



Support for a two-factor model of impulsivity and hazardous substance use in British and Australian young adults

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ABSTRACT

Multiple lines of evidence suggest impulsivity comprises two distinct components relevant to substance misuse. Reward drive reflects sensitivity to rewarding stimuli and subsequent approach motivation. Rash impulsiveness reflects the ability to inhibit such approach behavior in light of negative consequences. However, several studies suggest the latter trait to be a more robust predictor. This begs the question as to whether a less parsimonious two-factor model is necessary. This study employed structural equation modeling to compare the fit of one- and two-factor impulsivity models to alcohol and drug use data provided by British ($n = 183$) and Australian ($n = 271$) young adults. Results consistently supported the two-factor model and its cross-cultural consistency, with rash impulsiveness being the more robust predictor.

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1. Introduction

Recent work in the study of impulsivity and substance use suggests impulsivity is better conceptualized as comprising two dimensions: reward sensitivity/drive and rash impulsiveness, both of which convey risk (Belin, Mar, Dalley, Robbins, & Everitt, 2008; Dawe & Loxton, 2004; de Wit & Richards, 2004). However, few studies have empirically evaluated this two-factor model at the trait level, and none have explored whether it provides a better fit to the data than a one-factor model. Using structural equation modeling (SEM), this study adds to the literature by confirming the proposed two-factor structure of trait impulsivity and evaluating the hypothesized independent contributions of each factor to alcohol and illicit substance misuse. Additionally, the study sought to evaluate the generalizability of the model by comparing it across the United Kingdom (UK) and Australia.

There is little doubt that the personality trait of impulsivity is involved in the etiology of substance use disorders. Several large-scale, prospective studies have shown that impulsivity assessed during childhood or adolescence predicts alcohol and illicit drug use disorders in adulthood (e.g., Caspi, 2000; Fergusson, Boden, &

Horwood, 2008). However, recent research suggests that impulsivity is better conceptualized as comprising two distinct components, especially when considering substance use (Dawe & Loxton, 2004; de Wit & Richards, 2004). The *reward sensitivity/drive* component relates to an individual's sensitivity to, and subsequent motivation to acquire, rewards in the environment (Dawe, Gullo, & Loxton, 2004; Dawe & Loxton, 2004). Measures that more selectively tap this dimension include those designed to measure Gray's (1970) Behavioral Approach System (BAS), such as Carver and White's (1994) BAS scales, and the Sensitivity to Reward (SR) scale (Torrubia, Avila, Molto, & Caseras, 2001). The *rash impulsiveness* component is that component of impulsivity that also incorporates one's ability to inhibit/modify prepotent approach behaviors in light of negative consequences, particularly future negative consequences (Dawe & Loxton, 2004; Dawe et al., 2004). Measures that more selectively tap this dimension include Eysenck's Impulsiveness scale from the I₇ questionnaire (Eysenck, Pearson, Easting, & Allsopp, 1985), Cloninger's (1987) Novelty Seeking, Zuckerman's Sensation Seeking Scale (Zuckerman, Eysenck, & Eysenck, 1978), and the Barratt Impulsiveness Scale (Patton, Stanford, & Barratt, 1995). Importantly, the majority of large-scale, prospective studies concerning substance use have used self-report measures which reflect rash impulsiveness (Dawe et al., 2004; Quilty & Oakman, 2004; Zelenski & Larsen, 1999). Dawe and Loxton (2004) have argued that theoretical models of substance misuse could be

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enhanced by exploring the unique role of each impulsivity component.

Dawe and colleagues (Dawe & Loxton, 2004; Dawe et al., 2004; Gullo & Dawe, 2008) proposed a 2-Component Approach to Reinforcing Substances (2-CARS) model describing how both impulsivity components are involved in substance misuse. According to 2-CARS, an individual high in reward drive finds the rewarding aspects of alcohol/drug use as more salient, resulting in greater motivation to use such substances (Dawe et al., 2004). Additionally, they are also more susceptible to the reinforcing and incentive sensitization effects that follow actual substance use (Robinson & Berridge, 2001). This greater sensitivity to reward derives from a hyperfunctioning mesolimbic dopamine system, a key neurobiological substrate of the BAS (Gray, 1987; Pickering & Gray, 1999). The individual high in rash impulsiveness would find it more difficult to inhibit this approach motivation to use alcohol/drugs, even after considering the negative consequences of such use (e.g., serious health and legal consequences). This difficulty relates to hypo-functioning of the orbitofrontal and cingulate cortices (Dawe et al., 2004). In summary, the 2-CARS model proposes two separate components of trait impulsivity that convey risk for substance misuse through distinct mechanisms.

As mentioned earlier, while numerous large-scale, prospective studies have shown that measures of rash impulsiveness predict substance misuse, far fewer studies have been conducted with measures of reward drive. Only in recent years has research been conducted with measures specifically designed to assess reward sensitivity or drive, such as measures of Gray's (1970) BAS. Knyazev, Slobodskaya, Kharchenko, and Wilson (2004) investigated the role of BAS as a risk factor for substance use in a stratified randomized sample of 4501 Russian high-school students. Knyazev et al. reported BAS/reward drive to be the second most powerful predictor of substance use in adolescents, accounting for 10.5% variance. The most powerful predictor of substance use was drug offer (21.7%).

