment are rare, based on comparisons of various datasets and administrative records, including surveys and court records (see Keyes, Hatzenbuehler, and Hasin 2011).

[4]We code number of years lived with problem drinker(s) into categories because of the skewed distribution of the variable that includes a large number of zeroes. A continuous measure of number of years was also statistically significant (but small). Additional analyses using different cutpoints produced substantively similar results.

drinkers and adult mortality risk. We include two indicators of adult SES, educational attainment and family income. Education and income are continuous measures; the former reflects years of educational attainment and the latter captures the family income in dollars in the past year. For multivariate analyses, we take the log of family income to normalize the distribution.

The second group of mediators that we examine reflects adult family structure. We include a categorical measure of marital status at interview, coded as never married, married, divorced or separated, or widowed. We also include an indicator of whether the respondent had ever been married to or lived with a problem drinker or alcoholic as an adult (yes=1, no=0).[5]

Key indicators of adult health behaviors are body mass index (BMI), smoking, and drinking statuses. We code drinking status at the time of the interview as abstainer, former drinker, lifetime infrequent drinker, and current drinker. Among current drinkers, we code the average volume into less than 1, 1 to less than 2, 2 to less than 3, and 3 or more drinks per day (for similar coding, see Rehm, Greenfield and Rogers 2001). All statuses are compared to the category of the lightest current drinkers. Smoking is categorized into those who reported never smoking, having previously smoked, and being a current smoker. We use BMI as a proxy for diet and physical activity and calculate it as weight in kilograms divided by height in meters squared. BMI, a commonly-used measure of obesity, is based on self-reported height and weight.[6] We follow the World Health Organization (2000) guidelines to code BMI as underweight (less than 18.5), normal weight (18.5–24.9, the referent), overweight (25.0–29.9), and obese (greater than or equal to 30.0).

A few respondents were missing data on one or more mediators. We imputed missing data using the “mi” package in Stata 13 (Statacorp 2013), allowing us to retain all individuals in all analyses. Imputation models used an iterative Monte Carlo Markov Chain method and created 10 datasets. We impute 130 values (0.3 percent) for education, 5,246 values (12.7 percent) for income, 574 values (1.4 percent) for weight status, 42 values (0.1 percent) for smoking status, 1,051 values (2.5 percent) for drinking status, 41 values (0.1 percent) for marital status, and 347 values (0.8 percent) for having ever been married to a problem drinker values. All independent and dependent variables are used to inform imputation, as are auxiliary variables (income [less than $20,000, greater than or equal to $20,000], and region).

Analytic Plan

We employ Cox proportional hazards models to estimate the risk of death during the follow-up period from age and the predictor variables. The Cox models use a continuous measure of age as the time metric, using age at interview as entry. The duration of exposure is then the time elapsed from age at entry until age at death or censoring at the end of 2011. Examination of Schoenfeld residuals revealed that our main variable of interest, exposure to problem drinking in childhood, does not violate proportionality. We begin with a baseline

[5]Although measures of depression or psychological well-being could reflect additional pathways, such measures are not in the dataset.
[6]Although self-reported BMIs may slightly underestimate actual BMIs—because of tendencies for men to overstate their height and women to understate their weight (Rogers, Hummer, and Krueger 2003)—they are generally considered accurate (Stewart 1982), and capture additional mortality risks associated with body mass, especially among obese individuals.

model that includes the main predictor of interest—a binary indicator of childhood exposure to a problem drinker—along with basic control variables, sex (0=female, 1=male) and race/ethnicity (non-Hispanic white [omitted reference], non-Hispanic black, Hispanic, Non-Hispanic Asian, and other). We use a progressive adjustment approach (Mirowsky 2013) and sequentially add the three groups of hypothesized mediators (SES, family structure, and health behaviors) to assess the extent to which they attenuate the association between childhood exposure and adult mortality risk. We also test for moderating influences by examining interactions between childhood exposure to problem drinking and age, sex, SES, health behaviors, and family structure. A significant interaction would show, for example, a synergistic effect between childhood exposure to problem drinking and adult smoking on mortality. All analyses account for the complex sampling design of the NHIS-LMF. We also tested whether the results were sensitive to the length of mortality follow-up. Cox models using shorter follow-up periods produced nearly identical results to those presented here.

