

# Stressful life experiences, alcohol consumption, and alcohol use disorders: the epidemiologic evidence for four main types of stressors

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## Abstract

**Background** Exposure to stress is potentially important in the pathway to alcohol use and alcohol use disorders. Stressors occur at multiple time points across the life course, with varying degrees of chronicity and severity.

**Method** We review evidence from epidemiologic studies on the relationship between four different stressors (fateful/catastrophic events, child maltreatment, common adult stressful life events in interpersonal, occupational, financial, and legal domains, and minority stress) and alcohol consumption and alcohol use disorders.

**Results** Studies generally demonstrate an increase in alcohol consumption in response to exposure to terrorism or other disasters. Research has demonstrated little increase in incident alcohol use disorders, but individuals with a history of alcohol use disorders are more likely to report drinking to cope with the traumatic event. Childhood maltreatment is a

consistent risk factor for early onset of drinking in adolescence and adult alcohol use disorders, and accumulating evidence suggests that specific polymorphisms may interact with child maltreatment to increase risk for alcohol consumption and disorder. Stressful life events such as divorce and job loss increase the risk of alcohol disorders, but epidemiologic consensus on the specificity of these associations across gender has not been reached. Finally, both perceptions of discrimination and objective indicators of discrimination are associated with alcohol use and alcohol use disorders among racial/ethnic and sexual minorities.

**Conclusion** Taken together, these literatures demonstrate that exposure to stress is an important component in individual differences in risk for alcohol consumption and alcohol use disorders. However, many areas of this research remain to be studied, including greater attention to the role of various stressors in the course of alcohol use disorders and potential risk moderators when individuals are exposed to stressors.

**Keywords** Alcohol · Stress

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Exposure to stress in varying forms is an integral life experience that can provoke a variety of reactions. The last four decades have seen great growth in knowledge concerning the relationship between exposure to stressful life events and various types of psychopathology, including alcohol-related outcomes. Despite the accumulated research findings, however, many questions remain. To inform the development of effective prevention and treatments, investigators across disciplines, including epidemiologists, are involved in research to better understand the connection between stress and alcohol consumption, abuse, and dependence.

In psychiatric and substance abuse research, the term “stress” is often understood to indicate an experience denoting adversity (Dohrenwend 2000). Stress exposures consist of external stimuli that are threatening or harmful, that elicit fear, anxiety, anger, excitement, and/or sadness, and that are negative in impact and outcome (Sinha 2001; 2008). As has been noted (Lazarus 1999; Levine 2005; McEwen 2007; Selye 1976; Sinha 2008), mild to moderate levels of stress can present challenges that are within the capabilities of an individual to overcome, producing a sense of mastery and accomplishment that eventually result in a positive outcome. However, adverse experiences that exceed the coping abilities of the individual increase the risk for psychopathology.

Just as individuals vary in their capabilities, stress exposures can be viewed as varying across several dimensions. Shown in Fig. 1 is an array of dimensions upon which stressful exposures can be conceptualized. One dimension is severity, which can range from the mild, e.g., the daily hassles of family and job among healthy individuals whose basic needs are met, to the severe, e.g., extreme adversity that threatens the life, physical integrity, health and home of oneself and one's loved ones. Other dimensions, not necessarily orthogonal to each other, include whether the stressor occurred during childhood or maturity, the degree to which the stressor is acute or chronic, expected or unexpected, emotional or physical threat, and the difficulty of discerning whether the stressor was the cause or consequence of the health outcome under consideration.

When excess or pathological drinking is the health outcome of interest in epidemiologic research on stress, some aspects of the research context are useful to keep in mind. First, most research on stress and alcohol has been conducted in developed countries where alcohol is widely available and its consumption generally normative. In general population samples in these contexts, alcohol consumption is common

(Keyes et al. 2008), as are alcohol use disorders (Hasin et al. 2007b), defined as maladaptive patterns of drinking associated with impaired control that affect major life domains such as health, interpersonal, and socio-occupational functioning. Second, unlike research on stress responses such as post-traumatic stress disorder or depression, research on stress and alcohol involves access to an exogenous substance. Thus, an individual must have access to alcohol and in most cases have the means to purchase alcohol in order for alcohol consumption to be an option as a stress response.

In this review, we consider the epidemiologic evidence for a relationship between stress and alcohol outcomes, including alcohol consumption, problem drinking, and alcohol use disorders as defined in DSM-IV. We focus on the epidemiologic evidence for a relationship between alcohol outcomes in response to four types of stressful experiences: (1) fateful/catastrophic events, (2) child maltreatment, (3) common adult stressful life events in interpersonal, occupational, financial, and legal domains, and (4) minority stress. These four types of stressors all have a considerable body of literature, and exemplify life stressors that vary across a number of the dimensions listed in Fig. 1. To be included in this review, studies must have included a defined measure of exposure to these stressors. As much as possible, we focus the review on studies with large, representative samples since these are most likely to produce clearly interpretable results. Recent reviews have elucidated the literature on biomarkers that indicate the mechanisms through which stress has an effect on substance use from animal and clinical human studies (Koob 2006; Sinha 2008); in this review, we focus on epidemiologic data for the overall effect of stress on alcohol use in non-treated human populations. We divide our review of each class of stressful experiences into three sections: first, we review the placement of each category of stressor along the dimensions described in Fig. 1; second, we describe methodological issues in terms of both measurement and study design in epidemiologic studies of each stressor; and third, we describe major epidemiologic findings regarding the relationship between each stressor and alcohol consumption as well as alcohol use disorders and highlight important directions for future research.