While this study provided important insights concerning how reward drive-related traits contribute to risk for substance misuse, there is other evidence suggesting rash impulsiveness may be the stronger predictor. In a representative community sample of 1803 young adults (ages 19–21 years), Johnson, Turner, and Iwata (2003) found that BAS-Fun Seeking, but not BAS-Reward Responsiveness or BAS-Drive, predicted lifetime drug and alcohol use disorder diagnoses. Similarly, in a large Australian community sample ($N = 2725$), Jorm et al. (1999) reported that BAS-Fun Seeking and BAS-Drive, but not BAS-Reward Responsiveness, was significantly associated with hazardous drinking. Factor analytic studies have shown that BAS-Fun Seeking, unlike BAS-Reward Responsiveness and BAS-Drive, taps elements of rash impulsiveness in addition to reward drive (Dawe & Loxton, 2004; Zelenski & Larsen, 1999). This suggests that measures of rash impulsiveness may be more robust predictors of substance use problems. Indeed, Franken and Muris (2006) found that while both BAS-Drive and BAS-Fun Seeking were associated with illicit drug experimentation, BAS-Fun Seeking was the stronger predictor. Additionally, they reported that only BAS-Fun Seeking was associated with binge drinking. A study by Loxton et al. (2008) found that while club drug-users scored higher on BAS-Drive and the Disinhibition subscale of the Sensation Seeking Scale, only the latter (a measure of rash impulsiveness; Dawe & Loxton, 2004) was associated with polydrug use.

Taken together, the evidence suggests that while both traits convey risk for substance misuse, rash impulsiveness is a stronger direct predictor of problem use. This begs the question as to whether there is a need to consider a second impulsivity component. The adoption of a less parsimonious, two-factor model like 2-CARS is only warranted if doing so provides a better explanation of substance use than a one-factor model. While the evidence re-

viewed above is not compelling in this regard, these large-scale studies suffer from two important limitations. First, they did not explore the role of impulsivity traits in addiction-related mechanisms. Second, they did not examine the direct association of each trait with substance misuse while controlling for variance explained by the other. Each of these will be discussed in turn.

Several recent studies suggest that trait reward drive and rash impulsiveness are related to distinct mechanisms involved in addictive behavior. Kambouropoulos and Staiger (2004) found that only reward drive predicted alcohol cue-elicited urge to drink in social drinkers. Furthermore, only Rash impulsiveness predicted increased positive affect after alcohol cue presentation. Gullo, Jackson et al. (2010) studied the role of reward drive and rash impulsiveness in the reversal learning of hazardous drinking college students. The reversal learning task used in this study required participants to learn that a previously reinforced approach response was no longer being rewarded and required modification/inhibition. This form of learning has been shown to rely on the integrity of the orbitofrontal cortex, and to be diminished in substance abusers (Fellows & Farah, 2003; Hildebrandt, Brokate, Fink, Muller, & Eling, 2008). Consistent with 2-CARS, Gullo, Jackson et al. (2010) found that while both traits predicted more hazardous drinking, only rash impulsiveness was related to poor reversal learning. Reward drive actually predicted better learning, again suggesting dissociation between traits.

Reward drive and rash impulsiveness have also been linked to distinct types of drug-related cognition. Egan, Kambouropoulos, and Staiger (2010) found that ecstasy users were higher than non-users in reward drive and rash impulsiveness, but the traits were related to different drug use motivations. Reward drive was associated with motivation to use ecstasy to enhance social cohesion. By contrast, rash impulsiveness was associated with motivation to use the drug in order to reduce negative affect. Gullo, Dawe et al. (2010) also found that the two traits were independently associated with distinct substance-related cognitive mechanisms. In separate samples of college drinkers and alcohol-dependent inpatients, they found that reward drive, but not rash impulsiveness, predicted higher positive alcohol expectancies (e.g., "I'm a better lover after a few drinks"; Goldman, Greenbaum, & Darkes, 1997). Additionally, they reported that only rash impulsiveness predicted (lower) drinking refusal self-efficacy (i.e., perceived ability to refuse alcohol in a cued situation). In sum, when examining specific mechanisms, a two-factor model seems indicated.

The second limitation is that most self-report studies have not investigated the unique association of reward drive and rash impulsiveness with substance misuse. That is, examining the direct association of each trait with substance misuse while controlling for variance explained by the other. This is an important limitation given the significant overlap found between measures of reward drive and rash impulsiveness (between 5% and 38% shared variance, depending on the measures; Dawe & Loxton, 2004). The use of structural equation modeling (SEM) could assist in this regard as it allows the testing of associations between multiple latent factors (Kline, 2005). That is, by analyzing only the variance shared between several measures of reward drive on the one hand, and rash impulsiveness on the other, a less biased index of each trait could be derived to explore the unique aspects of each trait in substance misuse. Another advantage of SEM is that it allows the comparison of competing models while taking model parsimony into account (Kline, 2005). That is, it could be used to examine whether a two-factor model of impulsivity provides a better fit to the data than a one-factor model. SEM also allows the comparison of a model across different populations. This cross-cultural generalizability has also not been directly tested.

The present study sought to evaluate the 2-CARS model of impulsivity and substance misuse by investigating the unique role

of reward drive and rash impulsiveness in both alcohol and illicit drug use. It also sought to test the cross-cultural generalizability of the model, by comparing the magnitude of predicted associations across a British and Australian sample. It was hypothesized that the 2-CARS model would provide a better fit to the data than a one-factor model of impulsivity, and that both reward drive and rash impulsiveness would contribute unique variance to the prediction of alcohol and illicit drug misuse. Structural invariance of alcohol and illicit drug models across the two sites was also hypothesized. That is, that the magnitude of association of each trait with substance misuse would not differ by country.