Results

Table 1 shows descriptive statistics of exposure to problem drinking during childhood and adulthood. Nearly one in five adults (18.1 percent) reported having lived with a problem drinker during their first 18 years of life. It is much more common to have lived with one rather than two or more problem drinkers during childhood. Parents were the most likely problem drinking relative during a person's first 18 years of life. Respondents had lived with a problem drinker for an average of about 28 months. This number reflects the influence of the approximately 82 percent of the sample that did not report living with a problem drinker and therefore had a value of 0 months; the average is considerably higher among those who reported ever living with a problem drinker (154.4 months or nearly 13 years [results not shown]).

Table 1 also presents variations in problem drinking by other covariates. Individuals were likelier to have lived with problem drinkers during their childhood if they were younger rather than older; female rather than male; non-Hispanic white rather than other race/ethnic groups; divorced rather than married, widowed, or single; low- rather than high-income; current rather than never or former smokers; and former or current heavy drinkers rather than abstainers or infrequent drinkers. Lastly, nearly a third of those who have ever married or lived with a problem drinker had childhood exposure to a problem drinker. Although these results are informative, they do not control for other covariates. Next, we turn to multivariate analyses.

Table 2 displays the risk of death associated with exposure to problem drinking in childhood. Compared to respondents who had not lived with a problem drinker during their formative years, respondents who had done so experienced 17 percent higher risk of death (hazard ratio [HR]=1.17; $p<.001$) over the follow-up period, with controls for sex and race/ethnicity (Model 1). This elevated risk declines modestly to 16 percent ($p<.001$) with additional controls for adult SES. Marital status is significantly associated with mortality risk but does not reduce the increased risk for those exposed to problem drinkers. Considering whether the individual ever married or lived with a problem drinker slightly reduces the elevated risk to 14 percent ($p<.001$; Model 4). Weight status is significantly

associated with mortality risk and slightly dampens the increased risk for those exposed to problem drinkers to 13 percent (p<.001; Model 5). The risk further attenuates with controls for respondents' current drinking and smoking status (Models 6 and 7). The ln(HR) for living with a problem drinker during childhood is reduced by 14.6 percent with controls for drinking ((ln[1.13] − ln[1.11])/ln[1.13] * 100) and an additional 26.2 percent with smoking status controls. The full model (Model 7) shows that the risk of death associated with living with a problem drinker during a person's first 18 years of life is elevated by 8 percent (p<.01), net of adult demographic, socioeconomic, behavioral, and family structure controls and mechanisms. Overall, the full set of controls and mechanisms reduces the risk of mortality related to exposure to problem drinking in childhood by half (from a HR of 1.17 in Model 1 to a HR of 1.08 in Model 7). We also tested for but did not find significant interactions between adult circumstances and childhood exposure to a problem drinker (results not shown).[7]

Table 3 illustrates the influence of different types of exposure to problem drinking in childhood. As in the previous table, modeling begins with a baseline model that controls for sex and race/ethnicity, and then progressively adds controls for adult SES (Model 2), family structure (Models 3 and 4), and health behaviors (Models 5–7). Importantly, most measures of exposure to problem drinking—including the number of problem drinkers, the individual's relationship with them, and the duration of exposure—elevate a person's risk. Controlling only for sex and race/ethnicity, and compared to those who did not live with a problem drinker during childhood, those who did experienced 15 percent higher risk of death (p<.001), and those who lived with two or more problem drinkers experienced 28 percent higher risk of death over the follow-up period (p<.001; Panel A, Model 1). The elevated mortality associated with living with two more problem drinkers (2.7 percent of the population) did not remain significant once we controlled for all mediators (see Model 7). Smoking and unhealthy drinking appear to be a key pathway for these individuals.

Panel B of Table 3 examines the relationship to the problem drinker. Compared to adults who did not live with a problem drinker in childhood, those who lived with a mother problem drinker experienced 23 percent higher risk of death (p<.05), those who lived with a father problem drinker experienced 14 percent higher risk of death (p<.001), and those who lived with two problem drinker parents experienced 39 percent higher risk of death over the follow-up period (p<.01), net of sex and race/ethnicity (Model 1). These differences were attenuated to marginal significance in the full model (Model 7), so that compared to adults who did not experience problem drinking in the childhood home, those with problem-drinking fathers experience 6 percent higher risk of death (p<.10), and those with problem-drinking parents experienced 22 percent higher risk of death (p<.10) over the follow-up period.