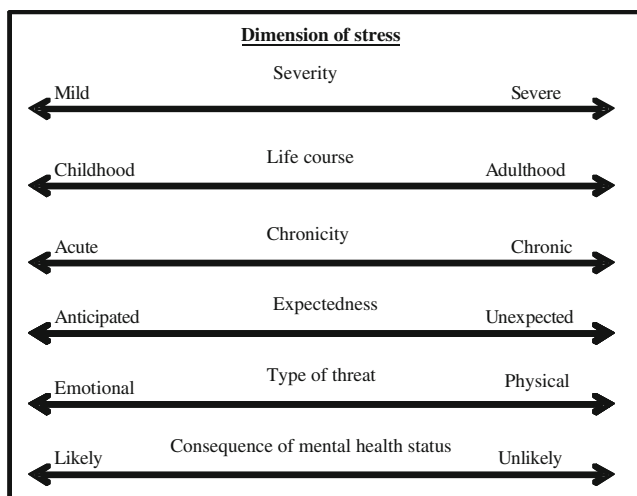


Fig 1 Dimensions of stressful experiences

### Part I. The relation between fateful/catastrophic events and alcohol use disorders

Considering the various correlated dimensions of stress in human populations described in Fig. 1, fateful/catastrophic events such as direct exposure to disaster and/or terrorism typically lies on the more extreme end of the severity continuum. These stressors are usually acute, unexpected, and exposure is very unlikely to result from an individual's

alcohol consumption. Fateful/catastrophic events can involve both physical threat to one's life and emotional threat (e.g., knowing someone lost or killed in the fateful/catastrophic incident, fear of additional exposures), and are generally not limited to a particular point in the life course.

#### Methodological issues in the study of fateful/catastrophic events

Exposure to natural and man-made disasters including terrorism is unlikely to be caused by having an alcohol use disorder, a limitation in the study of other adult stressful exposures. Thus these exposures are often considered "fateful" (Dohrenwend 2000; Dohrenwend et al. 1978; Hasin et al. 2007a; Schiff et al. 2007), creating a quasi-experimental design. In the quasi-experimental design, individuals who are exposed to a disaster should be comparable to individuals who are not exposed on all other factors associated with alcohol consumption and alcohol use disorders. Such designs make causal inference concerning the relationship between exposure and outcome much stronger (Glymour 2006). However, interpretation may depend on the specific circumstances of the event. For example, studies of exposure to nightclub disasters (fires and terrorist attacks; Kennedy et al. 2005; Mahoney et al. 2005) involve individuals who are younger and more likely to consume alcohol than the general population. The study of such events may still provide important information, but the type of individuals involved and the appropriate control group must be considered carefully. Also, some exposures that could be viewed as fateful (e.g., traumatic experiences during combat) are non-random, given evidence that joining the military and volunteering for combat are partly heritable (Lyons et al. 1993) and may be related to traits that are also related to alcohol outcomes, such as risk-taking and sensation-seeking.

#### Substantive findings regarding the relationship between fateful/catastrophic events and alcohol

##### *Studies of alcohol consumption in response to disaster*

Both in the USA and internationally, many studies have addressed the relationship between different types of natural and man-made disaster and alcohol consumption. These have included studies of natural disasters such as exposure to flood (North et al. 2004), volcano (Adams and Adams 1984), earthquakes (Shimizu et al. 2000), and hurricanes (Cerdeña et al. 2011; Kohn et al. 2005). Studies have also addressed man-made disasters such as exposure to mass shootings (North et al. 1994; Smith et al. 1999), fire/exposure to grotesque death (Green et al. 1985; Reijneveld et al. 2003; Sims and Sims 1998), ferry disaster (Joseph et al. 1993), and nuclear accidents (Kasl et al.

1981). In studies whose timeframe is a year or less, these studies consistently indicate post-disaster increases in alcohol consumption (Joseph et al. 1993; Kasl et al. 1981; Kohn et al. 2005; Reijneveld et al. 2003; Sims and Sims 1998; Smith et al. 1999). Studies with multiple and/or longer follow-ups generally find attenuation of this relationship over time (Joseph et al. 1993).

A number of studies have also addressed alcohol consumption in response to exposure to terrorism. Substantial research on mental health generally and alcohol consumption specifically has been conducted after the terrorist attacks on the World Trade Center in New York and the Pentagon in Washington D.C. on September 11, 2001 ("9/11"), indicating that alcohol consumption generally increased in both New York City and elsewhere in the short term following the attacks. Increased alcohol use was found among the following groups: survivors of the attack on the Pentagon (Grieger et al. 2003); residents of Manhattan in the 1 and/or 6 months following the attack (Ho et al. 2002; Vlahov et al. 2004; Vlahov et al. 2002); residents in the tri-state area of Connecticut, New York, and New Jersey (Melnik et al. 2002); and adults from a nationally representative sample (Stein et al. 2004). Longer-term studies show increased alcohol consumption 1 and 2 years later among New Yorkers at greater exposure levels to the attack (Boscarino et al. 2006). Few studies examined alcohol and terrorism exposure outside the USA, but two studies of adolescents in different cities in Israel showed that geographic proximity to terrorist attacks was associated with greater quantity, frequency and binge drinking (Schiff et al. 2006, 2007). A recent meta-analysis of 27 studies assessing substance use in response to terrorism included studies whose follow-up times ranged from 1 week to more than 2 years (DiMaggio et al. 2009). This meta-analysis found a pooled effect indicating that the population level of alcohol consumption is increased following a terrorist attack (DiMaggio et al. 2009).

The studies noted above are limited by lack of ability to control for pre-disaster alcohol consumption or alcohol use disorders measured before the attack. Three studies that overcame this limitation documented an increase in alcohol consumption following exposure to disaster independent of alcohol consumption levels measured prior to the exposure (Cerdeña et al. 2011; Hasin et al. 2007a; Richman et al. 2004). For example, among community heavy drinkers in New Jersey who were assessed in detail at two time points before 9/11/01, participants within five miles of the World Trade Center at the time of the 9/11 attack had significantly higher levels of alcohol consumption in the subsequent 3 months than participants who were further away, even after controlling for pre-9/11 alcohol disorders (Hasin et al. 2007a). Work is underway to better understand cognitive factors associated with post-9/11 drinking in this sample

(Beseler et al. 2011). Thus, the available evidence indicates that the population level of alcohol consumption increased following 9/11, at least over several months, with some evidence for longer-term increases in alcohol consumption among individuals with high exposure levels.

