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Risk-taking and Alcohol Use Disorders Symptomatology in a Sample of Problem Drinkers

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Abstract

The relationship between risk-taking behavior and alcohol use disorder (AUD) symptoms is poorly understood. This study employed a modified version of a behavioral measure of risk-taking, the Balloon Analogue Risk Task (BART), to examine its relationship to alcohol use and related symptoms in a community sample of individuals with or at risk for AUD. A total of 158 (71.9% male) participants completed a testing battery that included the BART, a structured diagnostic interview for AUD, and measures of alcohol use and related problems. Estimates of IQ and working memory were assessed as covariates. Results indicated that the relationship between risk-taking propensity, as assessed by the BART, and alcohol problems was significant and negative. Individuals with higher symptom count made fewer pumps per trial on the BART, indicating less risk-taking. Importantly, this relationship was attenuated when controlling for estimated IQ and working memory span. Further examination demonstrated that IQ and age mediated the relationship between risk-taking propensity and symptom count. The main negative relationship observed between risk-taking on the BART and alcohol use and AUD symptomatology in this sample stands in contrast to the positive relationships observed in adolescent and non-clinical samples. Together, these findings highlight the need to consider development and the course of addiction in order to fully elucidate the effects of risky-decision making on AUD liability. Furthermore, our results demonstrate the importance of inclusion of neurocognitive covariates (IQ) as well as demographic variables (age) when using this task.

Keywords

BART; risk-taking; decision-making; impulsivity; alcohol dependence

Previous research has suggested that risk-taking, as a personality trait, may serve as a liability factor for substance use disorders, including alcohol use disorders (AUD, e.g., (Bechara & Damasio, 2002; Lejuez et al., 2002; Stout, Rock, Campbell, Busemeyer, & Finn,

2005; Zuckerman, Ball, & Black, 1990). Risk-taking reflects the ability of an individual to weigh costs and benefits during decision-making with potentially negative outcomes. These processes are likely related to genetically-determined individual differences in psychological processes (Kuhnen & Chiao, 2009) such as sensitivity to reward and impairments of inhibitory (impulse) control (Kreek, Nielsen, Butelman, & LaForge, 2005). Advances in assessment have resulted in objective, quantitative, behavioral measures of risk-taking tendencies, such as the Iowa Gambling Task (Bechara, Damasio, Damasio, & Anderson, 1994) and the more recently developed Balloon Analogue Risk Task (BART; Lejuez et al., 2002). Unlike previously used self-report measures of risk-taking (Zuckerman et al., 1990; Zuckerman, Eysenck, & Eysenck, 1978), these behavioral tasks are thought to allow for the assessment of risk-taking as an objective behavior.

During performance on the BART, participants make decisions on each trial about the amount of risk they are willing to accept in order to inflate a computer-generated balloon and obtain a reward. They successively press a “pump” button to accept risk in order to obtain larger reward accruals (and inflate the on-screen balloon) or a “cash out” button to avoid further risk and to obtain the reward accrued thus far. If the risk function of the program is exceeded, participants will observe a trial failure (i.e., balloon explosion) with reward forfeiture. Across trials, the number of “pump” presses made before “cashing out” represents a behavioral measure of risk-taking, with individuals who are risk-prone pressing a greater number of times on an average trial (Lejuez et al., 2002). As including data from trials in which the balloon exploded biases the mean (Lejuez et al., 2002), the commonly used metric in the literature and in this report is an adjusted mean. Performance on the BART appears to be trait-like as it has demonstrated test-retest reliability (White, Lejuez, & de Wit, 2008) and moderate heritability in adolescent males (Anokhin, Goloshevkin, Grant, & Heath, 2009). A recent three year longitudinal study found that risk-taking propensity increased across development in adolescents, and that the magnitude of such increases was associated with alcohol use (MacPherson, Magidson, Reynolds, Kahler, & Lejuez, 2010). These results indicate that BART performance may change across development and that the degree of increase in risk taking on the BART may be most predictive of “real-world” risk-taking behaviors, including alcohol use.

