Alcohol Expectancies and Drinking Refusal Self-Efficacy Mediate the Association of Impulsivity With Alcohol Misuse

Matthew J. Gullo, Sharon Dawe, Nicolas Kambouropoulos, Petra K. Staiger, and Chris J. Jackson

Background: Recent work suggests that 2 biologically based traits convey risk for alcohol misuse: reward sensitivity/drive and (rash) impulsiveness. However, the cognitive mechanisms through which these traits convey risk are unclear. This study tested a model predicting that the risk conveyed by reward sensitivity is mediated by a learning bias for the reinforcing outcomes of alcohol consumption (i.e., positive alcohol expectancy). The model also proposed that the risk conveyed by rash impulsiveness (RI) is mediated by drinkers’ perceived ability to resist alcohol (i.e., drinking refusal self-efficacy).

Methods: Study 1 tested the model in a sample of young adults (n = 342). Study 2 tested the model in a sample of treatment-seeking substance abusers (n = 121). All participants completed a battery of personality, cognitive, and alcohol use questionnaires and models were tested using structural equation modeling.

Results: In both studies, the hypothesized model was found to provide a good fit to the data, and a better fit than alternative models. In both young adults and treatment-seeking individuals, positive alcohol expectancy fully mediated the association between reward sensitivity and hazardous alcohol use. For treatment seekers, drinking refusal self-efficacy fully mediated the association between RI and hazardous drinking. However, there was partial mediation in the young adult sample. Furthermore, neither trait was directly associated with the other cognitive mediator.

Conclusions: The hypothesized model was confirmed on a large sample of young adults and replicated on a sample of treatment-seeking substance abusers. Taken together, these findings shed further light on the mechanisms through which an impulsive temperament may convey risk for alcohol misuse.

Key Words: Impulsivity, Alcohol Expectancy, Drinking Refusal Self-Efficacy, Alcohol, Reward.
recently, expectancy challenges have also been shown to reduce young children’s positive perceptions of alcohol (Cruz and Dunn, 2003). Importantly, the acquisition of positive expectancies (PE) regarding alcohol does not require direct experience with alcohol, as these expectancies could arise through vicarious learning or modeling (e.g., Bandura, 1977). Indeed, alcohol expectancies have been reported in young children with no alcohol experience (Anderson et al., 2005b; Miller et al., 1990).

Several studies have also investigated the relative contribution of impulsivity and outcome expectancies to alcohol misuse. Smith and Anderson (2001) initially proposed a model in which trait impulsivity/disinhibition influenced learning by creating a bias in which the positive outcomes of a previous drinking occasion (or observed drinking occasion) are more likely to be encoded into memory. This, in turn, was proposed to influence the likelihood of future drinking occasions. They hypothesized that such expectancies influence future drinking and may be a mechanism through which trait impulsivity conveys risk. However, there has been mixed support for this model (e.g., Anderson et al., 2003; McCarthy et al., 2001). The other key cognitive variable that has been identified as having a central role in alcohol treatment outcome is refusal self-efficacy (Adamson et al., 2009; Kavanagh et al., 1996; Solomon and Annis, 1990).

Drinking refusal self-efficacy has been found to contribute unique variance in the prediction of alcohol use over-and-above expectancies. Oei and Jardim (2007) found that alcohol expectancies and drinking refusal self-efficacy each contributed unique variance to the prediction of alcohol consumption in college students. Finally, Young and colleagues (2006) found drinking refusal self-efficacy contributed significant unique variance to the prediction of alcohol consumption over-and-above positive and negative alcohol expectancies in a sample of 174 college students.

In comparison with studies reporting on nondependent samples, studies that have examined the differential contribution of positive alcohol expectancy and drinking refusal self-efficacy in alcohol-dependent populations tend to find less consistent results. Connor and colleagues (2007) reported that poor drinking refusal self-efficacy was significantly related to higher levels of quantity, frequency, and alcohol-related problems in a sample of alcohol-dependent patients participating in a detoxification program ($N = 143$). Alcohol expectancy was only predictive of alcohol-related problems. In contrast, in a large sample of recently detoxified alcohol-dependent patients ($N = 296$), Oei and colleagues (2007) found no relationship between alcohol expectancy or drinking refusal self-efficacy and volume or frequency of alcohol consumption. Furthermore, in an equally large sample of recently detoxified patients ($N = 300$), Hasking and Oei (2002) reported that alcohol expectancy predicted concurrent frequency of alcohol consumption, but not quantity of consumption and that refusal self-efficacy was not related to either index of consumption. Thus, to date, there is considerable evidence linking impulsivity to substance use and misuse, evidence that outcome expectancies are associated with the initiation and ongoing use of alcohol, and that self-efficacy is linked to treatment outcome. However, any theoretical model of alcohol misuse that attempts to integrate these 3 constructs must also take into account recent research concerning the structure of impulsivity.

There is now an emerging consensus that impulsivity is not a simple, 1-dimensional trait, but rather comprises at least 2 separate facets relevant to addictive behavior (Belin et al., 2008; Dawe and Loxton, 2004; Swann et al., 2002; de Wit and Richards, 2004). At the trait level, the first component manifests as differences in sensitivity to rewarding stimuli and the strength of motivation to obtain them (sensitivity to reward or “reward drive,” SR; Dawe and Loxton, 2004). In more general models of personality, this dimension is reflected in traits such as extraversion, agency, positive emotionality, and Gray’s (1970) Behavioral Approach System (BAS; Depue and Collins, 1999; Gullo and Dawe, 2008). Indeed, the evidence suggests that SR lies at the core of each of these traits (Depue and Collins, 1999; Lucas and Diener, 2001). Furthermore, cross-sectional studies have reported significant associations between SR and severity of alcohol problems in dependent samples (Franken et al., 2006; Johnson et al., 2003).

The second component, at the trait level, manifests as individual differences in inhibitory control and the propensity to act without forethought (RI; Dawe and Loxton, 2004). In more general models of personality, this dimension is reflected in traits such as constraint, conscientiousness, and even psychoticism1 (Depue and Collins, 1999; Gullo and Dawe, 2008). While each conceptualization has its own unique emphasis, the evidence suggests that inhibitory control lies at the core of each of these traits (Depue and Collins, 1999; Rawlings and Dawe, 2008). A significant relationship between RI and vulnerability to substance abuse has also been consistently reported in both cross-sectional and prospective clinical studies (Moeller et al., 2002; Tarter et al., 2003).

Several studies have linked SR and RI to distinct behavioral processes involved in hazardous drinking. Self-report measures of SR have been associated with stronger physiological responses to alcohol, as well as stronger conditioning to alcohol cues (Brunelle et al., 2004; Glautier et al., 2000). High scores on measures of SR have also been associated with greater craving and approach motivation in response to alcohol cues (Franken, 2002; Kambouropoulos and Staiger, 2001). By contrast, RI has been linked to inhibitory deficits in alcohol-dependent individuals (Finn et al., 2002) and appears to mediate the behavioral disinhibition associated with a family history of alcoholism (Lovallo et al., 2006; Saunders et al., 2008). Thus, recent behavioral studies support a differential role for SR and RI in hazardous drinking.