#### *Studies of problem drinking in response to disaster*

The research described above focuses on any alcohol consumption after disaster. Studies of alcohol use disorders and problem drinking following major disasters have been less consistent. Following the Oklahoma City bombings in 1995, North et al. reported no increase in incident alcohol use disorders, either in survivors of the attack (North et al. 1999) or rescue workers (North et al. 2002). Survivors of other disasters such as Hurricane Andrew (David et al. 1996), flooding (Green et al. 1992; North et al. 2004), jet crash (Smith et al. 1990), a combined sample of survivors from Oklahoma City terrorist bombings as well as the Nairobi bombing (North et al. 2005) also showed no evidence of incident alcohol use disorders. After 9/11, neither living near the attack site nor knowing someone lost or killed was associated with incident alcohol problems 6 months following the attack (Vlahov et al. 2006), and exposure to 9/11 was not associated with the trajectory of alcohol use and binge drinking in the 3 years following the attack (Cerdeña et al. 2008). In a recent pooled analysis of data from 10 different disasters including exposure to flooding, shootings, and plane crashes, North et al. again reported no evidence of increased risk for incident alcohol use disorders after these events (North et al. 2010). However, individuals with pre-existing alcohol disorders were more likely to report increased drinking after these events (North et al. 2010).

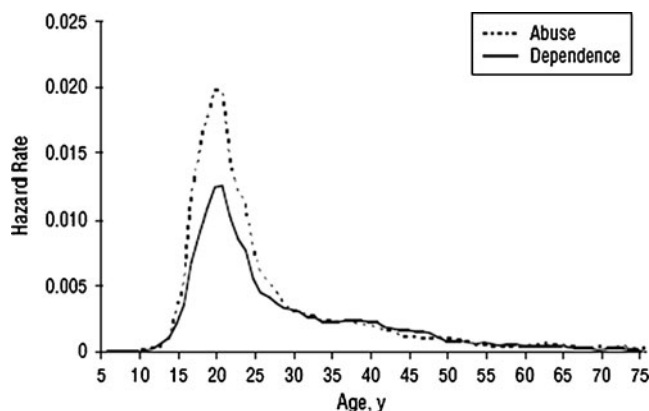
Several studies contradict the above evidence, however. Evidence from survivors of Hurricane Katrina indicates elevated rates of alcohol problems compared to national and local pre-disaster averages (Flory et al. 2009), and increases in binge drinking were found among those most exposed to the hurricane, controlling for pre-hurricane alcohol use (Cerdeña et al. 2011). Among New Yorkers interviewed 1 and 2 years after 9/11, greater exposure levels predicted binge drinking at 1 but not 2 years, and an increase in alcohol dependence at both time points (Boscarino et al. 2006). Further, 7 months after the Mount St. Helens volcano eruption, alcohol center referrals and liquor law violations had increased compared to the pre-eruption period (Adams and Adams 1984). Survivors of the Beverly Hills Supper Club fire appeared to have an increase in alcohol abuse more than 2 years after the fire (Green et al. 1985). Thus, the literature is inconsistent on the role of fateful traumatic events in the development of alcohol disorders. Note that studies of incident alcohol use disorders after major disasters were conducted in adult

populations, wherein the risk for an incident alcohol use disorder is low (Hasin et al. 2007b). As shown in Fig. 2, the hazard rates for the age of onset of alcohol abuse and dependence in the general population (Hasin et al. 2007b) are highest in the late teens and early 20s, and negligible after age 30. Since early-onset drinking is strongly and consistently related to the risk for later alcohol use disorders (Grant and Dawson 1997), studies of incident alcohol use disorder risk following exposure to disaster in adolescent and young adult populations are necessary to comprehensively understand the relation between disaster and alcohol use disorders.

Taken together, there remain questions about the role of traumatic event exposure in alcohol consumption and the development of alcohol use disorders. Accumulating evidence suggests increases in alcohol consumption in the short term following exposure to trauma even among individuals with no history of alcohol use disorders, but there is less consistent evidence on the duration of this relationship at the population level. Few incident alcohol use disorders occur in response to traumatic events among adults. However, individuals with pre-existing or past alcohol use disorders may be more likely to experience relapse to problematic drinking or alcohol use disorders after trauma exposure. Such relapses may carry as much personal and societal burden as incident disorders. Thus, public health efforts following disasters should include assessment and proper referral of individuals at risk for post-disaster problematic drinking.

## **Part II. The relation between child maltreatment and alcohol use disorders**

Childhood maltreatment includes many adverse exposures (sexual, emotional and physical abuse, emotional or physical neglect) during the first 18 years of life.



**Fig 2** Hazard rates for age of onset of DSM-IV alcohol abuse and dependence. Reprinted from Hasin et al. (2007b)



Considering the various correlated dimensions of stress in human populations described in Fig. 1, childhood maltreatment experiences range from mild (e.g., occasionally saying hurtful things) to severe (e.g., chronic physical and/or sexual abuse). Further, these stressors can be acute but are often chronic throughout childhood, and are implausibly a consequence of alcohol consumption as they typically occur before drinking initiation. Childhood maltreatment can involve both physical threat (e.g., physical and sexual child abuse, physical neglect of needs) and emotional threat (e.g., emotional abuse and neglect). These experiences are common, and may account for a significant proportion of all adult psychopathology (Afifi et al. 2008; Green et al. 2010). Further, events frequently co-occur (Dong et al. 2004; Dube et al. 2002; Edwards et al. 2003; Finkelhor et al. 2007), such that exposure to one type of childhood maltreatment increases the risk of exposure to others.