The association of the BART with “real world” risk-taking and substance use behaviors has been supported in several studies. Specifically, the average number of “pump” responses made by subjects on the BART has been positively associated with substance use and other health-related risk-behaviors (Lejuez et al., 2002), such as having ever tried a cigarette (Lejuez, Aklin, Bornoalova, & Moolchan, 2005) or having ever tried ‘ecstasy’ (Hopko et al., 2006). Alcohol consumption was positively related to greater “pump” responses on the BART among individuals with low novelty seeking and low harm avoidance (Skeel, Pilarski, Pytlak, & Neudecker, 2008). Recently, performance on the BART predicted alcohol consumption in a college student sample over and above delayed reward discounting and response inhibition (Fernie, Cole, Goudie, & Field, 2010). A recent study of adolescent/young adults found that smokers were less likely than nonsmokers to take risks on the BART, even in situations where risk-taking in the task is considered adaptive (i.e., low risk balloons; (Dean, Sugar, Hellemann, & London, in press). However, no studies to date have examined the relationship between performance on the BART and clinical indices of AUD

among clinical samples of individuals reporting AUD symptomatology. Should the relationship between risk-taking on the BART and AUD remain positive across development and after the onset of alcohol problems, higher pump responses on the BART are expected to be associated with higher alcohol use and problem severity. This would support the idea that risk-taking contributes to the maintenance and severity of alcohol dependence. To that end, the present study will test the relationship between risk-taking, measured by the BART, and alcohol use and related problems in a large community sample of problem drinkers.

Although the BART represents a promising behavioral measure of risk-taking, several aspects of performance on the BART remain unexplored in the human literature. For example, pre-clinical research has demonstrated that inter-trial variability may be a distinct phenotype measured within the task (Jentsch, Woods, Groman, & Seu, 2010); that is, some subjects choose to accept a more stable level of risk throughout the task, while others explore various levels of responding, or may be more reactive to recent trial outcomes. These two performance characteristics (average pumps and variability of pumps) were shown to rely on separate brain circuitry in the rat, further corroborating their distinction as separate factors (Jentsch et al., 2010). Although separate, they are both important components that underlie the utility of an organism's behavior when faced with decisions made under risk, with highly variable behavior nearly always detracting from ideal outcomes.

Another interesting metric that emerges from the task is the average number of pumps on trials immediately following a balloon explosion. Unlike the more general mean pumps measure typically used in analysis, this post-failure measure allows insight into specific influences of negative feedback on the decision-making process as it is occurring. One crucial diagnostic component of alcohol dependence consists of continued use of alcohol despite knowledge of adverse physical, emotional, or social consequences directly due to alcohol use. Thus, one might expect that compulsive alcohol use may be associated with reduced reactivity to negative feedback in the BART. With this in mind, the present study will examine dimensions of the BART beyond average number of pumps, including response variability and post-failure responding.

In summary, the present study utilizes a clinical sample of adult non-treatment seeking problem-drinkers to: (a) test the association between decision-making under risk with AUD symptomatology in a clinical sample using the BART; (b) examine sub-dimensions of BART performance such as inter-trial variability and post-failure reactivity as they relate to AUD measures; and (c) account for neurocognitive variables likely associated with performance on the BART, such as IQ and working memory. Together, these analyses seek to further elucidate the association between performance on the BART and risky alcohol use in the real world.

Methods

Participants

Non-treatment seeking heavy drinkers ($N = 158$) were recruited from the Los Angeles community through flyers, print, and online advertisements as part of a larger, ongoing alcohol administration study. Inclusion criteria were: (1) age between 21 and 65; (2) self-identification of “problems with alcohol;” (3) telephone endorsement of consuming a minimum of 48 standard drinks per month. Exclusion criteria were: (1) current treatment for alcohol problems, history of treatment in the 30 days prior to enrollment, or currently seeking treatment; (2) not having an alcoholic drink within 21 days of the telephone screening interview; (3) history of bipolar disorder or psychotic disorder, or positive evaluation for these disorders during a structured diagnostic interview. Participants were compensated \$40 for participation in the face-to-face assessment procedure, as well as up to an additional \$5 based on performance on the BART (outlined below). The average age of the sample was 30.29 ($SD = 10.49$, range 21 to 63), with a majority of participants being male (71.9%). The ethnic background of the sample was as follows: White (46.7%), African American (20.4%), Asian (6.6%), Latino (11.8%), Other/Mixed-Ethnicity (11.2%), and ethnicity not given (3.3%). The average number of years of education was 14.8 ($SD = 2.26$).

Procedures

Interested individuals called the laboratory and completed an initial telephone screening interview for the inclusion and exclusion criteria outlined above. During this telephone interview, participants were asked about quantity and frequency of drinking and if they had ever been diagnosed with one of the exclusionary psychiatric disorders, namely bipolar disorder or any psychotic disorder. Participants were also asked whether they wanted to receive any treatment now or had received any treatment for alcohol problems (including formal treatment and/or use of self-help groups) in the past 30 days and were excluded for positive answers. Treatment seekers were excluded as the next phase of the study included an alcohol administration. Those who did indicate a desire for treatment were provided with a referral packet. Eligible participants were invited to a face-to-face assessment session, which included the BART, as well as the individual differences and neurocognitive measures described below. During the in-person testing session prior to the assessment procedures, all participants provided written informed consent upon receiving a complete explanation of the study. Blood alcohol concentration (BAC) equal to 0.00, as verified by a Breathalyzer test (Dräger, Telford PA), was required before assessment commenced. All procedures were approved by the Institutional Review Board of the University of California, Los Angeles.

