

**Impulsivity-related cognition in alcohol dependence: Is it moderated by DRD2/ANKK1
gene status and executive dysfunction?**

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This is the preprint version of a published journal article. Please cite as:

Gullo, M. J., St. John, N., Young, R. McD., Saunders, J. B., Noble, E. P., & Connor, J. P. (2014). Impulsivity-related cognition in alcohol dependence: Is it moderated by DRD2/ANKK1 gene status and executive dysfunction? *Addictive Behaviors*. doi:10.1016/j.addbeh.2014.02.004

Abstract

Perceived impaired control over alcohol use is a key cognitive construct in alcohol dependence that has been shown to predict treatment outcome. It has also been shown to mediate the risk for problem drinking conveyed by impulsivity in non-dependent drinkers. The aim of the current study was to evaluate whether the risk conveyed by high impulsivity also operated through this pathway in alcohol-dependent drinkers. Furthermore, the extent to which this hypothesized relationship was moderated by genetic risk (A₁ allele of the D2 dopamine receptor DRD2 gene) and verbal fluency as an indicator of executive cognitive ability (Controlled Oral Word Association Test) was also examined. A sample of 143 alcohol-dependent inpatients provided an extensive clinical history of their alcohol use, 10 milliliters of blood for DNA analysis, as well as completed self-report measures of impulsivity, impaired control and severity of dependence. As hypothesized, perceived impaired control (partially) mediated the association between impulsivity and dependence severity. This risk mechanism was not moderated by the DRD2 polymorphism or verbal fluency. These results suggest that, in alcohol dependence, perceived impaired control is a cognitive mediator of impulsivity that is unaffected by DRD2 and neurocognitive processes underlying the retrieval of verbal information.

Keywords: impulsivity, impulsive cognition, impaired control, DRD2, alcohol dependence, cognitive dysfunction

1. Introduction

Beliefs about control over drinking are key predictors of problematic alcohol use and dependence severity (Connor, George, Gullo, Kelly, & Young, 2011; Heather, Tebbutt, Mattick, & Zamir, 1993; Leeman, Patock-Peckham, & Potenza, 2012; Oei & Jardim, 2007; Young, Connor, Ricciardelli, & Saunders, 2006). The formation and strength of these alcohol-related cognitions are influenced by dispositional factors, including trait impulsivity and genetics (Smith & Anderson, 2001; Young, Lawford, Feeney, Ritchie, & Noble, 2004). Impulsive drinkers hold different beliefs about alcohol compared to non-impulsive drinkers, believing it to be both more rewarding and more difficult to resist (Gullo, Dawe, Kambouropoulos, Staiger, & Jackson, 2010). Indeed, mediational analyses have demonstrated that the influence of impulsivity on alcohol use is mediated, in part, through cognition (Gullo et al., 2010; Kabbani & Kambouropoulos, 2013; Leeman et al., 2012), although these have mostly been with non-dependent college drinkers. A greater understanding of the role of *impulsive* cognition in alcohol dependence is likely to have important implications for improving treatment for impulsive drinkers.

The confidence to achieve a certain goal, or *self-efficacy*, lies at the core of human agency (Bandura, 1986). Self-efficacy is not only the product of past experience, but is influenced by a range of biological, cognitive, and environmental factors (Bandura, 1999). Recent systematic reviews highlight self-efficacy as one of the most consistent predictors of treatment outcome (Adamson et al., 2009). Increasing self-efficacy is a key mechanism identified in cognitive-behavioral approaches that reduces relapse (Litt, Kadden, Kabela-Cormier, & Petry, 2008). Self-efficacy is a multi-faceted construct and understanding the factors that influence self-efficacy beliefs concerning drinking control could inform treatment for alcohol dependence.