2. Method

2.1. Participants

The sample comprised a total of 499 participants. Of the 213 participants in the UK sample, 159 (74.6%) were female and 54 (25.4%) were male. The age range of participants was 18–23 years. Of the 286 participants in the Australian sample, 202 (70.6%) were female and 84 (29.4%) were male. The age range of the sample was 18–56 years, with the vast majority (85.6%) aged 18–23 years. There was a significant difference in age between the two sites, with Australian participants ($M_{\text{years}} = 21.01$, $SD = 5.66$) being older than UK participants ($M_{\text{years}} = 19.02$, $SD = 0.99$), $t(294.37) = -5.66$, $p < .001$. Age correlated significantly with level of illicit drug use in both samples ($r_{\text{UK}}[181] = .27$, $p < .001$; $r_{\text{Aust}}[269] = .18$, $p = .002$). However, controlling for age did not affect parameter estimates or overall model fit. Therefore, it was not included in the final model.¹

In the UK sample, participants were first-year undergraduate students. For their participation, psychology students were offered partial course credit, and non-psychology students were offered £5.00 for their participation (\approx \$AU10.25 at the time of recruitment). In the Australian sample, psychology students were offered partial course credit for their participation. Non-psychology students were offered \$AU10.00 for their participation. Informed consent was obtained from each participant and the study was approved by all relevant ethics committees.

2.2. Materials

Reward drive was measured using Carver and White's (1994) BAS scales as indicators of a latent reward drive factor. The BAS-Drive scale (BAS-D) includes four items assessing the persistent pursuit of desired goals (e.g., "When I want something, I usually go all-out to get it"). It has been reported to have a Cronbach's α of .74–.87 and an 8-week test-retest reliability of .66 (Carver & White, 1994; Cooper, Gomez, & Aucote, 2007; Heubeck, Wilkinson, & Cologon, 1998; Leone, Perugini, Bagozzi, Pierro, & Mannetti, 2001; Miller, Joseph, & Tudway, 2004). The BAS-Reward Responsiveness scale (BAS-RR) includes five items assessing positive responses to reward/anticipated reward (e.g., "When I get something I want, I feel excited and energized"). It has been found to have a Cronbach's α of .68–.89, and a test-retest reliability of .59 (Carver & White, 1994; Cooper et al., 2007; Heubeck et al., 1998; Leone et al., 2001; Miller et al., 2004). The BAS-Fun Seeking scale (BAS-FS) includes four items assessing the desire for new rewards and a willingness to pursue them on the spur of the moment (e.g., "I am always willing to try something new if I think it will be fun"). The measure has been reported to have a Cronbach's α of .66–.88 and an 8-week test-retest reliability of .69 (Carver & White,

1994; Cooper et al., 2007; Heubeck et al., 1998; Leone et al., 2001; Miller et al., 2004). Factor analytic studies have found BAS-D and BAS-RR to load on a reward drive factor with other self-report measures of BAS sensitivity, supporting their convergent validity (Dawe & Loxton, 2004). The BAS-FS scale has been found to load on both reward drive and rash impulsiveness factors, suggesting the measure is tapping both constructs (Dawe & Loxton, 2004; Zelenski & Larsen, 1999). Therefore, this measure served as an indicator for rash impulsiveness as well.

Rash impulsiveness was assessed using the novelty seeking scale of the Tridimensional Personality Questionnaire (TPQ-NS; Cloninger, 1989), I_7 (Impulsiveness), and BAS-FS as indicators of a latent rash impulsiveness factor. Cronbach's α for TPQ-NS has been reported to be approximately .74, and test-retest reliability of .85 (Cloninger, 1987; Cloninger, Przybeck, & Svrakic, 1991). Cronbach's α for I_7 (Impulsiveness) has been reported as .84 (Corulla, 1987; Eysenck, Easting, & Pearson, 1994). Furthermore, Luengo, Carillo-de-la-Pena, and Otero (1991) reported the I_7 (Impulsiveness) scale to have a 1-year test-retest reliability of .76. Factor analytic studies have found each of these measures to load on a rash impulsiveness factor with other self-report measures of the construct, supporting their convergent validity (Dawe & Loxton, 2004; Zelenski & Larsen, 1999).

2.2.1. Hazardous substance use

The Alcohol Use Disorders Identification Test (AUDIT; Saunders, Aasland, Babor, de la Fuente 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, with Cronbach's α ranging from .80 to .94. It has a 6-week test-retest reliability of .88 (Daeppen, Yersin, Landry, Pecoud, & Decrey, 2000). The AUDIT has been consistently found to be more sensitive to non-dependent problem drinking than other commonly used screening instruments and several studies support the measure's validity as an index of hazardous drinking in college students (Dawe, Loxton, Hides, Kavanagh, & Mattick, 2002; Kokotailo et al., 2004; Roche & Watt, 1999).

The Alcohol, Smoking and Substance Involvement Screening Test (ASSIST; Ali et al., 2002), version 3.0, is an eight-item, interviewer-administered questionnaire designed to screen for the presence and severity of psychoactive substance use (alcohol, tobacco, and illicit drugs). The ASSIST was developed, and has been validated for, international use in primary care settings (Humeniuk et al., 2008). The alcohol specific substance involvement (SSI Alcohol) and the global continuum of illicit drug risk composites were used for the purposes of this study. The illicit drug composite includes items assessing frequency of use over the past 3 months of illicit substances the participant has indicated ever using. It also contains items relating to the adverse social, health, and legal effects of their use, as well as items relating to dependence symptoms (loss of control over use, failure to meet expectations). Illicit substances assessed include cannabis, cocaine, amphetamine-type stimulants, inhalants, hallucinogens, and non-prescription use of sedatives and opioids. The composites have been shown to discriminate between non-problematic use and abuse of alcohol and illicit drugs, as well as between abuse and dependence diagnoses (Humeniuk & Ali, 2006). The SSI Alcohol and illicit drug risk composites have been shown to have a 3-month test-retest reliability of .70 and .81, respectively (Humeniuk & Ali, 2006). Additionally, both composites have been shown to have good internal consistency (Cronbach's α : SSI Alcohol = .84, illicit drug risk = .91).