Measures

Demographic information was collected, including age, sex, ethnicity, and education. In-depth assessment of alcohol use and alcohol-related symptoms was performed, along with the BART and the following neurocognitive and individual differences measures.

Alcohol Use and Related Symptoms—Alcohol dependence and the exclusionary psychiatric diagnoses (i.e., bipolar disorder or psychotic disorder) were assessed using the

Structured Clinical Interview for DSM-IV (SCID; (First, Spitzer, Gibbon, & Williams, 1995) by bachelor's-level interviewers or graduate students under the training and supervision of a licensed clinical psychologist (last author). Alcohol abuse and dependence symptoms were recorded, for a total of 11 possible DSM-IV symptoms. The 30-day timeline follow-back (TLFB) interview (Sobell & Sobell, 1980) was used to assess drinking behavior including detailed data on the quantity and frequency of alcohol use over a 30-day period. An alcohol binge was defined as consuming 4 or more drinks on a given episode for a woman and 5 or more drinks for a man. The following measures of alcohol use were derived from the 30-day TLFB and used in the analyses: (a) total drinking days; (b) average drinks per drinking day; and (c) total binge drinking days.

The BART—A modified version of the BART (Lejuez et al., 2002) was administered as follows. Participants were presented with a picture of a balloon on the computer screen and could press two keys: one to inflate the balloon (“pump”), and one to end the trial (“cash out”) and move on to the next trial. Each balloon trial began at a value of \$0.01. With each pump, the participant could earn a small amount of money (\$0.003) that was tallied continuously, and the balloon would near-instantly inflate on-screen by a small amount. Participants chose at each pump whether to continue to inflate the balloon or to press the “cash out” key to end the trial, add the money to the guaranteed “bank”, and begin the next trial. However, a certain amount of risk is applied to each pump, such that inflation to a certain point will cause the balloon to visibly explode on the screen resulting in a loss of money earned so far on that trial. Risk of balloon explosion was distributed following a normal distribution with a mean at the midpoint of possible pumps (32 of 64 possible pumps) and with a standard deviation of 20; this value is half that of several previous studies employing the BART, which allowed a full range of 128 pumps. Each session consisted of 72 trials, which is more than used previously (e.g., (Lejuez et al., 2002) (MacPherson et al., 2010) (Fernie et al., 2010) (Lejuez, Aklin, Zvolensky, & Pedulla, 2003). These modifications were made to decrease the possibility of participant fatigue on individual trials, while still obtaining data from a large number of trials for reliability purposes. Because we were interested in pursuing sub-measures of the task (including the mean of pumps occurring after a trial failure), it was necessary to ensure that across the session enough explosions would occur to generate reliable data even among the more conservative participants. The task was administered in MATLAB (v7.5) on a Macbook laptop.

Participants earned up to an additional \$5.00 based on their performance on the BART. This amount was chosen as a function of the proportion of time spent on the task in comparison to the full assessment visit, as only about 12 minutes of the three-hour visit were spent on the BART. Because inclusion of pumps made in trials that resulted in explosions may negatively bias the mean, the adjusted mean pumps (AMP) was used as a primary variable of risk-taking propensity (Lejuez et al., 2002). This is the standard adjusted average used in other studies using the BART. In addition to AMP, a measure of response variability was derived using the within-subject inter-trial variability of pumps at “cash out”, divided by AMP (as variability will naturally increase with the mean). This measure was included based upon recent pre-clinical reports that within-subject response variability in the BART is an indicator of optimized performance that is mechanistically-dissociable from mean

pumps per trial (Jentsch et al., 2010). Hereafter, this variability measure will be referred to as VARAMP. The average number of pumps on trials immediately following a trial failure (balloon explosion) was also calculated and will be referred to as post-failure mean pumps (PFMP).

Neurocognitive and Individual Difference Measures—The following neurocognitive and individual differences measures of high relevance to BART performance were administered: (1) The Shipley Institute for Living Scale (Zachary, 1986) is a brief self-report measure which provides both verbal, performance, and total IQ estimates that are moderately correlated with the WAIS-derived full scale IQ; (2) The Digit Span Task is a working memory task in which participants are asked to recall various sequences of numbers, either recalling the numbers from first to last (Digits Forward) or recalling the numbers from last to first (Digits Backward). This is a classic working memory task that captures individuals' abilities to cognitively retain and manipulate information. Norm-referenced scores from the Shipley scale and digit span task were used in the analyses as estimates of IQ and working memory, respectively.