Perceived impaired control over drinking is one type of self-efficacy relevant to alcohol dependence (Bandura, 1999; Oei & Baldwin, 1993). The Impaired Control Scale (ICS; Heather et al., 1992) is the most widely used instrument to measure this construct. It comprises three subscales: 1) the degree to which control has been attempted in the past 6 months (ICS: Attempted Control); 2) the extent to which these attempts have not been successful (ICS: Failed Control), and; 3) the drinker's beliefs about their potential to control future drinking (ICS: Perceived Control). The third subscale provides a measure of self-efficacy concerning future drinking control, while the other two provide an assessment of previous instances of impaired control. Both the Failed Control and Perceived Control subscales show robust associations with alcohol consumption, problems, dependence severity, and treatment outcome (Heather, Booth, & Luce, 1998; Kabbani & Kambouropoulos, 2013; Leeman et al., 2012; Marsh, Smith, Saunders, & Piek, 2002). Uniquely, perceived impaired control can discriminate between dependent drinkers with an abstinence versus moderation treatment goal and is amenable to cognitive-behavioral therapy (Heather et al., 1998, 1993). For example, refusal skills training and expectancy challenges have the potential to influence a patient's perceived control over future drinking. This makes perceived impaired control a relevant treatment target and, due to its subjective nature, is likely to be influenced by various factors, including impulsivity.

Perceived impaired control and poor drinking refusal self-efficacy are key cognitive mechanisms through which impulsivity conveys risk for alcohol problems (Gullo et al., 2010; Kabbani & Kambouropoulos, 2013; Leeman et al., 2012). Impulsivity consists of two core components, rash impulsiveness and reward drive (Dawe & Loxton, 2004). Perceived impaired control (partially) mediates the risk conveyed by rash impulsiveness, the component of impulsivity related to poor inhibitory control and acting without forethought (Dawe & Loxton, 2004; Kabbani & Kambouropoulos, 2013; Leeman et al., 2012). By

contrast, reward drive, the other major component of trait impulsivity related to reward sensitivity and appetitive motivation, conveys risk through heightened positive alcohol expectancies (Dawe, Gullo, & Loxton, 2004; Gullo et al., 2010; Kabbani & Kambouropoulos, 2013).

Several studies have reported that perceived impaired control mediates the association between rash impulsiveness and problem drinking (Kabbani & Kambouropoulos, 2013; Patock-Peckham, King, Morgan-Lopez, Ulloa, & Filson Moses, 2011; Patock-Peckham & Morgan-Lopez, 2006). The magnitude of the mediation effect ranges from $\beta = .09$ (Kabbani & Kambouropoulos, 2013) to $\beta = .20$ (Patock-Peckham & Morgan-Lopez, 2006). Kabbani and Kambouropoulos found full mediation, while the two studies by Patock-Peckham and colleagues reported partial mediation. The difference is likely due to the inclusion of alcohol expectancies and reward drive in Kabbani and Kambouropoulos' model, which both predicted variance in alcohol problems. Therefore, their smaller (full) mediation effect may be a more accurate estimate of the true effect unique to impaired control and rash impulsiveness. Indeed, Gullo et al. (2010) estimated a (partial) mediation effect of similar magnitude for drinking refusal self-efficacy in college drinkers ($\alpha\beta = .09$). However, none of these studies controlled for past consumption, nor previous attempts and failures of drinking control.

Past studies investigating mediation have sampled non-dependent, college drinkers. While important, the generalizability of these findings may be limited when applied to alcohol dependence. Dependent drinkers are more likely to carry candidate risk polymorphisms predictive of heavy alcohol use, such as the A1 allele of the Taq1A polymorphism of the dopamine D2 (*DRD2*) receptor gene (Munafò et al., 2007), and the presence of this genetic risk has been previously associated with lower self-efficacy beliefs. There is also evidence that newly abstinent dependent drinkers possess deficits in key

cognitive domains, including deficits in the ability to store and search for verbal information (verbal fluency; Chertkow & Bub, 1990; Stavro, Pelletier, & Potvin, 2013). Such deficits could also affect the role that explicit beliefs have in ‘impulsive’ consumption in treatment-seeking, dependent drinkers. Across two studies that included both college students and those with substance use disorders, the effect of rash impulsiveness mediated by explicit cognition was approximately two-thirds that observed in treatment-seeking inpatients ($\alpha\beta = .06$, Gullo et al., 2010, Study 2).

