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Risky Cannabis Use is Associated with Alexithymia, Frontal Lobe Dysfunction and Impulsivity  
in Young Adult Cannabis Users

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## Abstract

Risky or problematic alcohol use by young adults has been found to be associated with factors such as alexithymia, frontal lobe dysfunction, reward sensitivity and impulsivity. One interpretation is that these factors reflect inherent traits that predispose to risky substance use in general, a notion examined in the present study. Alexithymia, everyday frontal lobe functioning, sensitivity to reward and punishment, and impulsivity were examined in 138 young adult cannabis users who were divided into Low Risk ( $n = 99$ ) and Risky ( $n = 39$ ) users according to their Cannabis Use Disorder Identification Test (CUDIT) scores. Risky cannabis use was significantly positively associated with alexithymia, multiple signs of frontal lobe dysfunction in everyday life, and impulsivity. A broader pattern of dysfunction was indicated for risky cannabis use than for risky alcohol use in this sample. Findings are interpreted as likely reflecting not only inherent traits that predispose to risky substance use in general, but also perhaps to residual effects of recent heavy cannabis use in the Risky user group. Longitudinal research is needed to disentangle these competing possibilities.

**Keywords:** cannabis, marijuana, alexithymia, impulsivity, frontal lobe

A variety of research findings have supported the proposed role of a constellation of linked trait factors in risky or harmful alcohol consumption, including reward sensitivity, impulsivity, frontal lobe dysfunction and alexithymia (Kauhanen, Julkunen & Salonen, 1992; Lyvers, Czerczyk, Follent, & Lodge, 2009; Lyvers, Duff & Hasking, 2011; Lyvers, Onuoha, Thorberg & Samios, 2012; Thorberg et al., 2009, 2011a,b). Although signs of frontal cortical dysfunction and related behavioural deficits in alcoholics have long been attributed to cumulative neurotoxic sequelae of chronic alcoholism (Lyvers, 2000; Noel et al., 2001; Oscar-Berman & Marinkovic, 2007), substantial evidence also points to frontal lobe related traits such as impulsiveness, disinhibition, and executive cognitive dysfunction as inherent factors that may promote or predispose to excessive or problematic use of alcohol or other substances by young adults (Chen et al., 2007; Iacono, Carlson, Taylor, Elkins, & McGue, 1999; Lyvers et al., 2011, 2012; Spinella, 2003). Alexithymia, which refers to difficulties identifying and describing feelings, a paucity of fantasy life as well as an externally oriented thinking style (Sifneos, 1973), has also been linked to alcohol and cannabis use disorders (Dorard et al., 2008; Thorberg et al., 2009, 2011a,b; Troisi, Pasini, Saracco, & Spalletta, 1998) as well as behavioural signs of frontal lobe dysfunction (Lyvers et al., 2012), and appears to persist in substance dependent individuals following extended abstinence as revealed by stable Toronto Alexithymia Scale (TAS-20; Bagby, Parker, & Taylor, 1994) scores (Pinard, Negrete, Annable & Audet, 1996). The latter findings support a trait rather than state conception of alexithymia, although other evidence does support a state interpretation (Haviland, Hendryx, Shaw & Henry, 1994; Honkalampi, Hintikka, Saarinen, Lehtonen & Vilnamaki, 2000). Of course, trait and state aspects of alexithymia are not mutually exclusive notions.

As in other Western countries the most widely used and accepted illicit intoxicant in Australia is cannabis (National Drug Strategy, 2011). Indices of impulsivity, disinhibition, reward sensitivity, executive dysfunction and alexithymia have been reported to be higher in cannabis users than in non-users (Caracuel et al., 2008; Clark, Roiser, Robbins & Sahakian, 2009; Croft,

Mackay, Mills & Gruzelier, 2001; Dafters, Hoshi & Talbot, 2004; Daumann, Pelz, Becker, Tuchtenhagen & Gouzoulis-Mayfrank, 2001; Dorard, et al., 2008; Fletcher et al., 1996; Fontes et al., 2011; Liraud & Verdoux, 2000; Verdejo-Garcia, Bechara, Recknor & Perez-Garcia, 2006; Whitlow et al., 2004), however the variation in such measures within samples of cannabis users as a function of their level of use has received less attention. Troisi et al. (1998) found that prevalence and severity of alexithymia increased as a function of increasing frequency of cannabis use in their sample of cannabis users. Bolla, Brown, Eldreth, Tate and Cadet (2002) found that even after 28 days of abstinence, heavy cannabis users showed poorer executive cognitive performance than light users, suggesting either a predisposing trait or enduring drug-induced impairment interpretation.