#### Methodological issues in the study of child maltreatment

Concerning measurement, most studies use retrospective reporting of adverse childhood events among adults to determine exposure status as this is the most feasible means for data collection (Green et al. 2010; Hardt and Rutter 2004). However, several studies have used court and social service records as the exposure measurement (Kaufman et al. 2007; Thornberry et al. 2001; Widom et al. 1995, 2007b). Child abuse self-reports, including childhood sexual abuse, are unstable over time (Fergusson et al. 2000; Johnson et al. 1999; Maughan and Rutter 1997; Widom 1996, 1997; Widom et al. 1999). Reporting instability appears related to several factors, including the context, salience, and severity of the event(s), closeness to the perpetrator, time since the event(s) occurred, and social desirability in the context of the interview (Hardt and Rutter 2004; Widom 1996, 1997). Available evidence from longitudinal data with multiple assessments of maltreatment and latent class analyses of report status (Fergusson et al. 2000) and comparison between court records and self-report (Widom 1996, 1997) indicate that few reports are false positives, indicating that the main result of unstable reporting is likely to be underestimation of the prevalence of adverse childhood events which could weaken but not bias apparent associations. Further, evidence from the Christchurch Health and Development study showed that underestimation is unrelated to psychiatric diagnosis at the time of the interview, and that relative risks for the association between childhood maltreatment and alcohol use disorders remained stable when maltreatment was assessed at multiple time points (Fergusson et al. 2000). Thus, the associations between maltreatment and alcohol use are unlikely to be fully explained by biased retrospective reporting.

Substantive findings regarding the relationship between child maltreatment and alcohol consumption and disorders

Epidemiologic studies addressing the role of adverse childhood events on alcohol consumption and alcohol use disorders have employed several types of designs, including cross-sectional studies of adults with retrospective assessment of adverse childhood events, prospective cohort studies, and twin and other genetically informed samples. Studies have generally shown that most forms of child maltreatment are related to higher risk of adolescent alcohol consumption (Bensley et al. 1999; Hussey et al. 2006; Sartor et al. 2007; Thornberry et al. 2001) and adult alcohol consumption and alcohol disorders (Anda et al. 2002; MacMillan et al. 2001; Molnar et al. 2001; Nelson et al. 2006). A review documented that childhood maltreatment and other childhood stressors were associated with earlier onset of adolescent alcohol consumption and with alcohol use disorders in adulthood (Enoch 2010). However, two issues may either confound or modify the apparent relationship between early maltreatment and adult alcohol use disorders: (1) family history or genetic vulnerability to alcohol problems, and (2) comorbid psychopathology.

Family history of alcoholism may confound the relationship between early maltreatment and adult alcohol use disorders because child maltreatment is more likely to occur among children of alcoholics (Gilbert et al. 2009), who may not only engage in harmful parenting practices (Kettinger et al. 2000; Stanger et al. 2004; Suchman et al. 2007, 2008) but who may also pass along genes increasing risk of alcohol disorders to their offspring. While studies using animal model overcome this difficulty and suggest extended stress in early life leads to later self-administration of alcohol (Cruz et al. 2008; Miczek et al. 2008), some epidemiologic studies suggest that the relationship between maltreatment and alcohol disorders may be at least partially confounded by family history of alcohol problems. For example, a prospective cohort study of Midwest court-recorded cases of abuse and neglect compared to matched community controls found no remaining association between early abuse and adult DSM-III-R alcohol use disorders after controlling for family history of alcohol problems among men (Widom et al. 1995, 2007b), although among women, physical neglect remained associated with alcohol use disorders. A confounding role of family history was also suggested by Sher et al. (1997) in a prospective convenience sample of college freshman. Finally, a population-based longitudinal study of adult female twins concordant for a positive history of sexual abuse indicated no residual association between childhood sexual abuse and alcohol disorders (Bulik et al. 2001).

However, several studies that controlled for family history of alcoholism indicate a persistent relationship

between childhood adverse events and adult risk for alcohol use disorders, including parental divorce (Pilowsky et al. 2009; Thompson et al. 2008), and death of a parent or foster home placement (Pilowsky et al. 2009; Kendler et al. 1996). Further, one study documented strong and significantly increased odds of alcohol disorders based on retrospective assessment of childhood sexual abuse among same-sex twins in Australia (Nelson et al. 2002), even after controlling for family background variables including parental alcohol problems. Finally, recent data from a population-based study of twins in Virginia reported that participants who reported any maltreatment were 1.74 times as likely to experience an alcohol use disorder in adulthood; while controlling for family level risk factors substantially attenuated the observed association, a direct effect after control remained (Young-Wolff et al. 2011). Thus, the literature is inconsistent on whether family history of alcohol problems confounds the association of child maltreatment and adult alcohol use disorders, suggesting continued attention to this issue in further studies.

Information about possible modifying effects of genetic vulnerability comes from studies of specific genetic variants. The mapping of the human genome enables investigation of this possible source of variation through analysis of specific genetic polymorphisms. The finding that functional polymorphisms in the monoamine oxidase A enzyme (Caspi et al. 2002) interact with childhood maltreatment to predict antisocial behavior in adulthood stimulated research on whether this effect generalizes to substance use disorders, but thus far, the findings did not replicate (Young et al. 2006). The serotonin transporter promoter variant, 5-HTTLPR, has been studied in interaction with stressful experiences in a wide variety of psychiatric outcomes in several studies after initial publication of such an interaction result for major depression (Caspi et al. 2003). For example, youth with court-documented maltreatment were at higher risk for early onset alcohol use if they had the heterozygous (*s/l*) genotype compared to the *ll* genotype (Kaufman et al. 2007). In another youth study, the effect of the same heterozygous genotype on increased risk for substance use was attenuated in families providing involved-supportive parenting (Brody et al. 2009a). In an innovative step involving random assignment of the environment, the investigators then randomized at-risk families to an intervention designed to increase involved-supportive parenting or a control condition (Brody et al. 2009b). Among those with the heterozygous 5-HTTLPR genotype, children in treated families had less substance use at follow-up compared to children of the control families (Brody et al. 2009b). Taken together, these studies suggest that the risk for later alcohol outcomes is affected by an interaction of stressful early home environments and genetic vulnerability.

Psychiatric comorbidity may also confound the relationship between early maltreatment and alcohol disorders because maltreatment affects multiple psychiatric disorders (Green et al. 2010; Kendler et al. 2000; Kessler et al. 1997; Widom et al. 2007a); and alcohol use disorders are highly co-morbid with other forms of psychopathology (Hasin et al. 2007b). Data from the National Comorbidity Survey indicated that no adverse childhood experiences were associated with alcohol dependence in adulthood once comorbid psychopathology was controlled, although repeated molestation and a physically/verbally aggressive mother were associated with alcohol abuse (Kessler et al. 1997). The propensity to express psychopathology increases with the number of events experienced, and associations are strongest with risk for psychiatric disorders in early adulthood rather than with their persistence (McLaughlin et al. 2010a). Thus, careful examination of the effects of comorbidity is necessary in studies of early stressful exposures and adult outcomes.

