Stress, January 2013; 16(1): 3–15

ISSN 1025-3890 print/ISSN 1607-8888 online
DOI: 10.3109/10253890.2012.671397

informa
healthcare

Self-reported impulsivity, but not behavioral choice or response impulsivity, partially mediates the effect of stress on drinking behavior

KRISTEN R. HAMILTON[3], EMILY B. ANSELL[3], BRADY REYNOLDS[2], MARC N. POTENZA[1,3,4], & RAJITA SINHA[1,3]

[1]*Child Study Center, Yale University School of Medicine, New Haven, CT, USA,* [2]*Research Institute at Nationwide Children's Hospital and Department of Pediatrics, Ohio State University, Columbus, OH, USA,* [3]*Department of Psychiatry, Yale University School of Medicine, New Haven, CT, USA, and* [4]*Department of Neurobiology, Yale University School of Medicine, New Haven, CT, USA*

(*Received 29 September 2011; revised 23 February 2012; accepted 26 February 2012*)

Abstract
Stress and impulsivity contribute to alcohol use, and stress may also act via impulsivity to increase drinking behavior. Impulsivity represents a multi-faceted construct and self-report and behavioral assessments may effectively capture distinct clinically relevant factors. The present research investigated whether aspects of impulsivity mediate the effect of stress on alcohol use. A community-based sample of 192 men and women was assessed on measures of cumulative stress, alcohol use, self-reported impulsivity, and behavioral choice and response impulsivity. Data were analyzed using regression and bootstrapping techniques to estimate indirect effects of stress on drinking via impulsivity. Cumulative adversity exhibited both direct effects and indirect effects (via self-reported impulsivity) on drinking behavior. Additional models examining specific types of stress indicated direct and indirect effects of trauma and recent life events, and indirect effects of major life events and chronic stressors on drinking behavior. Overall, cumulative stress was associated with increased drinking behavior, and this effect was partially mediated by self-reported impulsivity. Self-reported impulsivity also mediated the effects of different types of stress on drinking behavior. These findings highlight the value of mediation models to examine the pathways through which different types of stress increase drinking behavior. Treatment and prevention strategies should focus on enhancing stress management and self-control.

Keywords: *Alcohol use, self-reported impulsivity, choice impulsivity, response impulsivity, mediation, stress*

Introduction

The association of excessive alcohol use with public health problems and mortality underscores the importance of examining factors that impact alcohol use (Room et al. 2005). Stress and impulsivity are associated with increased drinking behavior (Evenden and Ryan 1999; Sinha 2008; Lejuez et al. 2010), and stress may also act via impulsivity to influence alcohol consumption (Fox et al. 2010). Stress has been associated with increases in impulsivity (Glass and Singer 1972; Cohen 1980; Muraven and Baumeister 2000; Sinha 2001; Tice et al. 2001). Because stress has been associated with increases in impulsivity, and impulsivity is associated with increased alcohol use, impulsivity may mediate the effect of stress to increase alcohol use. Determining whether impulsivity mediates the stress and alcohol use relationship is important for treatment and prevention efforts. If impulsivity mediates the effect of stress to increase alcohol use, then treatments targeted to reduce impulsivity may diminish the effect of stress on hazardous drinking.

In the past decade, research attention has focused on impulsivity because of its role in detrimental behaviors, including excessive alcohol use (Lejuez et al. 2010). Impulsivity, a tendency to act rapidly with diminished regard for future consequences, can be inferred psychometrically from responses on

Correspondence: K. R. Hamilton, Department of Psychiatry, Yale University School of Medicine, 1 Church Street, New Haven CT 06511, USA. Tel: 203 737 6232. Fax: 203 737 3591. E-mail: kristen.hamilton@yale.edu

self-report questionnaires and behaviorally from responses on laboratory tasks (Moeller et al. 2001; Lane et al. 2003; Reynolds et al. 2006). Choice impulsivity is indexed behaviorally by a diminished ability or willingness to delay gratification on behavioral tasks that measure delay of reward. Response impulsivity, on the other hand, is indexed behaviorally by a diminished ability to inhibit prepotent responses on behavioral response inhibition tasks. Self-reported impulsivity does not always correlate or factor with either type of behavioral impulsivity (Lane et al. 2003; Meda et al. 2009). Furthermore, the two types of behaviorally assessed impulsivity do not typically correlate with each other (Lane et al. 2003; Reynolds et al. 2006), suggesting the measures may assess different impulsivity dimensions. The dimension of impulsivity captured by self-report measures may reflect a characteristic that individuals are able to identify in their own behavior (Lane et al. 2003), while the behavioral impulsivity laboratory tasks assessing individuals' ability to inhibit a response or to delay gratification at the time of measurement rely less on self-perception. The effect of stress on drinking behavior may be differentially mediated by the various dimensions of impulsivity, and knowledge of such differences could result in improved treatment and prevention strategies for individuals based on their impulsivity characteristics.

Different types of impulsivity were associated with different stages of alcohol use, abuse, and dependence (Lejuez et al. 2010). Early alcohol use was related to greater delay discounting, poorer response inhibition, and increased self-reported impulsivity (Lejuez et al. 2010). Current and early problem alcohol use and alcohol use disorders were associated with greater delay discounting and self-reported impulsivity (Lejuez et al. 2010). Hazardous drinking, a type of excessive drinking related to public health problems (Room et al. 2005), also was associated with self-reported impulsivity (Hamilton et al. in press).

Longitudinal studies are valuable in examining changes in alcohol use that correlate with changes in impulsive tendencies. In a longitudinal study that followed men and women from the ages of 18–35, decreases in self-reported trait impulsivity were associated with decreases in problematic alcohol use (Littlefield et al. 2010). From this large sample, five trajectory groups were identified that differed in baseline levels of self-reported impulsivity and developmental patterns of change. Interestingly, the trajectory group that demonstrated the sharpest decline in the trait also had an accelerated decrease in alcohol use from ages 18 to 25 (Littlefield et al. 2010). In a longitudinal study of Alcoholics Anonymous participants, decreases in self-reported trait impulsivity mediated reductions in alcohol-related problems (Blonigen et al. 2011). In light of these findings, it is conceivable that self-reported impulsivity may mediate the effect of stress on alcohol use.

Stress is experienced when organisms perceive that a challenge exceeds their resources for coping (Baum et al. 1997, 1981, 1982), setting in motion a cascade of physiological events involving the hypothalamic–pituitary–adrenal axis and the sympathetic nervous system in an attempt to regain homeostasis (McEwen 2000). Cumulative stress is a risk factor that can increase individual vulnerability for a wide range of addictive behaviors (Sinha 2008). Physiological responses to stressors may alter brain motivational pathways, such as those involving the medial prefrontal cortex, a region implicated in self-control and the inhibition of impulses (Arnsten and Goldman-Rakic 1998; Sinha 2008). Over time, such neurobiological alterations may predispose individuals to a behavioral style characterized by increased impulsiveness, which could lead stressed individuals to engage in addictive behaviors like alcohol consumption (Sinha 2008). The effects of stress on the neurobiology of impulse control support the possibility that impulsivity may mediate the relationship between stress and alcohol use, a possibility that has not been previously examined.