Panel C of Table 3 shows that the more years that a person had lived with a problem drinker in childhood, the greater the mortality risk. For instance, compared to adults who had not lived with a problem drinker, those who had done so for 1 to 5 years experienced 13 percent

[7]Models separately interacted adult age, sex, race/ethnicity, marital status, education, income, smoking status, and drinking status with childhood exposure. Out of 19 interaction terms across eight models, none were statistically significant at p<0.05.

higher risk of death (p<.10), those who had done so for 6 to 11 years experienced 15 percent higher risk of death (p<.05), and those who had done so for 12 or more years experienced a 18 percent higher risk of death (p<.001) over the follow-up period, net of adult demographic factors (Model 1). The mortality risk associated the number of years lived with a problem drinker attenuated with the full set of controls (Model 7).

Table 4 displays the association between childhood exposure to problem drinking and specific causes of death. The baseline model (Model 1) shows that individuals who lived with a problem drinker during childhood were more likely to die from many of the major causes we examined, including cancer (with a 17 percent higher risk of death; p<.01), chronic lower respiratory diseases (28 percent higher risk; p<.05), and especially accidents (79 percent higher risk; p<.001).[8] Models 2–7 progressively control for adult socioeconomic conditions, family structure, and health behaviors. Poorer health behaviors of individuals exposed to problem drinkers in childhood explains a large portion of their elevated risks of death (compare Models 7 and 5). After controlling for all three groups of adult mediators, the elevated risk remains significant for accidents (HR=1.60, p<0.001), and marginally significant for kidney disease (HR=1.40, p<0.10), and “all other” causes (HR=1.11, p<0.10). Taken together, these findings indicate that exposure to problem drinking in childhood elevates mortality risk in adulthood in large part through health damaging behaviors, such as smoking, heavy drinking, and reckless driving.

Discussion and Conclusions

Childhood exposure to problem drinkers is not only fairly common (nearly 1 in 5 U.S. adults report being exposed), it also has enduring consequences on the risk of death throughout adulthood. This study contributes to the growing literature on the importance of childhood circumstances for adult well-being, providing evidence that risky family environments are prevalent and problematic. For instance, adults aged 21 and older who were exposed to problem drinkers in childhood had a 17 percent greater risk of death (p<.001) compared with peers who were never exposed, net of sex and race/ethnicity. Early life exposure to problem drinking increases the risk of death throughout the life course through multiple mechanisms.

This study contributes to the application of CI theory by demonstrating that the duration, intensity, and source of childhood adversity lead to increased risk of death. The findings generally demonstrate a dose-response relationship. Longer exposure to problem drinkers in childhood contributes to increased mortality risk, which suggests that chronic exposure is

[8]The current NHIS-LMFs follow mortality through 2011 but restrict the number of cause-of-death categories. Using the previous files, which linked mortality through 2006 and provided 113 causes of death (NCHS 2013), we examined the risks of death due to chronic liver disease, accidents, suicides, and homicides. We found that individuals who lived with a problem drinker were 96 percent more likely to die from chronic liver disease and cirrhosis of the liver over the follow-up period, net of demographic controls, and 60 percent more likely to die with the full set of controls. The heightened risk of liver disease mortality may reflect different drinking patterns beyond drinking status, such as earlier initiation to drinking, heavy episodic drinking, or greater likelihood of drinking problems (Rogers et al. 2015). Two categories of external causes—transport and non-transport accidents—also displayed striking risks. Compared to individuals who never lived with a problem drinker in childhood, those who did experienced a 73 percent higher risk of death from transport accidents and 55 percent higher risk of death from nontransport accidents over the follow-up period, net of demographic controls. Roughly 93 percent of transport accidents are motor vehicle crashes. In the full model, the risk of transport accidents was still 60 percent higher, but the risk of nontransport accidents was no longer statistically significant. The risk of suicide and homicide mortality, considered both separately and jointly, was not statistically significant, in part because of the small number of deaths.

especially problematic (see also Burke, Schmied and Montrose 2006). In addition, we find that being exposed to two or more problem drinkers elevated mortality risk marginally more than being exposed to one problem drinker, which is consistent with Dube et al.'s (2001) finding that individuals who grew up with both parents who abused alcohol were much more likely to experience adverse childhood events than those who grew up with one parent who abused alcohol. And source of exposure matters. Exposure to parental drinking has a stronger effect than sibling or other relative drinking, which supports Elder's (1998) notion of linked lives.