These data suggest that the association between childhood maltreatment and adult alcohol use disorders is complex to measure, analyze, and interpret. While many studies document a strong relationship between maltreatment and alcohol use, those studies that control for family history of alcohol problems and/or co-morbid psychopathology find attenuation of the relationship. Genetic epidemiologic findings suggest that genetic vulnerability may interact with rather than confound the association between maltreatment and alcohol disorders; however, suggesting that further investigation of these pathways in genetically informed samples is crucial for advancing this literature.

### **Part III. The relation between interpersonal, occupational, financial, and legal stressful life events and alcohol use disorders**

Stressors in adulthood related to interpersonal relationships, occupation, personal finance, and legal problems can have a profoundly negative impact on mental health (Greeley and Oei 1999). However, these stressful events could as easily be consequences or risk factors of alcohol disorders, creating ambiguity in interpreting associations between these stressors and alcohol outcomes. Compared to other stressors we discuss in this review and considering the dimensions described in Fig. 1, the events described in this section generally lie on the milder end of the severity continuum, though with considerable variability in severity. Further, these stressors are most often acute, although some may be chronic (e.g., unemployment during economic recessions). The events often

involve emotional threat (e.g., divorce) but could involve physical threat as well (e.g., being the victim of a violent crime).

Methodological issues in the study of interpersonal, occupational, financial, and legal stressful life events

While the measurement of stressful life events in psychiatric epidemiologic research has been a source of debate for decades (Brown 1993; Dohrenwend 2000, 2006; Wheatron 1994), the most commonly used method of measuring interpersonal, occupational, financial, and legal stressful life events is through subjective response in the form of a checklist or inventory. Typically, these questionnaires include 10 to 20 experiences thought to be stressful that are self-reported by the respondent (e.g., Cohen et al. 1983; Dohrenwend et al. 1978; Grant et al. 2004; Holmes and Rahe 1967; Paykel et al. 1971; Sarason et al. 1978). Shown

in Box 1 is a list of common interpersonal, occupational, financial, and legal stressors that are often examined in epidemiologic studies, e.g., divorce, death of a relative, and job loss. Research participants indicate whether these experiences happened to them and in what time frame. Test–retest reliability studies of these scales indicate fair reliability (Klein and Rubovits 1987; Rahe et al. 1970; Ruan et al. 2008). As noted by Dohrenwend (2006), however, these scales have a number of problems, including “intracategory variability,” the lack of specific meaning within categories of stressful life events (Dohrenwend 2006). For example, losing a job is often included on stressful life event checklists, but the meaning of the lost job is different for one who experiences a plant shut down after 30 years in the same position compared to an artist or a musician accustomed to temporary work. Similarly, divorce can be devastating to one member of a couple and a relief to the other.

### **Box 1. Acute stressful life events often used in epidemiologic research of stress and alcohol**

Death or serious illness in a family member and/or close friend

Moved residence

Fired or laid off from job

Major financial crisis, bankruptcy, or problems paying bills

Trouble with boss or coworker

Separation, divorce, or breakup of a romantic relationship

Serious interpersonal problems with a friend, neighbor, or relative

Trouble with police, got arrested or sent to jail

Victim of a violent crime

Additionally, self-reported stressful life events may be subject to biased reporting, as negative events are more likely to be recalled and may be described as more salient when individuals are experiencing a psychiatric disorder (Brown 1989; Dohrenwend et al. 1984; Kihlstrom et al. 2000; Meyer 2003a; Schwarz 2007). Given the issues involved with self-reported stressful life events, some investigators have worked with methods to measure stressful events more objectively, or independently, of the participant’s perceptions or appraisals of the event as stressful by including assessments by independent raters (Dohrenwend 2000; Lazarus 1999; Meyer et al. 2008; Monroe 2008; Wethington et al. 1995). Despite the limitations of subjective self-report measures of stress, however, they capture how individuals respond to, cope

with, and adapt to stressful experiences, which might be important for the development of psychiatric disorders (Clark et al. 1999; Williams et al. 2003). Thus, while imperfect, subjective self-reports of stress provide useful information and are thus commonly used in epidemiologic studies.

Further, daily interpersonal stress exposures such as problems at work, trouble with the police, or breakup of romantic relationships are likely to be influenced by having an alcohol disorder. While these exposures are likely to be stressful for anyone experiencing them, they can be as much a consequence of an alcohol use disorder as a cause. Stressful life events often co-occur, and individuals who experience multiple stressful life events may be different than individuals who do not experience such events on an

underlying factor that also influences the risk for excess alcohol consumption and alcohol use disorders. For example, stressful experiences and alcohol use disorders are both heritable, and some have suggested a common genetic vulnerability to both of these experiences (Lyons et al. 1993; Sher et al. 2005). Thus, teasing apart the temporal and causal directions of relationships between these adult stressors and alcohol is a difficult task in general population epidemiologic samples. Despite these limitations, research into the effects of these exposures is important from a public health perspective as they are common in the general population.

Substantive findings regarding the relationship between interpersonal, occupational, financial, and legal stressful life events and alcohol

Surprisingly few large-scale epidemiologic studies have examined the association of interpersonal, occupational, financial, and legal stressful life events with alcohol use and disorder. Various studies in small to medium adult community samples have found that counts of perceived stressful life events are associated with alcohol consumption and problem alcohol use (not necessarily alcohol use disorders; Cole et al. 1990; King et al. 2003), though a population-based longitudinal study of older adults (mean age, 61) did not demonstrate long-term effects (i.e., 1 year or greater) of acute stressful life events on patterns of alcohol consumption (Skaff et al. 1999). A national prospective study of 3,006 women found increased risk of alcohol abuse after being an assault victim, and no evidence of reverse causation (that alcohol consumption alone contributed to the risk for assault) (Kilpatrick et al. 1997). Further, in a larger, longitudinal study of college students ( $N=295$ ) in which reports of stressful events, alcohol, and drugs were reported daily for 2 years on the internet, students homozygous for the *s* allele of 5-HTTLPR were at substantially increased risk for heavy drinking and drug use if they experienced a high level of stressful life events (Covault et al. 2007). Thus, consistent with studies on gene by environment interaction in early maltreatment and alcohol outcomes, evidence suggests that these relationships may be modified by genetic factors, suggesting value in further studies of this type among varied samples.