Longitudinal studies of stress and alcohol use provide empirical support for a role for stress in alcohol use. In a sample of university students followed longitudinally during their transitional year after graduation, higher levels of perceived stress and stressful life events predicted greater consumption of alcohol and a greater frequency of intoxication (Sadava and Pak 1993). A variety of socially relevant stressful life events increase drinking (Veenstra et al. 2006). The transitions of both retirement (Perreira and Sloan 2001) and divorce (Romelsjo et al. 1991; Perreira and Sloan 2001) were associated with increased alcohol use in two large samples of men and women followed longitudinally. Marital stress (Brennan et al. 1999) and becoming widowed (Perreira and Sloan 2001) were associated with increased alcohol use in men, while moving away from a friend or the death of a friend were associated with increased drinking in women (Glass et al. 1995). In addition, financial stressors and more negative life events were associated with increased drinking in women (Brennan et al. 1999). Interestingly, some types of stressors, particularly health-related stressors, were associated with decreased alcohol consumption (Glass et al. 1995; Brennan et al. 1999). For this reason, it is important to consider stressor type when examining the relationship between stress and alcohol use. Although it was not examined in these studies, it is possible that elevated impulsivity mediated the relationship between stress and increased alcohol use reported in many experiments.

To examine mediation, statistical associations may be used within a cross-sectional sample to determine

statistically whether associations with retrospectively assessed stressful life events support theoretically predicted relationships. Although the cross-sectional design does not allow for the examination of impulsivity, stress, and alcohol use over time, associations of stress and impulsivity with alcohol use have been established in studies with longitudinal designs. For this reason, it is reasonable to model retrospective reports of stressful life events over the course of the lifespan, as measured in the present study, and examine their relationships to impulsivity and alcohol use.

The present research was conducted to determine whether self-reported impulsivity, choice impulsivity, and response impulsivity statistically mediate the effects of stress on alcohol use. Self-reported impulsivity was assessed using the Barratt impulsiveness scale, version 11 (BIS-11), choice impulsivity was assessed by performance on the experiential discounting task (EDT), and response impulsivity was assessed by performance on the go/no-go task (GNG) in a community-based sample of 192 men and women. Based on previous research in which self-reported impulsivity and choice impulsivity were associated with current alcohol use, early alcohol problems, and alcohol use disorders (Lejuez et al. 2010), it was hypothesized that self-reported impulsivity and choice impulsivity, but not response impulsivity, would statistically mediate the effect of stress on alcohol use (see Figure 1).

Methods

Participants

Recruitment and eligibility. One hundred ninety-two individuals (90 men and 102 women) were recruited from the greater New Haven community via advertisements placed either online or in local newspapers and community centers. Eligibility was ascertained via an initial phone screen. All participants were required to be between the ages of 18 and 50 years and able to read and write in English to at least a sixth grade level, and to meet stringent health requirements as determined by a specialist research nurse. Exclusion criteria included Diagnostic and Statistical Manual of Mental Disorders, fourth edition (DSM-IV) dependence for any drug other than alcohol or nicotine. Participants using prescribed medications for any psychiatric or medical disorders were also excluded, and all individuals underwent stringent medical assessments including electrocardiography and laboratory tests of renal, hepatic, pancreatic, and hematopoietic and thyroid function. Participants were administered breath alcohol testing and urine toxicology screens to verify self-reported drug and alcohol information. Participants were required to have normal values on all blood work and lab results, and were excluded if they tested positive for drugs of abuse other than alcohol or nicotine. All participants gave both written and verbal informed consent, and the study was approved by the Human Investigation Committee of the Yale University School of Medicine.

Procedure

Assessments

Alcohol use disorders test (Babor et al. 2001). Drinking behavior was assessed using the alcohol use disorders test (AUDIT). The AUDIT is a 10-item screening instrument that identifies drinking behaviors and distinguishes between low-risk drinkers and individuals with hazardous or risky patterns of alcohol use. In the AUDIT screen, individuals endorse one of five responses (i.e. 0 = never, 1 = less than monthly, 2 = monthly, 3 = weekly, 4 = daily or almost daily) to items such as, "How often during the last year have you been unable to remember what happened the night before because you had been drinking?" The AUDIT accurately assesses severity of problematic alcohol use behaviors across age, gender, and cultures (Allen et al. 1997). AUDIT scores have been correlated with those on the Michigan alcohol screening test (Bohn et al. 1995) and the Cut down, Annoyed, Guilty, Eye-opener (CAGE) alcohol screening instrument (Hays et al. 1995) as well as with future indicators of alcohol-related problems (Claussen and Aasland 1993). The AUDIT has high test–retest reliability (Daeppen et al., 2000) and internal consistency reliability (Ivis et al. 2000). AUDIT scores also were used to classify men and women as hazardous drinkers using gender-based cutoff scores

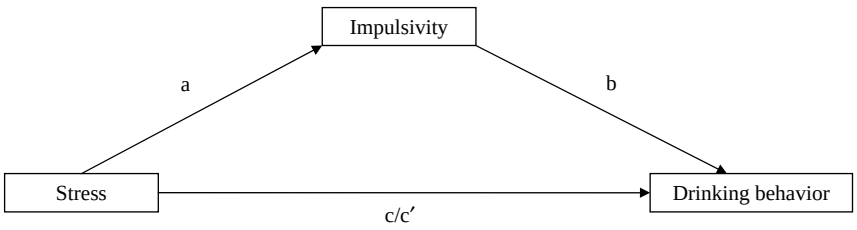

Figure 1. Mediation model of stress, impulsivity, and drinking behavior.

(Conigrave et al. 1995; Bradley et al. 1998; Steinbauer et al. 1998; Babor et al. 2001; von der Pahlen et al. 2008). Women with an AUDIT score of 4 or greater and men with an AUDIT score of 8 or greater were categorized as hazardous drinkers.

The cumulative adversity interview (Turner and Wheaton 1995). This 140-item event interview is a comprehensive measure of cumulative adversity that covers major life events, life trauma, and chronic stressors. Recent life events are also included. The *recent life events* section is composed of a checklist of 33 items referring to discrete stressful events occurring in the previous 12 months. These are broadly divided into items referring to exits from the social field (e.g. death, divorce, relationships ending) and undesirable events, both interpersonal and financial (e.g. being attacked, financial crises, robberies). The *major life events* section includes 11 items relating to social adversities, not typically violent in nature, but which differ from standard life events due to their severity and potentially long-term consequences (Turner and Lloyd 2003). Examples of items are parental divorce and failing a grade in school. The *life traumas* section comprises 34 items relating to life trauma, witnessed violence, and traumatic news. Life trauma includes events which imply force or coercion and include physical, emotional, and/or sexual abuse, such as rape and being injured with a weapon. Witnessed violence items involve being present in dangerous or upsetting situations, such as seeing someone get shot or attacked with a weapon. Traumatic news items involve not being present, but instead hearing news about someone else being killed, abused, or injured. The *chronic life events* section composed of 62 items relating to continuous stressors or ongoing life problems. Items refer to longer-term interpersonal, social, and financial relationships and responsibilities including work and home environment and relationships with family and significant others. *Total scale:* a total scale score was computed by standardizing each subscale and summing the scores. This approach ensured that each category of events was weighted equally in the final score. In all cases, a higher score relates to a higher number of stressful events.