Lyvers et al. (2009, 2010, 2011, 2012) evaluated university student and community samples of young adult social drinkers using the Alcohol Use Disorders Identification Test, or AUDIT (Babor, Higgins-Biddle, Saunders, & Monteiro, 2001), to identify risky drinkers, who displayed elevated scores on indices of alexithymia, reward sensitivity, impulsivity and frontal lobe dysfunction compared to low risk drinkers. The comparable measure of risky cannabis use is the Cannabis Use Disorders Identification Test, or CUDIT (Adamson & Sellman, 2003). In the present study, the CUDIT was administered to a community sample of young adult cannabis users along with the same measures of impulsiveness, alexithymia, frontal lobe dysfunction, and sensitivity to reward and punishment as administered previously in conjunction with AUDIT by Lyvers et al. in young adult social drinkers. Based on the notion that the differences found in risky drinkers reflected inherent traits that predispose to risky substance use in general, rather than alcohol in particular, we anticipated that risky cannabis users would be similarly differentiated from low risk cannabis users (as defined by CUDIT) on these same trait measures. AUDIT scores were also taken in order to assess possible differential correlates of AUDIT and CUDIT in the present sample of cannabis users, most of whom also used alcohol to varying degrees, as well as correlates of concomitant heavy use of both substances.

## Method

### *Participants*

There were 138 cannabis user participants aged 18-35 years ( $M = 22.56$  years,  $SD = 4.28$ ), including 49 males (35.5%) and 89 females (64.5%). Of these cannabis users 89.4% also reported using alcohol. The sample consisted mostly of university students, with 87% of participants reporting they were currently enrolled in university studies. Participants responded to publicly displayed posters and flyers distributed in locales known to be frequented by cannabis users in the northern New South Wales and Southeast Queensland region. Participation was requested in an online survey through *Survey Monkey* comprising a test battery assessing factors relating to cannabis and alcohol use. The stated inclusion criteria were that all participants had to be current cannabis users, non-intoxicated during survey completion and aged 18-35 years. There was no incentive for participation.

### *Materials*

The online survey, containing a combination of the measures assessing the key variables of interest, was administered via *Survey Monkey*, an online, business-related survey generation program. The demographics questionnaire and six questionnaires comprising the online survey are outlined below. All participants were required to read and agree to the explanatory statement posted at the start of the online survey before proceeding further, and were asked not to provide any personally identifying information in order to ensure their anonymity.

The *Demographics Questionnaire* requested basic personal information including age, gender, whether individuals are currently enrolled in university, whether they use alcohol and/or cannabis, and if they had done so within the past week.

The *Alcohol Use Disorders Identification Test* (AUDIT; Babor, Higgins-Biddle, Saunders, & Monteiro, 2001) is a 10-item scale developed by the World Health Organization measuring the frequency and severity of alcohol use and prevalence of alcohol use disorders. Scores range from 0 to 40; scores above 0 but less than 8 indicate Low Risk drinking, whereas

scores of 8 or above indicate Risky drinking. The psychometric properties of this widely used measure have demonstrated sound reliability and validity (see Reinert & Allen, 2007).

The *Cannabis Use Disorders Identification Test* (CUDIT; Adamson & Sellman, 2003) is a 10-item scale, based on the AUDIT, measuring the frequency and severity of cannabis use. As in the AUDIT, scores can range from 0 to 40 and the cut-off score differentiating Low Risk from Risky cannabis use is 8. Like the AUDIT, the CUDIT demonstrates sound psychometric properties (see Piontek, Kraus & Klempova, 2008).

The *Barratt Impulsiveness Scale 11* (BIS-11; Patton, Stanford, & Barratt, 1995) is a 30 item self-report scale measuring trait impulsiveness. All items are scored on a 4-point Likert scale, whereby 1 = “Rarely/Never”, 2 = “Occasionally”, 3 = “Often” and 4 = “Almost Always/Always”. External validity has been demonstrated in clinical populations (Swann, Andersen, Dougherty, & Moeller, 2001) and construct and convergent validity have been shown with related behavioural and psychometric instruments (Carrillo de la Pena, Otero & Romero, 1993). Elevated scores on the BIS-11 were positively correlated with brain imaging signs of prefrontal cortex dysfunction in a schizophrenic sample (Hoptman et al., 2002) and have shown correlations with other indices of frontal dysfunction in previous work (Lyvers et al., 2011).

