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Rash Impulsiveness and Reward Sensitivity in Relation to Risky Drinking by University Students: Potential Roles of Frontal Systems

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Abstract

Background. Two forms of impulsivity, rash impulsiveness and reward sensitivity, have been proposed to reflect aspects of frontal lobe functioning and promote substance use. The present study examined these two forms of impulsivity as well as frontal lobe symptoms in relation to risky drinking by university students. **Methods.** University undergraduates aged 18-26 years completed the Alcohol Use Disorders Identification Test (AUDIT), Barratt Impulsiveness Scale (BIS-11), Sensitivity to Punishment and Sensitivity to Reward Questionnaire (SPSRQ), Frontal Systems Behavior Scale (FrSBe), and a demographics questionnaire assessing age, gender, and age of onset of weekly drinking (AOD*).* **Results.** AUDIT-defined Harmful drinkers reported earlier AOD and scored higher on BIS-11, the Sensitivity to Reward (SR) scale of the SPSRQ, and the Disinhibition and Executive Dysfunction scales of the FrSBe compared to lower risk groups. Differences remained significant after controlling for duration of alcohol exposure. Path analyses indicated that the influence of SR on AUDIT was mediated by FrSBe Disinhibition, whereas the influence of BIS-11 on AUDIT was mediated by both Disinhibition and Executive Dysfunction scales of the FrSBe. **Conclusions.** Findings tentatively suggest that that the influence of rash impulsiveness on drinking may reflect dysfunction in dorsolateral prefrontal and orbitofrontal systems, whereas the influence of reward sensitivity on drinking may primarily reflect orbitofrontal dysfunction. Irrespective of the underlying functional brain systems involved, results appear to be more consistent with a pre-drinking trait interpretation than effects of alcohol exposure.

Research on the etiology of risky or problematic alcohol use has pointed to a complex interaction of genetic, developmental and environmental factors. Findings have implicated neurobiological and personality variables that preceded alcohol exposure as well as neurobehavioral deficits attributed to the duration and severity of alcohol exposure (Kreek, Nielsen, Butelman, & LaForge, 2005; Lyvers, 2000; Simons, Gaher, Correia, Hansen, & Christopher, 2005; Varma, Basu, Malhotra, Sharma, & Mattoo, 1994; Verdejo-García, Rivas-Péreza, López-Torrecillasa, & Pérez-García, 2006; Volkow & Li, 2004). Deficits of frontal lobe functioning and associated cognitive and behavioral manifestations have been attributed by some researchers to the cumulative effects of chronic alcohol misuse (Lyvers, 2000; Oscar-Berman & Marinkovic, 2007; Verdejo-García, Bechara, Recknor, & Pérez-García, 2006) and/or an early onset age for excessive alcohol use at a vulnerable time of cortical development (Crews, He, & Hodge, 2007; Pitkänen, Lyyra, & Pulkkinen, 2005). However, an alternative case can be made that to some extent such deficits may have predated the exposure to alcohol and may have predisposed to problematic drinking (Dawe, Gullo & Loxton, 2004; Lyvers, Czerczyk, Follent & Lodge, 2009; Lyvers, Duff & Hasking, 2011). Such factors may include an inherited imbalance in the neural interactions between the prefrontal cortex and subcortical regions involved in reward and risk processing (Dawe et al., 2004; Spinella, 2003; Van Leijenhorst et al., 2010), as well as inherent personality traits such as appetitive impulsivity and aversive neuroticism (Hair & Hampson, 2006; Kambouropoulos & Staiger, 2007; Schmidt, Buckner, & Keough, 2007; Zuckerman & Kuhlman, 2000).

Impulsivity is a trait known to be linked to frontal lobe functioning (Berlin, Rolls, & Kischka, 2004; Chen et al., 2007; Elliott & Deakin, 2005; Franken, van Strien, Nijs, & Muris, 2008; Schoenbaum & Shaham, 2008; Yacubian et al., 2007) as well as problematic drinking and other forms of substance misuse (Dawe et al., 2004; Dawe & Loxton, 2004; Hanson, Luciana, & Sullwold, 2008; O'Connor & Colder, 2005; Simons et al., 2005; Spinella, 2004). Dawe et al. (2004) distinguished between two forms of impulsivity that promote excessive substance use: reward sensitivity and rash impulsiveness. Reward sensitivity refers to the degree to which behavior tends

to be motivated by the prospect of positive reinforcement, and according to Dawe et al. can be measured by the SR scale of the Sensitivity to Punishment and Sensitivity to Reward Questionnaire (SPSRQ; Torrubia, Ávila, Moltób, & Caseras, 2001). Rash impulsiveness refers to acting without due regard for negative consequences, and according to Dawe et al. can be measured by the Barratt Impulsiveness Scale (BIS-11; Patton, Stanford, & Barratt, 1995). Different brain systems were proposed to underlie these two forms of impulsivity, i.e., the mesolimbic dopamine system for reward sensitivity and the orbitofrontal cortex and anterior cingulate for rash impulsiveness. Dawe et al. suggested that reward sensitivity may play a major role in the onset of regular substance use, whereas rash impulsiveness may promote ongoing excessive or problematic substance use despite adverse outcomes. Dawe et al.'s distinction between two forms of impulsivity that promote risky or problematic alcohol or other substance use has recently been supported in multiple large samples (Gullo, Dawe, Kambouropoulis, Staiger & Jackson, 2010; Gullo, Ward, Dawe, Powell & Jackson, 2011).