The present study sought to examine impulsive cognition in an alcohol dependent sample, and investigate the moderating role of genetic and neurocognitive risk factors. It was hypothesized that perceived impaired control would mediate the association between rash impulsiveness and severity of alcohol problems in alcohol dependent drinkers. We predicted that this mediation effect would be at the lower end of the range reported in previous studies ($.06 \leq \alpha\beta \leq .09$). It was also predicted that the role of perceived impaired control as a mediator of impulsivity would be significantly reduced in *DRD2* A1+ allele carriers and those with low verbal fluency (see Figure 1).

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2. Method

2.1 Participants

A total of 143 alcohol dependent inpatients (93 men; 65%) were recruited from a public hospital drug and alcohol treatment unit. The mean age of the sample was 41.14 years ($SD = 10.45$, range: 19 – 65) and all identified as being of Caucasian descent. Patients were approached to take part in the study within 2 – 5 days of admission to the unit. Inclusion criteria were: 1) met DSM-IV diagnostic criteria for alcohol dependence; 2) judged to be

physically and mentally well enough to undertake a clinical interview; 3) provided written consent to participate in the clinical interview and for a blood sample to be taken for DNA analysis. Exclusion criteria were: 1) current polysubstance misuse, excluding nicotine and less-than-monthly cannabis use; 2) severe alcohol-related medical complications; 3) being heavily sedated with a benzodiazepine or other medication, and 4) acute alcohol-related withdrawal. Approximately 44% of consecutive admissions met exclusion criteria. Data from this sample and some of the alcohol use measures and genetic data have previously been reported in Connor et al. (2008). Impulsivity, impaired control, and verbal fluency measures have not previously been analyzed or reported, nor has moderated mediation involving the genetic data been conducted.

2.2 Measures

2.2.1 Alcohol Consumption. Frequency of alcohol consumption was measured by asking participants to indicate their average number of drinking days in a typical week over the past month (0 – 7). Quantity of alcohol consumption was measured by the self-reported number of standard drinks consumed per drinking occasion during a typical week, over the past month. To facilitate accurate reporting, participants were provided pictures of common alcoholic beverages with their alcohol content measured in Australian standard drinks (1 standard drink = 10g ethanol).

2.2.2 Alcohol Dependence Severity. Severity of alcohol dependence was assessed using the 25-item Alcohol Dependence Scale (ADS; Skinner & Allen, 1982). This self-report measure has been shown to accurately classify alcohol abuse or dependence with 94% accuracy (Ross, Gavin, & Skinner, 1990).

2.2.3 Impulsivity. The 12-item Psychoticism and Extraversion scales from the Short-form Eysenck Personality Questionnaire-Revised (Eysenck & Eysenck, 1994) were used as

measures of rash impulsiveness and reward drive, respectively. While originally intended as a measure of ‘psychosis-proneness’, Eysenck’s psychoticism scale has long been regarded as more accurately measuring impulsivity and antisociality (Depue & Collins, 1999; Digman, 1990; Gullo, Dawe, & McHugh, 2011; Rawlings & Dawe, 2008; Zelenski & Larsen, 1999). Factor analytic studies have shown psychoticism to load with other measures of rash impulsiveness, supporting its construct validity (Dawe & Loxton, 2004; Gullo et al., 2010; Zelenski & Larsen, 1999). The short-form psychoticism scale has a 2-year test-retest reliability of .61 - .72 (Heath, Cloninger, & Martin, 1994).

Across three studies, Lucas and Diener (2001) demonstrated that individual differences in reward sensitivity lie at the core of trait extraversion. Similarly, factor analytic studies have shown measures of extraversion load with other measures of reward drive, further supporting its construct validity (Dawe & Loxton, 2004; Gullo et al., 2010; Quilty & Oakman, 2004; Zelenski & Larsen, 1999). The short-form extraversion scale has a 2-year test-retest reliability of .75 - .87 (Heath et al., 1994).

