Foundation for Alcohol Research & Education

Frontal Lobe Functioning in Relation to Risky or Harmful Alcohol use by Young Adults

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BRINGING AMBITION TO LIFE

About the Foundation for Alcohol Research and Education

The Foundation for Alcohol Research and Education (FARE) is an independent charitable organisation working to prevent the harmful use of alcohol in Australia. Our mission is to help Australia change the way it drinks by:

- helping communities to prevent and reduce alcohol-related harms
- building the case for alcohol policy reform and
- engaging Australians in conversations about our drinking culture.

Over the last ten years FARE has invested more than $115 million, helped 750 organisations and funded over 1,400 projects addressing the harms caused by alcohol misuse.

FARE is guided by the World Health Organization's Global Strategy to Reduce the Harmful Use of Alcohol1 for addressing alcohol-related harms through population-based strategies, problem-directed policies, and direct interventions.

Summary of findings

Initial data collected from 124 university students of both genders aged 18-26 years were consistent with expectations based on previous research (Lyvers et al., 2009, 2011) for the trait measures of frontal lobe related functions. The research found:

- drinking level as assessed by the Alcohol Use Disorders Identification Test (AUDIT), trait impulsivity as assessed by the Barratt Impulsiveness Scale (BIS-11), and Frontal Systems Behavior Scale (FrSBe) indices of frontal lobe dysfunction were significantly elevated in offspring of alcoholics as determined by the Children of Alcoholics Screening Test (CAST-6).

- FrSBe Disinhibition as well as the BIS-11 measure of impulsivity and the Sensitivity to Reward (SR) scale of the Sensitivity to Punishment and Sensitivity to Reward Questionnaire (SPSRQ) were all significantly positively related to harmful drinking as assessed by the AUDIT, and were inversely related to age at which respondents began drinking on a weekly basis.

In other words, crucial trait measures of poor impulse control, poor self-regulation and high reward-driven behaviour were linked to harmful levels of drinking and earlier drinking onset as well as parental alcoholism in this young adult university student sample.

However, contrary to predictions the three neuropsychological measures of executive cognitive performance, using tests which were designed to assess behavioural changes reflecting frontal lobe damage in brain-injured patients (described below), were unrelated to AUDIT scores or age at onset of weekly drinking (AOD) in the university sample. Because university students are selected for university on the basis of high problem-solving skills, we suspected that a ceiling effect might account for this failure to find the expected relationships. Therefore we recruited and tested a further 47 young adults who were *not* university students from the general community, and the expected findings emerged.

- Poorer neuropsychological test performance on the Delis-Kaplan Executive Function System (D-KEFS) Tower Test was significantly related to harmful drinking as measured by AUDIT in the community sample of young adults.

- Poorer neuropsychological test performance on the Iowa Gambling Task (IGT) was also significantly related to harmful drinking in this group.

- Poorer performance on the Wisconsin Card Sorting Test (WCST) was however only marginally related to harmful drinking.

In other words, for two out of three crucial and well-validated problem-solving tests of frontal lobe executive functioning, young adults who drank at a harmful level by AUDIT criteria performed significantly worse than those who drank at lower levels, and for the third test the difference came close to being significant.

These findings in the community sample of young adult social drinkers are mostly consistent with our hypothesis that inherent deficiencies of frontal lobe related executive cognitive functioning promote risky or harmful alcohol consumption.

These research findings will inform both prevention and treatment efforts aimed at harmful alcohol consumption.

Contents

Acronyms

ANOVA	analysis of variance
AOD	Age and Onset of weekly Drinking
AUDIT	Alcohol Use Disorders Identification Test
BIS-11	Barratt Impulsiveness Scale
BUHREC	Bond University Human Research Ethics Committee
CAST-6	Children of Alcoholics Screening Test
COA	Children of Alcoholics
D-KEFS	Delis-Kaplan Executive Function System
FrSBe	Frontal Systems Behavior Scale
IGT	Iowa Gambling Task
MANCOVA	multivariate analysis of covariance
MANOVA	multivariate analysis of variance
PE	Perseverative Error
PEBL	Psychological Experiment Building Language
SP	Sensitivity to Punishment (SPSRQ measure)
SR	Sensitivity to Reward (SPSRQ measure)
SPSRQ	Sensitivity to Punishment and Sensitivity to Reward Questionnaire
WCST	Wisconsin Card Sorting Test

Background

This research project tested the hypothesis that inherent deficiencies of frontal lobe functioning promote alcohol-related risk in young adults. Deficits of frontal lobe functioning and associated cognitive and behavioural impairments are well known correlates of chronic alcoholism and are commonly interpreted as reflecting cumulative effects of high alcohol exposure (Lyvers, 2000). However, such associations may alternatively reflect traits predating alcohol exposure which predispose to risky or problematic drinking in young people, and which may also be associated with familial alcoholism history. For example, university students have a higher prevalence of alcohol use as well as high risk drinking than do non-students of the same age, yet not all students drink at high or risky levels, suggesting that excessive alcohol use by young adults may be influenced to some extent by underlying trait factors in addition to social environmental factors.