Lyvers et al. (2011) recently assessed a community sample of social drinkers on the SR and BIS-11 measures of reward sensitivity and rash impulsiveness, respectively, as well as on the Frontal Systems Behavior Scale (FrSBe; Grace & Malloy, 2001). The FrSBe has three scales designed to assess behavior changes associated with damage to three prefrontal systems: the Apathy scale (anterior cingulate dysfunction), the Disinhibition scale (orbitofrontal dysfunction), and the Executive Dysfunction scale (dorsolateral prefrontal dysfunction). As predicted by Dawe et al. (2004), both SR and BIS-11 were positively associated with risky drinking as defined by the Alcohol Use Disorders Identification Test (AUDIT; Babor, de la Fuente, Saunders, & Grant, 1992). However, contrary to Dawe et al.'s model there was no association of risky drinking with the FrSBe Apathy scale implicating the anterior cingulate; rather, both the Disinhibition and Executive Dysfunction scales of the FrSBe were positively associated with risky drinking, potentially implicating orbitofrontal and dorsolateral prefrontal dysfunction respectively. Findings were consistent with the hypothesis that inherently poorer frontal lobe functioning, manifesting as high

levels of sensitivity to reward and rash impulsiveness, may reflect risk factors for problematic alcohol consumption. The FrSBe Disinhibition scale was strongly related to both SR and AUDIT, consistent with evidence that patients with orbitofrontal damage exhibit abnormally elevated sensitivity to reward (Hornak et al., 2004) and are at increased risk of problematic substance use (Spinella, 2003). Further, consistent with the hypothesized role of reward sensitivity, Lyvers et al. (2011) found an inverse relationship between SR and the age at which an individual started drinking weekly (AOD), suggesting that over-responsiveness to reward contingencies influences the drinking-related choices made at younger ages (Crews et al., 2007; Loxton & Dawe, 2001; Monti et al., 2005; Pardo, Aguilar, Molinuevo, & Torrubia, 2007; Volkow & Li, 2004). By contrast the FrSBe Executive Dysfunction scale was strongly related to BIS-11 and AUDIT, consistent with a theoretical link between impaired executive control, rash impulsiveness and problematic drinking (Lyvers, 2000). Alcoholics have been reported to exhibit high levels of rash impulsiveness (Ketzenberger & Forrest, 2000) and tend to make excessive errors on the Wisconsin Card Sorting Test, a neuropsychological task sensitive to dorsolateral prefrontal cortical functioning (Dolan, Bechara & Nathan, 2008; Smith, Perdices, O'Sullivan, Large & Barrett, 1997).

The majority of the community sample recruited by Lyvers et al. (2011) across a broad age range of 18-68 years consisted of Low Risk drinkers by AUDIT criteria, which compromised the sensitivity of the study to factors associated with AUDIT-defined Harmful drinking (only 9% of their sample). The present study utilised the same measures in an Australian university student sample aged 18-26 years, a group known to be characterised by high levels of both Hazardous and Harmful drinking (Hasking, Lyvers & Carlopio, 2011; Lyvers et al., 2009; Lyvers, Hasking, Hani, Rhodes & Trew, 2010). Relationships of the Disinhibition and Executive Dysfunction indices of the FrSBe with the two dimensions of impulsivity (as proposed by Dawe et al., 2004) and drinking were explored with a view to identifying how these dimensions of impulsivity may be related to harmful drinking as an expression of dysfunction in frontal systems. Based on the recent findings of Lyvers et al. (2011) we expected to obtain evidence that AUDIT-defined Harmful drinking is

related both to high reward sensitivity and rash impulsiveness in young adult social drinkers. We further hypothesized that the relationship between the SR index of reward sensitivity and drinking as assessed by AUDIT scores would be mediated via the FrSBe index of orbitofrontal dysfunction, the Disinhibition scale, whereas the relationship between the BIS-11 index of rash impulsiveness and AUDIT scores would be mediated via the FrSBe index of dorsolateral prefrontal dysfunction, the Executive Dysfunction scale.

Method

Participants

The 124 university student participants were 84 females and 40 males who all reported drinking alcohol at least occasionally. These psychology and marketing undergraduate students were all recruited at Bond University and participated for course credit. Ages ranged from 18 to 26 years (M = 20.08 years, SD = 1.72). The sample was characterized by very low rates of both illicit drug use and smoking.