2.2.4 Perceived Impaired Control. The Impaired Control Scale (ICS; Heather, Tebbutt, Mattick, & Zamir, 1992) comprises three subscales: 1) The ICS-Attempted Control scale (5-items) measures the extent to which an individual has attempted to control their drinking over the past 6 months; 2) ICS-Failed Attempts (10 items) measures the extent to which these attempts were unsuccessful (over past 6 months); 3) The ICS-Perceived Control scale (10 items) assesses the individual’s beliefs about their ability to control future drinking. All items are rated on a 5-point Likert-type scale. In alcohol-dependent inpatients, the ICS scales have a 1-week test-retest reliability of .81 - .91 (Heather et al., 1993) and its factor structure has been independently validated using Confirmatory Factor Analysis (Marsh et al., 2002). Whilst cognitions concerning one’s perceived level of drinking control were of interest in the present study, all three scales were administered so that previous attempts and

their success/failure could be controlled for. This would allow for a more valid assessment of the possible influence of alcohol-related cognition independent of drinking history.

2.2.5 Verbal Fluency. The Controlled Oral Word Association Test (COWAT; Benton & Hamsher, 1976) is the most widely-used measure to assess the retrieval of verbal information, and is a common screening tool for executive neurocognitive impairment more broadly (Spren & Strauss, 1991). It involves 3 trials, each requiring participants to generate as many words as possible in 60 s that begin with a particular letter (*F*, *A*, then *S*). The COWAT has a 6-month test-retest reliability of .74 (Ruff, Light, Parker, & Levin, 1996) and is sensitive to dysfunction in the frontal and temporal brain regions (Baldo, Shimamura, Delis, Kramer, & Kaplan, 2001; Chertkow & Bub, 1990; Troyer, Moscovitch, & Winocur, 1997).

2.2.6 Genotyping. Details of genetic extraction have been described elsewhere (see Connor et al., 2008). Blood samples (10ml) were taken via venipuncture and DNA was extracted using standard techniques and used as a template for the determination of *Taq1 A DRD2* alleles by the polymerase chain reaction (Noble, Berman, Ozkaragoz, & Ritchie, 1994). All genotyping was carried out blind to participants' responses. Genetic data were not available for 37 participants who discharged themselves prior to venipuncture. Of the remaining 106 participants, 35 (33%) were A1+ allele carriers and 71 (76%) were A1- allele.

2.3 Analyses

Mediation was tested using the *Joint Significant Test*, which is currently regarded as the optimal method for assessing mediation (MacKinnon, Fairchild, & Fritz, 2007; MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002). First, it involves testing the statistical significance of the ' α path' representing the association between the independent variable (rash impulsiveness) and proposed mediator (impaired control). This is followed by

testing the statistical significance of the ‘ β path’, which represents the association between the proposed mediator (impaired control) and the dependent variable (severity of alcohol dependence). If both the α and β paths are significant, there is evidence for mediation (MacKinnon et al., 2002). To evaluate the magnitude of the mediated effect, confidence intervals based on the distribution of the unstandardized product ($\alpha\beta$) were calculated using the PRODCLIN program (MacKinnon, Fritz, Williams, & Lockwood, 2007). Meehl’s (1990) Corroboration Index (C_i) was used to test the accuracy of hypothesized point-estimate predictions for the mediation effect. The C_i is jointly determined by the accuracy of the point-estimate itself and the narrowness of the hypothesized prediction interval. For example, a ‘weak’ hypothesis that the mediation effect is merely statistically significant and of any positive value (i.e., $0 < \alpha\beta \leq 1$) would, if supported by the data, result in a $C_i = .50$. By contrast, a ‘strong’ hypothesis with a point-estimate prediction for the mediation effect that allowed no margin of error (e.g., $\alpha\beta = .23$) would, if supported by the data, yield a $C_i = 1.00$. Moderated mediation was then assessed by examining whether the strength of the α and β paths were conditional on *DRD2* allele status (A1+ versus A1-) or verbal fluency (see Figure 1). This was tested using the MODMED SPSS macro (version 3.1) developed by Preacher, Rucker, and Hayes (2007).