The *Toronto Alexithymia Scale* (TAS-20; Bagby et al., 1994) is the most frequently used alexithymia measure. There are 20 items rated on 5-point Likert scale (1 = strongly disagree to 5 = strongly agree) yielding a total alexithymia score as well as three subscale scores: Difficulty Identifying Feelings (DIF), Difficulty Describing Feelings (DDF), and Externally Oriented Thinking (EOT). Higher scores indicate a greater degree of alexithymia. Scores below 52 are interpreted as indicating no alexithymia, whereas scores from 52 to 60 suggest borderline alexithymia and scores of 61 or above indicate clinical levels of alexithymia. The TAS-20 has demonstrated good internal consistency, test-retest reliability, and factorial validity (Berthoz & Hill, 2005; Parker, Taylor & Bagby, 2003; Thorberg et al., 2010; Thorberg, Young, Sullivan, Lyvers, Hurst, Connor et al., 2010).

The *Frontal Systems Behavior Scale* (FrSBe; Grace & Malloy, 2001) is a 46-item scale designed to detect everyday functional behavioural deficits in frontal brain damaged patients. There are three subscales to assess deficits associated with each of three prefrontal cortical systems: Apathy (amotivation, lethargy, disinterest and diminished affective disposition; reflects anterior cingulate dysfunction, 14 items), Disinhibition (impaired inhibitory control, socially unacceptable behaviour and unregulated or excessive emotional responses; reflects orbitofrontal dysfunction, 15 items), and Executive Dysfunction (impairments of judgement, abstract reasoning, attention, working memory and cognitive flexibility; reflects dorsolateral prefrontal dysfunction, 17 items). Questions are based on a 5-point Likert-scale where 1 = “Almost never”, 2 = “Seldom”, 3 = “Sometimes”, 4 = “Frequently”, and 5 = “Almost always”. There are 14 reverse-scored items, for example, “I use strategies to remember important things (e.g., write notes to myself).” The validity of the FrSBe has been demonstrated in brain damaged populations (Stout, Ready, Grace, Malloy & Paulsen, 2003) as well as in substance abusers (Spinella, 2003), and the subscales have all demonstrated good internal consistency as has the total score (Grace & Malloy, 2001). The FrSBe was designed to assess pre and post-lesion functional differences in brain damaged patients, but as the present study tested a non-clinical community sample, only current levels of functioning were assessed.

The *Sensitivity to Punishment and Sensitivity to Reward Questionnaire* (SPSRQ; Torrubia, Avila, Molto & Caseras, 2001) is comprised of two separate subscales: Sensitivity to Punishment (SP) and Sensitivity to Reward (SR). Each subscale has 24 items measuring an individual’s motivational propensity to engage in either approach (SR) or avoidance (SP) behaviour in the presence of either rewarding or punishing stimuli, respectively. Items are answered via dichotomous yes (1) or no (0) responses, which are summated for each subscale. The SPSRQ is based on Gray’s (1987) Reinforcement Sensitivity Theory (RST), such that the SR scale was designed to evaluate the RST’s Behavioural Activation System (BAS) and the SP scale was designed to evaluate the RST’s Behavioural Inhibition System (BIS). The SPSRQ has been



established as a valid and reliable measure of the theoretical components of RST (Corr, 2004; Loxton & Dawe, 2001). SR and SP have demonstrated high internal consistency, test–retest reliability, and convergent, construct and discriminate validity (see Caseras, Avila, & Torrubia, 2003).

### *Procedure*

Following ethical approval from the Bond University Human Research Ethics Committee (BUHREC), participants were recruited via posters and flyers requesting participation in the online survey via *Survey Monkey* with the web address for the survey provided. Posters were placed, and flyers distributed, in areas known to be frequented by cannabis users in northern NSW and southeast Qld Australia. The questionnaire battery took approximately 15 minutes to complete. There was no incentive for participation.

### Results

Of the initial 225 individuals who completed the online survey, 71 cases were excluded due to data entry errors, missing data, or not meeting study criteria (i.e., there was one non-user of cannabis, and seven cases did not meet age criteria). In addition 16 multivariate outliers were identified via Boxplots and removed, leaving 138 cases for analysis.

Correlations among the variables were then examined. As can be seen in Table 1, TAS-20 total alexithymia score, as well as the DIF subscale score, were significantly positively correlated with BIS-11 impulsivity, SR reward sensitivity, all three frontal lobe dysfunction subscales of the FrSBe, AUDIT scores and CUDIT scores, and were negatively related to age. BIS-11, SR, Disinhibition and Executive Dysfunction FrSBe subscales and AUDIT scores were all intercorrelated as found in previous work (Lyvers et al., 2009, 2010, 2011, 2012). CUDIT scores were significantly positively correlated with all three TAS-20 alexithymia subscales, all three FrSBe frontal lobe dysfunction subscales, BIS-11 and AUDIT scores, and negatively correlated with SP, in this sample of cannabis users.