Materials

The *Alcohol Use Disorders Identification Test* (AUDIT; Babor et al., 1992) contains 10 questions consisting of 3 quantity/frequency questions (e.g. “How often do you have a drink containing alcohol?”), 3 dependence-related items (e.g. “How often during the last year have you failed to do what was normally expected of you because of drinking?”), and 4 alcohol-related consequences or harm questions (e.g. “Have you or someone else been injured because of your drinking?”). Every AUDIT question is scored from 0 to 4, with an overall score ranging from 0-40. The suggested cut-offs are 1-7 for Low Risk drinking, 8-15 for Hazardous drinking and 16+ for Harmful drinking (Babor et al.). Internal consistency is high ranging from α =.80 (Kane, Loxton, Staiger, & Dawe, 2004) to α =.94 (Pal, Jena, & Yadav, 2004). Temporal stability is also high ranging from r =.87 over one week (Rubin et al., 2006) to r =.93 and .95 over four weeks (Bergman & Källmén, 2002; Dybek et al., 2006). Convergent validity with the Michigan Alcoholism Screening Test has been established (Pal et al., 2004).

The *Frontal Systems Behavior Scale* (FrSBe; Grace & Malloy, 2001)) is a 46-item scale assessing everyday behaviors associated with dysfunction in three major prefrontal cortical systems. The FrSBe has three corresponding subscales: Apathy (poor initiation, reduced drive and interest, e.g., "Sit around doing nothing"; anterior cingulate dysfunction), Disinhibition (restlessness, risk taking, socially inappropriate behavior, e.g., "Do or say embarrassing things"; orbitofrontal dysfunction), and Executive Dysfunction (problems with learning, sequencing, working memory, and mental flexibility, e.g., "Make the same mistakes over and over, do not learn from past experience"; dorsolateral prefrontal dysfunction). The standard version of the self rating form of the FrSBe measures behavioral change by obtaining pre- and post-lesion ratings. For the purposes of this study and in keeping with previous research (Lyvers et al., 2009, 2011; Lyvers, Onuoha, Thorberg, & Samios, 2012; Spinella, 2003; Verdejo-García, Rivas-Péreza et al., 2006) only current self-ratings were obtained. Items are rated on a 5 point Likert-type scale from 'almost never' to 'almost always'. The first 32 items represent deficits and are rated accordingly, with the final 14 positively stated items reverse scored. Scores are summated in each subscale to indicate the degree of impairment. Factor analyses of the FrSBe in clinical populations have supported the construct validity of the subscales (Stout, Ready, Grace, Malloy, & Paulsen, 2003). Evidence also supports reliability (Velligan, Ritchab, Suia, DiCoccoa, & Huntzingerab, 2002) with high internal consistency ($\alpha = .88$ to $\alpha = .91$) as well as three month temporal stability ($r = .78$). Convergent and divergent validity for each subscale have been established with the Wisconsin Card Sorting Test, the Social and Occupational Functioning Scale and the Functional Needs Assessment, and diagnostic validity has been confirmed for detecting levels of frontal lobe dysfunction in clinical samples (Chiaravalloti & DeLuca, 2003; Stout, Wyman, Johnson, Peavy, Salmon, 2003; Velligan et al., 2002), including substance abusers (Spinella, 2003).

The *Sensitivity to Punishment and Sensitivity to Reward Questionnaire* (SPSRQ; Torrubia et al., 2001) is a 48 item questionnaire containing two scales: Sensitivity to Punishment (SP; 24 items, e.g. "Are you often afraid of new or unexpected situations?"), and Sensitivity to Reward (SR; 24

items, e.g. “Do you sometimes do things for quick gains?”). Dichotomous responses of either ‘yes’ (1) or ‘no’ (0) produce a score for each scale which is a summation of all affirmative responses. The magnitude of the score indicates the level of SR and SP. Both scales demonstrate acceptable levels of internal consistency (SP, α = .81 to .83; SR, α = .73 to .76) and three month test–retest reliability (SP, *r* = .89; SR, *r* = .87) (O'Connor & Colder, 2005; Torrubia et al., 2001). Construct validity has been demonstrated in a two factor solution (Caci, Deschaux, & Bayle, 2007; O'Connor, Colder, & Hawk, 2004) with concurrent validity assessment finding SR positively related to extraversion (*r* =.48) and Gray’s Behavioral Activation Scale (*r* =.43) and SP related to neuroticism (*r* =.70) and Gray’s Behavioral Inhibition Scale (*r* = .50) (Sava & Sperneac, 2006).