Based on the tenets of CI theory, we seek to determine what kinds of resources or behaviors can alter the effects of childhood exposure to problem drinking. We find that the main mechanism through which exposure to problem drinkers in childhood elevates overall and cause-specific mortality risk in adulthood is risky behaviors. Adults who had been exposed to problem drinking in childhood were more likely to smoke and drink. Adolescents and young adults may cope with the stress and trauma associated with problem drinking within the childhood family by overeating, smoking, abusing alcohol, and engaging in reckless behavior, including careless driving (see Miller, Chen, and Parker 2011).

Several factors could explain why adults exposed to problem drinkers in childhood disproportionately engage in risky behaviors, including, importantly, family context. Children may imitate their parents' behaviors (Bandura 1977). Risky behaviors may also be an externalizing response to being raised in harsh, cold, chaotic, or abusive family environments (Felitti et al. 1998; Taylor, Repetti and Seeman 1997). And these behaviors may signal biological embedding of early environments (Hertzman 1999; Odgers and Jafee 2013). For example, prenatal exposure to alcohol can lead to fetal alcohol spectrum disorders, which are permanent and include a range of conditions including intellectual disabilities, impaired vision and hearing, compromised heart and kidney function, and such behavioral problems as poor impulse control (CDC n.d.). Impulsiveness is, in turn, associated with a host of risky behaviors including marijuana use (Simons and Carey 2002), higher levels of alcohol use (Patock-Peckham and Morgan-Lopez 2006), and aggressive and reckless driving (Dahlen et al. 2005).

Favorable adult circumstances (e.g., high education, high income, marriage) did not ameliorate the deleterious consequences of childhood exposure to problem drinking on adult mortality risk. Light to moderate alcohol consumption differs from most other health behaviors in that it is positively associated with SES: compared to adults with lower incomes and levels of educational attainment, individuals with higher incomes and levels of educational attainment are more likely to be current regular drinkers and less likely to abstain from drinking or drink infrequently (Rogers et al. 2013). Yet we found a slight inverse relationship between SES and exposure to problem drinking. For example, the percentage of respondents who lived with a problem drinker during childhood was 19.2 among those earning less than $10,000 per year but 15.3 among those earning $50,000 or more per year. We speculate that the higher prevalence of regular drinking and the lower prevalence of infrequent drinking and abstention among higher SES individuals may confer a slight health benefit for higher SES adults on average, but may also dampen what would otherwise be a larger SES gap in childhood exposure to problem drinking. Thus, whereas

such dire economic conditions in childhood as poverty and food insecurity may contribute to cumulative inequality, exposure to problem drinking in childhood may have less of an effect on SES disparities, but a much greater impact on adult longevity.

The results inform the development of strategies and policies to reduce the collateral damage or second-hand effects of problem drinking (see Giesbrecht, Cukier and Steeves 2010). Our results indicate that the approximately 80,000 deaths attributed to alcohol each year (U.S. DHHS n.d.) are underestimated because they do not take into account the indirect effects of problem drinking—including exposure to problem drinkers—on individuals connected to the drinker. Policymakers should consider ways to reduce the injurious effects of exposure to problem drinking among children, such as targeting problem drinking among parents or within family environments. For example, interventions that improve family functioning, provide external supports to the family, and teach parenting skills can soften the impact of parental problem drinking (see review in Burke, Schmied and Montrose 2006).

These policies are particularly important given our results which suggest that it is difficult to mitigate the elevated mortality risk associated with childhood exposure to problem drinking when those individuals become adults. Reducing exposure or the effects of exposure could reduce alcohol-related deaths. And interventions could be beneficial throughout the pathways from exposure to death, including reducing exposure to problem drinking in childhood; reducing such risky but coping behaviors as smoking, overeating, and drinking to excess; early identification and treatment of diseases that may be more common among this vulnerable population; intervention and accommodation of comorbid conditions, functional limitations, and disability; and intervention to maintain social connections and support (see also Felitti et al. 1998). It is important to break any cycle in the intergenerational transmission of problem drinking, especially in households with young, impressionable, vulnerable children. Furthermore, reducing adverse childhood experiences can contribute to improved health and survival of both children and adults.[9]