In the current sample, 71.7% ( $n = 99$ ) were defined by their CUDIT scores as Low Risk

cannabis users (CUDIT score < 8) whereas 28.3% ( $n = 39$ ) were defined by CUDIT as Risky cannabis users (CUDIT score 8+). Further, by AUDIT criteria 52.2% ( $n = 72$ ) reported being Low Risk drinkers (AUDIT score < 8) whereas 47.8% ( $n = 66$ ) reported being Risky drinkers (AUDIT score 8+). As expected there was considerable overlap between CUDIT and AUDIT risk groups in the present sample,  $\chi^2(1) = 18.45$ ,  $p < .0001$ , such that 63% of the Low Risk cannabis users were likewise Low Risk drinkers, and 77% of the Risky cannabis users were likewise Risky drinkers. As determined by TAS-20 cut-off scores, 79.7% ( $n = 110$ ) reported being non-alexithymic (TAS-20 score < 52), whereas 20.3% ( $n = 28$ ) reported being either alexithymic or borderline alexithymic (TAS-20 score 52+) in the present cannabis using sample. Chi-square analyses showed that CUDIT risk group was significantly associated with TAS-20 alexithymia group,  $\chi^2(1) = 8.19$ ,  $p = .004$ . Of the Low Risk cannabis users, 14% were alexithymic or borderline alexithymic, whereas of the Risky cannabis users 36% were alexithymic or borderline alexithymic. By contrast the association between AUDIT risk group and TAS-20 alexithymia group was not significant,  $\chi^2(1) = 2.34$ ,  $p = .126$ .

A two-way (CUDIT risk group X gender) between-subjects multivariate analysis of covariance (MANCOVA) was performed on scores obtained from the TAS-20 DIF, DDF and EOT subscales, FrSBe Apathy, Disinhibition and Executive Dysfunction subscales, SP and SR scales from the SRSPQ, and the BIS-11. Age was the covariate due to its significant correlations with some of the variables of interest (see Table 1). Box's M test was not significant using  $p < .01$  as the criterion due to the sensitivity of this test to unequal cell sizes. After FrSBe Disinhibition scores were subjected to log10 transformation Levene's test of homogeneity of variances was nonsignificant for all outcome measures. According to Pillai's Trace the effect of CUDIT group on the combined dependent measures was significant,  $F(9, 125) = 4.44$ ,  $p < .0001$ ,  $\eta^2 = .242$ , observed power = .997. Neither the multivariate effect of gender nor the interaction was significant. Univariate effects of CUDIT risk group were significant for DIF,  $F(1, 133) = 24.43$ ,  $p < .0001$ ,  $\eta^2 = .155$ , observed power = .998; DDF,  $F(1, 133) = 5.38$ ,  $p = .022$ ,  $\eta^2 = .039$ ,

observed power = .634; EOT,  $F(1, 133) = 7.32, p = .008, \eta^2 = .052$ , observed power = .766; BIS-11,  $F(1, 133) = 19.27, p < .0001, \eta^2 = .127$ , observed power = .992; FrSBe Apathy,  $F(1, 133) = 9.52, p = .002, \eta^2 = .067$ , observed power = .865; FrSBe Executive Dysfunction,  $F(1, 133) = 21.94, p < .0001, \eta^2 = .142$ , observed power = .996; and the log transformed FrSBe Disinhibition,  $F(1, 133) = 21.80, p < .0001, \eta^2 = .141$ , observed power = .996. The relevant untransformed group means are presented in Table 2.

For comparison purposes a two-way between-subjects MANCOVA was also performed comparing AUDIT risk groups and genders on the same dependent measures, with age again being the covariate. Box's M and Levene's tests were again nonsignificant indicating no violation of assumptions. According to Pillai's Trace the effect of AUDIT risk group on the combined dependent measures was significant,  $F(9, 125) = 4.79, p < .0001, \eta^2 = .256$ , observed power = .999. Univariate effects of AUDIT group were significant for DIF,  $F(1, 133) = 9.28, p = .003, \eta^2 = .065$ , observed power = .856; BIS-11,  $F(1, 133) = 22.25, p < .0001, \eta^2 = .143$ , observed power = .997; FrSBe Executive Dysfunction,  $F(1, 133) = 11.71, p = .001, \eta^2 = .081$ , observed power = .925; and the log transformed FrSBe Disinhibition,  $F(1, 133) = 31.20, p < .0001, \eta^2 = .190$ , observed power = 1.00. These findings were all consistent with previous work using different young adult samples of social drinkers (Lyvers et al., 2009, 2010, 2011). The relevant untransformed group means are presented in Table 3.