¹ Similarly, excluding Australians over age 23 did not affect parameter estimates or model fit. Therefore, all Australians were retained for analysis.

2.3. Procedure

In the UK sample, all personality questionnaires were completed by participants prior to attending the testing session in which the ASSIST interview was conducted and the AUDIT self-report measure was administered. For psychology students, the personality measures were administered in classes as part of a standard research assessment battery. For non-psychology students, personality measures were posted out 1–2 weeks prior to their scheduled testing session.

In the Australian sample, participants accessed a secure website which administered all measures as online questionnaires. While the two testing sites differed in the method in which the questionnaires were administered, there is substantial evidence demonstrating little difference in pencil-and-paper versus internet administration of self-report instruments (Gosling, Vazire, Srivastava, & John, 2004). Studies have shown that personality measures retain their psychometric properties when administered online (Buchanan & Smith, 1999; Chuah, Drasgow, & Roberts, 2006), including measures of impulsivity (Aluja, Rossier, & Zuckerman, 2007). Comparative studies have also shown that alcohol and drug use assessments retain satisfactory psychometric properties when administered as an online questionnaire compared to an interview or pencil-and-paper questionnaire, even when participants do not complete the entire measure in one sitting (Brodey et al., 2004; Miller et al., 2002; Thomas & McCambridge, 2008). Meta-analyses have also shown little-to-no negative effect of social desirability/impression management on internet administered questionnaires, compared to pencil-and-paper questionnaires or interviews (Dwight & Feigelson, 2000; Richman, Kiesler, Weisband, & Drasgow, 1999).

2.4. Data analysis

All models were tested with structural equation modeling (SEM) using maximum likelihood estimation. Stringent criteria were used for evaluation of model fit. In accordance with Hu and Bentler (Bentler, 2007; Hu & Bentler, 1999), the χ^2 test was used as a statistical test of model fit ($\alpha = .05$). The comparative fit index (CFI), root mean-square error of approximation (RMSEA), and standardized root mean-square residual (SRMR) were also used (Bentler, 2007).

As a general rule-of-thumb, Hu and Bentler (1999) suggested the following cut-offs for “good” fit: CFI $\geq .95$; RMSEA $\leq .06$; SRMR $\leq .08$; and these were adopted for the present study. However, such cut-offs should be regarded as guidelines rather than “golden rules” when assessing model fit (Marsh, Hau, & Wen, 2004). In addition to reporting SRMR, Bentler (2007) recommended reporting the largest several standardized residual covariances. None/few of these values should exceed 2.58 ($p < .01$). Lastly, the fit of the hypothesized model was compared to that of non-hypothesized alternatives (McDonald & Ho, 2002). The Akaike Information Criteria (AIC) was selected to assist in model comparison, where smaller values indicate a model is better-fitting and more parsimonious.

The hypothesized model for hazardous alcohol use included reward drive as a latent variable with three indicators (BAS-D, BAS-RR, and BAS-FS), rash impulsiveness as a latent variable with three indicators (I_7 [Imp], TPQ-NS, and BAS-FS), and hazardous alcohol use as a latent variable with two indicators (SSI alcohol and AUDIT total score). The BAS-FS scale was specified as an indicator of both impulsivity traits, consistent with previous research (Dawe & Loxton, 2004; Zelenski & Larsen, 1999). It was hypothesized that reward drive and rash impulsiveness would both significantly predict hazardous alcohol use, such that higher levels of these traits would be associated with more hazardous use. The reward drive and rash impulsiveness latent factors were allowed to covary.

The hypothesized structural model for illicit drug misuse was identical to that proposed for alcohol misuse. Illicit drug misuse was measured using the global illicit drug risk composite from the ASSIST. All models were tested as multi-group models to explore possible cross-cultural differences.

3. Results

3.1. Data screening and assumptions

Prior to analysis, all variables of interest were examined for accuracy of data entry, missing values, and fit between their distributions and the assumptions of SEM using maximum likelihood estimation. The original dataset contained responses from 499 participants. Twenty-five participants (5.0%) failed to answer any item on more than two measures and were thus excluded, leaving 474 cases for further analysis. In addition to this, four participants (<0.1%) were missing data on age, 16 participants (3.4%) were missing data on I_7 (Imp), 38 participants (8.0%) were missing data on TPQ-NS, 14 participants (3.0%) were missing data on the AUDIT, and two participants (0.4%) were missing data on the ASSIST. Little's Missing Completely at Random (MCAR) test was not significant ($\chi^2 [242] = 275.54, p = .07$), suggesting the data were Missing Completely at Random. Missing data were estimated using Expectation Maximization (EM), a robust estimation technique suitable for SEM (Newman, 2003; Schafer & Graham, 2002).