Despite the many strengths of NHIS-Alcohol for our analyses, the study has four main limitations. First, we cannot control for potential left censoring. Some children who were adversely affected by problem drinking of others in the family may have become institutionalized (in prisons, jails, mental institutions, or drug rehabilitation facilities), homeless, or died and therefore missed by the survey (see also Brown et al. 2009). Second, we do not have time-varying covariates. We have information about whether the respondent had ever been married to or lived with a problem drinker, but we do not know whether the respondent was still living with the problem drinker at the time of the survey, had divorced, or had been widowed. This may be a minor limitation because it would lend itself to conservative parameter estimates. Third, the retrospective questions may have been influenced by recall bias. For instance, some respondents may not accurately recall exposure

[9]Problem drinkers may produce harmful drinking-related effects that inspire some children to abstain from, quit, or consume low amounts of alcohol as adults. For example, the percentage of adult abstainers was 11 among those who had lived with a problem drinker in childhood, and 20 among those who had not lived with a problem drinker. Compared to the general adult population, abstainers who lived with a problem drinker in childhood were more likely to be female, non-white, never smokers, and to have lower SES (results not shown). A better understanding the intergenerational transmission and rejection of problem drinking is an important topic that merits further research.

to problem drinking, which could result in conservative estimates of the effects of childhood exposure on problem drinking. While this concern should not be ignored, studies of the reliability and validity of retrospective recalls of childhood experiences support their judicious use in population research (Haas 2008). Finally, although our analyses capture many of the main social and behavioral determinants of health and mortality, we lack information on other childhood exposures such as family dissolution and socioeconomic strain. While this information could be valuable, the effects of parental alcohol abuse on children's health and well-being in adulthood persist even when controlling for childhood SES (e.g., Christoffersen and Soothill 2003; Danese and Tan 2014; Fisher et al. 2010; Friedman et al. 2015; Thomas, Hyppönen and Power 2008).

Our findings show how early life circumstances shape adult longevity. Risky behaviors may be central factors related to higher mortality risk of adults exposed to problem drinking in childhood. Just as second-hand smoking harms those exposed to the smoke, so too can childhood exposure to alcohol abuse (e.g., Giesbrecht, Cukier and Steeves 2010). Such information underscores the need for health researchers to consider a broader range of childhood exposures than just low SES, especially given the high prevalence of living with a problem drinker. Additionally, health research can gain important insight from incorporating CI theory to understand how different statuses and experiences early in the life course shape subsequent health and well-being. Overall, problem drinking, including exposure to problem drinking in childhood, is far-reaching, enduring, pernicious, and associated with shorter lives.

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Biographies

Richard G. Rogers is a Professor of Sociology and the Director of the Population Program in the Institute of Behavioral Science at the University of Colorado at Boulder. His research examines differences in adult and early life mortality by demographic characteristics, socioeconomic status, health behaviors, and health conditions. Rogers has published in such journals as *American Sociological Review*, *Social Forces*, *Demography*, *Journal of Health and Social Behavior*, and *Social Science & Medicine*.

Elizabeth M. Lawrence is a postdoctoral fellow in the Carolina Population Center at the University of North Carolina – Chapel Hill. Her research examines social inequality and health, with a focus on how individuals' educational and health trajectories develop together over the life course. She has published in journals such as *Journal of Health and Social Behavior*, *Social Science & Medicine*, *Demography*, *Social Science Research*, and *Advances in Life Course Research*.

Jennifer Karas Montez is an Assistant Professor in the Department of Sociology at Syracuse University. Her research examines inequalities in health and longevity at the intersections of gender, educational attainment, and geography. Her current work focuses on the structural conditions behind the falling international ranking of U.S. women's longevity. She has published on these topics in *Social Forces*, *Demography*, *Journal of Health and Social Behavior*, *Journals of Gerontology*, and *American Journal of Public Health*.