Finally, although the size of one cell was small as expected, cell sizes were sufficient to combine CUDIT risk groups and AUDIT risk groups to create four groups for a oneway MANOVA: Low Risk AUDIT/Low Risk CUDIT ( $n = 63$ ); Low Risk AUDIT/Risky CUDIT ( $n = 9$ ); Risky AUDIT/Low Risk CUDIT ( $n = 36$ ); and Risky AUDIT/Risky CUDIT ( $n = 30$ ). Again Box's M test was nonsignificant and Levene's test was nonsignificant for all dependent measures after square root transforming BIS-11 scores. The MANOVA was exploratory given the small  $n$  for the Low Risk AUDIT/Risky CUDIT group; indeed we did not expect to find many risky cannabis users who were not also risky drinkers. Nevertheless the MANOVA on total TAS-20,

total FrSBe, BIS-11, SR and SP showed a significant effect of group according to Pillai's Trace,  $F(15, 396) = 4.16, p < .0001, \eta^2 = .136$ , observed power = 1.00. Significant univariate effects were obtained for TAS-20,  $F(3, 134) = 10.03, p < .0001, \eta^2 = .183$ , observed power = .998; FrSBe,  $F(3, 134) = 14.44, p < .0001, \eta^2 = .244$ , observed power = 1.00; transformed BIS-11,  $F(3, 134) = 14.64, p < .0001, \eta^2 = .247$ , observed power = 1.00; and SR,  $F(3, 134) = 3.07, p = .03, \eta^2 = .064$ , observed power = .708. MANCOVA controlling for age and gender yielded essentially the same findings, but MANOVA allowed use of post hoc Tukey HSD tests to determine which group means were significantly different. The conservative Tukey test showed that the Risky AUDIT/Risky CUDIT group scored significantly higher on TAS-20, FrSBe and BIS-11 than all three other groups,  $p < .01$ , and marginally higher on SR than the Low Risk AUDIT/Low Risk CUDIT group,  $p = .059$  (see Table 4). Examining the TAS-20 alexithymia classification, of the 30 Risky users of both drugs, 13 (43%) were either alexithymic or borderline alexithymic by TAS-20 cut-off criteria compared to only 11%-16% in the other three groups, a highly significant association,  $\chi^2(3) = 12.95, p = .005$ .

### *Path Analyses*

In a recent study (Lyvers et al., 2012) the relationship between total TAS-20 and AUDIT scores was fully mediated by FrSBe total scores in a community sample of social drinkers. A path analysis of the data from the present study showed identical results with regard to AUDIT; the same approach was taken with CUDIT scores instead of AUDIT scores. In order to test for mediation, the predictor variable needs to be significantly correlated with the dependent variable and also has to be significantly correlated with the mediator, and the mediator needs to be significantly related to the dependent variable even after controlling for the independent variable. If the predictor variable is no longer significantly associated with the dependent variable after controlling for the mediator, full mediation is established (Baron & Kenny, 1986). In the present study a standard regression analysis found a significant relationship between the predictor variable total TAS-20 score and the dependent variable CUDIT score,  $F(1, 136) = 21.79, p <$

.0001, accounting for 13.8% of the variance ( $p < .0001$ ). Second, a significant relationship was found between the predictor variable TAS-20 and the mediator FrSBe,  $F(1, 136) = 173.07, p < .0001$ , accounting for 56% of the variance ( $p < .0001$ ). Third, a multiple regression analysis was performed on CUDIT with TAS-20 and FrSBe to investigate the mediational effect of FrSBe,  $F(2, 135) = 16.70, p < .0001$ , indicating that only FrSBe ( $\beta = .37, t(2) = 3.19, p = .002$ ) showed univariate significance (see Table 4). As all four conditions were met and the Sobel test was significant ( $p < .01$ ) these findings indicate that FrSBe fully mediated the relationship between the TAS-20 total score and the CUDIT. However, TAS-20 predicted both AUDIT,  $F(1, 136) = 13.07, p < .0001$ , and CUDIT (see above), and AUDIT and CUDIT scores were highly correlated (see above), raising the possibility that the ability of TAS-20 to predict CUDIT was entirely mediated by AUDIT. Hierarchical regression showed that even after controlling for the significant effect of predictor AUDIT on CUDIT,  $F(1, 136) = 40.62, p < .0001$ , accounting for 23% of variance in CUDIT scores, TAS-20 still significantly predicted CUDIT scores beyond the ability of AUDIT scores to do so,  $Fchange(1, 135) = 10.96, p < .001$ , accounting for an additional 6% of variance.