The data were screened for outliers and this analysis revealed the presence of 17 (UK = 6, Australia = 11) univariate and three (UK = 1, Australia = 2) multivariate outliers. Most of these outliers were extreme scores (relative to the rest of the sample) on the illicit drug risk measure. The presence of these outliers severely affected multivariate normality, particularly for the illicit drug model. Thus, the outliers were removed, leaving a final 454 cases for analysis (UK: $n = 183$, Australia: $n = 271$). While this improved multivariate normality, there was still evidence that the assumption of multivariate normality was violated. Four of the measured variables (BAS-RR, AUDIT, SSI Alcohol, illicit drug risk) were significantly univariately skewed, $p < .001$. Mardia's Normalized coefficient indicated significant violation of multivariate normality in the UK alcohol model ($z = 4.03, p < .001$) and the Australian illicit drug model ($z = 2.03, p < .05$). To correct for the violation of this assumption, the Bollen–Stine bootstrap p , a bootstrap modification of model χ^2 , was also used as a significance test to evaluate the fit of the alcohol and illicit drug models. In addition, for these non-normal models, parameter estimates and standard errors were calculated using the bootstrap method (1000 samples) with bias-corrected 95% confidence intervals. Such methods have been shown to adequately correct for non-normality (Efron, 1988; Neal & Simons, 2007; Shrout & Bolger, 2002).

3.2. Descriptive statistics

The sample submitted for analysis comprised a total of 454 participants. Of the 183 participants in the UK sample, 137 (74.9%) were female and 46 (25.1%) were male. Of the 271 participants in the Australian sample, 192 (70.8%) were female and 79 (29.2%) were male. The sites did not significantly differ in the proportion of female/male participants, $\chi^2 (1) = 0.88, p = .35$.

In the UK sample, 145 participants (79.2%) reported consumption of alcohol within the last 3 months. In the Australian sample, 232 participants (85.6%) reported consuming alcohol within the last 3 months. There were no site differences in either lifetime prevalence ($\chi^2 [1] = 3.78, p > .05$) or 3-month prevalence ($\chi^2 [1] = 3.15, p > .05$) of alcohol use. According to SSI Alcohol scores derived from the ASSIST, 47 (25.7%) UK participants and 107

(39.5%) Australian participants were drinking at levels posing moderate or high risk to their health (score ≥ 11 ; Humeniuk & Ali, 2006). This difference was statistically significant, $\chi^2(1) = 9.28, p = .02$. According to the AUDIT, 62 (33.9%) UK participants and 136 (50.2%) Australian participants were drinking at hazardous or harmful levels (score ≥ 8 ; Saunders et al., 1993). This difference was also statistically significant, $\chi^2(1) = 11.81, p = .001$.

Descriptive statistics for the measured variables included in the models are presented in Table 1 and 2. As shown in Table 1, Australian participants reported significantly higher levels of hazardous alcohol use, as well as higher scores on all of the Carver and White (1994) BAS scales ($ps < .01$). However, there were no site differences in overall illicit drug risk. There were also no site differences in level of hazardous use of any specific illicit substance. As can be seen in Table 2, the reward drive indicator variables tended to have weaker relationships with drug and alcohol use in the Australian sample, compared to the UK sample.

3.3. The hypothesized alcohol model

The hypothesized model for alcohol misuse provided a very good fit to the data (see Table 3, Model 1). The largest standardized residual covariances were between I_7 (Impulsiveness) and BAS-D in the Australian sample ($z = 1.57$), and between TPQ-NS and BAS-D in the UK sample ($z = 1.26$). As predicted, greater rash impulsiveness predicted more hazardous alcohol use in both the UK sample (unstandardized coefficient = 0.76, $CI_{95}: 0.47-1.06, p < .001$) and the Australian sample (unstandardized coefficient = 0.61, $SE = 0.12,$

$p < .001$). There was a marginally significant association between reward drive and hazardous alcohol use in the UK sample (unstandardized coefficient = 0.54, $CI_{95}: -0.04-1.23, p = .07$), but not in the Australian sample (unstandardized coefficient = $-0.08, SE = 0.29, p = .78$). Additionally, reward drive was not related to rash impulsiveness in the UK sample (unstandardized coefficient = 0.55, $CI_{95}: -0.48-1.72, p = .29$) or Australian sample (unstandardized coefficient = 0.58, $SE = 0.44, p = .18$).

In the UK sample, the hypothesized model accounted for 26% ($CI_{95}: .13-.39$) of the variance in hazardous alcohol use. In the Australian sample, the hypothesized model accounted for 14% ($CI_{95}: .06-.24$) of the variance in hazardous alcohol use. When the model's structural paths were constrained to be equivalent across sites, there was no significant reduction in model fit, $\chi^2_{diff}(3) = 3.84, p > .05$. This suggests structural invariance across sites for the alcohol model. The hypothesized model with standardized coefficients is shown in Fig. 1.

Two alternative, non-hypothesized models were estimated for the purposes of comparison (McDonald & Ho, 2002). The first model included a one-factor impulsivity trait predicting hazardous alcohol misuse. This one-factor latent trait included all impulsivity measures as indicators. This model provided a poor fit to the data (see Table 3, Model 2). This suggested a two-factor model of impulsivity and alcohol misuse provided a better fit to the data. The second model was identical to the hypothesized model, but removed the hypothesized association between reward drive and hazardous alcohol use. This model provided a good fit to the data (see Table 3, Model 3). Furthermore, as shown in Table 3, the fit of this model to the data was not significantly different to that of the hypothesized model. This is in spite of the alternative model's significant χ^2 statistic, suggesting poorer fit. Inspection of the change in AIC also suggested the alternative model provided a poorer fit. Taken together, these results support the hypothesized model.

3.4. The hypothesized illicit drug model

The hypothesized model for illicit drug use provided a very good fit to the data (see Table 3, Model 4). The largest standardized residual covariances were between I_7 (Impulsiveness) and BAS-D ($z = 1.72$), and between I_7 (Impulsiveness) and illicit drug risk ($z = -1.38$) in the Australian sample. As predicted, greater rash impulsiveness predicted greater illicit drug risk in both the UK (unstandardized coefficient = 2.36, $SE = 0.44, p = .002$) and Australian samples (unstandardized coefficient = 0.87, $CI_{95}: 0.36-1.49, p = .001$). As hypothesized, reward drive was directly related to illicit drug use, but only in the UK sample (unstandardized coefficient = 1.28, $SE = 0.66, p = .05$). In the Australian sample, reward

Table 1
Means, standard deviations (SD), and Cronbach alpha coefficients of impulsivity and substance use measures for the UK ($N = 183$) and Australian ($N = 271$) samples.