3. Results

3.1 Data Screening and Assumptions

All analyses were performed using SPSS (version 21.0, SPSS Inc., Chicago, IL). All variables were examined for data entry errors, missing values, violation of distributional assumptions. Two cases (1.4%) had missing data on rash impulsiveness and reward drive, 11 (7.7%) on ICS-Failed Control and ICS-Attempted Control, 13 (9.1%) on ICS-Perceived Control. Thirty-eight cases (26.6%) had missing data on *DRD2* status and 44 (30.8%) had

missing data on verbal fluency. Data were Missing Completely At Random (MCAR) as indicated by Little's MCAR test, $\chi^2(160) = 187.41, p = .068$. Therefore, the missing data would not bias parameter estimates and missing data were imputed using Expectation Maximization (EM; Graham, 2009). One univariate and one multivariate outlier on ICS-Perceived Control were identified, but exclusion of these cases did not affect results of the main analyses. Therefore, they were retained. Examination of the residuals revealed no violation of distributional assumptions.

3.2 Descriptive Statistics

On average, participants reported consuming alcohol on 6 of the past 7 days ($M = 6.17, SD = 1.40$) and consumed approximately 19 standard drinks per drinking occasion ($M = 18.75, SD = 11.05$). Mean ADS score was 29.87 ($SD = 9.65$), with 79.7% of the sample scoring within the 'Substantial' or 'Severe' alcohol dependence range (Skinner & Allen, 1982). Descriptive statistics for predictors are presented in Table 1.

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3.3 Mediation Analysis

To test the relationship between rash impulsiveness and perceived impaired control (α path), a hierarchical regression was conducted with quantity and frequency of drinking entered as covariates on step one along with previous attempts and failures to control drinking over the past 6 months (ICS-Attempted Control and ICS-Failed Control, respectively). Rash impulsiveness, reward drive, *DRD2* status, and verbal fluency were then entered on the second step. As hypothesized, rash impulsiveness predicted stronger perceptions of impaired control over drinking, even after controlling for average quantity and

frequency of drinking history, and previous instances of impaired control (see Table 2). Additionally, A1+ allele status was significantly associated with greater perceptions of impaired control. By contrast, reward drive and verbal fluency were not significantly related to perceived impaired control. Approximately 30% of the variance in perceived impaired control was accounted for by the model, $F(8, 142) = 7.09, p < .001$.

-----INSERT TABLE 2 HERE-----

In order to test the relationship between perceived impaired control and alcohol dependence severity (β path), a second hierarchical regression was conducted. As before, drinking history and past instances of impaired control were entered on the first step. Rash impulsiveness, reward drive, *DRD2* status, verbal fluency, and perceived impaired control were then entered on the second step. As hypothesized, perceived impaired control was significantly associated with dependence severity, even after controlling for drinking history and past instances of impaired control (see Table 3). This provides evidence for partial mediation, as rash impulsiveness still predicted unique variance in dependence severity. The indirect effect of rash impulsiveness on alcohol dependence severity, mediated by perceived impaired control, was statistically significant ($\alpha\beta = .057$; unstandardized mediation effect 95% CI = 0.023, 1.060). The magnitude of the mediation effect fell slightly below that predicted, but nonetheless provided strong corroboration of previous findings ($C_i = .98$; Gullo et al., 2010; Kabbani & Kambouropoulos, 2013; Leeman et al., 2012). Overall, the model accounted for approximately 34% of the variance in alcohol dependence severity, $F(9, 142) = 7.67, p < .001$.