Table 1

Percentages of U.S. Adults Aged 21 and Over Who Lived with Problem Drinkers in Childhood, by Select Covariates, 1988

	Lived with problem drinker during first 18 yrs	Number of problem drinkers lived with			Problem drinker relation					# months lived with problem drinker
		0	1	2+	Mother	Father	Mother and father	Sibling	Other	
Population at the time of interview	18.1%	81.9%	15.4%	2.7%	1.7%	12.9%	1.6%	0.8%	1.1%	27.9
Demographic factors										
Age										
21–44	22.5%	77.5%	18.7%	3.7%	2.5%	15.6%	2.2%	0.9%	1.3%	33.2
45–64	16.2%	83.8%	14.2%	2.1%	1.1%	12.2%	1.3%	0.6%	1.0%	27.4
65+	8.4%	91.6%	7.7%	0.7%	0.2%	6.4%	0.2%	0.7%	0.9%	14.1
Sex										
Male	16.5%	83.5%	14.2%	2.3%	1.4%	11.7%	1.4%	0.8%	1.2%	25.1
Female	19.3%	80.7%	16.3%	3.0%	1.9%	13.8%	1.7%	0.8%	1.1%	30.1
Race/ethnicity										
Non-Hispanic white	18.6%	81.4%	15.8%	2.8%	1.7%	13.4%	1.7%	0.7%	1.0%	28.7
Non-Hispanic black	16.3%	83.7%	14.3%	2.0%	2.2%	10.3%	1.0%	0.8%	2.0%	24.7
Hispanic	17.9%	82.1%	15.5%	2.4%	1.2%	13.7%	0.8%	1.0%	1.1%	28.8
Non-Hispanic Asian	7.6%	92.4%	7.1%	0.5%	0.4%	4.7%	0.2%	1.2%	1.1%	11.7
Other	18.2%	81.8%	14.8%	3.4%	1.5%	13.0%	1.7%	0.6%	1.4%	27.0
Socioeconomic status										
Income										
<$10,000	19.2%	80.8%	15.9%	3.3%	1.9%	12.9%	1.7%	1.0%	1.7%	29.3
$10,000–$19,999	18.8%	81.2%	16.0%	2.7%	1.8%	13.4%	1.4%	1.0%	1.2%	28.8
$20,000–$29,999	18.8%	81.2%	15.9%	2.9%	1.5%	13.7%	1.6%	0.9%	1.1%	28.7
$30,000–$39,999	19.3%	80.7%	16.6%	2.7%	1.7%	14.0%	1.6%	0.9%	1.1%	29.4
$40,000–$49,999	18.9%	81.1%	15.9%	3.0%	1.6%	13.3%	2.2%	0.6%	1.2%	29.2
$50,000+	15.3%	84.7%	13.3%	2.0%	1.7%	11.2%	1.3%	0.4%	0.7%	24.5
Education										
<12 years	17.5%	82.4%	14.5%	3.1%	1.2%	12.4%	1.6%	1.0%	1.4%	27.6
12 years	19.7%	80.3%	17.0%	2.8%	1.7%	14.5%	1.6%	0.8%	1.1%	30.4
13–15 years	19.8%	80.2%	16.8%	2.9%	2.2%	14.0%	1.7%	0.8%	1.1%	29.9