The *Barratt Impulsiveness Scale* (BIS-11; Patton, Stanford, & Barratt, 1995) is a 30-item questionnaire designed to measure trait impulsivity. The BIS-11 items encompass three aspects of rash impulsiveness: non-planning impulsivity or the inability to plan and think carefully; 11 items, e.g.: “I plan tasks carefully” (reverse scored item); motor impulsivity or acting on the spur of the moment; 11 items, e.g.: “I do things without thinking”, “I buy things on impulse”; and attentional impulsivity or the inability to focus on the task at hand; 8 items, e.g. “I don’t pay attention.” Items are rated on a 4 point Likert-type scale ranging from rarely/never to almost always. Individual items are summed to create an overall score, with higher scores representing greater levels of rash impulsiveness. Research has established strong psychometric properties for the BIS-11 in both clinical (Barratt & Patton, 1983; Patton et al., 1995) and non-clinical populations (Spinella, 2007) with reliability coefficients ranging from α = .79 to α = .83 and temporal stability over one year of *r* = .60 (Luengo, Carrillo-de-la-Pena, & Otero, 1991). Convergent validity was established with neuropsychological measures sensitive to prefrontal cortical dysfunction (Spinella, 2004, 2007). Divergent validity was established with the Interpersonal Reactivity Index (*r* = -.20 to -.39) (Spinella, 2005).

Participants were also asked to complete *demographic questions* pertaining to age, gender, years of education, current age, illicit drug use, smoking and the age at which they started drinking weekly (Age of Onset of regular Drinking, or AOD).

Procedure

After obtaining ethical clearance from the Bond University Human Research Ethics Committee (BUHREC), recruitment of university students was made possible through poster advertisement and sign-up sheets which directed interested participants to a venue for supervised on-line completion of the survey. A commercial on-line survey program, Survey Monkey, was used to administer the questionnaires. Access to the questionnaires was only allowed after an explanatory statement had been read and online consent had been given. All participants were de-identified by the survey program which had been designed so that answers to the on-line survey could not be tracked to individual computer ISP addresses. The answers to questionnaires were automatically coded and collated into an anonymous data pool. A code word and number was used to link the online survey with performance tasks and maintain the anonymity of the participant.

Results

The data were analysed with the SPSS 16 Graduate Pack statistical package. Of the overall sample of 124 university students, 32.3% showed Low Risk drinking levels as defined by the AUDIT (n = 40), whereas 46.8% were defined as Hazardous drinkers (n = 58) and 21%% scored in the Harmful drinking range (n = 26). The mean drinking level for the entire sample was above the AUDIT score of 8, the Hazardous drinking cut-off (M = 10.75, SD = 6.43). Although these results were higher than the reported alcohol consumption by young adults according to the Australian Institute of Health and Welfare (2008), this was deemed to be consistent with the drinking patterns in university populations as reflected by other recent research (Hair & Hampson, 2006; Karam, Kypri, & Salamoun, 2007; Lyvers et al., 2009). A two-way chi-square goodness of fit test revealed that there was no relationship between gender and AUDIT risk group (Low Risk, Hazardous, Harmful), p = .64; i.e., AUDIT risk groups did not differ in gender breakdown. The mean ages of

the three AUDIT risk groups were an identical 20 years. The age at which the university students said they started drinking weekly (AOD) ranged from 12 to 21 years (M = 16.61 years, SD = 1.51) and varied between AUDIT risk groups as reported below.

Intercorrelations were conducted on variables AOD, AUDIT, FrSBe Apathy, Disinhibition and Executive Dysfunction, SPSRQ SP and SR, and BIS-11. These results are shown in Table 1. The important correlations to note in Table 1 are (1) the significant positive correlations of AUDIT with FrSBe Disinhibition, SR and BIS-11 scores, (2) the significant positive correlations of FrSBe Disinhibition and Executive Dysfunction with both SR and BIS-11, and (3) the significant negative relationships of AOD with AUDIT, BIS, and SR. These were all as expected based on previous work (Lyvers et al., 2009, 2011, 2012).

Comparison of Drinking Risk Groups on Trait Measures. A 3 (AUDIT risk group) x 2 (Gender) between-groups MANOVA was performed on AOD, FrSBe scales, BIS-11, and SPSRQ scales (SR and SP). Homogeneity assumptions were met according to the Box's M and Levene tests. With the use of Pillai's trace, the combined dependent variables were significantly affected by AUDIT risk level, $F(14, 226) = 3.38$, $p < .0001$, partial $\eta^2 = .17$, observed power = 1. Gender was also significant, $F(7, 112) = 2.37$, $p = .005$, partial $\eta^2 = .13$, observed power = .84. The interaction between Gender and AUDIT group was not significant, $p = .69$. When the unique effects of the AUDIT grouping variable on the dependent variables were considered, AOD, Disinhibition, Executive Dysfunction, BIS-11 and SR were all significant: AOD, $F(2, 118) = 7.21$, $p = .001$, partial $\eta^2 = .11$, observed power = .93; Disinhibition, $F(2, 118) = 14.38$, $p < .0001$, partial $\eta^2 = .20$, observed power = 1; Executive Dysfunction, $F(2, 118) = 4.16$, $p = .02$, partial $\eta^2 = .07$, observed power = .72; BIS-11, $F(2, 118) = 9.91$, $p < .0001$, partial $\eta^2 = .14$, observed power = .98; SR, $F(2, 118) = 6.62$, $p = .002$, partial $\eta^2 = .10$, observed power = .91. Tukey post-test ($p < .05$) revealed that the Harmful drinkers reported significantly earlier AOD and scored significantly higher on Disinhibition, Executive Dysfunction, BIS-11 and SR than the other two groups. The means for all variables demonstrating differences across the three AUDIT risk levels are shown in Table 2.