-----INSERT TABLE 3 HERE-----

3.4 Moderated Mediation Analysis

Moderated mediation was evaluated using the procedure outlined by Preacher et al. (2007), and tested using their MODMED SPSS macro (version 3.1). As in the mediation analysis reported above, drinking quantity and frequency, ICS-Attempted Control, ICS-Failed Control, and reward drive were included as covariates. The association between rash impulsiveness and perceived impaired control (path α) was not moderated by *DRD2* A1+ status ($B = -0.23, SE = 1.02, p = .82$) or verbal fluency ($B = 0.05, SE = 0.05, p = .30$). Furthermore, the association between perceived impaired control and alcohol dependence severity (path β) was not moderated by *DRD2* A1+ status ($B = -0.02, SE = 0.23, p = .92$) or verbal fluency ($B = -0.001, SE = 0.01, p = .88$). In summary, neither *DRD2* A1+ status nor verbal fluency moderated the (partial) mediation effect of rash impulsiveness on dependence severity through perceived impaired control.

3.5 Post-hoc Analyses

The lower than expected mediation effect may have resulted from our inclusion of past consumption and past drinking control failures as additional control variables. When the data were reanalyzed without controlling for past attempts and failures at drinking control, the mediation effect increased to $\alpha\beta = .07$. When reanalyzed without controlling for recent alcohol consumption or past impaired control, it increased further to $\alpha\beta = .08$. These increases were mostly due to a strengthening of the association between rash impulsiveness and perceived impaired control (α path).

While genetic risk did not moderate the observed mediation effect, *DRD2* A1+ status did account for unique variance in perceived impaired control (see Table 2). A post-hoc mediation analysis revealed that, like rash impulsiveness, *DRD2*'s association with

dependence severity was mediated by higher perceived impaired control ($\alpha\beta = .06$; unstandardized mediation effect CI 95% = 0.11, 2.89).

4. Discussion

To our knowledge, this is first study to investigate the mediating role of perceived impaired control in the association between rash impulsiveness and alcohol dependence. As hypothesized, perceived impaired cognition (partially) mediated the association between rash impulsiveness and severity of alcohol problems in alcohol-dependent inpatients. Contrary to expectation, the magnitude of the mediation effect was not moderated by the dopamine *DRD2* polymorphism or verbal fluency, suggesting that this aspect of impulsive cognition is independent of *DRD2* Taq1A related genetic risk for alcoholism and executive dysfunction.

Findings were consistent with those reported by Patock-Peckham and colleagues (Patock-Peckham et al., 2011; Patock-Peckham & Morgan-Lopez, 2006), and Kabbani and Kambouropoulos (2013) in non-dependent, college drinkers. The magnitude of the mediation effect was smaller in this sample of treatment-seeking inpatients, compared to college students (Kabbani & Kambouropoulos, 2013) and is consistent with Gullo et al. (2010), where drinking refusal self-efficacy was compared between college drinkers and dependent drinkers. As a secondary aim of the study, we formulated a point-estimate prediction for the mediation effect that was based on previous studies. The mediation effect observed in the present study ($\alpha\beta = .057$) fell just below the predicted interval range ($.06 \leq \alpha\beta \leq .09$), although still providing strong corroboration of past findings. To our knowledge, this is the first addiction study to incorporate point predictions that are evaluated using an index like Meehl's (1990) C_i .

Post-hoc analyses suggest the point-prediction's 'near miss' was the result of our conservative analytic approach. Unlike previous investigations into this impulsive cognition

pathway, the present study controlled for recent alcohol consumption and past instances of impaired control. This ensured associations involving impaired control in this study were specifically tapping into patients' subjective perceptions of drinking control, independent of recent behavior. Therefore, it should provide a more precise estimation of the association between impulsivity, perceived impaired control and dependence severity.

Unexpectedly, the role of impulsive cognition in alcohol dependence was not moderated by the *DRD2* polymorphism or verbal fluency. This was the first study to investigate moderated mediation in impulsive cognition and, as such, no point-estimate was predicted, only that significant moderation would be observed. These results suggest that the mediating role of perceived impaired control is robust to individual differences in patients' ability to process and retrieve verbal information, as well as certain aspects of dopamine neurotransmission. Therefore, there is no evidence to suggest that cognitive-behavioral treatments aimed at improving perceived drinking control and self-efficacy will be of less benefit to those at genetic risk of dependence or those with more severe cognitive impairment. As can be seen in Table 2, the *DRD2* polymorphism accounted for significant unique variance in perceived impaired control and a post-hoc mediation analysis revealed that its association with dependence severity was mediated by perceived impaired control. Thus, treatments that strengthening patients' beliefs in their drinking control could reduce some of the risk conveyed by *DRD2*. However, this post-hoc finding should be interpreted with caution until replicated.