The positive correlation of AUDIT with the Apathy subscale of the FrSBe was unexpected as previous research by Lyvers et al. (2009, 2010, 2011, 2012) did not find any association. The strong relationships observed in the present study between CUDIT and Apathy scores, as well between CUDIT and AUDIT scores, suggested that the AUDIT-Apathy relationship may have been fully mediated by CUDIT. A second path analysis was thus conducted to test this hypothesis. A standard regression found a significant relationship between the predictor variable Apathy and the dependent variable AUDIT,  $F(1, 136) = 8.09, p = .005$ , accounting for 5.6% of the variance ( $R^2 = .056, p = .005$ ). Second, a significant relationship was found between the predictor variable Apathy and the mediator CUDIT,  $F(1, 136) = 17.23, p < .0001$ , accounting for 11.2% of the variance ( $R^2 = .112, p < .0001$ ). Third, a multiple regression was performed on AUDIT with predictors Apathy and CUDIT to investigate the mediational

effect of CUDIT,  $F(2, 135) = 20.91, p < .0001$ , indicating that only CUDIT ( $\beta = .45, t(2) = 5.65, p < .0001$ ) showed univariate significance (see Table 5). The Sobel test was significant ( $p < .01$ ), thus these findings indicate that CUDIT fully mediated the relationship between FrSBe Apathy scores and AUDIT scores.

A final exploratory regression showed that TAS-20 DIF scores, which were specifically found to be positively related to AUDIT scores in alcohol dependent outpatients in a recent investigation (Thorberg et al., 2011a), were significantly predicted by both AUDIT ( $\beta = .21, t(2) = 2.50, p = .014$ ) and CUDIT ( $\beta = .28, t(2) = 3.12, p = .002$ ),  $F(2, 135) = 15.21, p < .0001$ , in the present cannabis-using sample.

### Discussion

In a community sample of cannabis users, cannabis-related risk as assessed by CUDIT was significantly positively correlated with all three TAS-20 alexithymia subscales as well as the overall score, all three FrSBe frontal lobe dysfunction subscales and the BIS-11 impulsivity index. CUDIT scores were significantly negatively correlated with the SP index of the SPSRQ but were unrelated to SR, whereas alcohol-related risk as assessed by AUDIT was significantly positively related to SR but unrelated to SP, as found in previous work (Lyvers et al., 2009, 2010, 2011, 2012). The previous findings were further supported in the present study such that AUDIT was positively related to the Disinhibition and Executive Dysfunction subscales of the FrSBe, and was also positively related to BIS-11 as well as the TAS-20 (especially the DIF subscale). AUDIT and CUDIT were highly positively correlated as expected. However the positive correlation of AUDIT with the Apathy subscale of the FrSBe was unexpected as the previous research cited above did not find such an association. A path analysis showed that the relationship between AUDIT and Apathy was fully mediated by CUDIT in the present cannabis-using sample. In other words, riskier drinkers tended to be riskier cannabis users in the present sample of cannabis users, but only riskier cannabis use was specifically associated with higher Apathy scores.