Data Analytic Plan

Prior to the main analyses, all variables were tested for the distributional assumptions and were transformed, including norm-based transformations, as needed. For t-tests, comparisons that violated the homogeneity of variance assumption were remedied using the Satterthwaite corrected degrees of freedom. All comparisons and analyses were performed using SAS (v9.2) running on a PC. Primary analyses employed Pearson Product Moment Correlations and regression analyses using the General Linear Model to test the association of the individual dimensions of BART performance and measures of alcohol consumption (TLFB) and symptoms of alcohol abuse and dependence (Symptom Count). Age, sex, IQ, and WM were added as covariates to the models regressing alcohol use and symptoms on BART indices. These covariance models were introduced when significant associations between BART performance and alcohol measures were obtained in order to further probe for the validity of the univariate findings. As these four variables fall into two separate domains (demographic variables and neurocognitive variables), two separate models were tested. The first tested for the explanatory contribution of the demographic control variables (age and sex) to the main relationship between AUD symptomatology and risk-taking, while the second model tested inclusion of neurocognitive control variables (IQ and WM).

Given the number of variables being compared, corrections for multiple comparisons were made following the recommendations of Dar, Serlin and Omer (1994). To that end, we adjusted for the number of hypotheses being tested as opposed to the number of variables indexing these hypotheses. Since we are testing for the relationships between three dimensions of the BART (i.e., mean adjusted pumps, response variability, and post-failure mean pumps) and two levels of alcohol misuse (alcohol use and alcohol problems), we have corrected for p-value for 6 planned comparisons/hypotheses. Thus, the p value required for significance was $p=0.0083$. Lastly, in order to further elucidate the multivariate nature of the associations among BART performance, alcohol use and problems, and the demographic and neurocognitive covariates, mediation and moderation models were examined where

appropriate following the steps endorsed by Baron and Kenny (1986). The Sobel test was used to determine mediation (Sobel, 1982) using available online software (Preacher & Leonardelli, March 2001).

Results

Descriptive Statistics

Six subjects were removed from the analyses as a result of positive assessments for either bipolar disorder or psychosis, as determined by the SCID, leaving a total of 152 subjects in the analyses reported herein. Of those, 72.3% met criteria for alcohol dependence, 15.5% met DSM-IV criteria for alcohol abuse only, 9.5% were diagnostic orphans (i.e., endorsed 1 or 2 dependence symptoms but did not meet diagnostic criteria for either alcohol abuse or dependence), and 2.7% did not endorse any symptoms of either alcohol abuse or dependence. Participants consumed an average of 6.49 standard drinks per drinking day ($SD = 4.5$, Range: 1.7 to 34.3), with an average of 128.4 drinks ($SD = 101.8$, Range: 5 to 549.4) over the past 30 days.

Means and standard deviations of BART performance and other study measures (e.g., norm-referenced IQ and digit span scores, alcohol consumption data) are presented in Table 1, along with correlations among them. Participants demonstrated a range of performance on the BART in terms of AMP ($M = 18.61$, $SD = 4.30$), with some being more variable between trials than others (VARAMP, $M = 1.23$, $SD = 0.89$). Nearly all participants were generally characterized as risk averse, falling on the lower half of potential pumps, consistent with previous samples (Hopko et al., 2006; Lejuez et al., 2003; Lejuez et al., 2002; Lejuez, Simmons, Aklin, Daughters, & Dvir, 2004; Skeel et al., 2008; White et al., 2008). Participants earned on average \$3.48 ($SD = \0.57) during the task and took an average of 12.33 minutes ($SD = 3.75$ minutes) to complete it. Across all 72 trials, participants exceeded the risk function resulting in a balloon explosion an average of 17.18 times ($SD = 8.96$).

Sex differences in all study variables were considered in an independent samples t-test (Table 2). Men and women did not significantly differ in BART performance in terms of the mean adjusted pumps or post-failure mean pumps. The only measures for which there was a significant sex effect were number of drinking days in the past 30 days, and number of binge days, suggesting heavier drinking in males versus females.

Participants' performance on the BART is illustrated in Figure 1, which relates AMP to money earned over the entire session. The optimal performance curve was calculated by determining the expected reward accrued given the expected percent of failed trials at any given level of risk-taking. As can be seen, most participants' mean responses fell to the left of the curve and many participants demonstrated suboptimal behavior by receiving much less money than would be expected. Split-half correlation of the means of pumps from the first and last half of trials showed that participants' behavior was consistent across the full session, $r(150) = 0.826$, $p < 0.001$. As an additional confirmation of within-subject reliability across the session, a paired samples t-test was conducted comparing AMP of the

first and second halves; participants showed no significant difference between these two halves, $t(151) = 0.76, p = 0.45$).