In an exploratory investigation, Lyvers, Czerczyk, Follent, and Lodge (2009) recruited 60 university undergraduates aged 18-25 years who were administered the Alcohol Use Disorders Identification Test (AUDIT), Frontal Systems Behavior Scale (FrSBe), and Sensitivity to Punishment and Sensitivity to Reward Questionnaire (SPSRQ). All participants reported they were at least occasional drinkers who rarely or never used illicit drugs and had no reported history of head injury or neurological problems. All were sober at time of testing as confirmed by alcohol breath test. AUDIT scores were positively correlated with FrSBe Disinhibition scores (an index of orbitofrontal cortex dysfunction) and SPSRQ Reward Sensitivity (SR) scores (which assesses the degree to which behaviour is influenced by the motivation to achieve gains). These measures were negatively correlated with age at onset of regular (weekly) drinking (AOD), such that higher Disinhibition and SR scores were associated with younger age at onset of regular alcohol use. High risk drinkers (as defined by AUDIT) of both genders had higher FrSBe Disinhibition and SPSRQ SR scores compared to low risk drinkers. The findings indicated that even in a highly selected subset of young adults – undergraduates attending a prestigious private university – associations are present between indices of frontal lobe functioning, reward sensitivity and alcohol-related risk, most likely reflecting traits that predispose to heavier drinking.

A subsequent investigation by Lyvers, Duff and Hasking (2011) extended the findings of Lyvers et al. (2009) by using a larger sample of 132 participants, including non-students recruited from the local community, and adding a screening test for familial alcoholism (the Children of Alcoholics Screening Test, CAST-6) as well as a measure of trait impulsivity (Barratt Impulsiveness Scale, BIS-11). SR and Disinhibition again differentiated Low Risk from Risky (Hazardous or Harmful) drinkers as defined by the AUDIT and Impulsivity (as measured by the BIS-11) and the Executive Dysfunction index of the FrSBe both differentiated Harmful drinkers from Low Risk and Hazardous drinkers. Both reward sensitivity and parental alcoholism predicted younger AOD, suggesting that greater inherent responsiveness to rewarding stimuli promotes both earlier and heavier alcohol use by youth. In contrast, sensitivity to punishment (SP) predicted later AOD, suggesting that trait anxiety may help delay the initiation of regular alcohol use in young people, perhaps due to health concerns and/or a desire to maintain self-control. CAST-6 scores were positively related to AUDIT, FrSBe Disinhibition and Executive Dysfunction, SR and BIS-11, and were negatively related to AOD (such that higher CAST-6 scores were associated with younger age of onset of weekly alcohol use). These associations were consistent with the hypothesis that inherently poorer frontal lobe functioning, and higher

sensitivity to reward and rash impulsivity, all constitute heritable risk factors for heavier or problematic alcohol consumption in young people.

Self-report is subject to various biases and distortions, making self-report measures inherently less desirable than laboratory procedures such as neuropsychological testing in evaluating the functional integrity of specific brain regions.

The present project thus extended the recent work described above by administering multiple validated neuropsychological tests of frontal lobe functioning to young adults aged 18-26 years, in order to determine whether deficiencies in frontal lobe function, as indicated by neuropsychological testing, are related to risky or harmful alcohol use by young adults as assessed by the AUDIT.

Methods

PARTICIPANTS

The investigation initially recruited 124 young adult university students of both genders, aged 18-26 years, from Bond University. The researchers excluded anyone who was intoxicated at time of testing (as shown by a breath test), had recently suffered from a head injury, used illicit drugs monthly or more often, did not drink alcoholic beverages, or was under the legal drinking age of 18.

Further data were subsequently collected from 47 community non-student young adult social drinkers aged 18-26 years in southeast Queensland and NSW (Sydney), who were recruited via advertisements in local newspapers. The same exclusion criteria applied.

Participants were paid $30 for their time.