Comparisons between risk groups as defined by CUDIT and AUDIT showed similar correlates of risky use for both substances, however a somewhat broader pattern of dysfunction was indicated for risky cannabis use than for risky drinking. This may reflect short-term residual effects of cannabis instead of, or in addition to, traits that predispose to heavier substance use in general, although the correlational nature of the present investigation cannot decide between these possibilities. We did ask participants if they had smoked cannabis recently (i.e., within the past week), however of those who responded “no,” 91% were in the Low Risk user group, hence recent use (more likely to have residual effects) was confounded by Risky use in the present sample. Heavy cannabis users have been reported to exhibit deficits in frontal lobe functioning (Fontes et al., 2011) that are present even after 28 days of abstinence (Bolla et al., 2002). Such deficits may reflect pre-drug traits that predispose to heavy substance use, but have also been attributed to reversible short-term neuroadaptive changes in the brains of heavy users (Chang, Yakupov, Cloak & Ernst, 2006) or to the enduring presence of THC in the body as a result of THC’s high lipid solubility and accumulation in fat cells with frequent use (Karschner et al., 2009; Pope & Yurgelun-Todd, 2004). Alcohol by contrast is metabolized and eliminated rapidly such that BAC is generally zero within 8-12 hours of heavy drinking cessation (Fleming, Mihic & Harris, 2001; Rohsenow et al., 2010). In this context the present finding that risky cannabis use, but not risky drinking, was positively associated with scores on the FrSBe Apathy subscale is especially intriguing. As the Apathy subscale was designed to assess everyday behavioural deficits following damage to the anterior cingulate, an area known to be strongly affected by THC intoxication (Martin-Santos et al., 2010), this relationship may reflect a residual effect of frequent or heavy cannabis use (D’Souza, Sewell, & Ranganathan, 2009; Hall & Solowij, 1998) due to the persistence of THC at active levels in Risky users (most of whom were also recent users). Interestingly, evidence suggests that THC intoxication lowers motivation on experimental tasks (Cherek, Lane & Dougherty, 2002), perhaps due to actions in the anterior cingulate.

A significant positive relationship was found between risky use of alcohol and reward

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sensitivity as measured by the SR scale of the SPSRQ, replicating previous work (Lyvers et al., 2009, 2010, 2011); yet despite the strong positive correlation between AUDIT and CUDIT in the present study, no relationship was found between CUDIT and SR scores. This was surprising given that both alcohol and THC stimulate dopamine release in the primary reward pathway (Gardner, 2005; Olive & Griffin, 2008), and both drugs are self-administered intravenously by laboratory primates in animal models of drug reward (Justinova, Tanda, Redhi & Goldberg, 2003). In any case, present results suggest that inherently high reward sensitivity or BAS activity is linked to risky use of alcohol but not cannabis among cannabis users, a surprising outcome that should be replicated before conclusions can be drawn. The other SPSRQ scale, the index of punishment sensitivity SP, was negatively correlated with use of cannabis but not alcohol. This negative relationship of cannabis use with SP in the present sample of cannabis users most plausibly reflects the purported protective effect of high SP against high levels of substance use (Lee, Wadsworth & Hotopf, 2006), although a residual anxiolytic effect of THC in heavy users that might dampen SP is an alternate possibility (Rubino et al., 2007).

When participants were divided into four groups by crossing CUDIT risk groups with AUDIT risk groups, the primary finding was that the Risky CUDIT/Risky AUDIT group scored significantly worse than the other three groups on TAS-20, FrSBe and BIS-11. In other words, risky users of both cannabis and alcohol reported more alexithymia symptoms, more everyday manifestations of frontal lobe dysfunction, and greater impulsiveness than Risky users of one drug but not the other, or Low Risk users of both drugs. Of course one might expect that concurrent risky users of two drugs would report worse psychopathology than risky users of one drug, or low risk users of both. However the direction of causation cannot be gleaned from evidence such as that obtained in the present investigation. Traits such as alexithymia, poor impulse control and executive dysfunction have all been proposed to promote substance abuse (e.g., Dawe, Gullo & Loxton, 2004; Lyvers et al., 2009, 2010, 2011, 2012; Spinella, 2003), but have also been linked to ongoing risky use with improvement following extended abstinence



(e.g., Bolla et al., 2002; Haviland et al., 1994). Of course, both directions of causation may apply; for example, those with inherently lower emotion regulation and executive “self-control” abilities may be more likely to abuse substances, and substance abuse itself may further impair executive functioning and self-restraint in a vicious cycle (Lyvers, 2000).

The correlational nature of the present findings is thus a limitation. The results were certainly consistent with the hypothesized constellation of traits that may predispose young adults to riskier use of cannabis as was previously found for alcohol; if anything risky cannabis use was associated with an even broader spectrum of dysfunction on the corresponding measures than was found for risky alcohol use in this and previous work. However, as discussed above, unlike alcohol the residual effects of frequent or heavy cannabis use may be far from negligible even days to weeks after last use, and may therefore account for at least some of the differences found between Risky and Low Risk cannabis users in the present investigation. Longitudinal research is thus needed before the competing influences of pre-drug traits and residual cannabis effects can be disentangled in terms of their respective influences on the outcome measures of this study in risky cannabis users.

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**Table 1.** Correlations among study variables (see text for variable definitions).