BART Performance Indices and AUD Symptoms

AUD symptom count was significantly and *negatively* correlated with AMP (Table 1). The observed correlation falls in the range of medium effect size (Cohen, 1988, 1992). AMP was not significantly associated with number of drinking days, drinks per drinking day, or number of binge days. PFMP was also significantly and negatively associated with symptom count, as well as with average drinks per drinking day, but not number of binge days. VARAMP was not associated with any alcohol measures. Complete results are presented in Table 1.

Probing the Relationship between BART and AUD: Demographic and Neurocognitive Controls

Analyses were conducted to further probe the univariate associations obtained. Specifically, significant associations between dimensions of the BART and alcohol use disorders symptoms and alcohol use were investigated after controlling for relevant demographic (gender and sex) and neurocognitive (IQ and WM) variables (see Table 3). Results for demographic control variables revealed that adding sex and age to the regression model attenuated the negative relationship between symptom count and AMP. However, the association between PFMP and symptom count remained significant after controlling for age and sex. Additionally, PFMP remained significantly associated with drinks per drinking day when sex and age were added to the model.

Analyses controlling for neurocognitive variables revealed that the negative relationship between symptom count and AMP was attenuated by adding estimated IQ and WM to the model. Unlike the relationship observed with AMP, the association between PFMP and symptom count remained significant when controlling for WM and IQ. Lastly, covariate analysis was conducted for the association between drinks per drinking day and PFMP. The relationship between PFMP and drinks per drinking day (DPDD) remained significantly, and negative in nature, even after controlling for IQ and WM indices.

Analyses of Mediation and Moderation Effects

In order to further elucidate the multivariate nature of the associations among BART performance, alcohol use and problems, and the demographic and neurocognitive covariates (i.e., age, sex, IQ, and working memory), mediation and moderation models were tested. Results revealed no significant moderation effects of age, sex, IQ, or working memory ($ps > .10$) in the relationships between performance on the BART and alcohol use and problems demonstrated above.

Mediation effects for age, sex, IQ and WM were examined as proposed by Baron and Kenny (1986). In brief, this approach consists of examining univariate relationships between (a) the independent variable (IV) and the proposed mediator, (b) the IV and dependent variable (DV), and (c) between the DV and mediator. Should all univariate paths of the model be significant, a multivariate model is tested in which both the IV and mediator are

simultaneously tested as predictors of the DV. Mediation is thought to occur when the magnitude of the IV to DV relationship is significantly attenuated by the mediator. A Sobel Test was used to formally test the mediation effects (Sobel, 1982) (Preacher & Leonardelli, March 2001).

As can be seen in Table 1, all univariate paths are significant for the associations between BART performance (IV; measured by AMP and PFMD), age (mediator), and symptom count (DV). As such, mediation analyses were justified for these variables. Multivariate analyses revealed that the association between BART performance and symptom count is significantly attenuated by adding age to the model, providing initial evidence of mediation. Sobel tests confirmed that age significantly mediated the relationship between AMP and symptom count (Sobel Test = -3.65, $p < .001$) and the relationship between PFMP and symptom count (Sobel Test = 3.61, $p < .001$). Moreover, analyses of IQ as a putative mediator revealed that it significantly explained the relationship between AMP and symptom count (Sobel Test = -3.22, $p < .01$), between PFMP and symptom count (Sobel Test = -3.05, $p < .01$), and between PFMP and drinks per drinking day (Sobel Test = -2.42, $p < .01$). All univariate relationships can be seen in Table 1. Together, these analyses suggest that age and IQ serve as mediators of the relationships between performance on the BART and alcohol problems in this sample.

Discussion

This study sought to examine, for the first time, the relationship between performance on the BART and AUD symptoms in a community sample of problem drinkers. These analyses also included relevant covariates in order to more fully characterize the main effects of BART performance on AUD symptomatology and drinking behavior. Analyses revealed that participants reporting more AUD symptoms were more *conservative* on the BART, as indexed by average pumps (AMP). The observed association was in the range of medium effect sizes (Cohen, 1988, 1992) and remained significant, albeit marginally, when controlling for working memory. Importantly, when controlling for IQ and demographic measures, the association between AMP and symptom count was no longer significant. Together, these results suggest that among individuals with AUD, higher risk-taking propensity, measured by the BART, is associated with lower symptom count, but that IQ and age account for a substantial proportion of the variance in BART performance and ultimately mediate this association. Thus, in order to fully understand the relationships between risk-taking and AUD symptomatology, age and IQ must be taken into account.