Barratt impulsiveness scale (Patton et al. 1995). The BIS-11 is a 30-item self-report questionnaire that assesses impulsivity and shows good test–retest reliability (Patton et al. 1995). In the BIS-11, participants endorse a response on the 4-point Likert-like scales (1 = rarely/never, 2 = occasionally, 3 = often, 4 = almost always/always) in response to each of 30 items (e.g. "I say things without thinking"). In addition to providing an overall impulsivity score, the BIS-11 also characterizes dimensions of impulsivity with three contributory subscales: attentional impulsivity, motor impulsivity, and non-planning impulsivity. The attentional impulsivity subscale measures tendencies related to attention and decision making, the motor impulsivity subscale measures tendencies to act without fully thinking through consequences of the action, and the non-planning impulsivity subscale measures tendencies not to plan ahead. The three BIS-11 dimensions are non-overlapping and demonstrate good reliability (Spinella 2007). General impulsivity, as well as the attentional, non-planning, and motor dimensions of impulsivity, was assessed in the present research. The BIS-11 is widely used in research and clinical settings, and elevated scores are typically observed in individuals with disorders with deficits in impulse control as a component, including substance abuse (Lejuez et al. 2007).

Experiential discounting task (Reynolds and Schiffbauer 2004). The EDT is a computerized real-time task in which subjects experienced chosen rewards at specified times throughout the assessment (Reynolds and Schiffbauer 2004). Participants completed four session blocks associated with different time delays, three of which involved choices between an adjusting and certain amount (initially, $0.15) that was delivered immediately or a standard amount ($0.30) that was delayed and probabilistic (35%). For the other session, there was no delay (0 s), and the reward ($0.30; probability, 35%) was delivered immediately. Choice options were indicated by the "illumination" of light bulbs on the screen. The adjusting immediate amount (right side of screen) was adjusted in value: the amount increased by a set percentage following a delayed standard choice but decreased following an immediate choice. The delayed standard amount (left side of screen) was fixed. The standard option choice resulted in a wait of a specified delay (0, 7, 14, and 28 s). If the money was delivered, it could be transferred to the "bank" by clicking on the "illuminated" bank building image, which resulted in coin delivery from a coin dispenser. For each choice block, subjects made choices until an indifference point was reached, defined as choosing each option (i.e. immediate and delayed) three times within six consecutive choice trials—thus holding the adjusting amount constant over those six choices. After an indifference point was established or the delayed option was chosen 15 times (reflecting minimal discounting), the session ended. The remaining sessions (i.e. 7, 14, and 28 s) were completed in ascending order. The EDT does not include an intertrial interval but controls for session time with an intersession interval (described in Reynolds and Schiffbauer 2004). Thus, a subject cannot end a choice block sooner by adopting any specific choice pattern. The subject has a predetermined timeframe within a choice block to reach an indifference point. However, because of certain program parameters, a subject making more immediate than delayed choices

or a subject making choices more quickly is not able to make more overall choices during a choice block. If the subject reaches an indifference point prior to the predetermined time frame elapsing (which occurs the vast majority of the time), the subject must wait for the remainder of the time allotted for that choice block. Thus, choice-session time is held constant across subjects. As described previously (Myerson et al. 2001), the average-area under the curve (AUC) value was used as the behavioral measure for EDT performance, with a smaller AUC reflecting steeper discounting and greater impulsivity.

GNG is a learning task designed to assess participants' ability to inhibit inappropriate responses. It consists of repeated presentations of eight numbers, of which four are designated "correct" and four "incorrect". Participants were informed as to the identities of the correct and incorrect letters before the task began. A different list of numbers was used for each session. Participants were required to respond as rapidly as possible to correct numbers, and withhold responses to incorrect numbers. They were rewarded for correct responses (+10 cents) and penalized for incorrect responses (−10 cents). Errors of omission (withholding a response when a "correct" stimulus is presented) and errors of commission/false alarms (responding to an "incorrect" stimulus) were recorded, and participants received money they earned at the end of the session.

Analyses. In order to test the proposed mediation model (Figure 1), ordinary least squares (OLS) and ordered logistic regressions were employed to test a, b, c, and c′ pathways. The a pathway represents unstandardized beta from the OLS regression of the stress scale on the proposed mediators, BIS-11, EDT, and GNG scores. The b pathway represents the ordered logistic regression of the mediators, BIS-11, EDT, and GNG, on AUDIT score, *controlling* for stress scale score. The ab pathway represents the effect of stress on AUDIT score via the effects of BIS-11, EDT, and GNG. This pathway is also known as the *indirect* effect of stress on AUDIT score. The c pathway represents the ordered logistic regression of the stress scale on AUDIT score without BIS-11, EDT, and GNG in the model. The c′ pathway represents the ordered logistic regression of the stress scale on AUDIT score with the effects of BIS-11, EDT, and GNG controlled. The c′ pathway is also called the direct effect of stress on AUDIT score as it represents the effects of stress on drinking behavior independent of self-reported, choice, and response impulsivity. Models were run for cumulative adversity interview (CAI) with EDT, GNG, BIS-11 overall impulsivity, and the three BIS-11 subscale scores as the mediating variables, AUDIT score as the dependent variable, and gender and IQ, which were positively associated with AUDIT score, as covariates. Positive findings were followed by analyses investigating the four CAI subscales (life trauma, major life events, recent life events, and chronic stressors) to identify the source of the findings. To test the significance of the indirect effects of stress scale (or subscales) on AUDIT score via EDT, GNG, and BIS-11 impulsivity, as well as the three BIS-11 dimensions of impulsivity, we employed the approach by Preacher and Hayes (2004) using the SPSS INDIRECT bootstrapping macro. As indirect effects do not meet normal assumptions for statistical analysis, bootstrapping was used to estimate the significance of the indirect effects. Corrected and accelerated 95% confidence intervals (CI) were computed using 10,000 bootstrapped re-samples for each indirect point estimate. CIs which do not contain a zero value indicate a significant effect.

Results

Demographics and mean scores on the scales for the sample are displayed in Table I. Participants on average were aged 30.7 years. The sample was 47% male and 63% Caucasian (25% African American, 5% Hispanic, 2% Asian, and 5% were classified as

Table I. Demographics of sample.

Subject variable	$N = 192$
Age in years*	30.42(9.3)
Years of education*	14.90 (2.2)
Gender (% male)	90 (46.9%)
Race	
African American (*N*% AA)	47 (24.5%)
Caucasian (*N*% Caucasian)	122 (63.5%)
Hispanic (*N*% Hispanic)	10 (5.2%)
Asian (*N*% Asian)	4 (2.1%)
Other (*N*% other)	9 (4.7%)
Marital status	
Never married (%)	131 (68.2%)
Married (%)	37 (19.3%)
Separated (%)	0 (0%)
Divorced (%)	22 (11.5%)
Cohabiting (%)	2 (1.0%)
Widowed (%)	0 (0%)
IQ (Shipley)*	112.8 (8.57)
Total self-reported impulsiveness (BIS-11)*	61.7 (12.1)
Non-planning impulsiveness	23.1 (5.0)
Attentional impulsiveness	16.9 (4.7)
Motor impulsiveness	21.8 (4.6)
Total choice impulsiveness (EDT)*	0.62 (0.2)
Total response impulsiveness (GNG)*	23.4 (11.1)
Total CAI*	12.4 (7.4)
Major life events*	2.2 (1.7)
Life traumas*	6.9 (4.7)
Recent life events*	3.3 (2.9)
Chronic stressors*	11.1 (6.6)
Alcohol dependent (%)	18 (9.4%)

*Denotes mean values (standard deviations); all other measures reported in frequency (percents). CAI total = standardized major life events + standardized life traumas + standardized recent life events.

"Other"). They had on average almost 3 years post high school education. Forty-four percent of the sample was classified as "hazardous drinkers," and 10% of the sample was alcohol dependent.