MATERIALS

The following tests were administered to all participants:

The *Tower Test* of the Delis-Kaplan Executive Function System (D–KEFS) (Delis, Kaplan, & Kramer, 2001) is a measure of executive functions, specifically assessing rule learning, spatial planning, inhibition of perseverative responding, inhibition of impulsive responding, and the ability to establish and maintain the instructional set (Delis et al., 2001). The D-KEFS Tower Test consists of a board with three vertical pegs, and five disks that vary in size from small to large. There are nine items in the D-KEFS Tower Test, and for each item a certain number of the disks are placed in a predetermined starting position, and a picture of the tower to be built is displayed to the examinee (i.e. showing the ending position of the disks). To complete each item the examinee is asked to build the target tower in the fewest number of moves possible by moving the disks across the three pegs. The examinee is required to follow two rules, including 1) move only one disk at a time using only one hand to move each disk, and 2) do not place a larger disk on top of a smaller disk. As the examinee progresses through to each item, the difficulty of completing the task increases, and therefore the minimum number of moves required to complete each target tower varies from one move (Item 1) to 26 moves (Item 9). Regarding scoring, a discontinue rule is followed based on a time limit, meaning that after three consecutive item failures the test is no longer administered.

Items are discontinued based on the following time limits: items 1 to 3 at 30 seconds, item 4 at 60 seconds, items 5 to 6 at 120 seconds, item 7 at 180 seconds, and items 8 to 9 at 240 seconds. A total achievement score is calculated by summing the total number of moves for all items administered, with bonus points allocated when the target tower is built within the time limit and within the minimum number of moves possible. A Total Achievement raw score can range from 0 (all items failed) to 30, which is then converted to a scaled score based on the age group of the examinee. The Total Achievement score is an overall measure of performance on the D-KEFS Tower Test.

The *Iowa Gambling Task* (IGT; Bechara, Tranel, Damasio, & Damasio, 1996) utilised in this study is a computerised version provided by the Psychological Experiment Building Language (PEBL) online psychological test battery, version 0.09 (Mueller, 2008). The penalty/reward structure in this version is identical to that specified by Bechara et al. with 40 cards per deck, and an identical reward structure. The IGT requires decision making and includes uncertainty, risk and evaluation of reward-punishment contingencies associated with four decks of cards. The participant is required to select repeatedly from four decks of cards (A-D). Each card selection can result in either the winning or losing of money. Decks A and B are characterised by large wins and occasional large punishments resulting in an overall loss of money. Decks A and B, therefore, are classified as risky or disadvantaged decks. Decks C and D are the advantaged decks and are associated with smaller wins and smaller losses so that repeated choices result in a profit outcome. Deck A differs from B and Deck C differs from D in the number of trials over which the losses are distributed: A and C have five smaller loss cards for every ten cards; B and D have one larger loss card for every ten cards. The main dependent variable used on this task was the difference between the number of advantageous and disadvantageous choices [(C+D) − (A+B)] on the last two of five blocks of 20 trials of the task. Research has demonstrated that for the later blocks a greater number of choices from decks A and B is associated with frontal lobe deficits (Bechara & Martin, 2004; Verdejo-García & Pérez-García, 2007). Poor IGT performance is observed in patients with damage to the dorsolateral prefrontal cortex and in patients who exhibit poor working memory and executive function impairments on the Wisconsin Card Sorting Task (Bechara & Martin, 2004).

The *Wisconsin Card Sorting Test -64* (Kongs, Thompson, Iverson & Heaton, 2000) is the abbreviated form of the standard Wisconsin Card Sorting Test (WCST; Heaton, 1981), and is designed to assess executive cognitive functioning (i.e. cognitive flexibility, abstract reasoning skills and planning). For ease of administration and to minimise procedural and scoring errors, the computerised version of the test was administered using PEBL. Tien et al. (1996) reported that there was minimal, if any, difference on PE and NPE measures between the original card version and the computerised version of the WCST. Successful performance of the WCST requires the participant to utilise the feedback provided by the program to continuously identify and sort the stimulus cards in accordance to the correct sorting rule, either colour, shape or number; the sorting principle varies from one set of trials to the next. The WCST takes approximately 10 minutes to administer. For the purposes of the present study, the percentage of Perseverative Errors (PEs) out of the total number of trials was the measure of interest. PE refers to the number of times the participant persisted in making an incorrect sorting choice that was previously correct, a type of WCST error that is specifically sensitive to frontal lobe damage.

The *Frontal Systems Behavior Scale* (FrSBe; Grace & Malloy, 2001) was designed to measure everyday behaviour associated with damage to the frontal lobes. The original form of the scale asks

for pre- and post-injury ratings from the participant and others, but as the present study did not examine brain-injured patients participants were asked to provide only current self-ratings as in previous work (Lyvers et al., 2009; Spinella, 2003; Verdejo-Garcia et al., 2006). The FrSBe provides indices of three distinct frontal lobe behavioural syndromes as manifested in everyday life: Apathy (anterior cingulate dysfunction), Disinhibition (orbitofrontal dysfunction), and Executive Dysfunction (dorsolateral prefrontal dysfunction). There are 46 items with each item rated on a 5-point Likert scale. Scoring yields scores on the three subscales of Apathy (14 items), Disinhibition (15 items), and Executive Dysfunction (17 items) as well as a total score.