1Despite Eysenck’s original intention, psychoticism, as currently measured, is now generally regarded as reflecting impulsivity and antisociality more than psychosis-proneness (Rawlings and Dawe, 2008).
Notably, the conceptualization of impulsivity discussed in Smith and Anderson’s (2001) model shares many of the features of RI, as it was based on Patterson and Newman’s (1993) model of disinhibition. This may explain, in part, the mixed empirical support for their model. While it is true that Patterson and Newman’s model proposes a pathway in which trait disinhibition (or RI) can reduce the likelihood of learning from aversive events, they also argue that extraverts and those high in BAS sensitivity are better at learning about rewards (such as alcohol) and, therefore, more motivated to acquire them. This is in line with the argument made by Dawe and colleagues that only the SR trait would be associated with reward-related learning and, by extension, positive alcohol expectancy (Dawe et al., 2004; Gullo and Dawe, 2008). Indeed, the findings of a recent study by Anderson and colleagues (Anderson et al., 2005a) are consistent with this argument. They reported that while extraversion was significantly related to positive alcohol expectancy in adolescents, Patterson and Newman’s disinhibition did not account for any additional variance. It is important to emphasize that Patterson and Newman’s model focuses more on reversal learning (in which a formerly rewarded response is now punished and should be inhibited) than the initial acquisition of conditioned associations. Furthermore, reversal learning is associated with RI (Franken et al., 2008). Based on these considerations, we proposed that positive alcohol expectancy would mediate the association of SR with alcohol misuse, and not the association of RI/disinhibition with misuse.

While no study has yet examined the relationship of impulsivity traits to drinking refusal self-efficacy, it is proposed that RI would be negatively related to such self-efficacy. Specifically, we proposed that individuals with poor inhibitory control should be more aware of their difficulty in refusing immediate rewards generally. This would then apply to alcohol drinking behavior just like any other reward. That is, the relationship is not predicted to result from any particular learning bias, but rather from direct experience with past goal-conflict situations (whether or not it required refusal of alcohol specifically). Our prediction is still consistent with Patterson and Newman’s (1993) disinhibition model in that we are simply proposing that high-RI individuals are more likely to be aware of their poor inhibitory control and, in turn, should expect to have greater difficulty resisting alcohol (like any other reward). In summary, the purpose of the proposed studies was to investigate these 2 cognitive pathways linking impulsivity to alcohol misuse in 2 samples: young college students at an earlier stage of their drinking history (study 2), and a sample of individuals seeking treatment for substance use problems (study 2).

**STUDY 1**

Based on recent work into the structure of impulsivity, a modification to Smith and Anderson’s (2001) model was proposed, in which only the association between SR and hazardous drinking was expected to be mediated by PE. In addition to this, the hypothesized model predicted that the association between RI and hazardous drinking would be mediated by drinking refusal self-efficacy. Both cognitive mechanisms were hypothesized to account for significant unique variance in hazardous drinking in a young college population. Positive alcohol expectancy was also expected to predict variance in refusal self-efficacy, in that, an individual who expects more positive outcomes from drinking is likely to experience more difficulty refusing the substance.

**MATERIALS AND METHODS**

**Participants**

The sample consisted of 342 college students (262 Caucasian, 45 Asian, and 35 “other”); 244 (71.3%) were female. Their mean age was 21.16 (SD = 5.19). Informed consent was obtained from each participant and the study was approved by the relevant university ethics committee.

**Materials**

**Reward sensitivity** was assessed using the Sensitivity to Reward scale (SR; Torrubia et al., 2001), BAS: Drive scale (BAS-D; Carver and White, 1994), and a modified version of the extraversion scale from the Eysenck Personality Questionnaire – Revised (EPQ-R; Eysenck and Eysenck, 1994). Each measure has been shown to have good internal and test–retest reliability, and factor analytic studies have found these scales to load on an SR factor with other SR-related self-report measures, supporting their convergent validity (Carver and White, 1994; Dawe and Loxton, 2004; Eysenck and Eysenck, 1994; Torrubia et al., 2001).

For the purposes of this study, the extraversion scale of the EPQR was modified to more selectively measure those aspects of extraversion related to agency. “Agentic extraversion” has been argued to be more related to SR and mesolimbic dopamine functioning (Depue and Collins, 1999; Gullo and Dawe, 2008). This involved the removal of items related to sociability, a component of extraversion that has been argued to be less related to the mesolimbic system (Depue and Collins, 1999). The 13-item ² agentic extraversion scale was found to have adequate internal consistency in the present study (Cronbach’s α = 0.76). The new measure was also subjected to a confirmatory factor analysis (CFA), which found the modified scale provided a good fit to the data, χ² (2, N = 327) = 2.87, p = 0.24, comparative fit index (CFI) > 0.99, standardized root mean-square residual (SRMR) = 0.02, root mean-square error of approximation (RMSEA) = 0.04 (CI90: 0.00 to 0.12).

**Reward impulsiveness** was assessed using the Impulsiveness scale of the I7 questionnaire (Eysenck et al., 1985), EPQ-R psychoticism scale (EPQ-P; Eysenck and Eysenck, 1994), and the Planfulness scale from the International Personality Item Pool (IPIP; Goldberg et al., 2006). The I7 (Impulsiveness) has a reported Cronbach’s α of 0.84, 1-year test–retest reliability of 0.76 (Eysenck et al., 1985; Luengo et al., 1991). Caruso and colleagues (2001) quantitatively reviewed the findings of 44 studies and found EPQ-P to have a median Cronbach’s α of 0.68. The scale has also been shown to have a 1-month test–retest reliability of 0.79. Factor analytic studies have found both EPQ-P and I7 (Impulsiveness) to load on an RI factor with other impulsivity measures, supporting their construct validity (Dawe and Loxton, 2004). It should be noted that the item on EPQ-P pertaining to drug use was not included in the calculation of the total score to avoid possible criterion contamination (Darkes et al., 1998).

²Individual item numbers are available from the authors upon request.
The IPPI planfulness scale is a 10-item scale designed to measure the construct of self-control, similar to the Control Scale of the Multidimensional Personality Questionnaire (MPQ; Tellegen, 1982). Example items from the planfulness scale include, “I jump into things without thinking” and “I make rash decisions.” The scale has been reported to correlate significantly with the MPQ-Control scale and to have adequate internal consistency (Cronbach’s ρ = 0.78; Goldberg et al., 2006).

Positive alcohol expectancy was measured using the 68-item Alcohol Expectancy Questionnaire (AEQ; Goldman et al., 1997). The AEQ contains 6 scales: Global Positive Changes, Sexual Enhancement, Social and Physical Pleasure, Social Assertiveness, Relaxation, and Arousal/Aggression. In a quantitative review of 26 studies, Kieffer and colleagues (2004) reported the validity and reliability of this questionnaire. The scale has been shown to have good internal consistency and test–retest reliability (r = 0.67 to 0.76) and mean internal consistency (Cronbach’s ρ = 0.70 to 0.80). However, the arousal/aggression scale was found to be less reliable, with a mean test–retest reliability of 0.45, and mean internal consistency of 0.59. A number of studies have shown the AEQ scales to predict drinking behavior, supporting its criterion validity (Jones et al., 2001).