Scale	Cronbach α		Mean (SD)		p	d
	UK	Australia	UK	Australia		
BAS-RR	.92	.65	14.24 (4.30)	16.60 (1.90)	<.001	0.76
BAS-D	.74	.72	10.06 (2.48)	10.69 (2.03)	.004	0.14
BAS-FS	.72	.74	10.74 (2.57)	11.41 (2.10)	.004	0.29
TPQ-NS	.71	.78	18.72 (4.82)	17.77 (5.43)	.06	0.09
I_7 (Imp)	.78	.82	8.18 (3.92)	8.17 (4.42)	.98	0.002
SSI Alc ^a	.75	.79	7.40 (7.70)	9.89 (8.30)	.001	0.31
AUDIT ^a	.81	.82	6.00 (5.39)	8.39 (6.06)	<.001	0.42
Illicit drug risk ^a	.86	.86	9.32 (13.45)	8.94 (13.07)	.90	0.03

Note: Bold p values are statistically significant. BAS-RR = BAS-Reward Responsiveness; BAS-D = BAS Drive; BAS-FS = BAS-Fun Seeking; TPQ-NS = Novelty Seeking; I_7 (Imp) = Eysenck Impulsiveness scale; SSI Alc = specific substance involvement – alcohol; AUDIT = Alcohol Use Disorders Identification Test.

^a Site differences tested using Mann-Whitney U .

Table 2
Intercorrelations of impulsivity and substance use measures for the UK ($N = 183$) and Australian ($N = 271$) samples.

Scale	BAS-RR	BAS-D	BAS-FS	TPQ-NS	I_7 (Imp)	SSI Alc	AUDIT	Illicit drug risk
BAS-RR	–	.52***	.62***	.08	.00	.20**	.19*	.14†
BAS-D	.42***	–	.52***	.14†	.13	.09	.14	.16*
BAS-FS	.32***	.41***	–	.30***	.25**	.27***	.24**	.30***
TPQ-NS	.01	.06	.50***	–	.58***	.27***	.33***	.42***
I_7 (Imp)	.06	.16**	.46***	.66***	–	.29***	.39***	.32***
SSI Alc	.06	.01	.21***	.29***	.27***	–	.79***	.49***
AUDIT	–.04	.01	.17**	.30***	.24***	.79***	–	.50***
Illicit drug risk	.02	.09	.18**	.23***	.08	.37***	.36***	–

Note: Values presented above the diagonal relate to the UK sample, values below refer to Australian sample. BAS-RR = BAS-Reward Responsiveness; BAS-D = BAS Drive; BAS-FS = BAS-Fun Seeking; TPQ-NS = Novelty Seeking; I_7 (Imp) = Eysenck Impulsiveness scale; SSI Alc = specific substance involvement – alcohol; AUDIT = Alcohol Use Disorders Identification Test.

† $p = .05$.
* $p < .05$.
** $p < .01$.
*** $p < .001$.

Table 3
Fit indices for hypothesized and non-hypothesized multi-group models ($N = 454$).

Model	χ^2 (df)	Bollen–Stine bootstrap p	CFI	SRMR	RMSEA (CI ₉₀)	AIC	χ^2_{diff} (df)	Δ AIC
<i>Alcohol</i>								
1. Hypothesized two-factor model	30.37 (20)	.11	.99	.03	.03 (.00–.06)	102.37		
2. One-factor impulsivity model	238.88* (26)	.001	.81	.14	.13 (.12–.15)	298.88		
3. Model with RD path removed	34.47* (22)	.09	.99	.06	.04 (.01–.06)	102.47		
Difference between Model 2 and Model 1							4.10 (2)	196.51
Difference between Model 3 and Model 1								0.10
<i>Illicit drugs</i>								
4. Hypothesized two-factor model	19.66 (12)	.06	.99	.03	.04 (.00 – .07)	79.66		
5. One-factor impulsivity model	232.64* (18)	.001	.68	.15	.16 (.14 – .18)	280.64		
6. Model with RD path removed	24.64* (14)	.025	.98	.05	.04 (.01 – .07)	80.64		
Difference between Model 5 and Model 4							4.98 (2)	200.98
Difference between Model 6 and Model 4								0.98

Note: CFI = comparative fit index; SRMR = standardized root mean-square residual; RMSEA = root mean-square error of approximation; AIC = Akaike Information Criterion. * $p < .05$.