Univariate effects of gender were only significant for SR, $F(1, 118) = 6.38$, $p = .01$, partial $\eta^2 = .05$, observed power = .71; as in previous work (e.g., Lyvers et al, 2009, 2011), males ($M = 15.78$, $SD = 4.11$) scored significantly higher on SR than females ($M = 13.49$, $SD = 4.10$).

The finding that AOD was significantly related to variables differentiating Harmful drinkers from the lower AUDIT risk groups raised the issue of whether the observed differences between young adults who drink at Harmful levels and those who drink at less risky levels might reflect a developmental influence of longer duration of alcohol exposure on the late developing prefrontal cortex, rather than inherent traits that predisposed to both earlier and heavier alcohol use. In an attempt to differentiate between these two competing possibilities, an AUDIT risk group X gender MANCOVA was conducted on the trait measures, controlling for the duration of alcohol exposure as the covariate. AOD was subtracted from current age to provide an estimate of the duration of alcohol exposure in years from the start of regular consumption to the present time. Perhaps surprisingly, the results were virtually unchanged. The multivariate effect of AUDIT risk group was again significant according to Pillai's Trace, $F(12, 226) = 3.21$, $p < .0001$, partial $\eta^2 = .15$, observed power = .99. Univariate effects were again significant for Disinhibition, $F(2, 117) = 14.29$, $p < .0001$, partial $\eta^2 = .20$, observed power = 1; Executive Dysfunction, $F(2, 117) = 4.22$, $p = .02$, partial $\eta^2 = .07$, observed power = .73; BIS-11, $F(2, 117) = 7.50$, $p = .001$, partial $\eta^2 = .11$, observed power = .94; and SR, $F(2, 117) = 6.29$, $p = .003$, partial $\eta^2 = .10$, observed power = .89. When AOD itself was the covariate to control for possible effects of early regular drinking on the developing brain, the results were again virtually unchanged. The multivariate effect of AUDIT risk group was once again significant according to Pillai's Trace, $F(12, 226) = 2.78$, $p = .001$, partial $\eta^2 = .13$, observed power = .98. Univariate effects were once again significant for Disinhibition, $F(2, 117) = 12.27$, $p < .001$, partial $\eta^2 = .17$, observed power = .96; Executive Dysfunction, $F(2, 117) = 4.51$, $p = .01$, partial $\eta^2 = .07$, observed power = .76; BIS-11, $F(2, 117) = 6.03$, $p = .003$, partial $\eta^2 = .09$, observed power = .88; and SR, $F(2, 117) = 4.88$, $p = .009$, partial $\eta^2 = .08$, observed power = .80.

Thus taking into account both earlier age at onset of regular drinking and longer duration of alcohol exposure had no impact on the results.

Regression on AUDIT. To test the hypothesis that each trait measure would differentially predict the AUDIT as a continuous variable, a sequential hierarchical regression was employed with total AUDIT scores as the criterion. Predictor variables were entered in the order of age and gender (step 1); BIS-11 scores (step 2); SPSRQ SP and SR scores (step 3); and FrSBe Apathy, Disinhibition and Executive Dysfunction scores (step 4). At step 1 the model was significant, $F(2, 121) = 6.08$, $p = .003$. Age and gender accounted for 9% of the variance in AUDIT, $R = .30$, $R^2 = .09$. At step 2, BIS-11 significantly improved prediction, explaining an additional 14% of the variance, $R = .48$, $R^2 = .23$, *F change* $(1, 120) = 21.24$, $p < .0001$. At step 3, the addition of SP and SR explained a further 7% of the variance in AUDIT scores, $R = .55$, $R^2 = .30$, *F change* $(2, 118) = 6.38$, $p = .002$. At step 4, the FrSBe subscales only marginally improved prediction by an additional 4% of variance, $R = .59$, $R^2 = .34$, *F change* $(3, 115) = 2.25$, $p = .09$. Table 3 displays the unstandardised regression coefficients (B), standardised regression coefficients (β), *t* scores and the R^2 change for all variables at each step to demonstrate the additional proportion of the variance uniquely explained by each set of variables at their point of entry. BIS-11 and SR made the strongest unique contributions to explaining AUDIT scores when the variance due to the other trait variables was controlled, consistent with Dawe et al.'s (2004) model.