Given the debate over the underlying structure of positive alcohol expectancy (e.g., context-specific domains vs. a single PE factor), 3 different measurement models were evaluated and compared to determine the optimal means of identification for inclusion in the main analysis (Goldman et al., 1997; Leigh, 1989). A 1-factor, non-hierarchical alcohol expectancy model was found to provide the most optimal fit to the data and was used in the main analysis, χ²(2, N = 327) = 7.66, p < 0.05, CFI = 0.99, SRMR = 0.05, RMSEA = 0.07 (CI90: 0.06 to 0.08), Consistent Akaike Information Criterion (CAIC) = 61.98 (see Data S1).

Drinking refusal self-efficacy was assessed using the Drinking Refusal Self-Efficacy Questionnaire – Revised (DRSEQ-R; Oei et al., 2005). This questionnaire comprises 3 scales: social pressure self-efficacy, emotional refusal self-efficacy, and opportunistic self-efficacy. Oei and colleagues (2005) confirmed the proposed 3-factor structure using CFA and also reported that the 3 scales loaded on a higher-order refusal self-efficacy factor. The 3 scales have been reported to have adequate internal consistency, Cronbach’s ρ = 0.83 to 0.95 (Oei and Jardim, 2007; Oei et al., 2005), and test–retest reliability, r = 0.84 to 0.93 (Young and Oei, 1996; Young et al., 1991).

Hazardous alcohol use. Three measures of hazardous alcohol use were included in the present study as indicators of a latent variable. The Alcohol Use Disorders Identification Test (AUDIT; Saunders et al., 1993) is a 10-item questionnaire designed to screen for hazardous and harmful levels of alcohol consumption. The AUDIT has been found, across a number of studies with various populations (e.g., university students, emergency room patients), to have good internal reliability and test–retest reliability (Deapen et al., 2000; Dawe et al., 2002). The AUDIT has been found to be more sensitive to nondependent problem drinking than other commonly used screening instruments and has even been found to better detect alcohol use and dependence than standard biochemical measures of alcohol consumption (Aertgeerts et al., 2001; Dawe et al., 2002).

The Alcohol, Smoking and Substance Involvement Screening Test (ASSIST; Ali et al., 2002), version 3.0, is an 8-item, interviewer-administered questionnaire designed to screen for the presence and severity of substance use. The alcohol-specific substance involvement composite (SSI Alcohol) was used for the purposes of this study. This composite has been shown to discriminate between nonproblematic alcohol use, abuse, and dependence (Humeniuk and Ali, 2006) and has been shown to have good internal consistency and test–retest reliability (Humeniuk and Ali, 2006). For the purposes of this study, the ASSIST was administered in self-report form, which has previously been shown to result in no detriment to its psychometric properties (Gullo, 2008).

The alcohol use section of the Addiction Severity Index (ASI)—“Lite” version (McLellan et al., 1997)—was also administered. This measure assesses the number of days in the past 30 that alcohol has been consumed (and consumed to intoxication) and number of years the substance has been used. A composite score representing severity of alcohol use can be calculated (McGahan et al., 1986). Alcohol use composites derived from the self-report version were reported to have a Cronbach’s ρ = 0.87 (Rosen et al., 2000). In addition to this, Brodey and colleagues (2004) reported that an online/internet-administered version of the ASI produced alcohol use composites that correlated highly with those derived from the interviewer-administered ASI (r = 0.93). These findings support the administration of the measure in an online, self-report form.

In addition to these measures, the EPQ-R Lie scale (EPQ-Lie) was also included as a measure of socially desirable responding. It has also been found to have good internal and test–retest reliability (Caruso et al., 2001; Eysenck and Eysenck, 1994).

Procedure

All measures were administered in an online, self-report format as there is now strong evidence supporting internet administration of self-report instruments generally (Gosling et al., 2004) and personality and alcohol use measures specifically (Brodey et al., 2004; Buchanan and Smith, 1999; Chuah et al., 2006; Miller et al., 2002), including impulsivity (Aluja et al., 2007).

RESULTS

Data Screening and Assumptions

Prior to analysis, all variables of interest were examined through SPSS (version 14.0.2, SPSS Inc., Chicago, IL) and AMOS (version 6.0, SPSS Inc., Chicago, IL) for accuracy of data entry, missing values, and fit between their distributions and the assumptions of structural equation modeling (SEM). The original dataset contained responses from 342 participants. Thirteen (3.8%) participants did not answer any item on more than 2 measures and were thus excluded, leaving 329 cases for further analysis. The variables of interest contained some missing data (range: 0.9 to 10.3%). The data were missing completely at random (MCAR), as suggested by Little’s MCAR test being not significant, χ²(775) = 798.43, p = 0.27. Missing data were estimated using Expectation Maximization, a robust estimation technique suitable for SEM (Newman, 2003; Schafer and Graham, 2002).

The data were screened for outliers and this analysis revealed the presence of 7 univariate outliers, 2 of whom were also multivariate outliers. The univariate outliers had no significant effect on model fit and were thus retained. However, the multivariate outliers significantly reduced multivariate normality, and overall fit of the models tested. Therefore, the multivariate outliers were removed, leaving 327 cases for further analysis. While this improved multivariate normality, there was still evidence that the assumption of multivariate normality was violated (Mardia’s Normalized coefficient = 6.77, p < 0.001).

Descriptive Statistics

The mean age at which participants reported having had their first full drink of alcohol was 14.18 (SD = 4.18).
Seventeen (5.2%) participants reported never having consumed a full drink of alcohol. Two hundred and ninety-one (89.0%) participants reported consumption of alcohol within the last 3 months. According to SSI Alcohol scores, 133 (40.7%) participants reported drinking at levels posing moderate or high risk to their health (score ≥11; Humeniuk and Ali, 2006). According to the AUDIT, 164 (50.2%) participants were drinking at hazardous or harmful levels (score ≥8; Saunders et al., 1993).

Descriptive statistics for measured variables are presented in Tables 1 and 2. As shown in Table 1, psychoticism had a relatively low Cronbach’s α, consistent with other studies (Caruso et al., 2001). The coefficient did not improve significantly when the drug use item was included. Table 2 shows EPQ-Lie was significantly correlated negatively with most self-report measures.

### Model Estimation and Evaluation

The model was tested using maximum likelihood estimation. Stringent criteria were used to ensure a rigorous evaluation of model fit. In accordance with the recommendations of Hu and Bentler (Bentler, 2007; Hu and Bentler, 1999), the $\chi^2$ test was used as a statistical test of model fit ($p < 0.05$). However, given this test can be overly sensitive in large samples, the “normed” $\chi^2$ was also examined. This statistic is calculated as $\chi^2$ divided by the model’s degrees of freedom (i.e., $\chi^2/df$). Obtained values of $\chi^2/df$ between 1.00 and 2.00 indicate good fit, and values between 2.00 and 3.00 indicate acceptable fit (Carmines and McIver, 1981). In addition to these indices, the CFI, RMSEA, and SRMR were also used to evaluate model fit (Bentler, 2007).