	DIF	DDF	EOT	TAS-20	BIS	SR	SP	Apathy	Exec. Dys.	Disinhibition	AUDIT	CUDIT
DIF	1											
DDF	.559**	1										
EOT	.311**	.479**	1									
TAS-20	.807**	.839**	.739**	1								
BIS	.478**	.315**	.520**	.557**	1							
SR	.269**	.112	.192*	.247**	.380**	1						
SP	.156	.151	-.016	.124	.020	.226**	1					
Apathy	.587**	.579**	.517**	.707**	.523**	.123	.314**	1				
Exec. Dys.	.648**	.444**	.520**	.685**	.718**	.346**	.225*	.714**	1			
Disinhibition	.613**	.404**	.464**	.631**	.711**	.377**	.154	.630**	.800**	1		
AUDIT	.354**	.156	.174*	.296**	.477**	.278**	-.089	.237**	.416**	.548**	1	
CUDIT	.382**	.172*	.311**	.372**	.456**	.078	-.220**	.335**	.415**	.439**	.480**	1
Age	-.180*	-.174*	-.102	-.192*	.063	-.106	-.144	-.020	-.074	-.092	-.038	.209*

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Note. \* $p < .05$ , \*\* $p < .001$

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Table 2.

*Low Risk versus Risky Cannabis Users compared on study variables (see text for definitions).*

Measure	Risk Group	<i>M</i>	<i>SD</i>
TAS-20**	Low Risk	40.69	9.73
	Risky	48.87	14.24
DIF*	Low Risk	12.40	4.61
	Risky	16.62	6.32
DDF**	Low Risk	10.74	4.39
	Risky	12.38	4.51
EOT**	Low Risk	17.55	4.15
	Risky	19.87	5.99
BIS-11**	Low Risk	57.19	7.22
	Risky	64.70	10.11
SR	Low Risk	12.58	4.42
	Risky	13.62	4.17
SP	Low Risk	11.54	5.30
	Risky	9.82	5.27
Apathy**	Low Risk	27.99	6.90
	Risky	32.51	8.01
Executive Dys.**	Low Risk	34.28	7.41
	Risky	41.49	9.59
Disinhibition**	Low Risk	29.85	6.22
	Risky	36.38	8.73

*Note.* \* $p < .05$  \*\* $p < .01$

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Table 3

*Low Risk versus Risky Alcohol Users compared on study variables (see text for definitions).*

Measure	Risk Group	<i>M</i>	<i>SD</i>
TAS-20**	Low Risk	39.78	9.90
	Risky	46.51	12.61
DIF**	Low Risk	12.00	4.54
	Risky	15.33	5.88
DDF	Low Risk	10.53	4.27
	Risky	11.94	4.59
EOT	Low Risk	17.25	4.48
	Risky	19.24	5.02
BIS-11**	Low Risk	55.83	7.19
	Risky	63.11	8.83
SR	Low Risk	11.86	4.16
	Risky	13.97	4.34
SP	Low Risk	11.40	5.56
	Risky	10.67	5.08
Apathy	Low Risk	27.83	7.58
	Risky	30.83	7.11
Executive Dys.**	Low Risk	33.45	7.48
	Risky	39.44	8.87
Disinhibition**	Low Risk	39.78	9.90
	Risky	46.51	12.51

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Note. \* $p < .05$  \*\* $p < .01$

Table 4

*Study Variables in Low Risk (LA) versus Risky (RA) Alcohol users crossed with Low Risk (LC) versus Risky (RC) Cannabis users (see text for variable definitions).*

Measure	Risk Group	<i>M</i>	<i>SD</i>
TAS-20 total	LA/LC	40.11	9.97
	LA/RC	37.44	9.57
	RA/LC	41.69	9.34
	RA/RC	52.30	13.70
FrSBe total	LA/LC	89.30	18.58
	LA/RC	90.44	16.30
	RA/LC	97.10	15.51
	RA/RC	116.37	23.16
BIS-11	LA/LC	55.67	7.38
	LA/RC	57.00	5.83
	RA/LC	59.86	6.14
	RA/RC	67.00	10.03
SR	LA/LC	11.97	4.31
	LA/RC	11.11	2.93
	RA/LC	13.64	4.47
	RA/RC	14.37	4.22

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Lyvers, M., Jamieson, R., & Thorberg, F. A. (2013). Risky cannabis use is associated with alexithymia, frontal lobe dysfunction, and impulsivity in young adult cannabis users. *Journal of Psychoactive Drugs*, 45(5), 394-403. <https://doi.org/10.1080/02791072.2013.844525>.

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SP	LA/LC	11.35	5.59
	LA/RC	11.78	5.70
	RA/LC	11.86	4.82
	RA/RC	9.23	5.09

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