This finding is in contrast to studies of subclinical and adolescent samples reporting a *positive* relationship between BART performance and risk-taking behaviors, including substance use and abuse (Hopko et al., 2006; Lejuez et al., 2005; Lejuez et al., 2002; MacPherson et al., 2010; Skeel et al., 2008). However, these results are consistent with a recent report showing that adolescent smokers were more conservative on the BART than nonsmoking controls (Dean et al., in press). Furthermore, AMP did not relate to alcohol consumption variables, such as drinking frequency, quantity, and binge drinking. It may be that there was not sufficient variability in drinking patterns to detect BART effects. However, there was considerable variability in the degree to which participants reported

problems associated with alcohol use, which afforded greater statistical power to detect associations with alcohol use disorders symptomatology. The effects of age on BART performance are in contrast with the prospective study documenting increasing risk-taking in BART performance over time in an adolescent cohort (MacPherson et al., 2010). In the current study, age was inversely correlated with risk-taking on the BART and mediated the relationship between AMP and symptom count. The average age of the sample was 30 years old; therefore, the current findings, in conjunction with the existing adolescent literature, suggest that developmental and age considerations may be central to understanding the predictive utility of risk-taking measured by the BART and alcohol use disorders.

Examination of Multiple Dimensions of the BART

The measure of inter-trial variability (VARAMP) was not associated with alcohol use and problems in this sample. This suggests that within this sample, variability in responding was not a useful phenotype for understanding AUD symptoms or drinking behavior. Although not predictive of alcohol problem severity among individuals currently exhibiting AUDs, these data support the hypothesis that the response variability phenotype is dissociable from the traditional AMP measure in human samples, which is consistent with recent pre-clinical findings (Jentsch et al., 2010). It remains to be seen if this lack of association is unique to this sample of problem drinkers or if this extends to social drinkers or younger samples of alcohol and drug users.

The measure of PFMP generally showed a larger effect size in relationship to alcohol use and related problems than did AMP. This includes a significant negative association with drinking behavior in terms of average alcohol units consumed per episode and number of binge days during the assessed period. Furthermore, this relationship survived control for demographics, IQ, and working memory. Indeed, IQ was also found to mediate this relationship. This indicates that responses to negative feedback (i.e., explosions) in the context of the BART may be a useful predictor of alcohol problems and consumption and that this association may be accounted for by differences in IQ. As with the main AMP measure, the relationship is negative, such that those who accepted more risk, on average, after a trial-failure had fewer problems and drank less. Thus, the ability to accept more risk after recent failures may be protective against alcohol problems, or more severe AUD symptomatology may associate with greater affective responses to failure. Interestingly, the recent study of adolescent smokers and nonsmokers also found a significant and positive association between verbal IQ and mean pumps on high risk balloons (Dean et al., in press), although mediation analyses were not conducted.

Given the cross-sectional nature of the study it is not possible to ascertain the direction of causality in these associations. For instance, it may be the case that the more risk-averse subset of heavy drinkers is more likely go on to develop AUD. This model would imply that in adulthood, risk-taking is a stable trait despite environmental influence, including alcohol use itself, and that a more conservative risk-taking profile represents a risk factor for the development of more severe AUD. Alternatively, it may be the case that the negative relationship between risk-taking on the BART and alcohol symptoms may be caused by the biobehavioral effects of extensive alcohol use itself. In other words, although higher risk-

taking measured by the BART may be a liability factor for the onset of (heavy) alcohol use in a subset of individuals (Lejuez et al., 2002; Macpherson, Magidson, Reynolds, Kahler, & Lejuez, in press; Skeel et al., 2008), as alcohol use progresses, the relationship may reverse direction. Thus, the trait of risk-proneness as assessed by the BART may change across development and upon chronic alcohol exposure.

The plausibility of this hypothesis is supported by recent work demonstrating changes in risk-taking propensity across adolescence and young adulthood (MacPherson et al., 2010), confirming that this trait is liable to change. This conceptualization may also be consistent with the allostatic model of addiction in which binge-intoxication represents the early, impulsive stages of the disorder (Koob, 2003). This is also consistent with developmental psychopathology models of alcohol misuse, in which risk-taking propensity plays an important role in alcohol use initiation and transition to heavy drinking in youth (Brown et al., 2008).

Two possible mechanisms may underlie such a change in risk-taking as alcohol dependence progresses. The first is a direct pharmacological effect of repeated alcohol intoxication. The second is a negative feedback mechanism, such that those with more severe dependence then develop a more conservative temperament as a result of the negative consequences experienced during the onset and continuation of dependence. Both of these would be important hypotheses to test in future studies. Indeed, pharmacologically-induced neurocognitive effects of chronic alcohol use are plausible as IQ estimates were negatively associated with symptom count; participants who drank more and had more problems due to alcohol use tended to have lower estimated IQ scores and WM span. Although this is an association and the causal relationship cannot be determined here, this further supports the plausibility that chronic alcohol exposure may result in neurocognitive deficits with behavioral ramifications.