The *Alcohol Use Disorders Identification Test* (AUDIT; Saunders et al., 1993) is a self-report measure widely used in screening for harmful and hazardous drinking in adolescent and adult populations. The AUDIT is comprised of 10 items answered on Likert scales. The AUDIT total score is devised by the summation of a participant's responses to all 10 items; possible scores range from 0-40. A score of 0-7 is indicative of 'Low Risk' a score of 8-15 suggests the respondent is drinking at a 'Hazardous' level, and a score of 16 or higher suggests the respondent is drinking at a 'Harmful' level.

The *Sensitivity to Punishment and Sensitivity to Reward Questionnaire* (SPSRQ; Torrubia et al., 2001) is a self-report measure assessing a participant's appetitive (SR) and aversive (SP) motivational system functioning levels in adolescent and adults. The SPSRQ is comprised of 48 items, of which 24 assess SR and 24 assess SP. Participants respond to all questions with either a yes or no response, with affirmative responses being summated to form SR and SP scores. SR tends to be elevated in patients with frontal lobe damage as well as in substance abusers.

The *Children of Alcoholics Screening Test* (CAST-6; Hodgkins et al., 1993) is a self-report measure that is widely used to screen for parental history of alcoholism.

The *Barratt Impulsiveness Scale* (BIS-11; Patton et al., 1995) is a widely used self-report index of rash impulsivity, a trait strongly linked to frontal lobe dysfunction.

The *Demographics Questionnaire* is comprised of 14 items. Questions pertain to age at onset of regular (weekly) drinking, current age, gender, student/non-student status, education level, illicit drug use history, and smoking history.

PROCEDURES FOR GAINING ETHICS CLEARANCE

The Bond University Human Research Ethics Committee (BUHREC) is committed to protecting the welfare and rights of human participants in research conducted by members of the Bond University community. Research by Bond University staff and students must be conducted in accordance with the National Health and Medical Research Council *National Statement on Ethical Conduct in Research Involving Human Participants* (1999). BUHREC promotes ethically, socially, scientifically and legally responsible research activity among all members of Bond University. University policy requires that approval from BUHREC must be sought and obtained *before* research commences. Bond University has an Ethics Policy and application process whereby researchers are required to complete an application form which is assessed by two reviewers who present their comments on the application to the BUHREC. The summary application is sent to all Committee members. Principal researchers are invited to attend the meeting to address any issues or concerns raised by the Committee. Approval, Conditional Approval or Not Approved will be conveyed to the applicant

within one week of the BUHREC meeting with specific recommendations where relevant. In accordance with AER University policy, ethics clearance for this research project was obtained prior to commencement of the project.

DATA ANALYSES

Initially correlational analyses were undertaken to determine relationships among all variables. Multivariate analyses of variance (MANOVA) were then used to compare Low Risk, Hazardous and Harmful drinker groups (as defined by AUDIT scores) on all measures. Regressions and path analyses were also undertaken for specific trait measures predictive of AUDIT as reported below.

DATA COLLECTION METHODS

After reading and signing the informed consent form, each participant was de-identified and given a unique code which was not traceable to their name as per the ethical requirement of anonymity. Participants completed a questionnaire battery comprised of the AUDIT, SPSRQ, BIS-11, FrSBe and demographic questions before undertaking the three selected neuropsychological tests for frontal lobe executive functions. Participants were given a breath test for alcohol in order to exclude any intoxicated participants from the study. After data collection, participants were paid $30 for their time.

STATISTICAL ANALYSES

Analyses included bivariate correlations, between-subjects multivariate analyses of variance (MANOVA), standard multiple regressions and pathway analyses.

DEMOGRAPHIC CORRELATES OF RISKY DRINKING IN THE UNIVERSITY SAMPLE

In the university sample of 84 females and 40 males aged 18-26 years, 32.2% showed Low Risk drinking levels as defined by the AUDIT (n=39), whereas 46.8% were defined as Hazardous drinkers (n=58) and 19.8% scored in the Harmful range (n=24). Although these results were higher than the patterns of alcohol consumption for individuals aged between 14 and 24 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 et al., 2007).

With α set at .05, a two-way chi-square goodness of fit test revealed no significant relationship between gender and AUDIT category group (Low Risk, Hazardous, Harmful). That is, men and women did not differ in the level of risky drinking by AUDIT criteria. The mean drinking level for the entire sample was above the AUDIT score of 8, the hazardous drinking cut-off (M=10.70, SD=6.39). The mean ages for the three AUDIT categories were analysed with a one-way analysis of variance (ANOVA) using α=.05 and the result was non-significant. That is, ages were not significantly different between Low Risk, Hazardous and Harmful drinkers in this sample. The age at which university students started drinking weekly ranged from 12 to 21 years (M=16.64, SD=1.51) in this sample.