The following cut-offs were used for “good” fit: CFI ≥0.95; RMSEA ≤0.06; SRMR ≤0.08 (Hu and Bentler, 1999). In addition to reporting SRMR, Bentler (2007) recommended reporting the largest standardized residual covariances. In a good-fitting model, none of these values should exceed 2.58 ($p < 0.01$). Lastly, the fit of the hypothesized model was compared to that of nonhypothesized alternative models. This is a highly recommended, but often ignored, component of evaluating model fit (Bentler, 2007; McDonald and Ho, 2002). The CAIC was selected to assist in model comparison, whereby smaller values indicate a model is better-fitting and more parsimonious (Bozdogan, 1987).

As the assumption of multivariate normality was violated, the Bollen–Stine bootstrap $p$, a bootstrap modification of model $\chi^2$, was also used to evaluate model fit. To further reduce the influence of nonnormality, standard errors of parameter estimates were calculated using the bootstrap bias-corrected method (1,000 samples), and significance tests were conducted using 95% confidence intervals derived from this method (Efron, 1988; Neal and Simons, 2007; Shrout and Bolger, 2002). Such methods have been shown to adequately correct for nonnormality of this sort (Efron, 1988; Neal and Simons, 2007; Shrout and Bolger, 2002).

The hypothesized structural model presented in Fig. 1 outlines the proposed role of impulsivity-related personality traits and alcohol-related cognitions in hazardous alcohol use. The 2 major hypotheses contained in the model specify mediation effects (Baron and Kenny, 1986; Holmbeck, 1997). Since the seminal work of Baron and Kenny, there has been much debate as to how best to evaluate mediation in psychological research (MacKinnon et al., 2007). In a comparison of 14 methods to test mediation, MacKinnon and colleagues (2002) found that the joint significance test was the best method in terms of power and type I error rate. This test involves assessing the statistical significance of the predicted relationship between the independent variable and the proposed mediator ($z$), and then assessing the predicted relationship between the proposed mediator and the dependent variable ($β$; Cohen and Cohen, 1983). If both of these tests are statistically significant, there is evidence for mediation (MacKinnon et al., 2007). That is, there does not need to be a significant direct association between the independent and dependent variables. MacKinnon and colleagues (2007) also recommend calculating confidence intervals from the bootstrap distribution of the mediation effect (i.e., $z × β$, or $zβ$) to evaluate the magnitude of the indirect or mediated effect. Cheung and Lau (2008) found that using bias-corrected bootstrap confidence intervals was the best method for testing such mediation effects in SEM.

In addition to the variables of interest, all structural models included a latent social desirability or “lie” variable. The latent social desirability variable included EPQ-Lie as a
Table 2. Intercorrelations of Impulsivity, Alcohol-Related Cognition, and Alcohol Use Measures in the College Sample (N = 327)

<table>
<thead>
<tr>
<th>Scale</th>
<th>1</th>
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<th>14</th>
<th>15</th>
<th>16</th>
<th>17</th>
<th>18</th>
</tr>
</thead>
<tbody>
<tr>
<td>BAS: drive—</td>
<td>1.00</td>
<td>0.26*</td>
<td>0.38*</td>
<td>0.43*</td>
<td>0.25*</td>
<td>0.24*</td>
<td>0.32*</td>
<td>0.42*</td>
<td>0.25*</td>
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<td>0.32*</td>
<td>0.42*</td>
<td>0.25*</td>
<td>0.24*</td>
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<tr>
<td>BAS: sensitivity to reward</td>
<td>0.04</td>
<td>1.00</td>
<td>0.18*</td>
<td>0.04</td>
<td>0.40*</td>
<td>0.17*</td>
<td>0.31*</td>
<td>0.03</td>
<td>0.16*</td>
<td>0.13*</td>
<td>0.64*</td>
<td>0.48*</td>
<td>0.31*</td>
<td>0.37*</td>
<td>0.38*</td>
<td>0.62*</td>
<td>0.31*</td>
<td>0.37*</td>
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<tr>
<td>BAS: social assertiveness</td>
<td>0.03</td>
<td>0.17</td>
<td>1.00</td>
<td>0.18*</td>
<td>0.05</td>
<td>0.14*</td>
<td>0.35*</td>
<td>0.16*</td>
<td>0.74*</td>
<td>0.50*</td>
<td>0.70*</td>
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<td>0.74*</td>
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<td>0.76*</td>
<td>0.77*</td>
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<tr>
<td>BAS: arousal—aggression</td>
<td>0.03</td>
<td>0.08</td>
<td>0.29*</td>
<td>1.00</td>
<td>0.10</td>
<td>0.21*</td>
<td>0.35*</td>
<td>0.15*</td>
<td>0.78*</td>
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<td>0.74*</td>
<td>0.75*</td>
<td>0.76*</td>
</tr>
<tr>
<td>BAS: emotion—relief</td>
<td>0.02</td>
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<td>0.19*</td>
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<td>1.00</td>
<td>0.14*</td>
<td>0.19*</td>
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<tr>
<td>BAS: opportunistic</td>
<td>0.04</td>
<td>0.02</td>
<td>0.14*</td>
<td>0.03</td>
<td>0.15*</td>
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<tr>
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<td>0.10</td>
<td>0.31*</td>
<td>0.29*</td>
<td>0.39*</td>
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<td>EPQ-Lie</td>
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<tr>
<td>ASI: alcohol</td>
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<td>0.20*</td>
<td>0.42*</td>
<td>0.35*</td>
<td>0.37*</td>
<td>0.31*</td>
<td>0.37*</td>
<td>0.37*</td>
<td>0.37*</td>
<td>0.37*</td>
<td>0.37*</td>
<td>0.37*</td>
</tr>
<tr>
<td>SSI: alcohol</td>
<td>0.08</td>
<td>0.10</td>
<td>0.31*</td>
<td>0.29*</td>
<td>0.39*</td>
<td>0.30*</td>
<td>0.30*</td>
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<td>0.30*</td>
<td>0.30*</td>
<td>0.30*</td>
<td>0.30*</td>
</tr>
<tr>
<td>DSREQ-R: social pressure</td>
<td>0.14*</td>
<td>0.18*</td>
<td>0.17*</td>
<td>0.14*</td>
<td>0.14*</td>
<td>0.14*</td>
<td>0.14*</td>
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<td>0.14*</td>
<td>0.14*</td>
<td>0.14*</td>
<td>0.14*</td>
</tr>
<tr>
<td>DSREQ-R: emotional relief</td>
<td>0.06</td>
<td>0.23*</td>
<td>0.16*</td>
<td>0.02</td>
<td>0.14*</td>
<td>0.14*</td>
<td>0.14*</td>
<td>0.14*</td>
<td>0.14*</td>
<td>0.14*</td>
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<td>0.14*</td>
<td>0.14*</td>
<td>0.14*</td>
<td>0.14*</td>
</tr>
<tr>
<td>DSREQ-R: opportunistic</td>
<td>0.04</td>
<td>0.02</td>
<td>0.14*</td>
<td>0.03</td>
<td>0.15*</td>
<td>0.14*</td>
<td>0.14*</td>
<td>0.14*</td>
<td>0.14*</td>
<td>0.14*</td>
<td>0.14*</td>
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<td>0.14*</td>
<td>0.14*</td>
<td>0.14*</td>
<td>0.14*</td>
</tr>
</tbody>
</table>

BAS, Behavioral Approach System; I 7 (Imp), Eysenck Impulsiveness Scale; AEQ, Alcohol Expectancies Questionnaire; DSREQ-R, Drinking Refusal Self-Efficacy Questionnaire

*p < 0.05.