Self-reported impulsivity

Cumulative adversity. The results for the mediation model examining the total cumulative stress score, BIS-11 impulsivity total score, and AUDIT score are presented in Table II. Gender [$r(190) = 0.243$, $p < 0.001$] and IQ [$r(190) = -0.19$, $p < 0.01$] were positively associated with AUDIT score, and were included as covariates in all analyses. Cumulative stress was positively associated with self-reported overall impulsivity and BIS-11 overall impulsivity was significantly associated with AUDIT score [Model $R^2 = 0.29$, F (4, 187) $= 18.76$, $p < 0.001$]. The indirect effect for cumulative stress total score on AUDIT score via BIS-11 total score also was significant [$a \times b = 0.10$, CI $= 0.06-0.17$] supporting a statistical mediation effect such that greater stress was associated with greater impulsivity, which was associated with higher scores on the AUDIT. The total effect of cumulative stress on drinking behavior was significant [$b = 0.29$, t (187) $= 4.80$, $p < 0.001$], and the direct effect, which controls for BIS-11, also was significant [$b = 0.19$, t (187) $= 3.10$, $p < 0.01$]. This suggests that the effects of cumulative stress on AUDIT score were not fully mediated by self-reported impulsivity score. Taken together, these meditational analyses suggest that self-reported impulsivity partially mediates the relationship between cumulative life stress and AUDIT score. Statistical mediation models of BIS-11 impulsivity total score were subsequently examined for each of the CAI subscales.

Life trauma. Life trauma was positively associated with overall self-reported impulsivity (see Table II) and overall impulsivity was positively associated with higher AUDIT scores (Model $R^2 = 0.29$, F (4,187) $= 19.50$, $p < 0.001$]. The indirect effect of life trauma on AUDIT score via self-reported impulsivity was significant [$a \times b = 0.14$, CI $= 0.08-0.23$], indicating that self-reported impulsivity partially mediated the effect of life trauma on AUDIT score. However, significant total [$b = 0.47$, t (187) $= 4.90$, $p < 0.001$] and direct effects [$b = 0.33$, t (187) $= 3.44$, $p > 0.001$] of life trauma on AUDIT score suggest that the impact of life traumas on drinking behavior occurs as a result of factors other than impulsivity.

Major life events. Major life events were positively associated with overall self-reported impulsivity and overall self-reported impulsivity was positively associated with drinking behavior, as assessed by AUDIT score (see Table II) [Model $R^2 = 0.25$, F (4,187) $= 15.59$, $p < 0.001$]. A significant indirect effect of major life events on drinking behavior via self-reported impulsivity suggested that self-reported impulsivity is a mediator of this relationship [$a \times b = 0.36$, CI $= 0.17-0.70$]. There were no significant total effects [$b = 0.29$, t (187) $= 1.05$, $p = 0.29$] or direct effects [$b = -0.07$, t (187) $= -0.25$, $p = 0.80$] of major life events on AUDIT scores.

Recent life events. Recent life events were positively associated with overall self-reported impulsivity, which in turn was associated with AUDIT score (see Table II) [Model $R^2 = 0.27$, F (4,187) $= 17.39$, $p < 0.001$]. The indirect effects of recent life events on AUDIT score by way of overall self-reported impulsivity were significant ($a \times b = 0.20$, CI $= 0.09-0.39$), suggesting that self-reported impulsivity mediates the relationship between recent life events and drinking behavior. The significance of the total effects [$b = 0.55$, t (187) $= 3.58$, $p < 0.001$] and direct effects [$b = 0.35$, t (187) $= 2.34$, $p < 0.05$] of recent life events on AUDIT scores suggested that recent life events impact drinking behavior by way of other factors that do not include self-reported impulsivity. These results indicate that overall self-reported impulsivity, partially mediates the relationship between recent life events and drinking behavior.

Chronic stress. Chronic stress, or the experience of difficult ongoing conditions of daily life, was positively associated with impulsivity, which was significantly associated with drinking behavior (see Table II) [Model $R^2 = 0.26$, F (4, 187) $= 16.02$, $p < 0.001$]. A significant indirect effect suggested that self-reported impulsivity mediates the relationship between chronic stress and drinking behavior ($a \times b = 0.10$, CI $= 0.15-0.18$). There was no significant direct effect [$b = 0.08$, t (187) $= 1.16$, $p = 0.25$] of chronic stress on problem drinking. A significant total effect [$b = 0.18$, t (187) $= 2.51$, $p < 0.05$] suggests that the relationship between chronic stress and AUDIT score depends on other factors, including self-reported impulsivity.

Non-planning, attentional, and motor impulsivity. The extent to which each of the three BIS-11 subscales of impulsivity, non-planning, attentional, and motor, statistically mediated the effects of stress on drinking behavior also was examined using statistical mediation models (Table III). The pattern of statistical

Table II. Mediation of association of stress with drinking behavior by self-reported, choice, and response impulsivity, with gender and IQ as covariates.

	Effect of IV on M (a)	Effect of M on DV (b)	Total effect (c)	Direct effect (c′)	Indirect effect ($a \times b$) (95% CI)	Gender on DV	IQ on DV
Self-reported impulsivity—BIS-11							
IV							
CAI total	0.62 *	0.16*	0.29*	0.19*	0.10* (0.06–0.17)	3.80*	−0.13*
Trauma	0.86*	0.16*	0.47*	0.33*	0.14* (0.08–0.24)	3.58*	−0.12*
Major life events	1.76*	0.20*	0.29	−0.07	0.36* (0.16–0.70)	3.67*	−0.18*
Recent life events	1.13*	0.18*	0.55*	0.35*	0.20* (0.09–0.42)	3.90*	−0.16*
Chronic stressors	0.52*	0.19*	0.18*	0.08	0.10* (0.05–0.18)	3.95*	−0.17*
Behavioral choice impulsivity—EDT							
IV							
CAI total	0.001	1.0	0.25*	0.25*	0.001 (−0.008 to 0.01)	4.00*	−0.12*
Trauma	−0.001	1.51	0.47*	0.47*	−0.001 (−0.03 to 0.02)	3.70*	−0.11*
Major life events	0.002	1.28	0.25	0.25	0.003 (−0.03 to 0.09)	3.93*	−0.17*
Recent life events	0.005	0.59	0.55*	0.54*	0.003 (−0.02 to 0.05)	4.15*	−0.17*
Chronic stressors	0.004	0.41	0.18*	0.18	0.002 (−0.02 to 0.03)	4.38*	−0.16*
Behavioral response impulsivity—GNG							
IV							
CAI total	−0.12	0.02	0.29*	0.29*	−0.002 (−0.03 to 0.007)	3.90*	−0.11*
Trauma	−0.24	0.02	0.47*	0.48*	−0.005 (−0.04 to 0.01)	3.57*	−0.10*
Major life events	−0.60	0.005	0.29	0.29	−0.003 (−0.08 to 0.04)	3.94*	−0.16*
Recent life events	0.03	0.001	0.55*	0.55*	0.001 (−0.02 to 0.03)	4.18*	−0.16*
Chronic stressors	0.03	−0.001	0.18*	0.18*	0.001 (−0.01 to 0.01)	4.44*	−0.15*

Notes: IV = independent variable (i.e. CAI total, trauma, major life events, recent life events, chronic stressors); M = mediator (i.e. self-reported impulsivity, choice impulsivity, response impulsivity); DV = dependent variable (AUDIT). The total effect is the effect of the IV on the DV without including the mediators in the model. The direct effect is the effect of the IV on the DV, controlling for the effects of the mediators. The indirect effect is the effect of the IV on the DV via the mediator. Gender and IQ were included as covariates in all analyses. *Denotes significance level of $p < 0.05$.