Of the 124 university participants only 23 were classified as children of alcoholics by the CAST-6. The mean drinking level for children of alcoholics was above the AUDIT cut-off score of 8 indicating Hazardous drinking (*M*=12.26) as was the mean for the other participants (*M*=10.34).

CORRELATIONS OF DRINKING MEASURES WITH TRAIT MEASURES

Intercorrelations were conducted on AOD, CAST-6, AUDIT, FrSBe Apathy, Disinhibition and Executive Dysfunction, SPSRQ SP and SR, and BIS-11. These are shown in Table 1. The important correlations to note in Table 1 are:

- the significant positive correlations of AUDIT with FrSBe Disinhibition, SR and BIS-11 scores, and
- the significant negative relationships between AOD and AUDIT, BIS, and SR.

These were all as per predictions.

TABLE 1: INTERCORRELATIONS AMONG TRAIT MEASURES.

	AOD	CAST-6	AUD	BIS	Ap	Dis	Exec	SP	SR
CAST-6	-.014								
AUD	-.390**	.120							
BIS	-.319**	.138	.359**						
Ap	.113	.133	.006	.303**					
Dis	-.169	.235**	.380**	.589**	.319**				
Exec	-.002	.168	.117	.682**	.634**	.632**			
SP	.102	.086	.040	.215*	.556**	.127	.420**		
SR	-.187*	.079	.438**	.359**	-.018	.523**	.326**	.156	

** p <.01 * p <.05

COMPARISON OF DRINKING RISK GROUPS ON TRAIT MEASURES.

A 3 (AUDIT risk group) x 2 (Gender) between-groups MANOVA was performed on AOD, CAST-6, FrSBe measures, BIS-11, and SPSRQ measures as well as the neuropsychological test scores. The assumption of homogeneity of variance-covariance matrices (Box's M) was not significant, indicating no violation. As the Levene's test was significant for two of the dependent variables, CAST-6 and SP, a more stringent alpha level was set (p < .01) (Tabachnik & Fidell, 2007). With the use of Pillai's trace, the combined dependent variables were significantly affected by AUDIT risk level, $F(20,214)=2.24$, $p<.01$, partial $\eta^2=.17$, observed power=.99. The interaction between Gender and the AUDIT was non-significant.

When the unique effects of the AUDIT grouping variable on the dependent variables were considered, only AOD, Disinhibition, BIS-11 and SR were significant: AOD, $F(2, 120)=6.35$, $p<.01$, partial $\eta^2=.09$, observed power=.89; Disinhibition, $F(2, 120)=11.19$, $p<.001$, partial $\eta^2=.16$, observed

power=.99; BIS-11, $F(2, 120)=8.62$, $p<.001$, partial $\eta^2=.13$, observed power=.97; SR, $F(2, 120)=5.36$, $p<.01$, partial $\eta^2=.09$, observed power=.83.

Tukey post-test ($p<.05$) revealed that the Harmful drinkers reported significantly earlier AOD and had significantly higher scores on Disinhibition, BIS-11 and SR than the other two groups. Further, Harmful drinkers scored significantly higher on FrSBe Executive Dysfunction than did Low Risk drinkers.

The means for all variables demonstrating differences across the three AUDIT risk levels are shown in Table 2, below.

TABLE 2: AGE OF ONSET OF DRINKING, DISINHIBITION, EXECUTIVE DYSFUNCTION, SENSITIVITY TO REWARD AND RASH IMPULSIVITY AS A FUNCTION OF AUDIT RISK LEVELS OF DRINKING.

Variable	AUDIT	Mean	Std Deviation	*n*
Age of Drinking Onset	Low risk	17.05	1.432	39
	Hazardous	16.74	1.433	58
	Harmful	15.75	1.511	24
Disinhibition	Low risk	30.08	6.192	39
	Hazardous	29.97	5.897	58
	Harmful	37.17	7.557	24
Executive Dysfunction	Low risk	34.18	7.980	39
	Hazardous	35.62	7.856	58
	Harmful	39.71	7.190	24
Sensitivity to Reward	Low risk	12.92	4.208	39
	Hazardous	13.79	3.933	58
	Harmful	16.96	3.747	24
Impulsivity	Low risk	60.15	10.893	39
	Hazardous	63.45	10.159	58
	Harmful	72.29	11.141	24

Note: Harmful drinkers significantly differed from Low Risk drinkers on all measures above, and also differed significantly from Hazardous drinkers on Age of Onset, Disinhibition, SR and BIS-11.