Path Analyses. To test the hypothesis that the influences of rash impulsiveness (as assessed by BIS-11 scores) and reward sensitivity (as assessed by SR scores) on drinking (as assessed by AUDIT scores) reflect dorsolateral prefrontal dysfunction (as assessed by FrSBe Executive Dysfunction) and orbitofrontal dysfunction (as assessed by FrSBe Disinhibition) respectively, path analyses were performed for both forms of impulsivity. With a significant relationship between SR and the AUDIT and BIS-11 and the AUDIT confirmed, three standard multiple regressions were performed in which variables were regressed on prior variables in the model. FrSBe Executive Dysfunction was regressed on BIS-11 and SR, and FrSBe Disinhibition was regressed on BIS-11

and SR followed by the regression of all prior variables (BIS-11, SR, Executive Dysfunction and Disinhibition) on AUDIT. Exploratory regression with a centred interaction term for both Executive Dysfunction and Disinhibition proved non-significant, ruling out moderation. Figure 1 shows that all the hypothesised paths were significant except the path from SR to Executive Dysfunction. The Sobel test statistic for the indirect pathway from BIS-11 to AUDIT through the intervening variable Executive Dysfunction was significant as predicted ($t = 2.57$, $p < .01$). The Sobel test statistic for the indirect pathway from SR to AUDIT through the intervening variable Disinhibition was also significant as predicted ($t = 1.87$, $p = .03$), as was the Sobel test statistic for the indirect pathway from BIS-11 to AUDIT through the intervening variable Disinhibition ($t = 2.36$, $p < .01$). The associations of BIS-11 (rash impulsiveness) and SR (reward sensitivity) with the AUDIT were thus mediated by Executive Dysfunction and Disinhibition for BIS-11 and by Disinhibition for SR.

Discussion

In the present study, young adults who were classed as Harmful drinkers on the basis of their AUDIT scores significantly differed from both Low Risk and Hazardous drinker groups on all dependent measures except SP, a trait dimension related to anxiety sensitivity and neuroticism (Sava & Sperneac, 2006), and Apathy, the FrSBe index of symptoms related to anterior cingulate dysfunction (Grace & Malloy, 2001). That is, Harmful drinkers reported earlier age of onset of regular drinking (AOD) and had higher scores on FrSBe Disinhibition, FrSBe Executive Dysfunction, BIS-11 and SR compared to the two lower risk groups according to the conservative Tukey post hoc test. By contrast, Hazardous and Low Risk drinkers did not differ on any of these measures by Tukey test.

Young adult university students have been characterised in previous studies by high levels of alcohol intake for both genders (e.g., Hair & Hampson, 2006; Hasking et al., 2011; Karam et al., 2007; Lyvers et al., 2009) which likely reflects the social context of university life – an influence that may account for the absence of any differences in gender composition between AUDIT risk groups in the present study. However, most university students do not drink at levels characterized

as Harmful by AUDIT criteria, hence individual difference variables are likely to distinguish those who choose to drink at Harmful levels at university from those who drink at less risky levels. The present findings suggest that rash impulsiveness and reward sensitivity are two such variables that may play crucial roles in promoting harmful levels of alcohol use in both genders, consistent with Dawe et al.'s (2004) model, and may reflect inherent dysfunction of frontal brain systems that predated alcohol exposure. The latter point was reinforced in the present study by the fact that all differences between Harmful drinkers and the lower risk groups remained highly significant even after statistically controlling for longer duration of alcohol exposure and earlier onset age of regular drinking. The virtually identical results would seem to render unlikely the possibility that earlier or longer alcohol exposure had adversely affected the developing adolescent brain in ways that caused the Harmful drinkers in this sample to differ from the lower risk groups on rash impulsiveness, reward sensitivity and FrSBe frontal lobe indices. On the other hand, intensity of past alcohol use was not assessed in the present study, so this possibility cannot be ruled out by the present findings.

The results of path analyses generally supported our hypothesis according to which the influence of rash impulsiveness on drinking may primarily reflect dorsolateral prefrontal dysfunction (as indirectly indexed here by FrSBe Executive Dysfunction scores), whereas the influence of reward sensitivity on drinking may primarily reflect orbitofrontal dysfunction (as indirectly indexed here by FrSBe Disinhibition scores). The BIS-11 measure of rash impulsiveness encompasses problems with attentional focus and foresight, aspects of executive control that have been linked to dorsolateral prefrontal functioning (Alvarez & Emory, 2006; Posner & Fan, 2008; Rothbart & Rueda, 2005), whereas interactions between the orbitofrontal cortex and dopaminergic reward system have been implicated in risky reward-driven behaviors (Galvan et al., 2006) including problematic drinking (Modell & Mountz, 1995). Thus the rash impulsiveness dimension of Dawe et al.'s (2004) model may reflect the functioning of top-down executive control systems, whereas the reward sensitivity (SR) dimension may reflect bottom-up subcortical-orbitofrontal interactions (Galvan et al.). However in the present study the path from the BIS-11 index of rash