	Lived with problem drinker during first 18 yrs	Number of problem drinkers lived with			Problem drinker relation					# months lived with problem drinker
		0	1	2+	Mother	Father	Mother and father	Sibling	Other	
16 years	14.2%	85.8%	12.1%	2.0%	1.9%	9.8%	1.4%	0.4%	0.7%	22.1
17+ years	13.9%	86.1%	12.1%	1.8%	1.7%	9.6%	1.2%	0.5%	0.8%	21.6
Family structure										
Marital status										
Married	18.7%	81.3%	15.9%	2.8%	1.6%	13.5%	1.7%	0.8%	1.1%	28.7
Widowed	9.3%	90.7%	8.4%	0.9%	0.4%	7.1%	0.4%	0.6%	0.8%	15.8
Divorced	22.2%	77.7%	18.4%	3.9%	2.4%	15.6%	2.5%	0.6%	1.1%	34.6
Never married	18.3%	81.7%	15.7%	2.6%	2.2%	12.6%	1.3%	1.0%	1.2%	28.0
Married to problem drinker										
Yes	31.9%	68.1%	25.5%	6.3%	2.9%	22.0%	3.7%	1.0%	2.3%	49.5
No	16.5%	83.5%	14.2%	2.3%	1.5%	11.9%	1.3%	0.7%	1.0%	25.4
Health behaviors										
Weight status										
Underweight	19.5%	80.5%	16.5%	3.0%	2.4%	13.4%	1.8%	1.0%	0.9%	28.8
Normal weight	18.1%	81.9%	15.3%	2.7%	1.9%	12.7%	1.7%	0.8%	1.0%	27.8
Overweight	17.6%	82.4%	14.9%	2.6%	1.3%	12.9%	1.4%	0.7%	1.2%	27.5
Obese	19.0%	81.0%	16.4%	2.6%	1.7%	13.7%	1.4%	0.7%	1.5%	29.5
Current drinking status										
Abstainer	11.1%	88.9%	9.6%	1.5%	0.8%	8.1%	0.5%	0.7%	1.0%	16.9
Lifetime infrequent	15.6%	84.4%	13.9%	1.7%	1.2%	11.4%	0.9%	1.0%	1.0%	24.6
Former	22.3%	77.7%	18.6%	3.6%	1.8%	16.1%	2.1%	0.8%	1.5%	36.4
Current (<1 drink/day)	19.1%	80.9%	16.2%	2.8%	2.0%	13.5%	1.8%	0.7%	1.1%	28.9
Current (1–<2 drink/day)	19.1%	81.0%	16.1%	3.0%	2.1%	13.2%	2.0%	0.8%	1.0%	28.0
Current (2 – <3 drink/day)	23.7%	76.3%	19.4%	4.3%	2.8%	15.4%	3.6%	0.6%	1.3%	32.3
Current (3+drink/day)	26.9%	73.1%	21.7%	5.2%	3.5%	17.8%	3.2%	0.9%	1.5%	41.7
Smoking status										
Never	14.7%	85.3%	12.8%	1.9%	1.4%	10.7%	1.0%	0.7%	1.0%	22.6
Former	17.6%	82.4%	15.0%	2.6%	1.6%	12.6%	1.7%	0.7%	1.0%	28.2
Current	23.9%	76.1%	19.7%	4.1%	2.3%	16.7%	2.4%	0.9%	1.5%	36.2

Source: 1988 NHIS Alcohol Supplement

Notes: The percentages for "problem drinker relation" sum to the percentage "live with problem drinker during first 18 years."

Percentages adjust for complex sampling design. N=41,308.

Table 2

Mortality Risk (Hazard Ratios) Associated with Childhood Exposure to Problem Drinking, U.S. Adults Ages 21 and Above, 1988–2011

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
Lived with problem drinker during first 18 years of life	**1.17** ***	**1.16** ***	**1.16** ***	**1.14** ***	**1.13** ***	**1.11** ***	**1.08** **
Demographic factors							
Male	1.48 ***	1.59 ***	1.62 ***	1.66 ***	1.70 ***	1.58 ***	1.51 ***
Race (non-Hispanic white)							
Non-Hispanic black	1.20 ***	1.01	0.99	0.99	0.98	0.99	0.97
Hispanic	0.87 **	0.77 ***	0.76 ***	0.76 ***	0.77 ***	0.77 ***	0.78 ***
Non-Hispanic Asian	0.56 ***	0.54 ***	0.54 ***	0.55 ***	0.55 ***	0.58 ***	0.60 ***
Other	1.08 *	1.03	1.03	1.03	1.03	1.03	1.01
Socioeconomic status							
Education		0.98 ***	0.98 ***	0.98 ***	0.98 ***	0.98 ***	0.99 **
Income (logged)		0.79 ***	0.82 ***	0.82 ***	0.82 ***	0.82 ***	0.83 ***
Family structure							
Marital status (married)							
Widowed			1.08 **	1.07 **	1.07 **	1.07 **	1.06 *
Divorced/separated			1.26 ***	1.22 ***	1.22 ***	1.20 ***	1.14 ***
Single			1.11 **	1.12 **	1.12 **	1.11 **	1.13 **
Ever married or lived with problem drinker				1.23 ***	1.22 ***	1.18 ***	1.12 ***
Health behaviors							
Weight status (normal)							
Underweight					1.51 ***	1.51 ***	1.40 ***
Overweight					0.94 **	0.94 **	0.98
Obese					1.25 ***	1.26 ***	1.33 ***
Drinking status (current, <1 per day)							
Abstainer						0.97	1.13 ***
Infrequent						1.03	1.11 ***

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
Former						1.22 ***	1.23 ***
Current, 1–<2 per day						1.15 **	1.08 +
Current, 2–<3 per day						1.68 ***	1.52 ***
Current, 3+ per day						1.87 ***	1.60 ***
Smoking status (never)							
Former							1.23 ***
Current							2.03 ***

Source: 1988–2011 NHIS-LMF.