Some epidemiologic studies suggest gender differences in the exposure to and effects of acute stressors. Women report more stressful life events than men (Kendler et al. 2001), and the stressful life events commonly reported by men and women differ (Kessler et al. 1985; Sieber 1974). Despite the greater number and different nature of stressful experiences in the lives of women, research has been inconsistent as to whether stressful experiences are more predictive of alcohol disorders in men or women. Smaller

community-based samples have demonstrated that the relationship between stressful life events and alcohol consumption is stronger in women than men (Allan and Cooke 1985; King et al. 2003; Rospenda et al. 2008), though men are more likely to report drinking to cope with distress (Nolen-Hoeksema and Harrell 2002) and drinking to relieve tension (Armeli et al. 2000). However, two larger, more representative samples have shown the opposite relationship: stressful life events are more strongly associated with alcohol consumption and alcohol use disorders in men compared to women (Dawson et al. 2005; San José et al. 2000). Data from the National Epidemiologic Survey on Alcohol and Related Conditions found that the number of past-year stressors was associated with alcohol consumption and binge drinking in both men and women, but that the relationship was stronger for men; each additional stressor increased the odds of heavy drinking by 24% (odds ratio for each additional stressor equaled 1.24) in men and 13% (odds ratio for each additional stressor equaled 1.13) in women (Dawson et al. 2005). Further, events such as divorce are consistently associated with an increase in drinking among men, but inconsistently associated with drinking among women. For example, Wilsnack et al. (1991) demonstrated an inverse relationship between divorce and drinking in a nationally representative survey of women (Wilsnack et al. 1991). Finally, several general population studies have found an increase in incident alcohol use disorders following job loss, particularly among men (Catalano et al. 1993; Crawford et al. 1987; Lahelma et al. 1995). Given that this literature remains inconsistent, more data from large-scale studies with bearing on these relationships would be beneficial to advance this literature.

#### **Part IV. The relation between minority stress and alcohol use disorders**

Minority stress is defined as exposure to specific stressors that result from minority status, especially prejudice and discrimination events (Meyer 2003b; Williams et al. 2003). These events range from mild (e.g., daily hassles such as being followed in a store) to more severe (e.g., being a victim of a violent crime), and include both emotional (e.g., workplace harassment; Waldo 1999) and physical (e.g., hate crimes; Herek 2009) threats to self. Minority status cannot be attributed to having an alcohol disorder, making one aspect of interpretation straightforward in studies in this area. Although minority stress can involve acute events, it is most frequently viewed as a chronic exposure that occurs across the entire life course (Williams et al. 2003). Finally, as depicted in Fig. 1, stressors vary with respect to whether they are expected. Research has indicated that while many stressors that members of minority groups confront are



unanticipated, one consequence of repeated exposure to discrimination is that individuals begin to expect rejection based on their stigmatized identity (Mendoza-Denton et al. 2002).

#### Methodological issues in the study of minority stress

Two salient methodological points are important in understanding the literature on minority stress and alcohol use. First, studies tend to assess discrimination due to a single aspect (e.g., race or sexual minority status or gender), but individuals have multiple domains of identity, all of which might be important in determining the amount of stress engendered by prejudice and/or discrimination (Meyer 2003b). For example, data from the National Epidemiologic Survey on Alcohol and Related Conditions indicated that among lesbians, gays, and bisexuals (LGBs), the risk of substance use disorders increased as a function of the number of domains in which discrimination was reported (McCabe et al. 2010). Thus, individuals who reported discrimination due to gender, race, and sexual orientation had a higher risk of substance use disorders compared to those who reported discrimination due to sexual orientation alone. Studies exploring the role of multiple identities in shaping the stress response and subsequent health outcomes, including alcohol use and abuse, are important to advance this literature.

Second, almost all studies described in this section use self-report of perceived discrimination experiences to measure exposure to discrimination. Although this research may capture how minority group members construe their experience of discrimination, these measures could be confounded with mental health status (Meyer 2003b), which may lead to biased associations between stress and alcohol use. Consequently, more recent research (discussed below) has attempted to utilize objective, ecologic measures of stress exposure that are outside the control of the individual, such as institutional discrimination (Hatzenbuehler et al. 2010).

Below, we briefly review the research for two specific minority groups: racial/ethnic minorities and sexual minorities (LGBs). While substantial research is available regarding alcohol use disorders among other minority groups (e.g., women, physically disabled, religious minorities), we focus on these two groups as they highlight some of the methodological issues and enduring paradoxes of this work. Although this section considers minority stress as one mechanism explaining alcohol use among racial/ethnic and sexual minority groups, we recognize that there are alternative pathways, such as social disadvantage (e.g., poverty level and cumulative disadvantage; Mulia et al. 2008) and social norms (e.g., Hatzenbuehler et al. 2008).

#### Racial/ethnic minorities

According to minority stress models, the stress resulting from prejudice and discrimination should lead to elevations in alcohol use among minority group members. Patterns of alcohol use among racial/ethnic minorities, however, fail to correspond to what would be predicted under a straightforward minority stress-psychopathology model. While Native Americans have higher rates of alcohol consumption and alcohol use disorders compared with non-Hispanic Whites (Hasin et al. 2007b), several large surveys indicate lower rates of alcohol consumption and alcohol use disorders among non-Hispanic Blacks, Asians, and Hispanics compared to Whites (Breslau et al. 2006; Hasin et al. 2007b; Kessler et al. 1994). These minority groups also have lower rates of other psychiatric disorders such as major depression, leading to what has been called the ‘minority paradox’ (Williams 2001) in mental health research: minority groups such as Blacks and Hispanics have lower rates of psychiatric and substance disorders despite greater exposure to institutional and interpersonal discrimination which have been shown to engender substantial stress via biological (Lewis et al. 2006) and psychological (Hatzenbuehler 2009) mechanisms. In contrast to findings from the between-group studies, within-group studies consistently show that perceived discrimination is associated with alcohol outcomes, including studies of Blacks (McLaughlin et al. 2010b; Richman et al. 1996; Taylor and Jackson 1990; Yen et al. 1999), Filipino-Americans (Gee et al. 2007), and Asian-American adolescents (Yoo et al. 2010).