The Hypothesized Structural Model

The hypothesized structural model provided a moderately good fit to the data (see Table 3, Model 1). The largest standardized residual covariance was between agentic extraversion and AEQ item parcel 4 (r = −1.84). Consistent with hypothesis, SR predicted greater positive alcohol expectancy (unstandardized coefficient = 1.00, CI95: 0.47 to 1.71, p = 0.001). However, positive alcohol expectancy was not found to have a significant direct association with hazardous alcohol use (unstandardized coefficient = 0.16, CI95: −0.09 to 0.40, p = 0.17). Instead, its effect on alcohol use was fully mediated by drinking refusal self-efficacy (unstandardized indirect effect = 0.58, CI95: 0.37 to 0.80, p < 0.01), which itself had a significant direct association with alcohol use (unstandardized coefficient = −0.68, CI95: −0.88 to −0.49, p < 0.01). Also, consistent with hypothesis, the significant direct association between RI and drinking refusal self-efficacy (unstandardized coefficient = −0.21, CI95: −0.43 to −0.03, p = 0.01) provided evidence for mediation. However, because RI still directly predicted variance in hazardous alcohol use (unstandardized coefficient = 0.26, CI95: 0.11 to 0.48, p = 0.01), this suggested only partial mediation. In total, the hypothesized model accounted for 68% (CI95: 0.56 to 0.76) of the variance in hazardous alcohol use.

Of note, the hypothesized structural model was also tested using Goldman et al.’s (1997) hierarchical conceptualization of positive alcohol expectancy (see Data S1). This model also provided a moderately good fit to the data, r2 (416, N = 327) = 0.956, 11, p < 0.001, Bollen–Stine bootstrap p = 0.001, CFI = 0.91, SRMR = 0.05, RMSEA = 0.06 (CI90: 0.06 to 0.07), CAIC = 1499.31. Parameter estimates were similar to those obtained in the hypothesized model. As suspected, there was some model misfit related to complexity of the measurement model (i.e., the inclusion of 3 item parcels as indicators for each of the 6 AEQ sub-domains). However, the results still supported the hypothesized model.

Three alternative, nonhypothesized models were estimated for the purposes of comparison (McDonald and Ho, 2002). The first model included a 1-factor impulsivity trait predicting the alcohol-related cognition variables and hazardous alcohol misuse. This model was identical to the hypothesized model, except it specified a 1-factor latent impulsivity variable that included all impulsivity measures as indicators. This model provided a marginally adequate fit to the data (see Table 3, Model 2). Inspection of the difference in CAIC reveals the hypothesized model provided a better fit to the data.

The second alternative model, based on the Smith and Anderson’s (2001) original hypothesis, was also tested for
comparison. This model was identical to the hypothesized model, but specified a direct association between RI and alcohol expectancy. This model also provided a moderately good fit to the data (see Table 3, Model 3). However, the \( \chi^2 \) difference test was not significant and the CAIC was larger, suggesting the hypothesized model provided a better, more parsimonious fit. Furthermore, neither of the 2 additional paths were statistically significant, suggesting SR had no direct association with drinking refusal self-efficacy (unstandardized coefficient = 0.32, CI95: −0.27 to 1.04, \( p = 0.27 \)), and RI had no direct association with positive alcohol expectancy (unstandardized coefficient = 0.08, CI95: −0.05 to 0.23, \( p = 0.23 \)). Taken together, these results support the hypothesized model.

A third alternative model was also tested. It was identical to the hypothesized model, but also included direct associations between SR and drinking refusal self-efficacy, and also between RI and positive alcohol expectancy (i.e., nonindependent effects). This model provided a moderately good fit to the data (see Table 3, Model 4). However, the \( \chi^2 \) difference test was not significant and the CAIC was larger, suggesting the hypothesized model provided a better, more parsimonious fit. Furthermore, neither of the 2 additional paths were statistically significant, suggesting SR had no direct association with drinking refusal self-efficacy (unstandardized coefficient = 0.32, CI95: −0.27 to 1.04, \( p = 0.27 \)), and RI had no direct association with positive alcohol expectancy (unstandardized coefficient = 0.08, CI95: −0.05 to 0.23, \( p = 0.23 \)). Taken together, these results support the hypothesized model.

DISCUSSION

Results of the present study support the hypothesized model. Specifically, SR and RI were found to be directly associated with separate cognitive mechanisms related to hazardous alcohol use. These findings support the proposed refinement to Smith and Anderson’s (2001) model. That is,
only those aspects of impulsivity related to SR, as opposed to RI/disinhibition, were found to be associated with positive alcohol expectancy. This is consistent with biological theories of personality that propose reward learning (and general reward sensitivity) is related to extraversion or the BAS (Gray, 1975).

The findings of the present study also help to clarify why previous research may have found inconsistent support for Smith and Anderson’s (2001) model. This inconsistency was likely the result of too broad an operationalization of impulsivity. For instance, inspection of Table 2 suggests that RI measures were associated with positive alcohol expectancy. However, when controlling for the variance these measures share with SR (as in the structural model), RI is no longer related to PE. This is similar to what Anderson and colleagues (2005a) reported in their sample of adolescents, in that only SR-related traits were associated with positive alcohol expectancy. Also of note, we obtained the same result regardless of whether expectancy was conceptualized (and modeled) as a single latent variable, or as the higher-order factor of a larger expectancy network (Goldman et al., 1997). Taken together, this suggests those aspects of impulsivity uniquely related to RI/disinhibition are not related to positive alcohol expectancy. Instead, RI was found to contribute to hazardous drinking through other cognitive mechanisms, such as drinking refusal self-efficacy.

The direct positive association between RI and alcohol misuse was partially mediated by lower drinking refusal self-efficacy. This study is the first to demonstrate an association between impulsivity and drinking refusal self-efficacy. The finding is consistent with the prediction that rash individuals expect to experience more difficulty refusing alcohol, perhaps due to awareness of their general difficulty in inhibitory control. Notably, this finding is also compatible with Patterson and Newman’s (1993) model of disinhibition. However, because of the cross-sectional nature of the present study, such a temporal sequence requires testing in a prospective study. It is possible that low refusal self-efficacy is solely an outcome of past refusal failures in alcohol-cued situations and plays no unique role in predicting future hazardous consumption.