Table III. Mediation of association of stress with drinking behavior by self-reported non-planning, attentional, and motor impulsivity, with gender and IQ as covariates.

	Effect of IV on M (a)	Effect of M on DV (b)	Total effect (c)	Direct effect (c′)	Indirect effect ($a \times b$) (95% CI)	Gender on DV	IQ on DV
Non-planning impulsivity—BIS-11							
IV							
CAI total	0.19⋆	0.25⋆	0.29⋆	0.24⋆	0.05⋆ (0.01–0.11)	3.78⋆	−0.10
Trauma	0.26⋆	0.26⋆	0.47⋆	0.40⋆	0.07⋆ (0.02–0.14)	3.51⋆	−0.09
Major life events	0.74⋆	0.34⋆	0.29	0.04	0.25⋆ (0.09–0.53)	3.63⋆	−0.14⋆
Recent life events	0.29⋆	0.30⋆	0.55⋆	0.47⋆	0.09⋆ (0.02–0.23)	3.90⋆	−0.14⋆
Chronic stressors	0.16⋆	0.31⋆	0.18⋆	0.13	0.05⋆ (0.01–0.11)	4.04⋆	−0.14⋆
Attentional impulsivity (BIS)							
IV							
CAI total	0.19⋆	0.38⋆	0.29⋆	0.22⋆	0.07⋆ (0.03–0.13)	4.09⋆	−0.14⋆
Trauma	0.24⋆	0.39⋆	0.47⋆	0.38⋆	0.10⋆ (0.04–0.17)	3.85⋆	−0.13⋆
Major life events	0.43⋆	0.47⋆	0.29	0.09	0.20⋆ (0.01–0.50)	4.07⋆	−0.18⋆
Recent life events	0.42⋆	0.42⋆	0.55⋆	0.38⋆	0.18⋆ (0.08–0.33)	4.23⋆	−0.18⋆
Chronic stressors	0.21⋆	0.44⋆	0.18⋆	0.09	0.09⋆ (0.04–0.18)	4.30⋆	−0.18⋆
Motor impulsivity (BIS)							
IV							
CAI total	0.25⋆	0.42⋆	0.29⋆	0.18⋆	0.11⋆ (0.06–0.18)	3.81⋆	−0.16
Trauma	0.37⋆	0.43⋆	0.47⋆	0.31⋆	0.16⋆ (0.09–0.28)	3.61⋆	−0.15⋆
Major life events	0.58⋆	0.54⋆	0.29	−0.02	0.31⋆ (0.14–0.55)	3.70⋆	−0.21⋆
Recent life events	0.46⋆	0.48⋆	0.55⋆	0.34⋆	0.22⋆ (0.10–0.40)	3.90⋆	−0.20⋆
Chronic stressors	0.17⋆	0.51⋆	0.18⋆	0.09	0.09⋆ (0.04–0.16)	4.00⋆	−0.20⋆

Notes: IV = independent variable (i.e. CAI total, trauma, major life events, recent life events, chronic stressors); M = mediator (i.e. non-planning impulsivity, attentional impulsivity, motor impulsivity); DV = dependent variable (AUDIT). The total effect is the effect of the IV on the DV without including the mediators in the model. The direct effect is the effect of the IV on the DV, controlling for the effects of the mediators. The indirect effect is the effect of the IV on the DV via the mediator. Gender and IQ were included as covariates in all analyses. ⋆denotes significance level of $p < 0.05$.

mediation of each of the subscales was similar to the pattern of mediation observed for total impulsivity. The subscale mediation results are tabulated in Table III, and are described in detail with statistical analyses in the supplemental materials section.

Choice impulsivity

The results for the meditation model examining the total cumulative stress score, choice impulsivity as assessed by AUC of the EDT, and AUDIT score are presented (see Table II). Cumulative life stress did not associate with the AUC of the EDT, and AUC did not significantly associate with AUDIT score (see Table II) [Model $R^2 = 0.21$, F (4, 185) = 12.61, $p < 0.001$]. There was no indirect effect of cumulative stress on drinking behavior via AUC ($a \times b = 0.00$, CI = −0.008–0.01), suggesting that AUC did not mediate the relationship between cumulative stress and drinking behavior. In fact, significant total [$b = 0.29$, t (185) = 4.69, $p < 0.001$] and direct effects [$b = 0.29$, t (185) = 4.66, $p < 0.001$] of cumulative stress on drinking behavior suggested that while a relationship existed between cumulative stress and drinking behavior, this relationship was completely independent of choice impulsivity as assessed by the EDT. No CAI subscale relationships with AUDIT scores were mediated by choice impulsivity (see Table II).

Response impulsivity

Cumulative life stress did not associate with response inhibition failures GNG, a measure of response impulsivity, and response inhibition failures did not significantly associate with AUDIT score [Model $R^2 = 0.22$, F (4, 187) = 12.86, $p < 0.001$] (see Table II). There was no indirect effect of cumulative stress on drinking behavior via response inhibition failures ($a \times b = -0.002$, CI = −0.03 to 007), suggesting that response inhibition failures did not mediate the relationship between cumulative stress and drinking behavior. In fact, significant total [$b = 0.29$, t (187) 4.80, $p < 0.001$] and direct effects [$b = 0.29$, t (187) = 4.80, $p < 0.001$] of cumulative stress on drinking behavior suggested that while a relationship existed between cumulative stress and drinking behavior, this relationship was completely independent of response impulsivity as assessed by the GNG. No CAI subscale relationships with AUDIT scores were mediated by response impulsivity (see Table II).

Hazardous drinking

Because hazardous drinking is associated with many public health problems (Room et al. 2005), it is important to understand factors that are related to the behavior. For this reason, all mediation analyses also

Table IV. Mediation of effects of stress on hazardous drinking by self-reported, choice, and response impulsivity.

	Effect of IV on M (a)	Effect of M on DV (b)	Total effect (c)	Direct effect (c′)	Indirect effect ($a \times b$) (95% CI)
Self-reported impulsivity—BIS-11					
IV					
CAI total	0.57*	0.05*	0.08*	0.05*	0.03* (0.01–0.05)
Trauma	0.80 *	0.05*	0.10*	0.06	0.04* (0.02–0.08)
Major life events	1.60*	0.06*	0.07	−0.02	0.10* (0.04–0.19)
Recent life events	1.09*	0.05*	0.20*	0.16*	0.06* (0.02–0.11)
Chronic stressors	0.46*	0.06*	0.05*	0.03	0.03* (0.01–0.05)
Behavioral choice impulsivity—EDT					
IV					
CAI total	−0.001	0.47	0.07*	0.07*	−0.001 (−0.01 to 0.002)
Trauma	−0.003	0.59	0.10*	0.10	−0.002 (−0.02 to 0.003)
Major life events	−0.002	0.38	0.06	0.07	−0.001 (−0.03 to 0.01)
Recent life events	0.004	0.14	0.20*	0.20*	0.001 (−0.008 to 0.02)
Chronic stressors	0.003	0.13	0.05*	0.05*	0.001 (−0.01 to 0.008)
Response impulsivity—GNG					
IV					
CAI total	−0.15	0.02	0.08*	0.08*	−0.002 (−0.02 to 0.002)
Trauma	−0.24	0.01	0.10*	0.10*	−0.01 (−0.05 to 0.01)
Major life events	−0.78	0.01	0.07	0.08	−0.008 (−0.05 to 0.01)
Recent life events	−0.07	0.01	0.20*	0.20*	−0.001 (−0.02 to 0.006)
Chronic stressors	−0.08	0.01	0.05*	0.05	−0.001 (−0.009 to 0.002)

Notes: IV = independent variable (i.e. CAI total, trauma, major life events, recent life events, chronic stressors); M = mediator (i.e. self-reported impulsivity, choice impulsivity, response impulsivity); DV = dependent variable (AUDIT). The total effect is the effect of the IV on the DV without including the mediators in the model. The direct effect is the effect of the IV on the DV, controlling for the effects of the mediators. The indirect effect is the effect of the IV on the DV via the mediator. *Denotes significance at $p < 0.05$.

were conducted with hazardous drinking as the dependent variable. All results that were reported when AUDIT score was the dependent variable were the same when hazardous drinking was the dependent variable (see Table IV). Self-reported impulsivity partially or fully mediated the effects of different types of stress on hazardous drinking.