#### Sexual minorities

In contrast to racial/ethnic minorities, LGB individuals have higher rates of substance use and substance use disorders than their heterosexual peers (Garofalo et al. 1998; Russell et al. 2002; Ziyadeh et al. 2007), including among adolescents (Eisenberg and Wechsler 2003; Hatzenbuehler et al. 2008) and adults (Burgard et al. 2005; Cochran et al. 2000; Drabble et al. 2005). Although research has tended to examine perceived discrimination as a risk factor for internalizing psychopathology such as depression and anxiety, recent studies have shown higher levels of alcohol use (Hatzenbuehler et al. 2011) and alcohol use disorders (McCabe et al. 2010) among LGBs who perceive that they have experienced higher levels of discrimination.

Because of the design of these studies, they are not able to rule out reverse causality (i.e., individuals with alcohol problems may perceive and report greater discrimination). In order to address some of these methodological limitations of subjective measures of discrimination, recent studies have developed novel measures for operationalizing objective stressors that LGB individuals confront, including

institutional forms of discrimination (e.g., anti-marriage laws, employment discrimination policies). Given that these institutional stressors occur outside the control of LGB individuals, they are not confounded with mental health status and therefore provide a stronger test of the effect of discrimination on mental health than measures of subjective stress. Literature is emerging documenting the relationship between these objective stressors and LGB health, including alcohol use. For example, a recent study examined the impact of state-level ballot initiatives banning gay marriage on the prevalence of psychiatric and substance use disorders in LGB populations (Hatzenbuehler et al. 2010). Results indicated that LGB respondents living in states that passed such bans in 2004 had significantly greater increases in psychiatric and alcohol use disorders than LGB respondents in states that did not pass such bans (Hatzenbuehler et al. 2010). This research demonstrates the potential importance of incorporating more objectively defined indices of social stress into research on alcohol use among minority populations. Indeed, an examination of how and why such social stressors contribute to the development and maintenance of alcohol use disorders within LGB populations represents a crucial avenue for future inquiry.

In sum, despite the fact that both Blacks and LGBs experience multiple minority stressors across the life course, only LGB populations appear to be at increased risk for psychiatric morbidity, including alcohol disorders (Schwartz and Meyer 2010). Studies are needed that explore the underlying reasons that can explain the apparent inconsistencies in the minority stress-alcohol literature. LGBs and racial ethnic minorities differ in multiple respects that might have important consequences for alcohol use, including the availability of social support and developmental timing of when these minority identities become salient. Indeed, some researchers have hypothesized that African-American populations have adopted strong resilience and coping mechanisms to combat minority stress (Clark et al. 1999) that may buffer against the development of substance use, perhaps as a result of racial socialization early in development (Hughes et al. 2006). In support of this hypothesis, a daily diary study of LGB and African-American young adults found that on days in which minority stressors were reported, African-Americans were more likely to engage in some adaptive coping strategies (e.g., seeking out social support) than were LGB respondents (Hatzenbuehler et al. 2009). Another dimension on which these two groups differ is the concealability of the stigma. In contrast to racial/ethnic minority status, sexual minority status is considered more concealable. On the one hand, this concealability might be associated with less discrimination and resulting stress, as individuals can choose when and with whom to reveal a sexual minority status. On the

other hand, those with concealed stigmas often experience greater stress than individuals with conspicuous stigmas (Pachankis 2007), perhaps because those with concealed stigmas have fewer opportunities for support from others who share their stigma (Frable et al. 1998). These and other potential explanations warrant further empirical testing.

#### *Implications and prescriptions for future research*

While the present review highlights many methodological challenges in conducting epidemiologic research on stress and alcohol, we also demonstrate remarkable consistency in the findings that major life stressors experienced by the general population are associated with increased risk of alcohol consumption and alcohol use disorders. Below we highlight the implications of these findings, and offer seven research areas in need of greater attention by the field of epidemiology.

First, as noted throughout this review, certain types of stressful life events are likely to be a cause as well as a consequence of alcohol consumption and alcohol use disorders. Below we describe several new methods aimed at resolving some of the temporality issues within existing study designs. Propensity analysis aims to mitigate the problem of reverse causation by carefully controlling confounding; these analyses have been applied to depression outcomes (Kendler and Gardner 2010), but have not yet been applied to alcohol outcomes. While propensity analysis deals with confounding issues, few studies have attempted to explore and model the interplay between stress and alcohol use disorders as iterative and dynamic processes. This type of analysis is difficult to conduct in traditional regression frameworks that do not allow for feedback loops and make assumptions about static processes. Dynamic systems models are an approach to modeling the dynamic interplay between stress and alcohol use. These are increasingly utilized by alcohol researchers to more accurately model dynamic processes other than stressful life events in human populations (Galea et al. 2010; Gruenewald and Johnson 2010; Gruenewald et al. 2010; Yu et al. 2009). No systems research has yet attempted to address the role of stress on alcohol consumption over time in the general population, but such research could aid in the etiologic investigation and specific estimation of effects of stressful life events on alcohol consumption, and also estimate the population-expected reduction in alcohol consumption and alcohol use disorders given the prevention of certain stressors. Such information can be useful for public health planning and resource allocation. None of these methods, however, can overcome the limitations of study design. Many of studies cited here are cross-sectionally collected samples with one-time measurement of stress and alcohol; tempo-

rality and reciprocity issues can only be resolved with prospective data and multiple time points of measurement.