While this study benefited from a relatively large clinical sample and conservative analytic approach, it has some limitations. The cross-sectional nature of the study limits causal inference and the ability to establish temporal precedence among mediation variables. Future studies that incorporate prospective data on patient outcomes, including changes in perceived impaired control and alcohol use, would provide a stronger test of mediation.

Additionally, investigation of other genetic markers of dependence risk and additional neurocognitive domains are necessary. While verbal fluency measures are sensitive to executive neurocognitive dysfunction and quick to administer, they comprise only one of several domains of neuropsychological functioning affected during early stages of alcohol abstinence (Stavro et al., 2013). Future studies examining genetic and neurocognitive moderation of drinking refusal self-efficacy is also warranted.

In summary, this study adds to a growing body of literature suggesting that a significant proportion of the risk for problem drinking conveyed by (rash) impulsivity is mediated by perceived impaired control. Findings provide strong corroboration of results previously obtained from non-dependent college samples. They also provide preliminary evidence that impulsive cognition is unaffected by executive dysfunction and genetic risk for alcohol dependence as measured by *DRD2* polymorphic status.

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Table 1

Descriptive Statistics for Continuous Predictors and Moderator (N = 143).

Variable	<i>M</i>	<i>SD</i>	Range
Rash Impulsiveness	8.97	1.15	7 – 12
Reward Drive	7.64	3.01	2 – 12
ICS-Perceived Control	27.92	7.60	0 – 40
ICS-Failed Control	29.15	6.60	4 – 40
ICS-Attempted Control	9.82	5.20	0 – 20

Note. ICS = Impaired Control Scale.

Table 2

Impulsivity predicting perceived impaired control (α path) (N = 143).

	ΔR^2	<i>B</i>	<i>SE B</i>	β	<i>sr</i> ²
<i>Step 1</i>	.24***				
Drinking Quantity		0.08	0.05	.11	.01
Drinking Frequency		0.38	0.42	.07	.004
ICS- Attempted Control		0.03	0.12	.02	< .001
ICS- Failed Control		0.51	0.09	.45***	.18
<i>Step 2</i>	.06*				
Rash Impulsiveness		1.00	0.50	.15*	.02
Reward Drive		-0.13	0.19	-.05	.003
Verbal Fluency		0.05	0.05	.08	.005
DRD2 A1+ Status		2.76	1.31	.17*	.02
Total <i>R</i> ²	.30***				

Note. ICS = Impaired Control Scale.

Table 3

Perceived impaired control (mediator) predicting alcohol dependence severity (β path) (N = 143).

	ΔR^2	<i>B</i>	<i>SE B</i>	β	<i>sr</i> ²
<i>Step 1</i>	.14***				
Drinking Quantity		0.23	0.07	.26**	.06
Drinking Frequency		-0.40	0.56	-.06	.003
ICS- Attempted Control		0.18	0.16	.10	.01
ICS- Failed Control		0.39	0.12	.27**	.07
<i>Step 2</i>	.20**				
Rash Impulsiveness		1.78	0.62	.21**	.04
Reward Drive		0.18	0.23	.06	.003
Verbal Fluency		-0.04	0.07	-.01	< .001
DRD2 A1+ Status		2.08	1.65	.10	.01
ICS-Perceived Control		0.49	0.11	.38***	.10
Total <i>R</i> ²	.34***				

Note. ICS = Impaired Control Scale.

Figure Legends

Figure 1. Diagrammatic representation of moderated mediation model tested in the present study. (*Note.* Dashed lines denote hypothesized moderation effects)