Alcohol-dependent participants

There were 21 alcohol-dependent individuals in the present sample. In order to ascertain whether the inclusion of alcohol-dependent individuals was driving the present results, all analyses also were performed with the 21 alcohol-dependent participants excluded from the mediation analyses. When they were excluded from the analyses, all major relationships remained the same. The only differences were that self-reported impulsivity became a full mediator of the effects of cumulative stress and trauma on drinking behavior, rather than a partial mediator, and IQ was no longer significant as a covariate. As the results did not differ regardless of their inclusion, the 21 alcohol-dependent participants were included in the final analysis because excluding them would reduce power and create a sample that did not represent the population from which it was taken.

Discussion

The present research was conducted to determine whether self-reported, behavioral choice, and behavioral response impulsivity mediate the relationship between stress and drinking behavior as assessed by the AUDIT. A variable may be called a mediator "to the extent that it accounts for the relation between the predictor and the criterion" (Baron and Kenny 1986). In the present research, self-reported impulsivity fully mediated the effects of some types of stress, and partially mediated the effects of other types of stress, on drinking behavior. Across models, the presence of greater stress was associated with higher levels of self-reported impulsivity, and this greater impulsivity was associated with drinking behavior. Behavioral measures of choice and response impulsivity, on the other hand, did not influence the relationship between stress and drinking behavior.

The separate contributions of impulsivity and stress to increased alcohol use are well established (Sadava and Pak 1993; Lejuez et al. 2010) and the interactive effect of stress and impulsivity to predict alcohol use also has been reported (Fox et al. 2010). To our knowledge, this research was the first to determine whether different types of impulsivity mediate the relationship between stress and alcohol use. In the present research, self-reported impulsivity fully mediated the relationships between adverse major life events and drinking behavior, and chronic stress and drinking behavior. The effects of major life events and chronic stress on drinking behavior depended on self-reported impulsivity, and it is only through their association with self-reported impulsivity that major life events and chronic stress impact drinking behavior.

Full mediation refers to a case in which the independent variable (e.g. stress) no longer affects the dependent variable (e.g. drinking behavior) after controlling for the mediating variable (Baron and Kenny 1986). Partial mediation refers to a case in which the path from the independent variable (e.g. stress) to the dependent variable (e.g. drinking behavior) is reduced in absolute size but is still different from zero when the mediator is introduced (Baron and Kenny 1986). The full mediation by overall self-reported impulsivity and each dimension of self-reported impulsivity of the effects of chronic stress and major life events on drinking behavior demonstrates the importance of impulsivity to addictive behaviors. However, partial mediation by self-reported impulsivity of the effects of cumulative stress, life traumas, and recent life events on drinking behavior, when considering either the overall score or the scores on each of the contributory dimensions, reveals that it is not the only factor responsible for the effects of stress on drinking behavior. Significant direct effects of cumulative stress, life traumas, and recent life events on drinking behaviors, controlling for the effect of self-reported impulsivity, demonstrated the importance of stress itself as a construct impacting addictive behaviors, even without the influence of self-reported impulsivity. In addition, mediation of the effects of stress on drinking behavior by each dimension of self-reported impulsivity revealed that each impulsivity dimension contributes importantly to drinking behavior.

These findings support the stress-vulnerability theory (Sinha 2008) and suggest the possibility that stress might augment individual differences such as impulsivity. The positive association of stress with self-reported impulsivity in the present research is consistent with previous research in which stress was associated with increased impulsivity (Glass and Singer 1972; Cohen 1980; Muraven and Baumeister 2000; Sinha 2001; Tice et al. 2001). Direct and indirect pathways of stress on drinking behavior may reflect different pathways of effects of stress on reward sensitivity and its long-lasting effects on behavioral control. Effects of stress on early development, particularly highly stressful events including trauma, may affect brain development and alter stress and reward sensitivity (Meaney et al. 2002; Sinha 2008) and maturation of the prefrontal self-control systems (Gratton and Sullivan 2005). These effects may have lasting behavioral, physiological, and neuroendocrine effects on subsequent stressors and reward sensitivity, thereby increasing vulnerability for addictive behaviors. In addition, these early stressors may result in individual adaptations to stressful circumstances with negative implications for stress regulation, especially in prefrontal, executive cognitive functioning, and impulse control systems. This combined impact may represent the direct (alterations in limbic and striatal pathways) and indirect (prefrontal and self-control systems) effects observed in the cumulative adversity and distal (trauma and major life events) stress scales. Recent life events and chronic stressors, on the other hand, may exacerbate vulnerability for addiction via indirect effects, such as reduced self-control. Recent life events and chronic stressors might be considered "acute" stress, a type of stress that triggers neurochemical changes that impair prefrontal cortex functioning (Arnsten 2009). Therefore, the effects of cumulative stress on development of the prefrontal self-control systems may act in concert with the detrimental effects of acute stressors to further hinder prefrontal cortex functioning and impair self-control (Arnsten 2009).

Interestingly, while self-reported impulsivity either fully or partially mediated the relationships between different types of stress and alcohol use, behavioral choice and response impulsivity did not influence the relationships between stress and alcohol use. In fact, the statistical analyses indicate that the relationships between drinking behaviors and cumulative stress, trauma, recent life events, and chronic stressors were independent of choice and response impulsivity. Because relationships between alcohol use and choice impulsivity were previously reported (Lejuez et al. 2010), it was hypothesized that choice impulsivity, but not response impulsivity, would mediate the effects of stress on drinking behavior. This hypothesis was not supported, as neither type of behavioral impulsivity mediated the relationship between stress and drinking behavior. However, it should be noted that the correlations between the EDT and the more commonly used questionnaire-based measures (see Lejuez et al. 2010) were performed using k values, rather than the AUC values used in the present research. k values and AUC values are not linearly related to each other, and there is a lack of data on whether other measures of choice impulsivity are closely related using AUC values from the EDT. Differences in sample composition and in the measures used to assess discounting may contribute to apparent differences in results from research using k values and research using AUC. However, several researchers have used AUC values to examine delay discounting (Meda et al. 2009; Andrews et al. 2011). Several lines of research indicate that self-reported, behavioral choice, and behavioral response impulsivity are separate factors that do not correlate with each other (Lane et al. 2003; Reynolds et al. 2006; Meda et al. 2009). The results of the present research suggest that only self-reported impulsivity, which represents a self-perceived characteristic, mediates the effect of stress on drinking behavior.