TRAIT MEASURES PREDICTING DRINKING LEVELS

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* $(1, 120) = 21.24$, $p = .000$.
- 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 (overleaf) 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.

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

TRAIT MEASURES DIFFERENTIATING CHILDREN OF ALCOHOLICS

The hypothesis that parental alcoholism would be associated with elevated scores on FrSBe, BIS-11 and SR trait measures as well as AUDIT and earlier AOD was investigated via a 2 (CAST-6 category) X Gender between-groups MANOVA. The assumption of homogeneity of variance-covariance matrices (Box's M) was not significant, indicating no violation. With the use of Pillai's trace, the combined dependent variables were marginally significantly affected by CAST-6 category, p=.09. When the unique effects of the CAST-6 grouping variable on each of the dependent variables were considered individually, children of alcoholics scored significantly higher than children of non-alcoholic parents on AUDIT, F(1, 108)=5.35, p<.05, partial η^2=.05, observed power=.63; BIS-11, F(1, 108)=4.82, p<.05,

partial η^2=.05, observed power=.59; Disinhibition, $F(1, 108)=5.70$, $p<.05$, partial η^2=.05, observed power=.66; and Executive Dysfunction, $F(1, 108)=3.92$, $p<.05$, partial η^2=.04, observed power=.50. The means for significant variables across the two CAST-6 categories are shown in Table 4.

TABLE 4: SIGNIFICANT DIFFERENCES BETWEEN CHILDREN OF ALCOHOLICS (COA) AND NON-COAS.

Variable	CAST-6 category	Mean (SD)
AUDIT	COA	12.09 (8.21)
	Non-COA	10.54 (5.94)
BIS-11	COA	67.05 (13.22)
	Non-COA	63.62 (10.98)
Disinhibition	COA	34.05 (8.35)
	Non-COA	30.77 (6.56)
Executive Dysfunction	COA	38.91 (9.67)
	Non-COA	35.42 (7.62)

TRAIT MEASURES PREDICTING AGE OF ONSET OF DRINKING

Trait variables were examined as predictors of an early onset age of weekly drinking (AOD). Again a sequential hierarchical multiple regression was employed. Predictor variables were entered in the order of age and gender (step 1); BIS-11 (the rash impulsivity measure) (step 2); SPSRQ SP and SR scores (step 3); and FrSBe Apathy, Disinhibition and Executive Dysfunction (step 4).

- At step 1 the model was not significant, $F(2, 121)=1.36$, $p=.26$. Age and gender accounted for only 2% of the variance in AOD, $R=.15$, $R^2=.02$.
- At step 2, BIS-11 significantly improved prediction, explaining an additional 11% of the variance, $R=.36$, $R^2=.13$, *F change*$(1, 120)=15.09$, $p=.000$.
- At step 3, the addition of SP and SR only marginally contributed to prediction, explaining only a further 4% of the variance in AOD, $R=.41$, $R^2 = .17$, *F change*$(2, 118)=2.74$, $p=.07$.
- At step 4, the FrSBe subscales significantly improved prediction by an additional 7% of variance, $R=.49$, $R^2=.24$, *F change*$(3, 115)=3.51$, $p=.02$.

Table 5 (overleaf) shows the unstandardised regression coefficients (B), standardised regression coefficients (β), *t* scores and the R^2 change for each predictor at each step. BIS-11 made the strongest unique contribution to explaining AOD scores when the variance due to the other trait variables was controlled.

TABLE 5: REGRESSION COEFFICIENTS OF TRAIT MEASURES ON AGE AT ONSET OF WEEKLY DRINKING (AOD).

	Variable	B	β	t	R^2 change
Step 1	Age	.08	.09	.96	
	Gender	-.39	-.12	-1.34	.02
Step 2	Age	.09	.11	1.23	
	Gender	-.36	-.11	-1.30	
	BIS-11	-.04	-.33	-3.86***	.11
Step 3	Age	.10	.12	1.38	
	Gender	-.35	-.11	-1.25	
	BIS-11	-.05	-.35	-3.75***	
	SP	.06	.20	2.25*	
	SR	-.03	-.08	-.80	.04
Step 4	Age	.09	.10	1.26	
	Gender	-.53	-.16	-1.88	
	BIS-11	-.08	-.57	-4.77***	
	SP	-.02	.07	.70	
	SR	-.02	-.07	-.62	
	Apathy	.01	.02	.18	
	Disinhibition	-.01	-.07	-.53	
	Executive Dysfunction	.08	.41	2.63**	.07