Anticipation of more positive outcomes from drinking also strongly predicted lower refusal self-efficacy. Indeed, the data suggest this is an important pathway through which PE (and therefore, also SR) may influence hazardous drinking. The lack of any unique association between PE and hazardous alcohol use is inconsistent with the findings of Young and colleagues (2006). While Young and colleagues reported unique contributions from alcohol expectancy and drinking refusal self-efficacy to alcohol misuse, their study differed from the present investigation in 2 important ways. First, they administered the original DRSEQ, which Oei and colleagues (2005) have since revised because of psychometric shortcomings. The second difference between the 2 studies was the control of socially desirable responding. This study, unlike Young et al., measured and controlled for the influence of social desirability and this affected the results. When the hypothesized model was run without controlling for social desirability, positive alcohol expectancy did contribute unique variance to the prediction of hazardous drinking. Furthermore, the magnitude of this effect was comparable to that reported by Young and colleagues (standardized coefficient = 0.16, p = 0.04). This finding not only explains the divergent results, but underscores the importance of controlling for social desirability in self-report studies.

STUDY 2

Studies that have examined the differential contribution of positive alcohol expectancy and drinking refusal self-efficacy tend to find less consistent results in samples of dependent drinkers. This lack of consistency underscores the importance of testing models of alcohol-related cognition with clinical samples, in whom biological and behavioral factors play a more prominent role in alcohol use. The same argument applies to the role of temperament, specifically SR and RI. Indeed, the aim of study 2 was to attempt to replicate the hypothesized model, confirmed in study 1, in a sample of treatment-seeking alcohol users. The findings of this study would help to determine the applicability of the proposed model to clinical levels of alcohol use, and its potential to inform treatment interventions. The predictions of the hypothesized model were identical to those tested in study 1. However, because of the limited number of questionnaires that could be practically administered, single indicator latent factors were modeled in study 2. Additionally, in order for the nonhypothesized alternative models to be sufficiently “identified”, an additional parameter had to be constrained (Bollen, 1989). Therefore, the direct path from SR to hazardous drinking was removed, as this path was not significant in study 1.

MATERIALS AND METHODS

Participants

The initial sample consisted of 178 individuals seeking treatment at a residential rehabilitation program for alcohol and drug dependence. Given that our focus was on alcohol misuse, only individuals scoring higher than 15 on the AUDIT (> 15 considered an indicator of alcohol dependence; Babor et al., 2001) were included in the analysis (n = 121). Participants’ mean age was 34.1 (SD = 7.7), 67% (n = 81) were male and 86% were born in Australia/New Zealand whilst 11.5% were from Europe and 2.5% from the Americas. Informed consent was obtained from each participant, and the study was approved by the relevant university ethics committee.

Materials

Reward sensitivity. For practical reasons, individual differences in SR were assessed using the shortened, 17-item version of the Sensitivity to Reward scale (SR; Torrubia et al., 2001). This scale has been shown to correlate highly with the original (r = 0.96) and has equivalent internal reliability to the 24-item scale (O’Connor et al., 2004).

Rash impulsiveness was assessed using the Impulsiveness scale of the I3 questionnaire (Eysenck et al., 1985).

Positive alcohol expectancy. Leigh and Stacy’s (1993) Alcohol Outcome Expectancies (AE) questionnaire consists of 34 items rated on a
6-point Likert-type scale (from no chance to certain to happen). The scale has a 2-factor structure (positive and negative expectancies), with each factor comprising 4 subscales. Only the positive expectancies (PE) subscales were used in the present study. The PE subscales are: social facilitation, fun, sexual enhancement, and negative reinforcement/tension reduction. Cronbach’s alpha of 0.94 has been reported for the PE total score, as well as good test–retest reliability (0.87; Leigh and Stacy, 1993).

Drinking refusal self-efficacy was measured using the DRSEQ-R (Oei et al., 2005). Hazardous alcohol use was assessed using the AUDIT (Saunders et al., 1993). Scores above 15 are considered to be indicative of alcohol dependence (Babor et al., 2001).

RESULTS

Data Screening and Assumptions

Prior to analysis, all variables of interest were examined through SPSS (version 14.0.2) and AMOS (version 6.0) for accuracy of data entry, missing values, and fit between their distributions and the assumptions of SEM. The dataset contained responses from 121 participants and the variables of interest contained missing data from 7 of the cases. Little’s MCAR test was not significant ($\chi^2$ [1087] = 1116.58, $p = 0.26$), suggesting the data were MCAR. Given that the missing data consisted of only 5.8% of any variable, it was appropriate to conduct item-mean substitution (Tabachnick and Fidell, 2007). The data were screened for outliers; there were no multivariate outliers, and the univariate outliers had no significant effect on model fit and were thus retained.

Descriptive Statistics

Descriptive statistics for measured variables are presented in Tables 4 and 5. As shown in Table 4, the positive reinforcement scale of the alcohol expectancies measure had a low Cronbach’s $\alpha$. However, the positive alcohol expectancies total score retained good internal consistency. Table 5 shows that neither the SR nor the I7 (Impulsiveness) scale scores correlated with the AUDIT. However, both showed significant zero-order correlations with total positive alcohol expectancies.

Model Estimation and Evaluation

Because of the limited number of questionnaires that could be practically administered to this sample and the smaller sample size, the constructs of interest were modeled as single-indicator latent factors (Sass and Smith, 2006). As recommended by Bollen (1989), the constructs of interest were modeled as latent variables, each with a single indicator or measured variable with its measurement error set to SD$^2$ (1—Cronbach’s $\alpha$). The hypothesized model was tested using maximum likelihood estimation. The same stringent criteria for model fit employed in study 1 were also used in this study. This included comparing the fit of the hypothesized model to that of nonhypothesized alternative models. As in study 1, the joint significance test was used to test for mediation in the model. Unlike study 1, the assumption of multivariate normality was met for the present analysis. Therefore, model $\chi^2$ and standard errors of parameter estimates were evaluated without bootstrap modification.

The Hypothesized Structural Model

The hypothesized structural model provided a good fit to the data (see Table 6, Model 1). The largest standardized residual covariance was between SR and AUDIT ($z = -1.32$). As hypothesized, SR predicted greater positive alcohol expectancy (unstandardized coefficient = 0.92, $p = 0.01$) (Fig. 2). However, positive alcohol expectancy was not found to significantly predict hazardous alcohol use (unstandardized coefficient = 0.12, $p = 0.06$). Instead, its relationship with alcohol misuse was fully mediated by its association with drinking refusal self-efficacy, which itself was positively related to hazardous drinking (unstandardized indirect effect = 0.06, $p = 0.01$). Also, consistent with hypothesis, RI was negatively associated with drinking refusal self-efficacy (unstandardized coefficient = −1.17, $p = 0.05$). In fact, drinking refusal self-efficacy fully mediated the association between RI and hazardous drinking (unstandardized indirect effect = 0.09, $p = 0.047$).

Table 5. Intercorrelations of Impulsivity, Alcohol-Related Cognition, and Alcohol Use Measures in the Clinical Sample (N = 121)

<table>
<thead>
<tr>
<th>Scale</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Sensitivity to reward (SR)</td>
<td>−</td>
<td>0.38**</td>
<td>−</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. I7 (impulsiveness)</td>
<td>0.22*</td>
<td>0.24**</td>
<td>0.34**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Positive alcohol expectancies: total</td>
<td>0.25**</td>
<td>−0.12</td>
<td>−0.25**</td>
<td>−0.34**</td>
<td>−</td>
</tr>
<tr>
<td>4. DRSEQ-R: total</td>
<td>−0.05</td>
<td>0.08</td>
<td>0.26**</td>
<td>−0.32**</td>
<td>−</td>
</tr>
<tr>
<td>5. AUDIT</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

DRSEQ-R, Drinking Refusal Self-Efficacy Questionnaire – Revised; AUDIT, Alcohol Use Disorders Identification Test.