Despite these likely alcohol-related neurocognitive deficits, the inverse relationship between PFMP and symptom count remained significant even after controlling for WM span and IQ. Thus, over and above more global neurocognitive deficits associated with chronic alcohol use, it is possible that alcohol misuse itself may alter risk-taking behavior towards risk aversion in the BART context, possibly through a pharmacological mechanism. Lastly, an intriguing alternative explanation offered by Dean et al. (in press), consists of the notion that since most participants did not exceed the optimal reward/risk ration for the task (see Figure 1), participants who pumped more on the task often made more money, which in turn suggests that taking risks on the BART may represent an adaptive response. This conceptualization would be consistent with the meditational findings for IQ and suggest that individuals with higher IQ are better able to perform optimally on the BART (i.e., accept more “adaptive risk”), which in turn is negatively associated with AUD outcomes. Delineating the optimal and even adaptive level of risk-taking clearly warrants further investigation.

Design Considerations and Future Directions

Regardless of the direction of causality, it appears that having a high risk-taking trait (at least as can be measured in the laboratory by the BART) may not contribute to AUD

severity in the later stages of AUD development above and beyond its initial contribution to initiation of substance use. Prospective studies are certainly needed to empirically address the research questions raised by the study findings. These should consist of testing the pharmacological and negative feedback mechanistic hypotheses, as well as include age-matched non-AUD and abstinent community participants to consider the full range of alcohol use as it relates to the traditional AMP measure as well as the post-failure (PFMP) measure; these will greatly contribute to our understanding of how the risk-taking construct influences the development of AUD symptomatology, from initiation of alcohol use to severe dependence, as well as how this reflects underlying AUD etiology. Future studies should also include assessments of IQ and WM, as these are clearly important control variables when evaluating behavior in the BART.

This study has a number of strengths and limitations. Study limitations include the cross-sectional design and a sample comprised of individuals already exhibiting alcohol problems who are not seeking treatment. This may hinder direct comparison to other at-risk and non-dependent samples already phenotyped for risk-taking propensity and alcohol-related behavior. Next, the imbalance of sex may have reduced the power of detecting a sex effect on BART performance or moderation effects of sex, BART and AUD symptoms. However, the only such effect reported in the literature was found in subjects under acute stress (Lighthall, Mather, & Gorlick, 2009).

As the version of the BART employed in this study differed from the traditional version of the task, it is important to consider if the effects observed might be specific to this task variant. The BART used here had a larger number of trials (72), a shorter range of potential pumps within a trial (64), and a low cash value for a single pump (0.03 cents) while previous versions have typically included 30 trials on one type of balloon and a range of 128 pumps (e.g., Fernie et al., 2010; Lejuez et al., 2002; 2005; MacPherson et al., 2010; Skeel et al., 2008). Data from several variants of the task have already been published describing limited effects of task modifications. In the original task development study (Lejuez et al., 2002), balloons with potential ranges of 8, 32, and 128 pumps were concurrently tested and results revealed that the limited range balloons (8 and 32) showed no relationship with self-reported real-world risk behaviors as variability in performance was too limited. Furthermore, multiple task versions including modifications beyond number of trials and range of potential pumps have been published with no obvious or reported effects on participant behavior (Pleskac, Wallsten, Wang, & Lejuez, 2008; Zacny & de Wit, 2009). While the issue of task variants should be kept in mind when comparing results across studies, participants do show consistently risk-averse behavior, and the effects of varying the task generally appear to alter the variability observed in participant performance rather than altering the relationships per se.

Study strengths include the community sample of individuals with alcohol problems, including a large number of dependent individuals, as no studies addressing alcohol use and the BART published to date have sampled from a clinical population. In addition, this study employed a semi-structured diagnostic interview and included multiple relevant neurocognitive covariates, such as IQ and working memory, which appear to be critical to evaluating the relationship between risk-taking and AUD.

On balance, this study advances the understanding of risky decision making and AUDs by (1) employing a well-validated behavioral measure of risky decision making, the BART; (2) examining BART performance in a clinical sample phenotyped for diagnostic and neurocognitive measures of interest; and (3) considering multiple facets of BART performance, including response variability and post-failure reactivity. Results revealed a negative relationship between risk-taking on the BART and alcohol symptoms and problems, suggesting that although this behavior analog measure was positively associated with alcohol and substance use in non-clinical samples, the direction of the relationship may be reversed in clinical samples, such that individuals with more alcohol pathology are more risk averse. Importantly, IQ estimates were found to mediate the observed relationships between BART performance and AUD symptoms/alcohol use. Whether the observed reversal of the relationship between risk-taking and alcohol pathology reveals a cause or a consequence of chronic alcohol use, or rather reflects different stages of addiction, remains to be empirically determined.