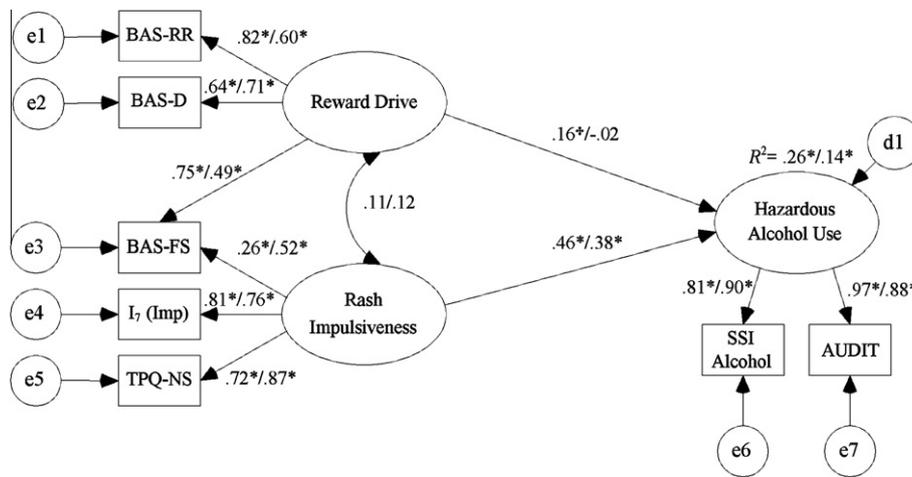


Fig. 1. Hypothesized two-factor model of impulsivity and hazardous alcohol use. Note: Values presented left of the slash are for the UK sample, values on the right refer to the Australian sample. Ellipses represent latent constructs, and rectangles indicate measured variables. Circles (e) reflect residuals or (d) disturbances; numbers above or nearby endogenous variables represent the amount of variance explained (R^2). BAS-RR = BAS Reward Responsiveness; BAS-D = BAS Drive; BAS-FS = BAS Fun Seeking; I₇ (Imp) = Eysenck Impulsiveness scale; TPQ-NS = Novelty Seeking; SSI Alcohol = Specific Substance Involvement - Alcohol; AUDIT = Alcohol Use Disorders Identification Test. * $p < .05$.

drive was not related to illicit drug use (unstandardized coefficient = 0.70, CI₉₅: -0.56 – 2.07 , $p = .29$). As in the alcohol use model, reward drive was not directly related to rash impulsiveness in the UK sample (unstandardized coefficient = 0.57, $SE = 0.44$, $p = .20$) or Australian sample (unstandardized coefficient = 0.50, CI₉₅: -0.28 – 1.49 , $p = .23$).

In the UK sample, the hypothesized model accounted for 27% (CI₉₅: .15–.41) of the variance in illicit drug use. In the Australian sample, the hypothesized model accounted for 6% (CI₉₅: .01–.13) of variance, significantly less than in the UK sample. When the model's structural paths were constrained to be equivalent across sites, there was a significant reduction in model fit, χ^2_{diff} (3) = 10.21, $p < .05$. Further analysis revealed significant site differences for the association between rash impulsiveness and illicit drug risk, χ^2_{diff} (1) = 8.87, $p < .05$. As shown in Fig. 2, rash impulsiveness had a much larger association with illicit drug risk in the UK sample (standardized coefficient = .47) compared to the Australian sample (standardized coefficient = .22), accounting for the difference in explained variance. This suggests only partial structural invariance for the illicit drug model.

Two alternative, non-hypothesized models were estimated for the purposes of comparison (McDonald & Ho, 2002). The first model included a one-factor impulsivity trait predicting illicit drug risk. This one-factor latent trait included all impulsivity measures as

indicators. This model provided a poor fit to the data (see Table 3, Model 5), suggesting a two-factor model of impulsivity and illicit drug use provided a better fit to the data. The second model was identical to the hypothesized model, but removed the hypothesized direct association between reward drive and illicit drug use. This model provided a good fit to the data (see Table 3, Model 6). Furthermore, as shown in Table 3, the fit of this model to the data was not significantly different to that of the hypothesized model. However, inspection of the change in AIC, and each models' χ^2 test, suggested that this alternative model provided a poorer fit than the hypothesized model. Taken together, these results support the hypothesized model.

4. Discussion

The results provide consistent support for the utility of a two-factor model of trait impulsivity and substance misuse by showing a differential role for these traits in predicting hazardous use. Results were largely consistent across two international sites, with the hypothesized 2-CARS model found to provide the best fit to the data for alcohol and illicit drug use. As predicted, rash impulsiveness emerged as a robust predictor of hazardous substance use. However, reward drive was less consistently associated with alcohol and illicit drug misuse.

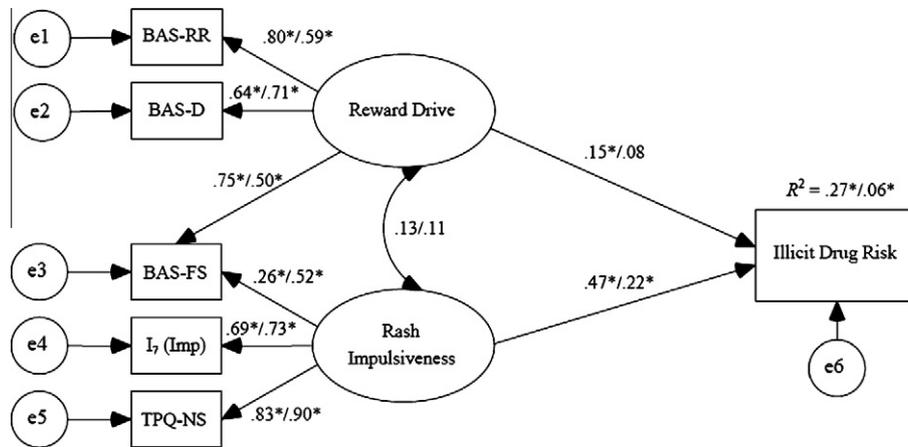


Fig. 2. Hypothesized two-factor model of impulsivity and illicit drug risk. *Note:* Values presented left of the slash are for the UK sample, values on the right refer to the Australian sample. Ellipses represent latent constructs, and rectangles indicate measured variables. Circles (e) reflect residuals or (d) disturbances; numbers above or nearby endogenous variables represent the amount of variance explained (R^2). BAS-RR = BAS Reward Responsiveness; BAS-D = BAS Drive; BAS-FS = BAS Fun Seeking; I7 (Imp) = Eysenck Impulsiveness scale; TPQ-NS = Novelty Seeking. $p < .05$.