Behavioral measures of impulsivity may be more sensitive than self-reported measures of impulsivity to current stress. In research examining the mediation by delay discounting of the effects of perceived stress on cigarette smoking in adolescents, only perceived stress

from the previous 30 days mediated the relationship between stress and cigarette smoking (Fields et al. 2009). By contrast, in the same research, stress from the previous 6 or 12 months did not mediate this relationship (Fields et al. 2009). These findings indicate that recency of stress exposure may be particularly relevant to understanding the relationships between stress, impulsivity, and tobacco smoking. Given these findings, future studies should examine a broader range of stress measures (based on recency) in investigating the relationships between stress, impulsivity, and drinking behaviors. In the present research, choice impulsivity and response impulsivity were assessed in the context of a neutral non-aroused or challenged state. Perhaps behavioral choice and response impulsivity assessed under stressed and non-stressed conditions would be more informative about the role of behavioral impulsivity in stress and alcohol use relationships, given the potential for stress to influence behavioral impulsivity. It is possible that choice and response impulsivity inferred from behavioral measures immediately after stress-induction, when participants were in a stressed state, would mediate the effects of stress on drinking behavior. Future research is needed to examine this possibility.

It is important to note that mediation analyses cannot prove causation. Results of mediation analyses can provide support for or against hypotheses, but cannot prove them (Preacher and Hayes 2004). Overall these findings extend previous research that has established the effects of stress and impulsivity on drinking behavior by examining statistical mediation models of stress with three dimensions of impulsivity on alcohol use. These findings support the stress-vulnerability theory and emphasize the impact that cumulative stress and adversity may have on impulsivity, a potential risk factor for increased drinking behavior and hazardous alcohol use. The results of this research can be used to inform the development of treatment and prevention strategies focused on enhancing both stress management and impulse control. Treatment and prevention strategies targeted toward impulsive individuals may be particularly valuable. This research emphasizes the importance of examining cumulative stress and adversity as experienced over the lifespan when developing and testing models of how and for whom stress affects drinking behavior. These findings do not preclude the examination of impulsivity as a moderator of chronic stressors on drinking behavior, nor do they preclude the reciprocal impact of drinking on individual differences in impulsivity. Additional factors, such as genetic variations, were not examined in this model but also may contribute to drinking risk.

An important limitation of the current findings is the cross-sectional nature of the sample. Potential causal mechanisms of change should be further studied in research with longitudinal designs to examine the indirect pathway of impulsivity on drinking behavior. The current cross-sectional analysis provides additional evidence that history of cumulative adversity is directly and indirectly associated with drinking behavior. Future research should be conducted with a longitudinal design to assess the effects of both cumulative stress and impulsivity on drinking behavior. An additional limitation is that there is a lack of research comparing EDT with other measures of delay discounting, which cites the need for more research in this area, particularly as behavioral assessments of choice impulsivity using AUC measures have been related to clinically relevant measures like treatment outcome (Krishnan-Sarin et al. 2007).

Declaration of interest: This work was supported, in part, by the NIH (T32 DA007238-21, K08 DA029641, RL1-AA017539, P20-DA027844, UL1-DE019586, and PL1-DA024859) and the NIH Roadmap for Medical Research Common Fund. The contents of the manuscript are solely the responsibility of the authors and do not necessarily represent the official views of any of the funding agencies. The authors report that they have no financial conflicts of interest with respect to the content of this manuscript. Dr Sinha is on the Scientific Advisory Board for Embera Neurotherapeutics and is also a consultant for Glaxo-Smith Kline Pharmaceuticals. Dr Potenza has received financial support or compensation for the following: Dr Potenza has consulted for and advised Boehringer Ingelheim; has consulted for and has financial interests in Somaxon; has received research support from the National Institutes of Health, Veteran's Administration, Mohegan Sun Casino, the National Center for Responsible Gaming and its affiliated Institute for Research on Gambling Disorders, and Forest Laboratories, Ortho-McNeil, Oy-Control/Biotie, Psyadon and Glaxo-SmithKline pharmaceuticals; has participated in surveys, mailings, or telephone consultations related to drug addiction, impulse control disorders, or other health topics; has consulted for law offices and the federal public defender's office in issues related to impulse control disorders; provides clinical care in the Connecticut Department of Mental Health and Addiction Services Problem Gambling Services Program; has performed grant reviews for the National Institutes of Health and other agencies; has guest-edited journal sections; has given academic lectures in grand rounds, CME events, and other clinical or scientific venues; and has generated books or book chapters for publishers of mental health texts.

References

Allen J, Litten R, Fertig J, Babor T. 1997. A review of research on the alcohol use disorders identification test (AUDIT). Alcohol Clin Exp Res 21:613–619.

Andrews MM, Meda SA, Thomas AD, Potenza MN, Krystal JH, Worhunsky P, Stevens MC, O'Malley S, Book GA, Reynolds B, Pearlson GD. 2011. Individuals family history positive for alcoholism show functional magnetic resonance imaging differences in reward sensitivity that are related to impulsivity factors. Biol Psychiatry 69(7):675–683.

Arnsten AF. 2009. Toward a new understanding of attention-deficit hyperactivity disorder pathophysiology: An important role for prefrontal cortex dysfunction. CNS Drugs 23(Supplement 1): 33–41.

Arnsten AF, Goldman-Rakic PS. 1998. Noise stress impairs prefrontal cortical cognitive function in monkeys: Evidence for a hyperdopaminergic mechanism. Arch Gen Psychiatry 55(4): 362–368.

Babor T, Higgins-Biddle J, Saunders J, Monteiro M. 2001. AUDIT. The alcohol use disorders identification test. Guidelines for use in primary care. 2nd ed., World Health Organization: Department of Mental Health and Substance Dependence.

Baron RM, Kenny DA. 1986. The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. J Pers Soc Psychol 51: 1173–1182.

Baum A, Gatchel RJ, Krantz DS. 1997. An introduction to health psychology. 3rd ed., New York: McGraw-Hill.

Baum A, Grunberg NE, Singer JE. 1982. The use of psychological and neuroendocrinological measurements in the study of stress. Health Psychol 1(3):217–236.

Baum A, Singer JE, Baum CS. 1981. Stress and the environment. J Soc Issues 37(1):4–35.

Blonigen DM, Timko C, Finney JW, Moos BS, Moos RH. 2011. Alcoholics anonymous attendance, decreases in impulsivity and drinking and psychosocial outcomes over 16 years: Moderated-mediation from a developmental perspective. Addiction 106(12):2167–2177.

Bohn M, Babor T, Kranzler H. 1995. The alcohol use disorders identification test (AUDIT): Validation of a screening instrument for use in medical settings. J Stud Alcohol Drugs 56: 423–432.

Bradley KA, Boyd-Wickizer J, Powell SH, Burman ML. 1998. Alcohol screening questionnaires in women: A critical review. JAMA 280(2):166–171.

Brennan PL, Schutte KK, Moos RH. 1999. Reciprocal relations between stressors and drinking behavior: A three-wave panel study of late middle-aged and older women and men. Addiction 94(5):737–749.

Claussen B, Aasland O. 1993. The alcohol use disorders identification test (AUDIT) in a routine health examination of long-term unemployed. Addiction 88:363–368.

Cohen S. 1980. Aftereffects of stress on human performance and social behavior: A review of research and theory. Psychol Bull 85: 858–866.

Conigrave KM, Hall WD, Saunders JB. 1995. The AUDIT questionnaire: Choosing a cut-off score. Alcohol use disorder identification test. Addiction 90(10):1349–1356.