impulsiveness to AUDIT through the mediator Disinhibition was also significant, consistent with Dawe et al. (2004) who proposed that the influence of rash impulsiveness on substance abuse reflects orbitofrontal dysfunction. Taken together, present findings thus tentatively suggest that the influence of rash impulsiveness on drinking potentially reflects dysfunction in both dorsolateral prefrontal and orbitofrontal systems, supporting earlier work on the involvement of frontal systems in addictions (see Lyvers, 2000, for a review). Further, although Dawe et al. (2004) originally proposed that reward sensitivity primarily reflects the activity of the subcortical dopaminergic reward system, the present findings tentatively implicate orbitofrontal involvement, consistent with the known interactions between dopaminergic and orbitofrontal systems (Volkow et al., 2011). Overall the present results support Dawe et al.'s notion of two distinct forms of impulsivity that influence substance use patterns in young adults. In addition, self-reported age at onset of regular drinking (AOD) was significantly negatively related to BIS-11 and SR scores, consistent with Dawe et al.'s model and other recent findings (Gullo et al., 2011; Lyvers et al., 2011) on factors pertaining to an early onset of substance use in adolescence.

There was no direct measure of frontal lobe functioning in the present study, which instead relied on the self-report FrSBe scales as indirect indices of dysfunction in frontal systems. The FrSBe scales were designed to detect changes in everyday functioning following frontal brain injury, and clinical studies have supported their validity (e.g., Chiaravalloti & DeLuca, 2003; Stout, Ready et al., 2003; Stout, Wyman et al., 2003; Velligan et al., 2002). Nevertheless our conclusions can only be regarded as tentative until there is direct evidence of dysfunction in the dorsolateral prefrontal and orbitofrontal brain regions of young adult university students who drink at harmful levels. In this regard functional MRI research has indicated that low frontal activation during an inhibition (go/no-go) task in adolescents prior to alcohol exposure predicts heavy drinking one year later (Norman et al., 2011) and is associated with familial alcoholism (Schweinsburg et al., 2004). Lower frontoparietal connectivity was also found to be associated with familial alcoholism in substance-naive youth in another recent functional MRI study (Wetherill et al., 2011). These brain

imaging findings are consistent with the general notion that some of the functional frontal lobe deficits associated with chronic alcoholism may reflect pre-alcohol traits rather than effects of chronic heavy drinking or adolescent alcohol exposure on the brain. However, the multiple frontal areas implicated in these studies overlap considerably and are not limited to the specific subregions said to be tapped by the FrSBe subscales. An additional issue with the present study concerns the cross-sectional nature of the present sample. Ideally an ambitious longitudinal study is needed to fully disentangle the relative contributions of pre-alcohol traits, early alcohol exposure during adolescence and other factors that may contribute to harmful alcohol use by young adults at university.

In any case the present findings are consistent with the notion of two forms of impulsivity that promote harmful alcohol use by young adults and that reflect frontal lobe functioning as inherent traits that predate alcohol exposure. Inherently poorer frontal lobe functioning could be the result of genetic factors underlying trait impulsiveness (Bevilacqua et al., 2010; Swann, Lijffijt & Scott, 2009) or alternatively effects of alcohol exposure *in utero* on the developing brain (Archibald, Fennema-Notestine, Gamst, Riley, Mattson & Jernigan, 2001). A recent study (Lyvers, Onuoha, Thorberg & Samios, 2012) found that children of alcoholic parents as defined by the Children of Alcoholics Screening Test (CAST; Jones, 1991) scored significantly higher on the FrSBe Disinhibition and Executive Dysfunction scales than did children of non-alcoholic parents; however the authors argued that, as alcoholism is far more common in men than in women, alcohol exposure *in utero* was unlikely to have been the primary basis of the observed group differences. Future research examining genetic influences on brain functioning and impulsive behavior will likely yield fascinating and informative results concerning the predisposition to early and risky substance use and the underlying neural mechanisms involved.

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Table 1

Intercorrelations among trait measures AUDIT (AUD), BIS-11 (BIS), FrSBe Apathy (Ap), FrSBe Disinhibition (Dis), FrSBe Executive Dysfunction (Exec), and SPSRQ SP and SR scales as well as age at onset of weekly drinking (AOD).