Consistent with the findings from animal research and human psychopharmacology (Belin et al., 2008; de Wit & Richards, 2004), the present study provides further support for a distinction between two aspects of trait impulsivity, as outlined in the 2-CARS model (Dawe et al., 2004; Dawe & Loxton, 2004; Gullo & Dawe, 2008). Rash impulsiveness was found to be the stronger and more robust predictor of problematic substance use, consistent with previous self-report studies. When controlling for rash impulsiveness, reward drive was less consistently associated with substance misuse.

While previous research with large samples support an association between reward drive measures and hazardous substance use, most have not examined its unique contribution independent of rash impulsiveness. In a sample of 276 college students, Franken and Muris (2006) found that both BAS-D and BAS-FS correlated with number of illegal substances ever used. Similarly, Egan et al. (2010) found ecstasy users reported higher levels of both reward drive (measured with the SR scale) and rash impulsiveness (measured with I7 Impulsiveness). Loxton et al. (2008) found that club drug-users scored higher on BAS-D, BAS-FS, and Disinhibition. Unfortunately, none of these studies examined the unique effects of the two traits, which may have inflated associations with reward drive.

In the present study, the structural models showed the relationship between reward drive and substance use was equivalent across countries. However, if one were to simply examine the zero-order correlations of each site, differences seemingly appear. The zero-order correlations were confounded by a lack of control for the variance shared between measures of the two traits. The British reward drive measures tended to show higher correlations with BAS-FS and rash impulsiveness, compared to the Australian data. However, when testing latent variables only the variance in a measure that is unique to the construct is analyzed. This allowed a more rigorous test of the role of reward drive and rash impulsiveness in substance use. Given the difficulties in constructing impulsivity measures that are entirely unrelated, future research needs to take this into account. Future studies should provide some assurance that only unique variance is being examined in the data analyzed, and relations are not artificially inflated.

While reward drive was less robustly associated with substance misuse in the present study, these findings do not imply the trait is unimportant to understanding such behavior, but rather that its influence is not as direct. Indeed, there is much evidence to suggest a role for reward drive in addiction-related mechanisms, such as through increased physiological response to alcohol (Brunelle

et al., 2004; Glautier, Bankart, & Williams, 2000), cue-elicited urge to drink (Kambouropoulos & Staiger, 2004), and positive outcome expectancies (Egan et al., 2010; Gullo, Dawe et al., 2010). These mechanisms in isolation do not equate to hazardous substance use, but do increase the risk for it. For example, a cue-elicited urge to drink, no matter how strong, will not lead to problem alcohol use so long as there is sufficient inhibitory control to prevent the individual from acting on it inappropriately. Rash impulsiveness may be the more robust predictor because it taps aspects of poor inhibitory control in addition to approach motivation.

Taken together, the results of this study and previous research suggest that rash impulsiveness is a more robust trait predictor of hazardous, problem substance use. This conclusion is consistent with the results of Loxton et al. (2008) and Johnson et al. (2003) who found only rash impulsiveness was associated with the more problematic use of illicit drugs and alcohol. Interestingly, this dissociation is also found in animals. Belin et al. (2008) reported in rats that only reward sensitivity/responsiveness predicted faster acquisition of cocaine self-administration. By contrast, only "impulsivity" predicted the transition to compulsive use; that is, persistent cocaine self-administration despite punishment.

The model's findings were robust across countries and substance classes. However, invariance testing did reveal a stronger association between rash impulsiveness and illicit drug misuse in the UK sample. It is possible that the difference in path strength was the result of site differences on some unmeasured variable related to both rash impulsiveness and drug use. Indeed, controlling for potential covariates/mediators has been shown to greatly affect observed relationships between rash impulsiveness measures and self-reported behavior (Fergusson et al., 2008; O'Connor & Jackson, 2008).

The main limitation of the present study is the sole reliance on self-report. While the use of latent variables reduces measurement error, this study did not explicitly measure and control for social desirability beyond assuring participants of confidentiality. Self-report measures are also prone to bias resulting from response styles and lack of insight (Reynolds, Ortengren, Richards, & de Wit, 2006). Future research, employing behavioral impulsivity measures, is therefore recommended. Another limitation is the study's recruitment of a mostly female sample of non-clinical substance users. Therefore, our findings may not generalize to all substance users, given the higher levels of use in males and in substance dependent individuals. Future research would need to investigate this. Due to the cross-sectional nature of the study, direction of causation

cannot be determined. However, there is a large body of evidence attesting to the predictive role of trait impulsivity (e.g., Fergusson et al., 2008). Lastly, while these findings provide evidence for two impulsivity traits in both alcohol and illicit drug use, there may be need to consider more (Jackson, 2008; Whiteside & Lynam, 2001) and future research should explore the extent to which additional traits add to the prediction of substance use behavior.

In conclusion, these findings add to the growing research literature supporting the utility of a two-factor model of impulsivity (such as 2-CARS) by demonstrating a differential role for these components in hazardous substance use. Results show consistent support for a direct association between trait rash impulsiveness and alcohol/illicit substance misuse, but less consistent support for an association between reward drive and substance misuse. The latter finding suggests the risk conveyed by reward drive is more indirect, and more dependent on the role of other mediating factors (Gullo & Dawe, 2008). Further research into the similarities and differences between these two impulsivity components would not only enhance the understanding of how they convey risk for addiction, but also better inform targets for early intervention in those placed at risk by virtue of an “impulsive temperament”.

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