Daeppen J, Yersin B, Landry U, Pecoud A, Decrey H. 2000. Reliability and validity of the alcohol use disorders identification test (AUDIT) imbedded within a general health risk screening questionnaire: Results of a survey in 332 primary care patients. Alcohol Clin Exp Res 24:659–665.

Evenden JL, Ryan CN. 1999. The pharmacology of impulsive behaviour in rats VI: The effects of ethanol and selective serotonergic drugs on response choice with varying delays of reinforcement. Psychopharmacology 146(4):413–421.

Fields S, Leraas K, Collins C, Reynolds B. 2009. Delay discounting as a mediator of the relationship between perceived stress and cigarette smoking status. Behav Pharmacol 20:455–460.

Fox H, Bergquist K, Gu P, Sinha R. 2010. Interactive effects of cumulative stress and impulsivity on alcohol consumption. Alcohol Clin Exp Res 34(8):1–10.

Glass DC, Singer JE. 1972. Urban stress: Experiments on noise and social stressors. New York: Academic Press.

Glass TA, Prigerson H, Kasl SV, Mendes de Leon CF. 1995. The effects of negative life events on alcohol consumption among older men and women. J Gerontol B Psychol Sci Soc Sci 50(4): S205–S216.

Gratton A, Sullivan RM. 2005. Role of prefrontal cortex in stress responsivity. In: Steckler T, Kahlin NH, Reul JMHM, editors. Handbook of stress and the brain. Dusseldorf: Elsevier. p 838.

Hamilton KR, Sinha R, Potenza MN. in press. Hazardous drinking and dimensions of impulsivity, behavioral approach, and inhibition in adult men and women. Alcohol Clin Exp Res.

Hays RD, Merz JF, Nicholas R. 1995. Response burden, reliability and validity of the CAGE, Short MAST, and AUDIT alcohol screening measures. Behav Res Method Instrum Comput 27(2): 277–280.

Ivis F, Adalf E, Rehm J. 2000. Incorporating the AUDIT into a general population telephone survey: A methodological experiment. Drug Alcohol Depen 60:97–104.

Krishnan-Sarin S, Reynolds B, Duhig AM, Smith A, Liss T, McFetridge A, Cavallo KM, Potenza MN. 2007. Behavioral impulsivity predicts treatment outcome in a smoking cessation program for adolescent smokers. Drug Alcohol Depen 88(1): 79–82.

Lane SD, Cherek D, Rhoades HM, Pietras CJ, Tcheremissine O. 2003. Relationships among laboratory and psychometric measures of impulsivity: Implications in substance abuse and dependence. Addict Disord Treat 2:33–40.

Lejuez CW, Bornovalova MA, Reynolds EK, Daughters SB, Curtin JJ. 2007. Risk factors in the relationship between gender and crack/cocaine. Exp Clin Psychopharmacol 15(2): 165–175.

Lejuez CW, Magidson JF, Mitchell SH, Sinha R, Stevens MC, de Wit H. 2010. Behavioral and biological indicators of impulsivity in the development of alcohol use, problems, and disorders. Alcohol Clin Exp Res 34(8):1334–1345.

Littlefield AK, Sher KJ, Steinley D. 2010. Developmental trajectories of impulsivity and their association with alcohol use and related outcomes during emerging and young adulthood. Alcohol Clin Exp Res 34(8):1409–1416.

McEwen BS. 2000. The neurobiology of stress: From serendipity to clinical relevance. Brain Res 886(1-2):172–189.

Meaney M, Brake W, Gratton A. 2002. Environmental regulation of the development of mesolimbic dopamine systems: A neurobiological mechanism for vulnerability to drug abuse? Psychoneuroendocrinology 27:127–138.

Meda S, Stevens MC, Potenza MN, Pittman B, Gueorguieva R, Andrews MM, Thomas AD, Muska C, Hylton JL, Pearlson GD. 2009. Investigating the behavioral and self-report constructs of impulsivity domains using principal component analysis. Behav Pharmacol 20(5-6):390–399.

Moeller FG, Barratt ES, Dougherty DM, Schmitz JM, Swann AC. 2001. Psychiatric aspects of impulsivity. Am J Psychiatry 158: 1783–1793.

Muraven M, Baumeister R. 2000. Self-regulation and depletion of limited resources: Does self-control resemble a muscle? Psychol Bull 126:247–259.

Myerson J, Green L, Warusawitharana M. 2001. Area under the curve as a measure of delay discounting. J Exp Anal Behav 76: 263–276.

Patton JM, Stanford MS, Barratt ES. 1995. Factor structure of the Barratt impulsiveness scale. J Clin Psychol 51:768–774.

Perreira KM, Sloan FA. 2001. Life events and alcohol consumption among mature adults: A longitudinal analysis. J Stud Alcohol 62(4):501–508.

Preacher KJ, Hayes AF. 2004. SPSS and SAS procedures for estimating indirect effects in simple mediation models. Behav Res Method Instrum Comput 36(4):717–731.

Reynolds B, Ortengren A, Richards JB, de Wit H. 2006. Dimensions of impulsive behavior: Personality and behavioral measures. Pers Indiv Differ 40:305–315.

Reynolds B, Schiffbauer R. 2004. Measuring state changes in human delay discounting: An experiential discounting task. Behav Processes 67(3):343–356.

Romelsjo A, Lazarus NB, Kaplan GA, Cohen RD. 1991. The relationship between stressful life situations and changes in alcohol consumption in a general population sample. Br J Addict 86(2):157–169.

Room R, Babor T, Rehm J. 2005. Alcohol and public health. Lancet 365(9458):519–530.

Sadava SW, Pak AW. 1993. Stress-related problem drinking and alcohol problems: A longitudinal study and extension of Marlatt's model. Can J Behav Sci 25(3):446–464.

Sinha R. 2001. How does stress increase drug abuse and relapse? Psychopharmacology 158:343–359.

Sinha R. 2008. Chronic stress, drug use, and vulnerability to addiction. Ann New York Acad Sci 1141:105–130.

Spinella M. 2007. Normative data and a short form of the Barratt impulsiveness scale. Int J Neurosci 117:359–368.

Steinbauer JR, Cantor SB, Holzer CE, 3rd, Volk RJ. 1998. Ethnic and sex bias in primary care screening tests for alcohol use disorders. Ann Int Med 129(5):353–362.

Tice D, Bratslavsky E, Baumeister R. 2001. Emotional distress regulation takes precedence over impulse control: If you feel bad, do it! J Pers Soc Psychol 80:53–67.

Turner RJ, Lloyd DA. 2003. Cumulative adversity and drug dependence in young adults: Racial/ethnic contrasts. Addiction 98(3):305–315.

Turner RJ, Wheaton B. 1995. Checklist measurement of stressful life events. In: Cohen S, Kessler R, Underwood GL, editors. Measuring stress. New York: Oxford University Press. p 29–58.

Veenstra MY, Lemmens PH, Friesema IH, Garretsen HF, Knottnerus JA, Zwietering PJ. 2006. A literature overview of the relationship between life-events and alcohol use in the general population. Alcohol Alcohol 41(4):455–463.

von der Pahlen B, Santtila P, Witting K, Varjonen M, Jern P, Johansson A, Sandnabba NK. 2008. Factor structure of the alcohol use disorders identification test (AUDIT) for men and women in different age groups. J Stud Alcohol Drugs 69: 616–621.

Supplementary material available online