Second, the literature addressing the interaction of genes and environment is still in early phases and studies are few. In this review, we note that allelic variation in risk genes modifies the relationship of childhood maltreatment or college-age daily stressors and alcohol consumption. Genetic data are increasingly collected in epidemiologic studies, so a greater variety of stressors and genetic variants can be tested in future studies to maximize our understanding of these relationships. In addition to examining biological factors (i.e., genetics) as effect modifiers, future research is also needed to identify biological factors as mediators linking stress to alcohol use and alcohol disorders. There is a growing literature showing that individuals exposed to highly stressful conditions have dysregulated physiological stress response systems (Chen et al. 2010; Evans and Kim 2007). In turn, HPA axis reactivity has been associated with multiple adverse health outcomes, including substance use (Daughters et al. 2009; Sinha 2008; Childs and De Wit 2009). These and other potential biological mechanisms warrant further investigation.

Third, checklists of stressful situations by respondents in epidemiologic surveys are a measurement approach that serves an important function due to their relative ease of administration and low cost. However, given the methodological issues outlined above, studies should utilize multiple measures of stressful life events whenever possible. For example, approaches designed by Brown et al. (Brown et al. 1995a,b) and used in epidemiologic research by Kendler et al. (2003) focus on more nuanced aspects of the stressful experience, categorizing each experience into non-mutually exclusive categories of loss, humiliation, entrapment, and danger. Research has shown differential relationships between each of these dimensions of a stressful experience and psychopathology, thus documentation of the effect of both the events themselves and the psychological dimensions of the events can be documented. Increasingly, researchers in epidemiology have been incorporating objective measures of stressful environments into research on substance use disorders. For example, the study described above using the objectively measured social context of LGBs shows how subjective, possibly biased aspects of stress reporting can be removed from the measure (Hatzenbuehler et al. 2010). Dohrenwend et al. (Dohrenwend 2006; Dohrenwend et al. 1993, 1978) advocate the use of independent assessments of respondent reported stressful events; this approach is more timely and costly than respondent-driven checklists of stressful events, but when feasible provides well-validated assessment of stress. Researchers in other health areas have used a transdisciplinary approach to characterizing stress; for example, neighborhood stress has been captured by medical

sociologists through observation and systematic recording of events and conditions of neighborhoods (Cohen et al. 2000; Sampson and Raudenbush 1999). Such an approach would be beneficial to add to subjective reports of stress to fully understand the relationships between objective and subjective forms of stress and alcohol.

Fourth, limited research has been conducted on the role of population-level economic recession on alcohol use and alcohol use disorders. A population-based study in Finland indicated that the relationship between job loss and alcohol consumption was greater during economic recessions than periods of economic growth (Luoto et al. 1998). Given the current worldwide economic downturn, the effects of economic stressors at the population-level on individual-level patterns of alcohol consumption and alcohol use disorders remain an important gap in the stress literature.

Fifth, meta-analyses of the effects of stressors on alcohol consumption are difficult to conduct given the wide variation in measurements of stress. However, an accumulation of studies has begun to facilitate this important type of review for exposure to terrorism (DiMaggio et al. 2009). Additional meta-analyses focused on specific types of stress or specific populations would be very useful contributions to summarize the accumulating epidemiologic data on stress and alcohol. Further, meta-analysis would allow for the estimation of pooled effect sizes for many of these exposures; given the large sample sizes reported here and the use of the odds ratio as the most common measure of association, it is possible that effects are statistically although not clinically significant. Studies examining the magnitude of these effects and the potential impact on public health of intervention are necessary.

Sixth, few large-scale epidemiologic studies have assessed the role of stressful life events on the course of alcohol use disorders after initial onset. Stress has primarily been examined as a risk factor for the adult incidence of alcohol disorders. Given that the age of onset sharply peaks in late adolescence and early adulthood (Fig. 2), those with a lifetime tendency to drink heavily or in a problematic manner, including after exposure to trauma, will mainly have already had their lifetime onset prior to adult trauma exposure. For these individuals, the risk for lifetime incidence is not what is clinically important, but rather, the potential for a relapse that might not otherwise have occurred. Clinical samples have documented that patients report stressful life events as a common trigger for relapse (Bottlender and Soyka 2005; Breese et al. 2005; Brown et al. 1990; Brown et al. 1995a,b; Vielva and Iraurgi 2001; Walter et al. 2006). Animal models have established that stress-related reinstatement of alcohol self-administration is possible (Gass and Olive 2007; Mason et al. 2009). However, few epidemiologic studies have addressed stressful events prospectively as a risk factor for exacerbation or

relapse of alcohol use disorders in the general population. Given the potential costs of such outcomes (loss of jobs, relationships, and even suicide; Hallberg and Mattsson 1990), we suggest that this issue receive greater attention in studies of post-disaster drinking and other types of stress exposures.

Finally, limited data exist to evaluate the effect of stress engendered by discrimination on the course of alcohol consumption and alcohol use disorders among racial/ethnic minorities. A small study showed that perceived discrimination is associated with an earlier onset of alcohol use among American Indian adolescents (Whitbeck et al. 2001). No published studies have thus far evaluated whether individuals at higher levels of stress due to perceived discrimination are less likely to seek treatment or remit in their alcohol use disorder, and important area for future research.

In conclusion, the psychological and psychiatric effects of stress remain an important mechanism for individual differences in all areas of mental health. We have demonstrated here the substantial evidence for effects on alcohol consumption and alcohol use disorders of fateful/catastrophic events such as exposure to disaster and terrorism, childhood adversities such as maltreatment, interpersonal stressors such as divorce and job loss, and chronic minority stress. While these data demonstrate the importance of stress in the development of alcohol problems in human populations, substantial work remains to be done in these areas. Refined measures of stress exposures, careful assessment of confounding and reverse causation, an examination of alcohol disorder course, including relapse, and the potentiating of stress effects by genetic vulnerability remain important topic areas in need of more epidemiologic study. By exploring the epidemiology of stress in human populations, we can integrate and translate work in experimental human and animal models in order to demonstrate the real world effects of these common yet often devastating exposures.

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