	AOD	AUD	BIS	Ap	Dis	Exec	SP	SR
AUD	-.390**							
BIS	-.319**	.359**						
Ap	.113	.006	.303**					
Dis	-.169	.380**	.589**	.319**				
Exec	-.002	.117	.682**	.634**	.632**			
SP	.102	.040	.215*	.556**	.127	.420**		
SR	-.187*	.438**	.359**	-.018	.523**	.326**	.156	

**p <.01 *p <.05

Table 2

Age of Onset of Drinking, Disinhibition, Executive Dysfunction, Sensitivity to Reward and BIS-11 Rash Impulsiveness as a Function of AUDIT Risk Levels of Drinking.

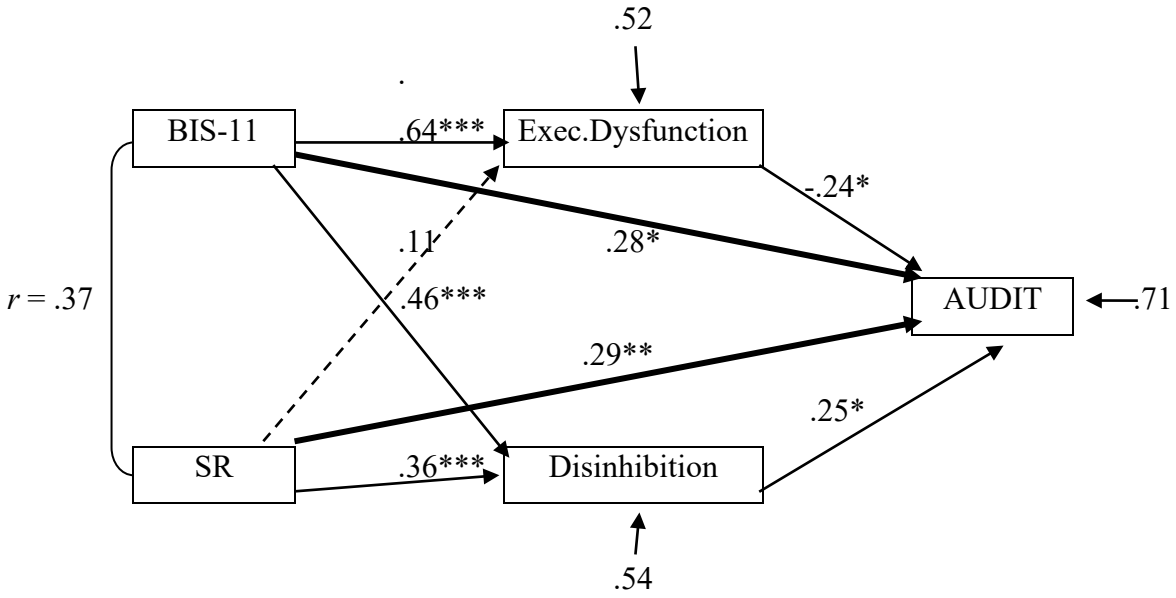
Variable	AUDIT	Mean	Std Deviation	N
Age of Drinking Onset	Low risk	17.02	1.42	40
	Hazardous	16.74	1.43	58
	Harmful	15.69	1.49	26
Disinhibition	Low risk	29.83	6.32	40
	Hazardous	29.97	5.90	58
	Harmful	37.85	7.68	26
Executive Dysfunction	Low risk	33.97	7.98	40
	Hazardous	35.62	7.86	58
	Harmful	40.62	7.62	26
Sensitivity to Reward	Low risk	12.95	4.16	40
	Hazardous	13.79	3.93	58
	Harmful	17.15	3.68	26
BIS-11	Low risk	60.05	10.77	40
	Hazardous	63.45	10.16	58
	Harmful	72.46	10.78	26

Table 3

Regression Coefficients of Trait Measures on the AUDIT.

	Variable	B	β	*t*	R^2 change
Step 1	Age	-.34	-.09	-1.04	
	Gender	3.95	.29	3.33***	.09
Step 2	Age	-.41	-.11	-1.37	.
	Gender	3.80	.28	3.46***	
	BIS-11	.21	.37	4.61***	.14
Step 3	Age	-.35	-.09	-1.19	.
	Gender	2.92	.21	2.67**	
	BIS-11	.16	.28	3.32***	
	SP	-.12	-.09	-1.19	
	SR	.45	.30	3.44***	.08
Step 4	Age	-.25	-.07	-.88	.
	Gender	3.32	.24	3.00**	
	BIS-11	.20	.34	3.08**	
	SP	.01	.01	.11	
	SR	.33	.22	2.16*	
	Apathy	-.04	-.04	-.35	
	Disinhibition	.22	.25	2.08*	
	Executive Dysfunction	-.22	-.28	-1.91	.04

***p <.001 **p <.01 *p <.05



***p <.001 **p <.01 *p <.05 N = 124

Figure 1. Path coefficients for the associations of BIS-11 and SR with AUDIT, mediated by FrSBe Disinhibition and Executive Dysfunction. Standardised betas are shown for all paths.