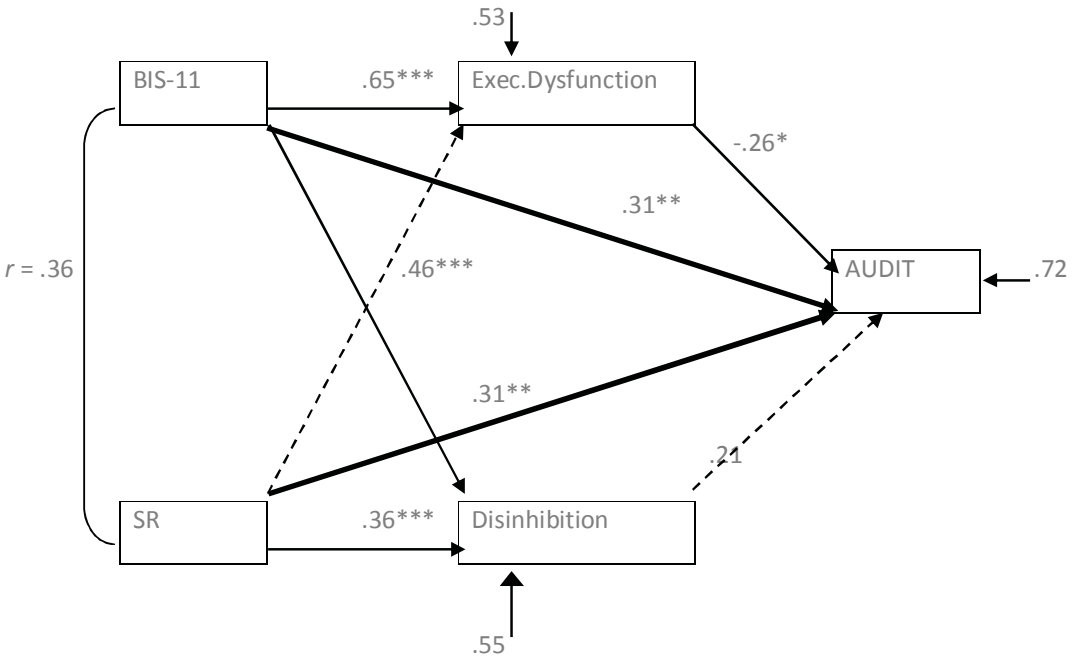
***p <.001 **p <.01 *p <.05

PATH ANALYSIS

To further investigate the extent to which the influence of rash impulsiveness (as assessed by BIS-11 scores) and reward sensitivity (as assessed by SR scores) on drinking behaviour were mediated by frontal lobe functioning, path analyses were performed for both variables. 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) onto 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 and the path from Disinhibition to AUDIT. The Sobel test statistic for the indirect pathway from BIS-11 to AUDIT through the intervening variable Executive Dysfunction was significant (t=2.72, p<.01) indicating evidence of strong mediation. Although the relationship between BIS-11 and AUDIT was noticeably diminished (β=.36 to β=.31) as was the relationship between SR and AUDIT (β=.44 to β=.31), this indicated only partial mediation (see Figure 1). The association of both BIS-11 and SR with the AUDIT is both a direct one unrelated to FrSBe frontal lobe functioning indices and an indirect one mediated by Executive Dysfunction for BIS-11 (rash impulsivity) and Disinhibition for SR (reward sensitivity).

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.



***p <.001 **p <.01 *p <.05 N = 121

These findings are fully consistent with the model of Dawe et al. (2004) according to which rash impulsivity (indexed here by BIS-11 scores) influences drinking via dorsolateral prefrontal executive dysfunction (as reflected on the FrSBe by Executive Dysfunction scores), whereas sensitivity to reward (indexed here by SR scores) influences drinking via orbitofrontal disinhibition of reward-driven responding (as reflected on the FrSBe by Disinhibition scores).

The results for the trait measures were all as predicted, however the failure to find the predicted relationships between risky or harmful drinking and poorer performance on neuropsychological tests of executive cognitive functioning was deemed likely to be due to the nature of the university student sample. As university students are selected on the basis of high levels of cognitive

performance and thus tend to be inherently good problem-solvers, we recruited a community sample of young adults who were not university students, hoping to get a sample with a broader spread of problem solving skills in order to provide a better test of our hypotheses concerning the relationship between risky drinking and neuropsychological test performance.