*a p < 0.05; **p < 0.01.

Table 4. Means, Standard Deviations (SD), and Cronbach’s $\alpha$ Coefficients for Impulsivity, Alcohol-Related Cognition, and Alcohol Use Measures in the Clinical Sample (N = 121)

<table>
<thead>
<tr>
<th>Scale</th>
<th>$\alpha$</th>
<th>Mean</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sensitivity to reward (SR)$^a$</td>
<td>0.73</td>
<td>9.99</td>
<td>3.89</td>
</tr>
<tr>
<td>I7 (impulsiveness)</td>
<td>0.83</td>
<td>12.21</td>
<td>4.41</td>
</tr>
<tr>
<td>Pos Exp: social</td>
<td>0.83</td>
<td>27.58</td>
<td>4.33</td>
</tr>
<tr>
<td>Pos Exp: fun</td>
<td>0.85</td>
<td>28.18</td>
<td>4.50</td>
</tr>
<tr>
<td>Pos Exp: sex</td>
<td>0.91</td>
<td>17.32</td>
<td>4.56</td>
</tr>
<tr>
<td>Pos Exp: negative reinforcement</td>
<td>0.61</td>
<td>13.66</td>
<td>2.54</td>
</tr>
<tr>
<td>DRSEQ-R: social pressure</td>
<td>0.89</td>
<td>86.74</td>
<td>12.01</td>
</tr>
<tr>
<td>DRSEQ-R: emotional relief</td>
<td>0.97</td>
<td>17.45</td>
<td>9.70</td>
</tr>
<tr>
<td>DRSEQ-R: opportunistic drinking</td>
<td>0.95</td>
<td>23.71</td>
<td>10.71</td>
</tr>
<tr>
<td>DRSEQ-R: total</td>
<td>0.97</td>
<td>53.84</td>
<td>25.27</td>
</tr>
<tr>
<td>AUDIT</td>
<td>0.79</td>
<td>31.09</td>
<td>7.40</td>
</tr>
</tbody>
</table>

$^a$17-Item version; contains no items pertaining to drug/alcohol use.
While SR was predicted to have no direct association with hazardous alcohol use, it was hypothesized to have an overall indirect effect. Indeed, as predicted, the data showed a significant indirect association between SR and hazardous drinking that was fully mediated by positive alcohol expectancy and drinking refusal efficacy (unstandardized indirect effect = 0.16, \( p = 0.01 \)). In total, the hypothesized model accounted for 17% (CI95: 0.05 to 0.31) of the variance in hazardous alcohol use. For the purposes of comparison with study 1, the hypothesized model was also tested with the direct path from SR to hazardous drinking included. This model was also found to provide a good fit to the data, \( \chi^2 (2) = 2.60, \text{CFI} = 0.99, \text{SRMR} = 0.04, \text{RMSEA} = 0.05, \text{CAIC} = 77.94 \). Furthermore, as in study 1, this path was not statistically significant (unstandardized coefficient = 0.37, \( p = 0.14 \)).

Two alternative, nonhypothesized models were estimated for the purposes of comparison (McDonald and Ho, 2002). The first alternative was based on Smith and Anderson’s (2001) model and was identical to the hypothesized model, except it specified a direct association between RI and positive alcohol expectancy. This model also provided a good fit to the data (see Table 6, Model 2). However, the direct path from RI to PE was not statistically significant (unstandardized coefficient = 0.55, \( p = 0.11 \)). Furthermore, the \( \chi^2 \) difference test was not significant, suggesting the hypothesized model provided a better, more parsimonious fit to the data. This is consistent with the difference in CAIC between the 2 models. Therefore, these results support the hypothesized model.

The second alternative model was identical to the hypothesized model, but also included direct paths from SR to drinking refusal self-efficacy, and from RI to positive alcohol expectancy (i.e., nonindependent effects). This model provided an adequate-to-good fit to the data (see Table 6, Model 3). However, the nonsignificant \( \chi^2 \) difference test suggested the fit of this model was not significantly better than the hypothesized model. Furthermore, neither of the 2 additional paths were statistically significant, suggesting SR had no direct association with drinking refusal self-efficacy (unstandardized coefficient = 0.29, \( p = 0.73 \)), and RI had no direct association with positive alcohol expectancy (unstandardized coefficient = 0.55, \( p = 0.11 \)). The difference in CAIC also

### Table 6. Fit Indices for Hypothesized and Alternative Structural Models in the Clinical Sample (\( N = 121 \))

<table>
<thead>
<tr>
<th>Model</th>
<th>( \chi^2 ) (df)</th>
<th>( p )</th>
<th>CFI</th>
<th>SRMR</th>
<th>RMSEA (CI95)</th>
<th>CAIC</th>
<th>( \chi^2 ) diff (df)</th>
<th>( \Delta \text{CAIC} )</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Hypothesized model</td>
<td>4.78 (3)</td>
<td>0.19</td>
<td>0.97</td>
<td>0.05</td>
<td>0.07 (0.00–0.18)</td>
<td>74.33</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Smith and Anderson (2001) model</td>
<td>2.26 (2)</td>
<td>0.32</td>
<td>0.99</td>
<td>0.03</td>
<td>0.03 (0.00–0.19)</td>
<td>77.60</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Non-independent effects model</td>
<td>2.14 (1)</td>
<td>0.14</td>
<td>0.98</td>
<td>0.03</td>
<td>0.10 (0.00–0.28)</td>
<td>83.28</td>
<td>2.52 (1)</td>
<td>3.27</td>
</tr>
<tr>
<td>Difference between model 2 and model 1</td>
<td>2.64 (2)</td>
<td>0.14</td>
<td>0.98</td>
<td>0.03</td>
<td>0.10 (0.00–0.28)</td>
<td>83.28</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Difference between model 3 and model 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

CFI, comparative fit index; SRMR, standardized root mean-square residual; RMSEA, root mean-square error of approximation; CAIC, Consistent Akaike Information Criterion.

*\( p < 0.05 \).

![Fig. 2. Hypothesized structural model of the relationship between impulsivity traits, alcohol-related cognition, and hazardous alcohol use in the clinical sample. Standardized parameter estimates are presented. All estimates are statistically significant at \( p < 0.05 \), except where indicated (ns).](image-url)
suggested that the hypothesized model was better fitting, and more parsimonious than the nonindependent effects model. Taken together, these results support the hypothesized model.

**DISCUSSION**

The results of study 2 replicate the hypothesized model in a sample of treatment-seeking alcohol abusers. That is, SR and RI were found to be directly associated with separate cognitive mechanisms related to hazardous alcohol use. Specifically, drinking refusal self-efficacy fully mediated the association between RI and hazardous drinking. Additionally, and consistent with the findings of study 1, the relationship between SR and alcohol misuse was more complex. Specifically, SR predicted greater positive alcohol expectancy which in turn predicted lower refusal self-efficacy, the latter of which directly predicted more hazardous drinking. That is, the association between positive alcohol expectancy and alcohol misuse was fully mediated by its association with drinking refusal self-efficacy. Importantly, these mediation effects were not only replicated in the present clinical sample, but also using a different measure of alcohol expectancy.