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Figure 1. Performance on the BART

Each dark square represents an individual participant. The curve represents an optimal function based on the chance of failure at any given mean of pumps and the expected amount of money accrued over an entire session.

Table 1

Means, Standard Deviations, and Correlations Among Study Variables.

Variable	Mean	SD	1	2	3	4	5	6	7	8	9
Demographic Information											
1. Age	30.01	10.41	-								
BART Performance Measures											
2. Adjusted Mean Pumps	18.61	4.32	-0.359**	-							
3. Variability of Pumps	1.25	0.84	0.038	-0.305**	-						
4. Post-Failure Pumps	17.92	3.86	-0.357**	0.873**	-0.114	-					
Individual Difference Measures											
5. IQ	100.20	20.56	-0.536**	0.449**	-0.164	0.395**	-				
6. Digit Span	10.38	3.07	-0.319**	0.345**	-0.151	0.318**	0.629**	-			
Alcohol Use and Pathology Measures											
7. AUD symptom count	5.36	2.93	0.457**	-0.228*	-0.022	-0.317**	-0.304*	-0.176	-		
8. Total Drinking Days	18.02	7.73	0.389**	-0.118	-0.068	-0.128	-0.238*	-0.126	0.416**	-	
9. Drinks per Drinking Day	6.95	4.52	0.212	-0.119	-0.085	-0.241*	-0.224*	-0.108	0.286*	0.068	-
10. Total Binge Drinking Days	12.30	8.52	0.440**	-0.096	-0.127	-0.188	-0.139	-0.049	0.547**	0.714**	0.413**

* Indicates significance at $p < 0.0083$.** at $p < 0.0001$.

The first value was chosen as the corrected significance level based on the number of planned comparisons (see Data Analytic Plan).

Table 2
Sex differences across study variables

Variable	Women Mean (SD)	Men Mean (SD)	df	t value
Age	28.52 (9.39)	30.65 (10.80)	143	-1.13
AMP	17.74 (3.14)	18.95 (4.67)	116	-1.81
VARAMP	1.30 (0.80)	1.23 (0.86)	140	0.45
PFMP	17.43 (2.64)	18.08 (4.24)	120	-1.10
IQ	98.56 (21.28)	101.25 (20.14)	142	-0.71
WM	9.82 (2.73)	10.65 (3.19)	142	-1.50
Count	4.66 (3.00)	5.66 (2.88)	144	-1.89
Drink Days	15.79 (6.87)	20.56 (7.74)	144	-2.40*
DPDD	6.00 (5.88)	7.37 (3.80)	59	-1.42
Binge Days	9.50 (6.83)	13.87 (8.72)	144	-2.96**

Independent samples t-tests comparing men and women across study variables.

* indicates $p < 0.05$ and

** $p < 0.001$.

AMP = adjusted mean pumps; VARAMP = inter-trial variability in pumps; PFMP = post-failure mean pumps; count = number of AUD symptoms obtained from SCID interview; drink days = number of days of alcohol consumption in last 30 days; DPDD = drinks per drinking day.

For comparisons violating homogeneity of variance, Satterthwaite corrected degrees of freedom were applied.

Table 3
Regression Models Including Control Covariates

Dependent Variable: Symptom Count			
<i>Demographic Controls</i>	β	Standard Error	<i>p</i> value
Model 1:			
AMP	-0.06	0.06	0.27
Sex	0.84	0.49	0.09
Age	0.11	0.02	< 0.001**
Model 2:			
PFMP	-0.14	0.06	0.03*
Sex	1.02	0.47	0.03*
Age	0.11	0.02	< 0.001**
<i>Neurocognitive Controls</i>			
Model 1:			
AMP	-0.08	0.06	0.21
IQ	-0.04	0.02	0.01*
WM	0.05	0.10	0.64
Model 2:			
PFMP	-0.17	0.07	0.01*
IQ	-0.04	0.02	0.006**
WM	0.07	0.10	0.64
Dependent Variable: Drinks per Drinking Day			
<i>Demographic Controls</i>	β	Standard Error	<i>p</i> value
Model 1:			
PFMP	-0.25	0.11	0.02*
Sex	1.68	0.83	0.04*
Age	0.055	0.04	0.17
<i>Neurocognitive Controls</i>			
Model 2:			
PFMP	-0.22	0.11	0.04*
IQ	-0.05	0.02	0.05*
WM	0.12	0.16	0.45

Regression models that included both demographic and neurocognitive covariates as control variables were tested. AMP = adjusted mean pumps; PFMP = post-failure mean pumps; WM = working memory.

* indicates $p < 0.05$,

** $p < 0.01$.