NEUROPSYCHOLOGICAL TEST PERFORMANCE AND DRINKING IN THE COMMUNITY SAMPLE

Data were collected from 47 community young adult social drinkers aged 18-26 years in southeast Queensland and NSW (Sydney). Pillai's Trace from an AUDIT group (Low Risk, Hazardous, Harmful) multivariate analysis of covariance (MANCOVA), controlling for age and gender, revealed a significant overall effect of AUDIT group on three well-known and validated neuropsychological tests of frontal lobe functioning: the WCST, the IGT and the Tower Test of the D-KEFS, $F(8, 32)=2.72$, $p=.02$, partial $\eta^2=.41$, observed power=.86. Individual effects were significant for total IGT gambling money earned, $F(2, 18)=4.21$, $p=.03$, partial $\eta^2=.32$, observed power=.66, and for the Tower Test total achievement score, $F(2, 18)=5.15$, $p=.02$, partial $\eta^2=.36$, observed power=.76. WCST perseverative errors (PEs), a measure found to be highly sensitive to acute drug effects in previous work (Lyvers & Maltzman, 1991; Lyvers et al., 1994; Lyvers & Tobias-Webb, 2010; Lyvers & Yakimoff, 1999), showed a nonsignificant trend ($p=.10$) toward more PEs in Hazardous and Harmful drinkers than in Low Risk drinkers. The relevant group means for the neuropsychological test measures in the community sample are shown in Table 6 below; note that the first measure (WCST-PE) is a measure of errors (thus higher values mean worse performance), whereas the subsequent two measures are measures of performance (thus higher values mean better performance).

TABLE 6: MEAN (SD) SCORES ON NEUROPSYCHOLOGICAL TESTS OF EXECUTIVE FUNCTIONING IN LOW RISK, HAZARDOUS AND HARMFUL DRINKERS AS DEFINED BY AUDIT FOR 47 NON-STUDENT YOUNG ADULTS.

Variable	Low risk	Hazardous	Harmful
WCST-PE	4.03 (7.25)	8.89 (11.86)	8.34 (6.89)
IGT total score	2,246.88 (1,054.70)	3,355 (1,129.95)	1,907.14 (482.34)
Tower Achievement	21.88 (3.27)	20.50 (2.92)	18.86 (2.19)

These three neuropsychological tests have been found to reliably distinguish patients with focal lesions of the frontal lobes from those with lesions of posterior regions as well as controls (Miller & Cummings, 2007), thus our results in a non-clinical sample are especially striking. As participants were all social drinkers from the community who reported never being in treatment for any alcohol or drug related problem, nor reported a history of head injury or neuropsychological or psychological disorder, the findings are consistent with our hypothesis that a significant portion of the executive function deficits reported in alcoholic samples (Lyvers, 2000) may reflect premorbid traits rather than neurotoxic sequelae of chronic alcohol exposure. Such premorbid executive function deficits are likely to predispose to risky or problematic alcohol use via mediating traits such

as impulsiveness, disinhibition and reward sensitivity. The present findings also point to caution concerning the common reliance of researchers on university student samples for research data, as the university sample failed to show the expected association between performance on neuropsychological tests of executive function and alcohol use, whereas the community sample of young adults did show this association in the present study as reported above. However the trait measures were significantly related to alcohol use in predicted ways in the university student sample.

Dissemination of Findings

The findings of this research project will be published in at least two articles in international addiction journals of high standing as they constitute a significant contribution to our understanding of the neuropsychological and trait correlates of risky drinking in young adults. We also anticipate presenting the findings at international conferences in the northern hemisphere summer of 2012, including the Congress on Problems of Drug Dependence in Palm Springs and the International Congress of Psychology in Cape Town.

Conclusions and Implications

In our university sample of young adults the findings for the trait measures were fully consistent with previous work (Lyvers et al., 2009, 2011). Trait indices of impulsiveness, disinhibition, executive cognitive dysfunction and reward sensitivity were all linked to harmful drinking, earlier onset of regular drinking, and parental alcoholism. These results suggest that an inherited predisposition to risky drinking may manifest as frontal lobe related traits. However in the university sample our three neuropsychological tests of frontal lobe function did not significantly differentiate Hazardous or Harmful drinkers from Low Risk drinkers (as defined by AUDIT), contrary to predictions. We speculated that this was due to the superior cognitive problem solving skills that are required for entry into university, restricting the range of cognitive function to a fairly high level in the university sample. To test this idea we recruited a second sample of non-student young adults from the general community; that sample did show the predicted association of poorer neuropsychological test performance with risky (Hazardous or Harmful) drinking compared to Low Risk drinking. Two of the three tests showed significant differences related to AUDIT-defined alcohol risk level, while the third showed a trend; all three tests are well-known and validated indices of the neuropsychological consequences of frontal brain damage, which makes the present findings rather striking.

This research funded by FARE has thus made a significant contribution to our knowledge of the trait factors linked to risky or problematic alcohol consumption by young adults as well as the underlying brain mechanisms that are likely involved. Future work in this area should ideally employ functional brain imaging techniques to make an even stronger case for functional frontal lobe deficiencies in young adults who drink at risky or harmful levels.

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