The findings support the role of cognitive mechanisms, particularly drinking refusal self-efficacy, as more proximal factors influencing alcohol-related problems. Although, one needs to be cautious in drawing any causal links based on cross-sectional data, it is plausible to speculate that individuals high on RI will have more difficulties in “stopping” and, in turn, this will undermine self-evaluation of their capacity to stop drinking. Interestingly, this study suggests that this process occurs independently of an individual’s positive beliefs about alcohol. Our finding of differential relationships between impulsivity traits and cognitive mechanisms enables a more sophisticated approach to treatment planning. For example, we have argued elsewhere that treatment for substance misuse needs to be refined by taking into account personality traits which convey greater risk for alcohol problems (Staiger et al., 2007). Our current results underscore the need for such an approach to also directly address drinking refusal self-efficacy, given it is likely that it plays a critical role in maintaining dependent drinking.

These findings are also consistent with a recent dismantling study by Litt and colleagues (2008) which found that the most efficacious treatments for cannabis dependence were those that increase refusal self-efficacy, irrespective of the specific techniques used to accomplish this (e.g., contingency management vs. motivational enhancement). It is possible that mindfulness training, with its emphasis on attentional control, may specifically address the rash-impulsive tendencies of the dependent drinker (Witkiewitz et al., 2005). Thus, learning to notice and “accept” urges to drink may reduce the likelihood of impulsive alcohol-seeking behavior which in turn will increase self-efficacy to refuse alcohol. However, this approach needs to be supported with direct attention to building self-efficacy through experiential learning tasks such as drink refusal role plays and/or tangible incentives such as those offered in contingency management (Beck et al., 1993; Litt et al., 2008).

There are 3 notable limitations to the findings of study 2. Firstly, as was the case in study 1, the cross-sectional design limits the extent to which one can infer causal effects and the direction of causation. Secondly, because of practical constraints, study 2 was unable to incorporate a measure of social desirability and control for the effects of socially desirable responding in the manner that study 1 did. Thirdly, severity of alcohol use was determined via the AUDIT self-report questionnaire, rather than a structured diagnostic interview. These limitations should be taken into account when considering the findings of study 2.

In summary, previous research suggests that low drinking refusal self-efficacy plays a critical role in maintaining hazardous alcohol use, and our data suggest it may be one mechanism through which RI conveys risk. Additionally, the results of study 2 are consistent with the notion that SR conveys risk through a “knock-on” effect whereby it increases the expectation of positive outcomes from drinking, which in turn reduces one’s confidence in their ability to resist drinking in cued situations (i.e., because the benefits seem so large). Put simply, SR may increase hazardous drinking by making the act seem more rewarding to the individual and, therefore, more difficult to resist. While this account is theoretically plausible, prospective studies are required to test our interpretation of the results. It could very well be that reduced refusal self-efficacy is merely an outcome of problematic drinking that has no causal role in future relapse. However, the results of Darkes and Goldman (1993) and Adamson and colleagues (2009) suggest it likely plays some role. Additionally, it seems even less plausible, on theoretical grounds, that low refusal self-efficacy could increase PE, which in turn increases one’s sensitivity to all rewards (a major dimension of personality). Indeed, research tends to suggest that greater sensitivity to drug rewards is actually associated with a lower sensitivity to other (nondrug) rewards (Koob and Le Moal, 2001; Lubman et al., 2009).

**GENERAL DISCUSSION**

The results of these studies suggest that drinking refusal self-efficacy is a strong, proximal predictor of hazardous drinking. Indeed, it mediated the effects of almost all other included variables. This further supports the importance of refusal self-efficacy in understanding alcohol misuse. It may be that refusal self-efficacy acts as something of a “gate-keeper” to the influence of other risk factors on hazardous drinking. This would be consistent with treatment studies demonstrating the importance of refusal self-efficacy to long-term abstinence (Adamson et al., 2009; Litt et al., 2008; Solomon and Annis, 1990). Clearly, a greater understanding of the factors that influence drinking refusal self-efficacy would have important implications for intervention efforts. Furthermore, when considering nonclinical populations, personality
traits could play a role in identifying at-risk groups prior to the onset of significant pathology.

For example, if one was to design a primary prevention protocol to delay alcohol initiation in children with a family history of alcohol dependence, individual differences in impulsivity traits could be utilized to help select intervention priorities. Specifically, high scorers on a measure of SR might benefit more from an intervention focusing on undermining positive alcohol expectancies, perhaps through expectancy challenges (e.g., Cruz and Dunn, 2003). In this case, more realistic expectancies could then serve to enhance refusal self-efficacy by reducing the perceived incentive of drinking. By contrast, high scorers on a measure of RI might benefit more from an intervention focusing on improving refusal self-efficacy directly, perhaps through carefully planned behavioral experiments, or role-playing refusal skills (Beck et al., 1993).

While study 1 controlled for social desirability by modeling it as a covariate, there are other limitations to note. In addition to social desirability effects, self-report measures are prone to bias resulting from lack of insight (Reynolds et al., 2006). Therefore, future studies could test the hypothesized model using behavioral measures of alcohol-related cognition, such as the Implicit Association Test (IAT; Greenwald et al., 1998) or “alcohol stroop” (Johnsen et al., 1994). Such measures have also been shown to prospectively predict drinking behavior (Field and Cox, 2008). Also of note, while the hypothesized model was replicated across the 2 samples, it focused solely on overall hazardous alcohol use. Future research should examine how well the model predicts actual patterns of use (quantity, frequency etc.) across different populations of drinkers. Lastly, it is important to note that while directions of effects are modeled in SEM, as in regression, such direction of causation cannot be adequately tested in nonexperimental, cross-sectional studies such as these. However, the consistency of results we have obtained across 2 studies with 2 different populations of drinkers clearly warrants longitudinal investigation of this model in future.

In conclusion, the findings of the reported studies extend those of previous research by offering some indication of how traits related to reward sensitivity and inhibitory control may convey risk for hazardous drinking at a cognitive level. Impulsivity traits have long been recognized as early indicators of risk and while being impulsive is not necessarily a “bad” thing, it can affect how one thinks about alcohol and, in turn, influence one’s drinking behavior (Gullo and Dawe, 2008). Intervention efforts can perhaps best use this knowledge not by targeting the traits themselves, but rather use them to target the more proximal cognitive mechanisms they influence.

REFERENCES


Saunders B, Farag N, Vincent AS, Collins FL, Sorocco KH, Lovallo WR

SUPPORTING INFORMATION
Additional Supporting Information may be found in the online version of this article:
Data S1. Identification/Conceptualization of Positive Alcohol Expectancy
Please note: Wiley-Blackwell is not responsible for the content or functionality of any supporting information supplied by the authors. Any queries (other than missing material) should be directed to the corresponding author for the article.