Notes: Referent in parentheses. Models adjust for complex sampling design. N=41,308 (with 13,293 deaths).

*** p < .001;

** p < .01;

* p < .05;

+ p < .10.

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Table 3

Mortality Risk (Hazard Ratios) Associated with Types of Childhood Exposure to Problem Drinkers, U.S. Adults Ages 21 and Above, 1988–2011

Problem Drinkers during First 18 Years of Life	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
Panel A: Number of problem drinkers (0)							
1	1.15 ***	1.15 ***	1.14 ***	1.13 ***	1.12 ***	1.10 ***	1.08 *
2+	1.28 ***	1.25 **	1.24 **	1.21 **	1.23 **	1.16 *	1.10
Panel B: Relationship to problem drinker (none)							
Mother	1.23 *	1.24 *	1.23 *	1.20 +	1.20 +	1.15	1.15
Father	1.14 ***	1.13 ***	1.13 ***	1.11 ***	1.11 ***	1.09 **	1.06 +
Mother and father	1.39 **	1.37 **	1.36 **	1.33 **	1.34 **	1.26 *	1.22 +
Sibling	1.18	1.17	1.17	1.15	1.15	1.15	1.10
Other	1.21 *	1.16 +	1.18 *	1.15 +	1.14	1.12	1.07
Panel C: Number of years lived with problem drinker(s) (0)							
1–5	1.13 +	1.14 +	1.14 +	1.13 +	1.12 +	1.09	1.04
6–11	1.15 *	1.17 *	1.17 *	1.15 *	1.15 *	1.13 *	1.13 +
12+	1.18 ***	1.17 ***	1.17 ***	1.14 ***	1.14 ***	1.11 **	1.09 *

Source: 1988–2011 NHIS-LMF.

Notes: Referent in parentheses. Models adjust for complex sampling design. Each panel reflects a separate model. The models within each panel progressively add in the following variables: (1) sex and race, (2) education and income, (3) marital status, (4) ever married to or lived with problem drinker, (5) weight status, (6) drinking status, and (7) smoking. N=41,308 (with 13,293 deaths).

*** $p < .001$;

** $p < .01$;

* $p < .05$;

+ $p < .10$.

Table 4

Cause-Specific Mortality Risk (Hazard Ratios) Associated with Childhood Exposure to Problem Drinking, U.S. Adults Ages 21 and Above, 1988–2011

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
Diseases of heart	1.05	1.04	1.05	1.03	1.03	1.02	1.00
Malignant neoplasms	1.17 **	1.15 *	1.15 *	1.13 *	1.13 *	1.09	1.06
Chronic lower respiratory diseases	1.28 *	1.26 *	1.25 *	1.21 +	1.22 +	1.14	1.08
Accidents (unintentional injuries)	1.79 ***	1.73 ***	1.72 ***	1.69 ***	1.69 ***	1.64 ***	1.60 ***
Cerebrovascular diseases	1.13	1.13	1.13	1.11	1.11	1.09	1.08
Alzheimer's disease	1.08	1.09	1.09	1.04	1.05	1.04	1.04
Diabetes mellitus	1.17	1.15	1.15	1.10	1.09	1.08	1.07
Influenza and pneumonia	1.13	1.13	1.14	1.16	1.16	1.13	1.11
Nephritis, nephrotic syndrome and nephrosis	1.48 *	1.48 *	1.47 *	1.45 +	1.44 +	1.42 +	1.40 +
All other causes	1.18 **	1.18 **	1.18 **	1.16 *	1.15 *	1.13 *	1.11 +

Source: 1988–2011 NHIS-LMF.

Notes: Each cause of death is analyzed separately. Models progressively add in the following variables: (1) sex and race, (2) education and income, (3) marital status, (4) ever married to or lived with problem drinker, (5) weight status, (6) drinking status, and (7) smoking. Models adjust for complex sampling design. N=41,308 (with 3,210 diseases of heart, 3,066 malignant neoplasms, 684 resipiratory, 382 accidents, 935 cerebrovascular diseases, 299 Alzheimer's disease, 367 diabetes mellitus, 402 influenza/pneumonia, 193 nephritis, nephrotic syndrome and nephrosis, and 3,706 deaths of other causes).

*** $p < .001$;

** $p < .01$;

* $p < .05$;

+ $p < .10$.